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COMMENTARY

Synergy, partner choice and frequency dependence: their integration into inclusive fitness theory and their interpretation in terms of direct and indirect fitness effects

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The aims of our Target Review (Lehmann & Keller, 2006) were to develop a simple model allowing us to delineate the conditions necessary for cooperation and altruism to evolve and argue that the models proposed so far can all be classified into four general categories depending on the selective forces at work. These are direct benefits to the Focal Individual (FI) performing a cooperative act, repeated interactions with direct or indirect information on the behaviour of the partner in previous moves, preferential interactions between related individuals and/or a linkage disequilibrium between genes coding for altruism and phenotypic traits that lead to assortment of individuals bearing altruistic genes (i.e. greenbeard effect). We were extremely pleased to see that authors of 14 of the 15 Commentaries found our framework useful and generally endorsed our views. Doebeli & Fletcher (2006) were the only authors who fundamentally disagreed with our classification. However, their criticisms mostly stem from a misunderstanding of our model and kin selection theory.

Several Commentaries raised similar general issues, in particular about the assumptions of our model and/or alleged limitations of Hamilton's rule. We therefore address these comments in the first four general sections. Following these, we address the criticisms raised by Fletcher and Doebeli (2006). Finally, in the two last sections, we discuss all the other specific comments raised in the Commentaries, and finish with a short conclusion.

Genetic assumption of our model

Queller & Strassmann (2006) pointed out that our model lacks rigour because it is 'built on principles of optimality and inclusive fitness rather than being based directly on gene frequencies'. Doebeli & Fletcher (2006) also suggested that 'we adjust the meaning of the fitness costs and benefits to highlight what we believe is fundamental'. Both these assertions are incorrect. Under the demographic conditions specified in the main text of our Target Review, and assuming an additive effects of genes underlying the level of helping, it is possible to express the change in allele frequency (p) over one generation of a mutant whose phenotype deviates only slightly (weak selection) from the phenotype expressed by a resident allele as:

$$\Delta p = (rb - c)p(1 - p). \quad (1)$$

(Rousset, 2004, pp. 108–109 and pp. 206–207). According to this equation, the direction of selection on the mutant allele is positive at all allele frequencies when: $rb - c > 0$, where $-c$ is the effect of a FI expressing the mutant genotype on its fitness, b is the effect of a partner bearing the mutant genotype on the FI's fitness and r is the genetic relatedness between the FI and its partner, which is evaluated in the absence of selection. Fitness, as specified in our Target Review, measures the expected number of offspring of a FI that reach adulthood, which is strictly equivalent to the definition of fitness given by Hamilton (1964). In other words, we derived Hamilton's rule from a population genetic model, classified behaviours as altruistic or cooperative as Hamilton did, and never modified the meaning of the cost ($-c$) and benefits (b) throughout the paper [see also Grafen's (2006) comments on this].

Demographic assumptions of our model

For the sake of simplicity, we only presented in the main text of our Target Review a simple social situation where interactions occurred between pairs of individuals in a large population of constant size. We also assumed that this population included only two kin classes and used Hamilton's rule to determine the direction of selection on the helping allele. In the Appendix, we considered other situations such as a spatially structured population with overlapping generations or with an explicit demography where helping can effect patch size. These examples were chosen to illustrate that our conclusions are robust to variation in demographic structure. However, van Baalen & Jansen (2006), Wild & Taylor (2006), Wenseleers (2006) suggested that there are demographic structures other than those that we considered. Although it is true that there are an infinity of demographic structures, such variations do not affect our conclusion that one of our four conditions needs to be fulfilled for cooperation or altruism to be selected for because the selective pressure on helping can always be broken down into direct and indirect effects of actors on the fitness of a FI. Indeed, when the type of social interaction is more complex than just pairwise interactions and/or involve several classes of actors (e.g. males and females, or several age classes) and/or if the population is geographically structured or of variable size, it is possible to express the change in allele

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frequency, as we originally detailed in the supplementary material of our Target Review, as:

$$\Delta p = \Delta W_{\mathrm{IF}} p(1-p), \qquad (2)$$

where ΔW_{IF} is again a measure determining the direction of selection at any allele frequency under weak selection (Rousset, 2004; Rousset & Ronce, 2004). When the population is of finite size, ΔW_{IF} determines the effect of selection on the probability of fixation of the mutant allele (Rousset, 2004). The measure of selection ΔW_{IF}, which is a weighted sum of the effects of all individuals in the population on the fitness of individuals bearing the mutant allele, fits with Hamilton's definition of inclusive fitness effect (Hamilton, 1964). In this case, the weights are the coefficients of relatedness between a FI bearing the mutant allele and the individuals affecting its fitness, the reproductive values of the FI's offspring and the frequencies of the various classes of individuals affecting the fitness of the FI. The inclusive fitness effect ΔW_{IF} can be interpreted as a generalization of the simple inclusive fitness effect ($rb - c$) used in the Target Review and reveals that the different selective forces (e.g. differential effects due to males and females and/or patch demography) influencing helping can always be broken down into direct and indirect effects on the FI's fitness. In other words, the incorporation of more complex life-histories does not change the general nature of the selective forces acting on helping, which can only evolve when at least one of our four conditions is fulfilled.

Strong selection, nonadditive gene effects and multilocus evolution

Several authors (Fletcher & Doebeli, 2006; Doebeli & Hauert, 2006; Queller & Strassmann, 2006; Wenseleers, 2006) pointed out that Hamilton's rule fails to provide the correct direction of selection on helping when there is strong selection and/or non-additive gene effects. In such situations selection is frequency dependent (e.g. Cavalli-Sforza & Feldman, 1978; Michod, 1982; Roze & Rousset, 2004). However, we shall show here that our conclusions are not affected by assuming more complex genetic underpinning of helping (for increased generality we also discuss the issue of multilocus evolution). Let us assume that genes at several positions in the genome affect the direct fitness of a FI and let designate by S these positions [a position is a locus in a particular context (see Kirkpatrick *et al.*, 2002, Fig. 1), for instance the place in the genome where the FI's helping genes reside or the place where the helping genes of a FI's relative reside]. The change in frequency p of a helping allele at a given locus can be expressed as:

$$\Delta p = \sum_{U \subseteq S} a_U D_U, \qquad (3)$$

where a_U is the intensity of selection on the helping allele resulting from the expression of the genes in the set of positions U [these positions can be in the FI's genome and/or in the genome(s) one or several other individuals], D_U is the genetic association between the genes in U and the genes residing at the helping locus of the FI, and the sum runs over all possible subsets of positions that can be constituted with the set of positions S (Kirkpatrick *et al.*, 2002). Inspection of Eqn 3 reveals that the selective pressure on a helping allele consists of (1) phenotypic effects of a set of genes (one or several genes residing in the FI and/or in different individuals) on the fitness of the FI and (2) genetic associations (covariance in the case of pairs of genes) between the genes affecting the FI's fitness and the genes at the FI's helping locus (see Appendix for an explicit example). Effects of genes on the FI's fitness and associations between genes can be evaluated to various orders of magnitude of phenotypic effects. These include first order effects (linear), second order effects (quadratic) or higher order effects. For instance, the effects of actors on fitness in Hamilton's rule are traditionally evaluated to first order phenotypic effects whereas relatedness is evaluated to the zero's order (i.e. in the absence of selection). But nothing prevents the evaluation of effects of actors on fitness to second order phenotypic effects and relatedness to the first order. That Hamilton's rule is evaluated to the lowest order only is not a weakness of inclusive fitness theory but of our own inability to exactly evaluate the direction of selection. Equation 3 also reveals that it is always possible to decompose the selective forces acting on helping into two categories, whatever the mode of gene action, intensity of selection, and number of loci affecting the FI's fitness. The first category consists in all effects dependent on the FI expressing its own genotype. These 'direct effects' can be conditional on whether the partners express genotypes identical to that of the FI at a given locus (which may be the basis of synergy for instance). The second category are indirect effects on the FI's fitness, which result from the expression of the genotype(s) of partner(s) and are unconditional on the FI's genotype. Because the selective pressure on helping can always be decomposed into direct and indirect effects of actors on the FI's fitness, we conclude that whatever the complexity of the genetic basis of helping, cooperation and altruism can evolve only when at least one of our four conditions is fulfilled.

ESS conditions and evolutionary branching

Doebeli & Hauert (2006) suggested that our classification is not applicable in cases where a population resides at an evolutionary branching point and that Hamilton's rule cannot be used at such points. In order to see why this is not true, we first explain how game theory relates to the inclusive fitness theory presented above.

Hamilton's rule, and more generally the inclusive fitness effect ΔW_{IF}, allows us to determine whether a

mutant helping allele will be selected for when rare and to establish the direction of selection at all gene frequencies under weak selection and additive gene effects (eqn 2). The inclusive fitness effect also allows us to locate candidate evolutionary stable strategies (ESS), which are found at the points where it is equal to zero (Day & Taylor, 1998; Rousset, 2004). The inclusive fitness effect further allows us to determine whether such a candidate ESS is convergence stable (i.e. an evolutionary attractor). However, the inclusive fitness effect ΔW_{IF} in itself does not allow us to determine whether a candidate ESS actually is an ESS (i.e. a final stop of evolution for the helping trait). The reason is that the inclusive fitness effect given in eqn 1 or eqn 2 is evaluated to the first order of phenotypic effects on fitness and is thus only a linear approximation of the selective pressure on the helping allele that neglects higher order effects. At an ESS point, the linear effects on fitness cancel each other so that quadratic effects can be used as the new approximation for determining the selective pressure on helping. After convergence to a candidate ESS, the further development of the evolutionary process can be determined by a weighted sum of the expected effects of all individuals in the population on the fitness of a FI bearing the mutant allele, where such effects on fitness are quadratic (Day, 2001; Ajar, 2003) and provide a good approximation under weak selection (Rousset, 2004). These effects can be fully interpreted in terms of inclusive fitness theory (Ajar, 2003, eqns 29–30), because they involve direct and indirect effects of the partners on the fitness of the FI of the same kind as the selection coefficient a_U in eqn 3. It is true that inclusive fitness effects do not allow us to predict what type of mutant will invade the population at a branching point. Whether it is a more or less cooperative mutant than the resident genotype that will appear depends on the type of mutations that occurred. However, at a branching point, no mutant allele can invade a resident allele unless it results in a greater inclusive fitness effect, hence implying that one of four conditions needs to be satisfied for the invasion of a mutant allele at a branching point.

Synergism and discrete/pure strategies

Several Commentaries raised the point that our model does not incorporate synergistic effects and that this is a problem with our classification because such effects are inherently frequency dependent (Doebeli & Hauert, 2006; Queller & Strassmann, 2006; Wenseleers, 2006). Although we did not discuss in details situations with synergetic effect we specified in the section direct benefits of our Target Review that 'the value of ζ will also depend on the behaviour of other group members when there are synergistic effects of cooperation'. The example of synergism that we had in mind was a situation where the synergistic benefit of helping for a FI was proportional to the product of its own investment and that of its partner. Consider for instance a one shot and random interaction between pairs of individuals in the same demographic setting as described in our Target Review (i.e. $x = 0$ and $\omega = 0$), where investment into helping results in a linear cost C for a FI bearing the helping allele, a benefit D to each partner increasing linearly with the product of the investment into helping of both the FI and its partner. Under such a situation, helping will spread when the inequality:

$$D\tau - C > 0 \tag{4}$$

is satisfied. In other words, helping is cooperative because the action (conditional on the action of the partner) results in increased fitness for both the FI (by $D\tau - C$) and its partner (by $D\tau$). In fact, this in eqn 4 for the evolution of helping is very similar to in eqn 6 in our Target Review and inspection of in eqn 4 reveals that when nobody in the population expresses helping in the first place ($\tau = 0$), investment into helping cannot be selected for and thus relies on kin selection for its initial emergence. The same conclusion was reached for the initial evolution of helping under repeated interactions (see eqn 18 of the supplementary material of the Target Review).

In our Target Review we did not consider the situation where helping is determined by discrete (or pure) strategies. In that case, the condition for the evolution of helping under synergistic effects is no longer given by eqn 4, yet it can be integrated in our framework. Consider the situation given in Wenseleers (2006) where an act of helping results in a cost C to a FI bearing the helping allele and in a benefit D to each partner when both the FI and its partner bear the helping allele. Assuming the same life-cycle as described in the main text of the Target Review, we find from eqn 3 and eqn 15 in the Appendix that the change in frequency p of the helping allele is positive when:

$$Dp - C > 0. \tag{5}$$

In other words, helping becomes a better strategy than the alternate option of defecting if the frequency of helpers in the population times the synergistic benefit exceeds the cost of helping. This condition of invasion is very similar to that in eqn 4 with the frequency of helpers p in the population playing the same role as the level of investment into helping ζ of an individual sampled at random from the population in the continuous helping strategy setup. And in the same way as in the continuous strategy case, helping is again cooperative because the act of helping results in an increase in fitness for both the FI (by $Dp - C$) and its partner (by Dp). Moreover, as in the continuous strategy situation, a positive relatedness between interacting individuals is required for helping to be selected for when helpers are initially rare in the population ($p \to 0$).

In conclusion, these analyses reveal that when individuals are not preferentially interacting with individuals

with above average relatedness, synergistic helping fall into our category of 'direct benefits' because an act of helping can evolve only when translating into increased direct fitness. Modelling synergistic effects can be readily done for situations of repeated interactions and nonrandom interactions between kin classes in the population when helping. For example, we derive in the Appendix a model where helping is a discrete strategy in a structured population. Importantly, however, the occurrence of synergism does not alter our point that at least one of our four conditions need to be satisfied for helping to be selected for.

Markets and partner choice

Several authors mentioned that we did not include the possibility of partner choice (Queller & Strassmann, 2006; Sachs, 2006), partner fidelity (Sachs, 2006) nor the possibility to terminate an interaction as a response to defection (Cant, 2006; Hammerstein & Leimar, 2006). This is correct and was done on purpose because these are all additional factors that can promote cooperation only if one of our four general conditions is met. The most common of these conditions is repeated interactions between individuals with direct or indirect information on the behaviour of the partner in the previous move(s). In that case it is correct that partner choice, partner fidelity and the possibility to terminate an interaction are potent mechanisms that will increase the probability that cooperative individuals preferentially interact with each other. Such effects could readily be integrated in the model presented in the main text of our Target Review by adding a new parameter quantifying the decreased likelihood of a cooperative individual engaging in an interaction with an individual that was previously not cooperative. This could be done for a situation where helping is a continuous strategy, as in the model in our Target Review, or a discrete strategy as discussed above for situation of synergism.

Partner choice, partner fidelity and the possibility to terminate an interaction as a response to defection could also play a role when information on whether a given individual is likely to be cooperative comes from reputation. As shown in the Appendix of our Target Review, this situation can be modelled in a similar manner to reciprocal altruism and, in this case, it would also be possible to add a new parameter quantifying the decreased likelihood of a cooperative individual to engage in an interaction with an individual that was previously uncooperative. Finally, one could also imagine that partner fidelity and the possibility to terminate an interaction as a response to defection could also play a role if individuals can assess the cooperative tendency of other individuals on the basis of a phenotypic or behavioural trait.

In conclusion, we agree that partner choice, partner fidelity and the possibility to terminate an interaction as a response to defection are important mechanisms that may promote cooperation and/or altruism. However, for these mechanisms to operate requires that at least one of four conditions is fulfilled.

Response to Fletcher and Doebeli

There is, unfortunately, much confusion in Fletcher & Doebeli's (2006) Commentary. Nevertheless, we welcome the opportunity to respond to their Commentary because it allows us to address common mistakes also made by a few other theoreticians who, unfortunately, have never made the effort to understand kin selection theory nor to familiarize themselves with the rich literature on this topic. Readers at ease with kin selection theory may want to skip this section.

We shall start by addressing four simple misunderstandings. First, F&D assert that we only used the most basic meaning of r (i.e. relatedness by descent). This is not correct as we also consider probabilities of identity in state of which identity by descent is only a particular case. Secondly, F&D assert that we modify the meaning of b and c. Unfortunately no specific equation is given. We strictly used Hamilton's (1964, 1970) definitions of fitness costs and benefits. As, Grafen (2006) pointed out: 'Lehmann and Keller rightly recognize that the b and c to be used in Hamilton's rule need to be appropriately derived'. Thirdly, F&D state that we implement a population-wide definition of altruism that leads to very unsatisfactory results in our classification. We do not know what F&D mean by a 'population-wide definition of altruism'. In any case, our definition is strictly equivalent to the definition of altruism given by Hamilton (1964, 1970) and there is a large literature justifying the use of such a definition (e.g. Grafen, 1985; Frank, 1998; Rousset, 2004). Finally, F&D provide a long discussion on the direct and inclusive fitness approaches. We are not clear what F&D wanted to state and refer readers to excellent discussions of Frank (1998) and Rousset (2004) on this issue. Importantly, our model is derived from a rigorous population genetic framework, and for simplicity, we either interpret our results by looking at the effect of a FI on the fitness of all members of the population (inclusive fitness approach) or by looking at the effect of all individuals in the population on the FI's fitness (direct fitness approach). Both approaches are perfectly correct and can be used interchangeably (Rousset, 2004, pp. 107–108). In the same section F&D also criticize Sachs *et al.* (2004) treatment of kin selection which, they suggest, 'confounds an accounting technique with a mechanism'. The strength of inclusive fitness theory is precisely that it provides both an accounting technique and a mechanism to explain the evolution of social behaviours. The treatment of kin selection theory of Sachs *et al.* (2004) is correct in our view.

Fletcher and Doebeli claim that the distinction between 'weak altruism', where the actor obtains a

benefit from its own act of helping (i.e. $-c > 0$ in eqn 1), and 'strong altruism' where there are no such direct benefits (i.e. $-c < 0$) is not fundamental. On the contrary, the vast majority of researchers interested in the natural phenomenon of cooperation and altruism, starting with Darwin himself, and including all the other authors who expressed their view on this matter in their Commentaries, understand that there is a fundamental distinction between 'weak altruism' and 'strong altruism' (see e.g. Ratnieks, 2006).

Fletcher and Doebeli assert that our framework tends to obscure the fundamental roles that assortment and nonadditivity play. This is a perplexing statement. Three of our categories are explicitly based on assortment. For example, in the conclusion of our Target Review, we stated that 'cooperation and altruism can evolve only when there are direct benefits to the FI performing a cooperative act, repeated interactions with direct or indirect information on the behaviour of the partner in previous moves, preferential interactions between related individuals and/or a linkage disequilibrium between genes coding for altruism and phenotypic traits that can be identified. In the three later cases helping evolves because there is a positive association between individuals at the genotypic and/or phenotypic levels'. Moreover, throughout the paper, and in all equations, we make clear that positive assortment is critical when there are no direct benefits.

Fletcher and Doebeli claim that when nonadditivity is present, altruistic behaviour can evolve even in the absence of positive assortment. This is wrong. Synergism can lead to cooperation without positive assortment but not to altruism (see the Synergism and discrete/pure strategies section).

Finally, F&D consider three models that they claim do not fit in our classification framework. The first is the grouping model of Avilés (2002) where grouping and cooperation are modelled as distinct co-evolving traits. In this model it is assumed that the fitness of an individual in a group increases with the number of helpers in the group raised to the power of some constant and decreases exponentially with the total number of individuals in a group. An analysis of the model reveals cycles in the level of helping with mutants having both a grouping and cooperative tendency being selected for when the population mostly consist in individuals with nongrouping tendencies. The dynamics of this model is very similar to Jansen & van Baalen's (2006) model. In both cases, frequency-dependent selection leads to variation over time in the level of altruism, the oscillations depending on the mutation rate. The model of Avilés (2002) is thus a special case of 'green beard' model as we correctly classified in the Table of our Target Review. Moreover, contrary to what is stated by F&D, synergy is probably not important. Although it is true that the model assumes that the fitness of an individual depends on the number of helpers in the group raised to the power of some constant (called synergy by F&D), the crucial assumptions of the model are that loners perform better when groups consist of defectors and the fitness of a helper increases with the number of helpers in its group. This situation can also occur when the fitness of individuals increases additively with the number of individuals within groups.

The second model is the so-called environmental feedback model by Pepper & Smuts (2002). In this model the authors consider two classes of individuals that feed on patches of plants that they leave when resources are depleted. The two classes are unrestrained eaters that quickly deplete local resources and restrained eaters, which depletes resources less quickly. Their model shows a positive assortment between unrestrained and restrained eaters but it does not investigate whether feeding restraint is a stable strategy in the population. In other words, although the simulations of Pepper & Smuts (2002) provide interesting information on possible mechanisms promoting positive assortment, they do not allow us to make any conclusion on the evolution of cooperation and altruism.

The third model is by Fletcher & Zwick (2004). F&D claim that this model provides an example where altruism can evolve between nonrelatives. An analysis of their model provides a perfect example of the contrary. The model by Fletcher & Zwick (2004) is a slightly different form of the group-selection model studied by Hamilton (1975) where individuals interact in randomly formed groups, except that several generations of reproduction occur before the stages of complete dispersal, regulation and formation of new groups. Such a life-cycle results in individuals of the same gene lineage interacting with each other and thus benefiting from the altruism of kin descending from the same parents. In the Appendix we show that helping cannot evolve if interactions between kin are prevented to occur. In conclusion, the model of Fletcher & Zwick (2004) falls perfectly into our classification scheme and provides a nice example of a failure to recognize kin selection.

Response to the other issues raised in the Commentaries

Queller & Strassmann (2006) suggested that one of their own models (Queller, 1992) already encompassed all the relevant forces presented in our model. This is correct. However, Queller's (1992) model does not partition the selective forces in terms of direct and indirect effects on fitness and does not distinguish between cooperation and altruism. Thus, although we agree that it is a useful model, it is not appropriate for establishing a simple classification of models of cooperation and altruism. The same comment holds for the group selection approach and multilevel selection models mentioned by Foster (2006).

Wenseleers (2006) raised concerns about possible limitations of Hamilton's rule under frequency depend-

ence which we addressed in the section Strong selection, nonadditive gene effects and multilocus evolution. Using eqn 3, we also present in the Appendix a model that allows us to show, in contrast to Wenseleers' (2006) suggestions, that cooperation in spatial game can be explained in terms of kin selection and that introducing discrete strategies does not limit the application of inclusive fitness theory.

Doebeli & Hauert (2006) listed two examples of situations that they claim do not fit in our framework. The first, taken from Hauert & Doebeli (2004) and is a situation where a FI investing x into helping plays with an individual investing y into helping. The fecundity of the FI is given by $B(x + y) - C(x)$, where B and C are monotonously increasing benefit and cost functions. Thus, the gradient of selection on helping is given by $B'(2x) - C'(x)$, where the primes denote derivatives. Accordingly, helping spreads when $B'(2x) - C'(x) > 0$. From this Doebeli & Hauert (2006) conclude that 'contrary to what seems to be implied in Lehmann and Keller, whether individuals receive net direct benefits from the act of cooperation is not determinant of whether cooperation is favoured'. This is an odd statement because it is completely contradicted by the analyses of the gradient of selection. Indeed, $B'(2x)$ is the marginal benefit of an individual investing into helping and $C'(x)$ is its marginal cost. Thus, $B'(2x) - C'(x)$ represents the net effect of a FI on its fitness when increasing investment into helping, which precisely corresponds to $-c$ in Hamilton's rule (eqn 1). When there are not direct benefits of helping, namely $B'(2x) = 0$, helping is counter-selected ($-c < 0$). Doebeli & Hauert (2006) thus provide a perfect example of a situation where helping spreads because it increases the FI's fitness. This example thus falls neatly into our category of direct benefits. In fact, that individuals should behave cooperatively under such situations has been recognized long ago as exemplified by the following quote by Adam Smith (1776): 'It is not from the benevolence of the butcher, the brewer, or the baker that we expect our dinner, but from their regard to their own interest. We address ourselves, not to their humanity but to their self-love, and never talk to them of our own necessities but of their advantages'.

Doebeli & Hauert (2006) also claim that the various formalizations of the evolution of altruism in 'spatial structuring models' of Nowak & May (1992), Killingback *et al.* (1999), Hauert & Doebeli (2004) do not fall into one of our categories as spatial structure promote cooperation because it leads to a positive assortment between cooperators. As explained in length in our Target Review these three models fall into our category kin selection. A good introduction to the topic of how spatial structure can promote the evolution of altruism by kin selection is given by Hamilton (1971).

van Baalen & Jansen (2006) were worried that we did not make sufficiently clear how kin selection relates to kin recognition and that our approach may perpetuate the misconception that kin selection requires discrimination of related individuals. We hope that our Target Review does not lead to such a misunderstanding. In the Target Review's Appendix we derived several models where kin selection can operate under some specific demographic situation without kin recognition. There are many other such situations (e.g. van Baalen & Rand, 1998; Taylor & Irwin, 2000; Lehmann *et al.*, 2006). van Baalen & Jansen (2006) also suggested that our model creates the impression that the costs, benefits and the relatedness structure are static properties of a populations. Although it is true that this was the case in the simple model presented in the main text, we also presented in the Appendix models where relatedness is a dynamical variable depending on population demography and where helping itself can affect the demography of the population. Finally, van Baalen & Jansen (2006) mention that, contrary to what we stated, greenbeard mechanisms are not inheritantly unstable. Here we shall acknowledge that we were somewhat imprecise by failing to explicitly stipulate the conditions under which greenbeard mechanisms are unstable. The situation that we had in mind was the simple case where evolution occurs in a panmictic population with interactions occurring between individuals that do not share any recent common ancestry. However, it is true that common descent in geographically structured population can counteract the erosion of linkage between a helping allele and a recognition allele, eventually leading to a stable level of altruism by a greenbeard effect (Axelrod *et al.*, 2004; Jansen & van Baalen, 2006).

Cant (2006) was concerned that we might have underestimated the role of punishment. In particular he pointed out that the option to terminate an interaction as a response to defection can be a powerful force favouring the spread of cooperation mutants in a noncooperative population. We agree that this can be a potent factor and have now discussed this issue in the Markets and partner choice section. As we made clear, the option of terminating an interaction as a response to defection, which can be classified in the category of reciprocity with direct or indirect information, may indeed play an important role when cooperation is established. However, the option to terminate an interaction as a response to defection cannot in itself help to promote cooperation in a noncooperative population.

Hammerstein & Leimar (2006) mentioned other interesting situations of cooperation, but these were mostly between species or between organelles. Although a framework similar to the one we developed could be used for interespecific interactions, we decided to concentrate on intraspecific interactions to avoid confusions. In their other Commentary, Leimar & Hammerstein (2006) provide an interesting historical view of the field of cooperation making the valid point that, unfortunately, there is a significant amount of models that are driven by their own properties rather than the aim to

understand cooperation and altruism in nature. This, together with the fact that a significant number of theoreticians fail to incorporate their work in a more general context of previously published work, was indeed the main reason that prompted us to write our Target Review. We hope that this debate will help gifted theoreticians to develop models aiming at understanding puzzling phenomena in nature rather than studying what Leimar & Hammerstein (2006) appropriately call 'highly stylized situations'. In their Commentary these authors also point out that we did not consider cultural evolution in our framework. Cultural evolution is undoubtedly an important mechanism allowing the transfer of information in humans, and as such, it plays a pivotal role in the evolution of cooperation and altruism (e.g. Cavalli-Sforza & Feldman, 1981; Boyd & Richerson, 1985). However, it should in principle always be possible to decompose the selective forces acting on cultural variants expressing helping according to an equation of the form of eqn 3. Accordingly, we are not aware of any model of cultural evolution, which cannot be classified into our four general categories.

Boyd (2006) correctly pointed out that even if the necessary conditions that we outlined for the evolution of cooperation under repeated interactions are satisfied this does not mean that reciprocity is an ESS. Boyd provides a clear explanation of why this may not be the case and we have nothing to add.

Sachs (2006) provides a detailed and very balanced discussion of the linked with other previously proposed frameworks. The only point meriting a remark is partner choice. For partner choice to select for cooperation requires that individuals can assess the cooperative tendencies of their partner hence leading to assortment between cooperative individuals. Hence, partner choice also requires information. Such information can come by several means, the most common in nature probably being repeated interactions (see Sachs *et al.*, 2004) for a detailed account of the many mechanisms that may allow one or several of our four conditions to be fulfilled).

Ratnieks (2006) makes several valuable comments on our framework and, contrary to Doebeli & Fletcher (2006), he feels that it is important to distinguish between 'weak altruism' and 'strong altruism' because these are biologically two very different situations. We fully agree with Ratnieks that weak altruism is a bad term as weak altruists, on average, enhance direct reproduction. This is why we use the term cooperation for acts that increase the fitness of the FI and its partner.

As stated by Michod & Herron (2006), our framework can also be useful to classify the forces involved in the evolution of individuality. However, there might be limitations, in particular when the transition involves partners of different species.

Grafen (2006) pointed out that introducing the parameter ζ to describe the fraction of benefit that return to the FI is unnecessarily complex because the net effect of an individual expressing helping on its fecundity is sufficiently described by a single parameter. The reason we introduced the parameter ζ was to illustrate that helping can be selected for because it results in a direct fitness benefit for a FI, without necessarily eliciting a cooperative response of the partner. This parameter can be useful to understand the selective forces, for example in the case given in Doebeli & Hauert (2006) that we discussed above. Grafen also expressed concerns about our claim that phenotype matching leads to uniform genetic similarity over the whole genome. While describing phenotype matching, we stated that: 'Since common genealogy generates phenotypic similarity for genetically determined traits, each trait can be used as an independent value to estimate average genetic identity. This is a process of statistical inference with arbitrary phenotypic traits being used as quantitative or qualitative variables. Importantly, both spatial recognition and phenotype matching lead to uniform genetic similarity over the whole genome'. What we meant by the later phrase is that if individuals sample a very large number of independent phenotypic traits in other individuals to estimate their genetic similarity with them, then the estimate of similarity is an indicator of average relatedness over the whole genome because common ancestry generates similarity between individuals at all loci. But we acknowledge that our statement was confusing and we wish to make clear that we where not stating that the evolutionary consequences of phenotype matching results in uniform genetic similarity. Although the evolution of recognitions systems and the consequence for the maintenance of genetic variability at matching loci is extremely complex and has to our knowledge not been fully worked out, we agree that kinship is probably the only biological factor that can produce uniform genetic similarity across the genome. Finally, Grafen also mentioned that contrary to what we asserted, greenbeards are not necessarily unstable. This is a valid point that we addressed in our response to van Baalen & Jansen (2006).

Conclusion

In this response we hope that we clarified issues that were not clear in our Target Review and addressed the many interesting points raised in the 15 Commentaries. In particular, we showed that our framework and classification are based on a population genetic model which does not depend on particular assumptions about the population structure and genetic underpinning of helping behaviour and that synergic effects can readily be incorporated in our model. Our classification is thus based on a robust framework allowing one to identity the conditions necessary for the evolution of cooperation and altruism. Clarifying the relationship between models and correctly classifying different situations belonging to the

same mechanism should facilitate communication, avoid duplication and focus the attention of theoreticians on biologically relevant phenomena.

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Appendix

In this Appendix we derive two different population genetic models where evolution occurs in a spatially structured population. First, we present a model where helping is a discrete strategy, which can result in synergistic effects. This model allows us to illustrate that contrary to what is asserted by Wenseleers (2006), cooperation in spatial game can be explained in terms of kin selection and that introducing discrete strategies does not limit the application of inclusive fitness theory. Then, we demonstrate that helping evolves in the model of Fletcher & Zwick (2004) by kin selection. We first present the general life-cycle, which is common to both models and then consider each specific case in turn. To derive these models we follow the direct fitness approach developed by Roze & Rousset (2005).

Life-cycle

Let us posit that evolution occurs in a population following Wright's infinite island model of dispersal. Individuals are haploid and live in demes where they have only one neighbour. Events of the life-cycle occur in the following order. (1) A one shot social interaction occurs between the two individuals living in a deme. (2) Each individual produces a very large number of juveniles and dies. (3) Each juvenile disperses independently from each other with probability m to another deme. (4) Regulation occurs with the effect that only two juveniles reach adulthood in each deme.

We consider a two allele model (say A and a) and assume that the fecundity of an individual depends on its own genotype and on the genotype of its partner. The change in frequency p of a helping allele A over one generation in the population can be written as:

$$\Delta p = E_{i,j}\left[w_{ij}\zeta_{(ij)}\right], \tag{6}$$

where w_{ij} is the expected number of adult offspring of individual j breeding in deme i and $\zeta_{(ij)} = p_{(ij)} - p$ is a centred variable with $p_{(ij)}$ designating the frequency (0 or 1) of allele A in that individual. The expectation in the equation for gene frequency change is taken over all individuals and all demes. The fitness of individual j in deme i depends on both its expected number of offspring reaching adulthood in deme i and on those reaching adulthood in other demes after dispersing. These two fitness components depend on fecundity that will be written under the form $1 + \delta f_{ij}$, where 1 is the baseline reproductive unit and f_{ij} is the phenotypic effect on the fecundity of individual j in deme i of the expressions of the genotype of that individual and the genotype of its neighbour. Accordingly, the fitness of individual j in deme i can be written as:

$$w_{ij} = \frac{(1-m)(1+\delta f_{ij})}{(1-m)(1+\delta f_i) + m(1+\delta f)} + \frac{m(1+\delta f_{ij})}{(1+\delta f)}, \tag{7}$$

where f_i is the effect of actors on the average fecundity of individuals in the focal deme and f is the effect of actors in the population on the average fecundity of individuals in other demes. Assuming weak selection (small δ), the change in frequency of allele A is given to the first order in δ by:

$$\Delta p = \delta E_{i,j}\left[\left(f_{ij} - (1-m)^2 f_i - m(2-m)f\right)\zeta_{(ij)}\right] + O(\delta^2), \tag{8}$$

where $O(\delta^2)$ is a remainder involving second and higher order terms.

Synergy

Let us denote by C the direct fecundity cost of bearing the helping allele, B the benefit of helping received by a neighbour bearing the helping allele and D the synergistic effect on the fecundity of each individual when both individual in a deme bear the helping allele. Under such conditions, the phenotypic effect of the genotypes of the two individuals in a deme on the fecundity of individual j in deme i is:

$$f_{ij} = -Cp_{(ij)} + Bp_{(ik)} + Dp_{(ij)}p_{(ik)} \tag{9}$$

where $p_{(ik)}$ is the frequency (0 or 1) of allele A in the individual j' neighbour in deme i (labelled here individual k). The effect of actors in deme i on the average fecundity of the two individuals in that deme is:

$$f_i = \frac{1}{2}f_{ij} + \frac{1}{2}f_{ik} \tag{10}$$

and the effect of actors on the average fecundity of individuals in different demes is:

$$f = \frac{1}{n_d}\sum_{h,h\neq i}^{n_d-1} f_h \tag{11}$$

where there is an infinite number of demes ($n_d \to \infty$). Following Kirkpatrick *et al.* (2002) and Roze & Rousset (2005), we express all the gene frequencies appearing in the fecundities given above in terms of centred variables ($p_{(ij)} = p + \zeta_{(ij)}$). The effect of actors on the fecundity of individual j in deme i becomes:

$$f_{ij} = (B-C)p + Dp^2 + (Dp - C)\zeta_{(ij)} + (B + Dp)\zeta_{(ik)} + D\zeta_{(ij)}\zeta_{(ik)}. \tag{12}$$

By expressing similarly f_i and f in terms of centred variables and substituting into eqn 8, we eliminate variables with repeated indices with the formula $\zeta_{(ij)}^2 = p(1-p) + \zeta_{(ij)}(1-2p)$ (Kirkpatrick *et al.*, 2002). By using $E_{i,j}[\zeta_{(ij)}] = 0$, $E_{i,j}[\zeta_{(ij)}\zeta_{(hk)}] = 0$ and $E_{i,j}[\zeta_{(ij)}\zeta_{(hj)}] = 0$ for all individuals from deme h different than i because individuals from different demes are unrelated and using $E_{i,j}[\zeta_{(ij)}\zeta_{(ik)}] = p(1-p)r$ for individuals from the same deme, where the coefficient of relatedness r is given here by Wright's measure of population structure ($r = F_{ST}$), we find that the change in gene frequency reads:

$$\Delta p = p(1-p)\left[-C + Br + D(r + (1-r)p) - (1-m)^2\left\{(B-C)\frac{(1+r)}{2} + D(r + (1-r)p)\right\}\right]. \quad (13)$$

The term in square brackets in this equation is the effect of all actors in a focal deme on the fitness of a FI bearing the helping allele. This effect on direct fitness is made of four different terms. First, the direct cost (C) resulting from the FI expressing helping. Secondly, the benefit B received by the FI from its neighbour, which is weighted by the relatedness r between actor and recipient. Thirdly, the synergistic effect D of helping which depends on the probability $r + (1-r)p$ that the neighbour of the FI also bears the helping allele. Fourthly, the increase in kin competition in the focal deme, which depends on the probability $(1-m)^2$ that a FI's offspring compete against another juvenile produced in the focal deme. This increase in kin competition depends on the increase of the number of juveniles produced in the focal as a result of the FI and its partner helping each other (effect of intensity $(B-C)(1+r)/2$) and as a result of the synergistic effect of helping on individual fecundity [effect of intensity $D(r + (1-r)p)$].

Inserting the equilibrium value of relatedness $r = F_{ST} = (1-m)^2/(1+2m-m^2)$, we find that all terms involving the benefit B cancel each other out, which is consistent with the results of Taylor (1992) and all our results given in the supplementary material of our Target Review when evolution occurs in a spatially structured population. Notice that the selective pressure for the Prisoner's dilemma (or repeated Prisoner's dilemma) can be found by substituting $C \equiv (S-P)$, $B \equiv (T-P)$ and $D \equiv P+R-S-T$ into eqn 2, where the payoffs are the so-called reward for mutual cooperation (R), temptation to defect (R), sucker's payoff (R) and punishment for mutual defection (P).

When the helping allele is rare ($p \to 0$), the net effect of the FI on its fitness is given by:

$$-c = -C + Dr - (1-m)^2\left(\frac{(B-C)}{2} + Dr\right), \quad (14)$$

which gathers all effects on fitness resulting from the FI expressing the helping genotype (such effects involve the effects that are conditional on whether the partner also expresses the helping genotype). Thus, the net direct effect on fitness depends on the cost C of expressing helping, the benefit B resulting from helping neighbours and the synergistic benefit of helping D, which is weighted by the coefficient of relatedness r.

When evolution occurs in a family structured population (e.g. interactions between siblings) and when there is no kin competition, the change in gene frequency (eqn 13) is given by:

$$\Delta p = p(1-p)(-C + Br + D\{r + (1-r)p\}), \quad (15)$$

which provides the same equilibrium frequency of the helping allele as obtained by Wenseleers (2006) and presented by his eqn A.8.

Fletcher and Zwick's altruism

The model analysed by Fletcher & Zwick (2004) is equivalent to the model studied by Hamilton (1975) except that several generations of reproduction occur before the stages of complete dispersal, regulation and formation of new groups. For simplicity, and without loss of generality, we investigate here analytically only the situation where there are only two individuals per group and only two successive events of reproduction before dispersal. The dynamics of the two phases of reproduction within groups is modelled by following the equations presented in Appendix A of Fletcher & Zwick (2004). However, we evaluate here the change in frequency of the helping allele with eqn 6, where w_{ij} is the fitness function giving the expected number of individuals in the lineage of individual j in deme i reaching adulthood after two events of reproduction (here m=1 in eqn 7 because dispersal is complete). The expected number of offspring descending from individual j in group i after two generations of reproductions can then be written as:

$$f_{ij} = \left(1 + B\left[\frac{(1 + Bp_{(ik)} - C)p_{(ij)}}{2} + \frac{(1 + Bp_{(ij)} - C)p_{(ik)}}{2}\right] - Cp_{(ij)}\right) \times (1 + Bp_{(ik)} - Cp_{(ij)}), \quad (16)$$

where $p_{(ij)}$ designates the frequency (0 or 1) of allele A in individual j of group i and in its offspring. The frequency (0 or 1) of allele A in the neighbour and in the offspring of the neighbour of individual j of group i is designated by $p_{(ik)}$. The second line of eqn 16 represents the number of offspring produced by individual j in group i. This value depends on the cost of helping C and on the benefit B that individual j in group i receives from its group mate. The first line in eqn 16 represents the number of offspring produced by the offspring of individual j in group i. This number depends on the cost of helping and on the benefit received by offspring of individual j in group i, which depends on the average number of altruists in the group after the first period of reproduction. Accordingly, the offspring of individual j in group i may receive benefits from siblings and from the offspring

of the neighbour of individual j in group i. Since dispersal is complete, there is no relatedness between the two individuals initiating a group and the relatedness r between two offspring descending from the same mother (or the relatedness between mother and offspring) is equal to one given that individuals are haploid. We rewrite eqn 16 in terms of centred variables and substitute it into eqn 8 and set $m = 1$. Taking the expectation over all groups in the population and individuals within groups we have $E_{i,j}[\zeta_{(ik)}\zeta_{(ij)}] = 0$, because dispersal is complete, $E_{i,j}[\zeta_{(ij)}\zeta_{(hk)}]=0$ and $E_{i,j}[\zeta_{(ij)}\zeta_{(hj)}] = 0$ for all individuals from group h different than i because individuals from different groups are unrelated and $E_{i,j}[\zeta_{(ij)}\zeta_{(ij)}] = p(1-p)r$ by our definition of relatedness. We find that the change in gene frequency is given by:

$$\Delta p = p(1-p)\Big[-C(1+p\{B-(1+B)C+B^2p\}) + r(1-C+p\{B-C\})\left(\frac{1}{2}\{B(1-C)-2C\}+pB^2\right)\Big], \quad (17)$$

where the first term in the square brackets is the effect of helping of a FI of the parental generation on its fitness. The second line of this equation is the effect of the offspring bearing the parental gene lineage on the fitness of the focal parent. If the relatedness between two offspring descending from the same parent were equal to zero ($r = 0$), the direction of selection on the helping allele is negative at all gene frequencies. However, because this relatedness is equal to one ($r = 1$) in the model of Fletcher & Zwick (2004), the change in gene frequency can be positive and is given by:

$$\Delta p = p(1-p) \times \left[\frac{B(1-C)^2}{2} - C(2-C) + pB(3B-3(1+B)C+C^2+2pB^2)\right]. \quad (18)$$

When the helping allele is rare ($p \to 0$), helping spreads when:

$$\frac{B(1-C)^2}{2} > C(2-C) \quad (19)$$

is satisfied. Helping spreads when helping results in a direct fitness benefit for the focal gene lineage. Thus, Doebeli & Fletcher (2006) are wrong when they assert that helping spreads in the model of Fletcher & Zwick (2004) for a reason that is not accounted by our framework. If one conducts an analysis of the selective pressure on helping [which Fletcher & Zwick (2004) do not do], it appears that helping evolves by kin selection in the model of Fletcher & Zwick (2004), hence proving that it is valuable to try to understand models within the framework given in our Target Review.

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