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Group selection and kin selection: Two concepts but one process

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In a recent paper, Traulsen and Nowak use a multilevel selection model to show that cooperation can be favored by group selection in finite populations [Traulsen A, Nowak M (2006) *Proc Natl Acad Sci USA* 103:10952–10955]. The authors challenge the view that kin selection may be an appropriate interpretation of their results and state that group selection is a distinctive process “that permeates evolutionary processes from the emergence of the first cells to eusociality and the economics of nations.” In this paper, we start by addressing Traulsen and Nowak’s challenge and demonstrate that all their results can be obtained by an application of kin selection theory. We then extend Traulsen and Nowak’s model to life history conditions that have been previously studied. This allows us to highlight the differences and similarities between Traulsen and Nowak’s model and typical kin selection models and also to broaden the scope of their results. Our retrospective analyses of Traulsen and Nowak’s model illustrate that it is possible to convert group selection models to kin selection models without disturbing the mathematics describing the net effect of selection on cooperation.

Traulsen and Nowak (1) (T&N) present a multilevel selection model and demonstrate that a mutant helping allele can be favored to fixation, when introduced as a single copy in a population monomorphic for selfishness if

$$\frac{b}{c} > 1 + \frac{N}{n_g} + \frac{\lambda}{q}, \quad [1]$$

where c is the cost of helping, b the benefit of helping for group members (excluding the actor), N the group size, n_g the number of groups, λ the migration rate between groups, and q the probability of group splitting (T&N, inequality ineq. 2). In their conclusion, T&N challenge the view that kin selection is an appropriate interpretation of their results and state that:

“It would be interesting to see how the mathematical methods of kin selection can be used to derive our central results given by eqs. 1–3 and what assumption are needed for such a derivation. The problem is that typical methods of kin selection are based on traditional considerations of evolutionary stability, which are not decisive for games in finite populations.”

Further, in a recent comment on the various possible mechanisms leading to the evolution of cooperation, Nowak (2) states that the group selection model of T&N results in a different process than kin selection. These are surprising statements, given that many authors have emphasized that group selection models are not different from kin selection models (3–8), and that kin selection theory has been extended to finite populations that can follow very diverse demographic regimes (8–14). To us, the mechanism favoring cooperation in T&N’s model is clearly kin selection. Indeed, kin selection operates whenever interactions occur among genetic relatives, that is, among individuals who tend to share a more recent common ancestor than individuals sampled randomly from the whole population. This may happen when interactions take place within families before the dispersal of offspring, or when dispersal is limited (population structure), so that relatives remain near each other. This is clearly the case in the “group selection” scenario

considered by T&N, because dispersal is limited, interactions occur among relatives.

In this paper, we first address T&N’s challenge and carry out a retrospective analysis of their model by deriving ineq. 1 using the kin selection approach for finite populations developed by Rousset (8, 15). Next, we consider a slightly different life cycle that resembles more the life cycles usually represented in classical kin selection formalizations. This allows us to highlight the few differences and broad similarities between T&N’s model and “typical” kin selection models. This second formalization suggests that ineq. 1 in fact holds for a large spectrum of life cycles, provided that $M_e \equiv \lambda/q$ is interpreted as the “effective number of migrants.”

Results

T&N’s Model. To derive ineq. 1 by using inclusive fitness theory (16), we endorse exactly the same assumptions as T&N. The population is subdivided into n_g groups, which grow in size as individuals within them reproduce. In any one time step, a single individual from the entire population is chosen for reproduction with a probability proportional to its payoff. When a group has reached a threshold size N , it either divides into two daughter groups with probability q (in which case a random group from the population is eliminated), or it does not divide (with complementary probability $1 - q$), in which case a random individual in the group is eliminated. Social interactions occur only among members of the same group, and individuals bearing a mutant allele (say A) express an act of helping, which decreases their payoff by c and generates a benefit b , which is shared by all other group members (thus excluding the actor). Selfish individuals then tend to replicate faster than helpers within groups, but groups comprising helpers grow faster and have a greater chance of dividing before going extinct. T&N also introduce migration between groups, by assuming that once an individual has reproduced, one of its offspring may migrate to another group with probability λ . If the group exceeds the critical size N after the arrival of the migrant, the group splits with probability q , or a random individual is eliminated from the group.

T&N analyze their model in the limiting case where both migration and group division are very rare ($q \ll 1$ and $\lambda \ll 1$), so they can assume in their calculations that the population behaves as if all its groups constantly remain at the threshold size N . With these assumptions, fixation within groups occurs on a faster time scale than migration and group division. The fixation probability π of a newly arisen mutant in the population can then be expressed as the product of the fixation probability π_g of a single mutant in a group (before migration or group division occurs) times the fixation

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Abbreviations: T&N, Traulsen and Nowak (ref. 1); ineq., inequality.

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probability π_p of the mutant at the level of the population conditional on its fixation at the level of a single group (e.g., T&N). The direction of evolutionary change of the mutant can then be determined by asking whether it has a larger or smaller probability of fixation than a single neutral mutant (8, 9, 17). The change of the fixation probability for a mutant with small phenotypic deviation δ relative to the resident allele (weak selection) is given by the derivative of the probability of fixation with respect to the phenotypic deviation δ , evaluated at $\delta = 0$. This yields

$$\frac{d\pi}{d\delta} = \frac{d\pi_g}{d\delta} \pi_p^\circ + \frac{d\pi_p}{d\delta} \pi_g^\circ, \quad [2]$$

where $\pi_g^\circ = 1/N$, and $\pi_p^\circ = 1/n_g$ are the fixation probabilities in the absence of selection (neutral case; that is, $\delta = 0$).

In the supporting information (*SI Appendix*), we show that Eq. 2 represents, in fact, a specific application of the inclusive fitness framework for finite populations of Rousset and coworkers (8, 9, 14, 15). Further, the effects of the mutant on its probability of fixation both at the level of a single group ($d\pi_g/d\delta$) and at the level of the population conditional on its fixation at the level of a single group ($d\pi_p/d\delta$) can be calculated by the direct fitness method for constructing kin selection models (6, 9, 18). In particular, Eq. 19 of *SI Appendix* reveals that the effect of the mutant on its probability of fixation at the level of a single group can be expressed as

$$\frac{d\pi_g}{d\delta} = \frac{\partial w_{ij}^g}{\partial z_{ij}} K_g, \quad [3]$$

where w_{ij}^g is a direct fitness function giving the expected number of individuals descending from individual j in group i (possibly including ij himself) between two reproductive events causing neither a migration nor a group division, z_{ij} is the phenotype of individual ij , and K_g is a positive constant that depends on demographic assumptions (e.g., iteroparity vs. semelparity, group size). Because reproduction occurs at a faster time scale than migration or group division, the function w_{ij}^g measures fitness on a “small” time interval, whereas migration or group division can be seen as being spaced by a “large” time interval. Eq. 3 informs us that the direction of selection on the probability of fixation of a single mutant at the level of the group depends only on the effect of the phenotype of individuals on their own fitness ($\partial w_{ij}^g/\partial z_{ij}$) and is independent of the behavior of the other individuals from the group. Because by definition the act of helping results in a fitness cost, selection disfavors the fixation of helping at the level of the group.

Eq. 21 of *SI Appendix* shows that the effect of the mutant on its probability of fixation at the level of the population can be written as

$$\frac{d\pi_p}{d\delta} = \left(\frac{\partial w_{ij}^p}{\partial z_{ij}} + \frac{\partial w_{ij}^p}{\partial z_i} R \right) K_p, \quad [4]$$

where w_{ij}^p is a direct fitness function giving the expected number of individuals descending from individual j in group i (possibly including ij himself) between two reproductive events causing either a migration or a group division. The function w_{ij}^p thus measures fitness on the “large” time interval, during which many reproductive events have occurred within groups, so that all groups are genetically monomorphic by the time migration or group division occurs. Eq. 4 also depends on z_i , which is the average phenotype of individuals in group i , R , which measures by how much two individuals randomly sampled from the same group are more related than two individuals sampled randomly from the whole population (Eq. 11 of *SI Appendix*), and K_p , which is a positive constant depending on demographic assumptions on the population (e.g., migration, population size).

Eq. 4 tells us that the effect of the mutant on its probability of fixation at the level of the population now depends both on the

effect $\partial w_{ij}^p/\partial z_{ij}$ of the phenotype of the individual on its fitness and the effect $\partial w_{ij}^p/\partial z_i$ of the mean group phenotype on the focal individual's fitness, weighted by the coefficient of relatedness among group members. Substituting the fitness effects (Eqs. 3 and 4) into the measure of selection $d\pi/d\delta$ (Eq. 2) reveals that this equation fits within Hamilton's definition of the inclusive fitness effect (16), which is a relatedness-weighted sum of the effects of the phenotypes of different actors on the fitness of a focal individual. The mutant allele is selected for when the inclusive fitness effect is positive (16), which is when $d\pi/d\delta > 0$.

A consequence of the assumptions of T&N's model is that relatedness is always equal to one ($R = 1$) and thus takes its maximal value. Indeed, because migration and group division are very rare, all lineages from a group trace back to a single ancestor during the time between two migration/division events. This result in a situation where the effect of migration affects not relatedness but only the fitness function w_{ij}^p and the weight K_p of the inclusive fitness effect. Following T&N's life cycle assumptions, we evaluate, in the *SI Appendix*, the fitness functions w_{ij}^g and w_{ij}^p ; and the two weights K_g and K_p . We find that the inclusive fitness effect is positive when

$$\frac{b}{c} > 1 + \frac{N + \frac{n_g \lambda}{q}}{n_g - 2 - \frac{\lambda}{Nq}}, \quad [5]$$

which is precisely ineq. 34 of T&N. This inequality represents their main and exact weak selection result, which holds for any group size and number (see Eq. 43 of *SI Appendix*). If $n_g \gg 1$ and $N \gg 1$, it simplifies to ineq. 1. Our derivation of ineq. 5 by the direct fitness method allows us to illustrate that it is possible to translate group selection models to kin selection models without disturbing at all the mathematics describing the net effect of selection on helping behaviors.

Typical Kin Selection Model. The assumptions used by T&N to obtain ineq. 1 imply that individuals are iteroparous, with no more than one individual dying per unit time, and that a breeding individual produces only one offspring (i.e., the Moran process). By contrast, traditional multilevel and kin selection models rely on different life-history assumptions, namely that all adult individuals produce at the same time a very large number of juveniles and then die (3, 8, 19–24). To complement T&N's analysis and to highlight the similarities and differences between their model and typical kin selection models, we derive a multilevel selection model for a finite population with nonoverlapping generations. We consider a population where individuals live in n_g groups of constant size N that are connected by migration. Each individual produces a large number of juveniles, the exact number depending on its own phenotype and the phenotypes of other group members. As in T&N's model, individuals bearing a mutant allele express an act of helping, which decreases their fecundity by c and increases the fecundity of their neighbors by b . All adults die, and juveniles from the same group assemble to form daughter groups of size N . The number of daughter groups produced by a group is proportional to the average fecundity in the group, and the individual contribution to a daughter group is proportional to individual fecundity relative to group fecundity. Groups then compete against each other to form the next generation of adults, which migrate randomly with probability λ to another of the n_g groups. As in T&N's model, the entire evolutionary dynamics is driven by individual fitness, and the effect of a mutant on its probability of fixation is given by

$$\frac{d\pi}{d\delta} = \left(\frac{\partial w_{ij}}{\partial z_{ij}} + \frac{\partial w_{ij}}{\partial z_i} R \right) K, \quad [6]$$

where w_{ij} is the fitness function giving the expected number of successful offspring of individual j in deme i , and K is a positive constant depending on the demographic assumptions on the population (Eq. 10 of *SI Appendix*). The function w_{ij} measures fitness between two reproductive events, which occur at exactly the same time scale as migration and group division. The crucial difference between Eq. 6 and the inclusive fitness effect obtained for T&N's model (Eq. 4) is the way relatedness affects selection on the mutant allele. Because of the separation of the time scale between fixation within groups and migration or group division, relatedness is always equal to one during the period of competition between groups in T&N's model. By contrast, relatedness depends on the demographic parameters of the population in the typical kin selection model, because fixation at the level of the group is very unlikely to precede migration or group division. For large deme size and low migration ($n_g \gg 1$ and $\lambda \ll 1$ and see Eq. 50 in *SI Appendix*), relatedness is given by

$$R = 1 - (N - 1) \left(\frac{1}{n_g} - 2\lambda \right). \quad [7]$$

Relatedness takes its maximum value when there is a very large number of groups ($n_g \rightarrow \infty$), and when migration is absent ($\lambda = 0$). Relatedness decreases as n_g decreases, because individuals from different groups may bear the same allele inherited from a common ancestor. Substituting the relatedness and the fitness function (Eq. 51 in *SI Appendix*) into the inclusive fitness effect (Eq. 6) reveals that, if $n_g \gg 1$ and $\lambda \ll 1$, helping spreads when

$$\frac{b}{c} > 1 + \frac{N}{n_g} + M_e, \quad [8]$$

where $M_e = 2N\lambda$ is the average number of migrants in a group. This condition of invasion is similar to ineq. 1, with the only difference that the number of migrants is given by $M_e = \lambda/q$ in T&N's model. This difference is explained by the differences in life cycles. Indeed, in our semelparous model, group splitting occurs in each generation (and all group splits), and all individuals can migrate. Relatedness then depends on the square of the migration rate and on group size (see Eq. 48 in *SI Appendix*), yielding $2N\lambda$ as the number of migrants, as is usually the case for haploid models with population structure (8, 25, 26). Our results suggest that provided M_e is interpreted as the "effective number of migrants", and that ineq. 8 applies to a continuum of life cycles, ranging from few group divisions and migrations occurring per unit time to frequent ones and from only one or a few individuals reproducing per unit time (overlapping generations) to all individuals in the population reproducing (non-overlapping generations). This is so because, in all these situations, competition occurs strictly and randomly between groups. Hence, the various within-group demographics will affect only quantitatively the condition under which selection favors helping. So long as competition occurs randomly between groups, ineq. 8 should also apply to simple metapopulation models with extinction and recolonization of groups, to other genetic systems (e.g., diploidy and haplo-diploidy), and to other mating systems (e.g., selfing and polygyny), because all these variations will affect only the effective number of migrants M_e , through the alteration of the effective migration rate and effective group size (8).

Our analyses also allow us to clarify why helping evolves in T&N's model, and how it relates to previous kin theoretical models for the evolution of helping in subdivided populations. Helping evolves in the two multilevel selection models analyzed in this paper, because competition occurs between groups *sensu stricto*. Indeed, the round of competition between groups that follows a group division event occurs at the level of the population, so that local competition between individuals for resources is prevented to occur. This results in a demographic situation where kin competition cannot hamper the spread of helping, a result that our typical kin selection model

suggests is true whatever the life-history assumptions concerning individuals within groups (e.g., overlapping generations vs. non-overlapping generations and haploidy vs. diploidy). By contrast, when competition occurs *sensu stricto* between individuals, genetically related neighbors are also more likely to compete for the same local resources because some individuals remain philopatric during the round of competition (see ref. 27 for a direct comparison between models involving competition between groups or between individuals). In this case, kin competition can at least partially offset the benefits of helping. For instance, helping at a fecundity cost to the actor is selected for under overlapping generations (28), but the fecundity benefits of helping are completely canceled out by the concomitant increase in kin competition under nonoverlapping generations (23, 29, 30).

Conclusion

We derived in this paper two multilevel selection models for the evolution of helping in finite populations. The first is a retrospective analysis of the model of T&N, who suggest that helping does not evolve through kin selection in their formalization. Using inclusive fitness theory (16), we recovered the main result of T&N as a specific application of kin selection theory for structured populations of finite size (8, 9, 14, 15). It is quite obvious that the mechanism that allows cooperation to evolve under T&N's life cycle is kin selection; interactions occur within groups, and individuals from the same group are related (i.e., they share a more recent common ancestor than individuals sampled randomly from the whole population). Hence, T&N's model falls into the scope of Hamilton's inclusive fitness theory, which is a general method for analyzing selection that can also be used to study the evolution of social interactions among nonkin (31, 32).

To illustrate the similarities and differences between T&N's life cycle with overlapping generations (the Moran scheme of reproduction) and more typical kin selection formalizations, we developed a multilevel selection model for nonoverlapping generations with frequent group divisions and arbitrary migration rates (3, 19–24). The analysis of this model suggests that ineq. 8 in fact holds for a variety of life cycles ranging from rare group divisions and migrations occurring per unit time to frequent ones and from overlapping to nonoverlapping generation situations, provided the parameter M_e is interpreted as the "effective number of migrants." Further, so long as competition occurs randomly and strictly between groups, ineq. 8 will also apply to simple metapopulation models with extinction and recolonization of groups and to other genetic systems and mating systems, because all these variations will affect only the average number of migrants M_e . To us, the two models investigated in this paper do not represent different mechanisms for the evolution of helping behaviors but only involve different sets of life-history assumptions and approximations, of which there are infinitely many.

The statement that helping evolves by a different mechanism ("group selection" or "multilevel selection") will only bring confusion in a domain of knowledge that was rather clear 20 years ago. This emphasizes the need for researchers to relate their work to the existing literature, especially when discussing the possible novelty of mechanisms leading to the evolution of cooperation and altruism (33, 34). To avoid semantic confusion both within and across disciplines (35), it appears more useful to reckon that, whenever interactions occur at a local spatial scale, and dispersal is limited, then interactions occur among genetic relatives, and thus kin selection is operating. Finally, it is also important to keep a tight link between modeling and biology and to develop models that are aimed at representing life cycles that occur in nature. As was pointed in a recent comment on social evolutionary theory (36), "For the study of cooperation and altruism, we think that the time has come to value work more highly that brings theory and observation into closer contact, compared with work that merely adds another twist to modeling."

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