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The selection of social actions in families: I. A collective fitness approach

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ABSTRACT

Here I analyse the basic properties of relatedness, inclusive fitness and kin selection, and derive alternative ways of formulating the selection of social behaviours. In particular, a 'collective fitness' rule is sought, which determines the direction of selection on a gene in terms of the effect an act has on the fitness of various individuals in a population, and the number of copies of the 'action' allele in various individuals in the population. The use of collective fitness allows for all the simplifications in kin-selection explanations of social behaviour that use inclusive fitness. In addition, it covers analyses of social behaviours in which factors other than kinship influence the distribution of genes in interacting individuals, without invoking an open-ended expansion of the concept of 'relatedness'. The treatment of social actions ends with a discussion of how far the concepts of relatedness, inclusive fitness and kin selection should be extended when factors other than kinship contribute to the distribution of genes to actors and recipients.

Keywords: collective fitness, inclusive fitness, kin selection, relatedness, social behaviour.

INTRODUCTION

Until 30 years ago, evolutionists thought of selection only as a process that maximizes the number of direct descendants that individuals or their genes leave – their lineal fitness. The scope of natural selection was broadened dramatically by the introduction of the concept of inclusive fitness by W.D. Hamilton (1963, 1964). Following earlier undeveloped insights by Fisher (1930) and Haldane (1932, 1955), Hamilton recognized that the behaviour of an individual can affect not only its own fitness, but also that of other conspecific individuals. If the other affected individuals are relatives, a proportion of them will inherit the same gene from their common ancestor by virtue of their kinship. Hamilton postulated that the selection of social behaviours takes these non-lineal fitness effects into account and maximizes the 'inclusive fitness' of individuals. Inclusive fitness comprises the base fitness of individuals in the absence of a social behaviour plus the effect on the fitness of the 'actor' and effects on the fitness of 'recipients' weighted by the genetic relatedness of the actor to the recipient. In its simplest and most common formulation as identity by descent (IBD), the relatedness, r, measures the probability that a gene in the actor will also be present in a recipient by descent from a common ancestor possessing the gene.

The concept of kin selection, as it was named by Maynard Smith (1964), received a large boost from its success in explaining why most eusocial animals with sterile castes are haplodiploids. In such species, female colony members are more closely related to their sibs ($r = 3/4$ if they are full sibs, since they have all their haploid father's genes and half their diploid mother's) than to their offspring ($r = 1/2$). Consequently, females propagate their own genes more effectively by aiding their sisters than by producing their own eggs.

Kin selection was soon applied to a variety of animal behaviours, including parental care, cooperative breeding, mimicry and many other postulated kinds of altruism or selfishness. There have been fewer applications of kin selection to plants. Considerable attention has been paid to the evolution of endosperm, the sterile tissue that nurtures developing angiosperm seeds (Charnov, 1979; Westoby and Rice, 1982; Queller, 1984). Applications of kin selection to other plant contexts, such as self-incompatibility (Hamilton, 1987) and parental care, have been more limited.

Kin selection has extended the scope of the theory of natural selection more than any other innovation since the publication of Charles Darwin's *Origin of Species* in 1859. Several factors have contributed to the impact that the concept of kin selection has made:

1. It broadened the applicability of natural selection by adding a new component, the effect that an individual's behaviour has on the fitness of relatives. This opened up a whole new area for selection studies, the social behaviour of plants and animals.
2. The results are often expressed as simple inequalities, such as Hamilton's Rule for altruism, which predict the direction of selection in terms of the costs and benefits to the interacting participants and only one other parameter, relatedness. The base fitness in the absence of the behaviour does not have to be stated or even known.
3. Kin selection primarily traces the selection of genes, because it is the distribution of genes that determines the relative importance of the effects of a social action on actors and recipients. When single copies of genes are assumed to act autonomously, the exact mode of inheritance of the genes responsible need not be specified explicitly.
4. Like other autonomous gene hypotheses, kin selection can be readily expressed in terms of the behaviour of individuals, as in Hamilton's original exposition. This is a tremendous boon, since evolutionary ecologists are usually more interested in the individuals carrying out a behaviour than in the genes controlling it.
5. The process of kin selection is (in the absence of complicating factors) independent of the dosage and frequency of the alleles controlling the behaviour. Thus one model serves for all allele frequencies.

Together, these factors give the theory of kin selection a degree of elegance and simplicity that is rarely attained in adaptationist hypotheses. A large variety of situations can be treated quantitatively with only three variables – the effects on the fitness of two kinds of interacting individuals and their relatedness. When one adds the ingenuity and novelty of the explanations, it is not surprising that kin selection has become a major part of the theory of adaptive strategies.

Here I analyse the basic properties of relatedness, inclusive fitness and kin selection, and derive alternative ways of formulating the selection of social behaviours. The treatment of social actions ends with a discussion of how far the concepts of relatedness, inclusive fitness and kin selection should be extended when factors other than kinship contribute to the distribution of genes to actors and recipients.

SOCIAL ACTS

In a social act, an actor performs a behaviour that affects directly the fitness of two or more genetically correlated individuals. At least one of the individuals must be someone other than the actor – a recipient. The actor is usually, but not necessarily, one of the affected individuals. Thus behaviours affecting only individuals *other than the actor*, such as offspring (as in parental investment strategies) or sibs (as in the nurturing behaviour of endosperm), are included here as social acts.

Social acts require special consideration only if the action alleles are more likely to be present in recipients than in the population as a whole – that is, the interactants are genetically correlated. If recipients have a random sample of alleles of action loci, the relative frequencies of the alleles in recipients are identical to the frequencies in the population as a whole. Then, the effects of the behaviour on the recipients cause no change in allele frequencies and are of no evolutionary significance. The most frequently treated cause of genetic correlations between actors and recipients is genetic relatedness, where genes are transmitted to both interactants from their common ancestor or ancestors. A correlation also arises when actors recognize their own phenotype (and genotype) in recipients, the 'greenbeard' phenomenon (Dawkins, 1982), as in the acts of cooperation. The pleiotropic action of an action allele might also cause a genetic correlation. For example, if carriers of action alleles tend to congregate in a particular environment, they may interact with each other non-randomly (Hamilton, 1978). Sources of correlations other than kinship have not received much attention, however.

The following accounts of social acts rely heavily on the concept of the collective fitness of an allele (Lloyd, 1992). This is simply the usual concept of the average fitness of an allele extended to social acts so that non-random effects on carriers other than the actor are incorporated. The collective fitness of an allele is obtained by tracing directly the copies of the allele in actors and recipients from the source of the genetic correlation, an ancestor in the case of interactions between relatives. To calculate collective fitness, the effects on the interactants are weighted by the number of allele copies present in each and added to the base fitness in the absence of the social act. In contrast, an inclusive fitness measure of average fitness weights the effect on an actor as one and that on recipients by a relative fraction, the relatedness.

The two measures of fitness are merely different ways of accounting. Collective fitness calculates the relative numbers of action alleles in actors and recipients by using the common ancestor as the reference point and calculating the distribution of alleles from the ancestor to each descendant directly, using the principles of inheritance. On the other hand, inclusive fitness does the corresponding calculation by starting at the actor, tracing inheritance back and forth in time from the actor to the ancestor then to the recipient, and encapsulating the relative distribution of action alleles to an actor and a recipient in a single measure of relatedness.

Collective fitness and inclusive fitness provide alternative, equally acceptable formulations of selection events, but they are not necessarily identical in economy, applicability or the insights they provide. Inclusive fitness has the merit that it looks at the joint inheritance of action alleles from the actor's point of view, seemingly simplifying the description of selection events. But it does so by invoking an additional parameter, relatedness, and we will see that the interpretation of relatedness is not always as straightforward as some commonly used explanations assume.

Both inclusive fitness and collective fitness allow simplifications of descriptions of selection by confining attention to the participants involved in a social act. In this respect, they differ from frequency-modulated fitness, which was introduced by Hamilton (1964) and incorporates the effects of a social act into calculations of the fitness of all members of a population in population genetics formulations of events.

The collective fitness of a gene is a convenient measure for combining the fitness effects of a social act on genes carried in actors and recipients. The use of collective fitness allows all the fortunate simplifications that were described above for kin selection explanations of social behaviour that use inclusive fitness. In addition, it covers the analysis of social behaviours in which factors other than kinship influence the distribution of genes to interactants, without invoking an open-ended expansion of the concept of 'relatedness'.

THE COLLECTIVE FITNESS RULE

We seek here to obtain an expression that determines the direction of selection on a gene in terms of the effect an act has on the fitness of various individuals in a population and the numbers of copies of the 'action' allele (the allele that controls the act) in the various individuals.

The fitness of an allele may be defined as the average number of descendants, exactly one generation later, of each copy of the allele in a population. It follows from the definition that a fitter allele (one with more descendant copies per original copy) will increase in frequency from one generation to the next at the expense of a less fit allele.

Suppose that, at a locus controlling an act, there is a 'null' allele with fitness w_0 in the absence of social acts, and an 'action' allele that when expressed has effects, e_i, on the fitness of the ith individual in the population. The effects are positive benefits or negative costs and are experienced by the actor expressing the action allele and/or by other conspecific individuals (i.e. recipients) who need not be relatives. The affected individuals may differ in reproductive value, v_i, the relative contribution (independent of the acts) that a class of individuals makes to the future gene pool of the population (Fisher, 1930). Suppose that in the ith individual, g_i copies of the action allele have their fitness affected by the social act. If m individuals are affected, the average (collective) fitness of the action allele is

$$\bar{w}_a = \bar{w}_0 + \frac{1}{m}\left(\sum_{i=1}^{m} e_i g_i v_i\right)$$

Collective fitness, like inclusive fitness, incorporates not only the lineal fitness effects on the actor, but also non-lineal effects on other individuals.

The action allele increases in frequency when its collective fitness is greater than that of the null allele. The allele has a fitness advantage, $\bar{w}_a - \bar{w}_0 > 0$, when

$$\sum_{i=1}^{m} e_i g_i v_i > 0 \qquad (1)$$

This is the 'collective fitness rule' for single acts. The action allele is selected if the net effect of the social act on its fitness, obtained by weighting the separate effects by the number of copies of the allele experiencing them, is positive. The rule shows that selection favours the allele with the highest advantage in collective fitness, $\bar{w}_a - \bar{w}_0$, but does not necessarily increase the absolute collective fitness, $\bar{w}$.

If there are a number, $j = 1, 2, \ldots, s$, of social acts and their effects combine additively,

$$\sum_{j=1}^{s} \sum_{i=1}^{m} e_{ij} g_{ij} v_{ij} > 0 \tag{2}$$

This is the collective fitness rule for multiple acts.

The outcome of selection on the collective fitness of alleles can be obtained by confining attention to the effects of the social action. The non-social fitness components common to all alleles disappear when the fitness advantage of the action allele is obtained by subtraction. This simplification parallels the description of selection as an inclusive fitness advantage in Hamilton's rule.

The collective fitness rule describes the outcome of selection in terms of its effects on *genes* in the first instance, but the explanations can also be worded in terms of individual actions when the conditions for phenotypic models (particularly constant gene effects) are met. The rule underlies the selection of all social behaviours, regardless of their nature or genetical basis. In many behaviours, including most of those analysed in the papers here, the reproductive values of the participating individuals are approximately equal or are of minor interest. If they are assumed to be equal, the v's drop out of the inequality, giving a simpler rule:

$$\sum_{i=1}^{m} e_i g_i > 0 \tag{3}$$

In the simplest case, the rule can be applied to non-social acts that affect only their carriers and random members of the population. Then, the individuals that perform a behaviour receive all the effects. There is no distinction between actors and recipients, and only one entity is considered. That is, for phenotypic or autonomous gene models of non-social behaviours, $g = 1$ and a phenotype or allele is selected if

$$\sum_{i=1}^{m} e_i > 0 \tag{4}$$

The net effect of a phenotype, or of the allele that determines it, is maximized.

Returning to social acts, an actor, a, and recipient, t, experience different effects on their fitnesses, e_{a} and e_{t}, and contain g_{a} and g_{t} copies of the action allele respectively. When a single act involving one actor and any number of recipients is performed and all participants have the same reproductive value, the collective fitness rule

$$\sum_{i=1}^{m} e_i g_i > 0$$

becomes

$$e_{\mathrm{a}} g_{\mathrm{a}} + \sum_{t=1}^{T} e_{\mathrm{t}} g_{\mathrm{t}} > 0 \tag{5}$$

where T is the number of recipients.

When the act is repeated and the multiple effects are additive, social acts are selected when

$$\sum_{j=1}^{s} \left(e_{\mathrm{a}j} g_{\mathrm{a}j} + \sum_{t=1}^{T} e_{\mathrm{t}j} g_{\mathrm{t}j} \right) > 0 \tag{6}$$

The principal interest lies in those situations in which one type of participant receives a benefit and the other a cost. In altruistic acts, actors receive a negative increment to fitness, a cost $-c_a$, while the recipient(s) receive a positive benefit, b_t. With a single act and recipient, (5) becomes

$$-c_a g_a + b_t g_t > 0$$

or

$$\frac{b_t}{c_a} > \frac{g_a}{g_t} \tag{7}$$

In selfish acts, the actor receives a benefit, b_a, while the recipient experiences a cost, $-c_t$. With a single act and recipient, (5) becomes

$$b_a g_a - c_t g_t > 0$$

or

$$\frac{b_a}{c_t} > \frac{g_t}{g_a} \tag{8}$$

The selection of altruistic or selfish acts is determined by the effects on the two participants and the relative numbers of controlling alleles they contain. The ratio of those numbers weights the two effects, so that they can be combined to determine the outcome of selection. We must therefore examine the determinants of that ratio.

WEIGHTING ACTOR AND RECIPIENT EFFECTS

Suppose that the recipients are relatives of the actors. Relatives are more likely than a randomly sampled member of the population to possess the action allele, because the allele may be transmitted to them from a common ancestor that gave the allele to the actor. Consider first a single act controlled by a rare allele. It is shown later that selection is frequency-independent when kinship alone controls the distribution of alleles. And, if the effects of repeated acts are constant (they combine additively), the outcome of interactions between particular relatives can be analysed by considering a single act. For a rare allele controlling one act towards a relative,

$$\frac{g_t}{g_a} = \left[\frac{\text{probability recipient gets action allele from the copy in the common ancestor}}{\text{probability actor has the action allele}} \right]$$

The denominator is 1, since the actor must have the action allele. The probability that an action allele in the recipient was inherited from the same copy in the common ancestor that provided the actor's copy depends on both the probability that the allele in the actor came from the common ancestor, $P(\mathrm{A})$, and the probability that the ancestor passed the same allele copy onto the recipient, $P(\mathrm{T})$. If the probability that a recipient inherits an action allele from an ancestor, $P(\mathrm{T})$, is independent of the probability that the actor does, the probability of both events happening jointly is the product of their separate probabilities – that is, $P(\mathrm{A}) \times P(\mathrm{T})$. Then

$$\frac{g_t}{g_a} = P(A) \times P(T) \tag{9}$$

When there is more than one common ancestor, the overall probability of the recipient getting an allele identical by descent (IBD) with that in the actor is the sum of the joint probabilities for each common ancestor (for complex pedigrees, each of the $c = 1, 2, \ldots, x$ connecting pathways in IBD calculations of relatedness) – that is, $\sum_c (P(A) \times P(T))$. Then

$$\frac{g_t}{g_a} = \sum_c [P(A) \times P(T)] \tag{10}$$

If it is further assumed that the only factors that determine the probability that any descendant gets an action allele from an ancestor are Mendel's Law of Segregation and the number of meioses between the ancestor and its descendant, then $P(A) = (1/2)$ and $P(T) = (1/2)$, where n_a and n_t are the numbers of generations separating the common ancestor from the actor and the recipient respectively. Then

$$\frac{g_t}{g_a} = \sum_c (½)^{n_a + n_t} = \sum_c (½)^n = r_{at} \tag{11}$$

where n is the total number of $(n_a + n_t)$ generations separating the actor and recipients through a common ancestor, and r_{at} is the IBD relatedness of the actor to the recipient.

In deriving equation (11), we have shifted from counting the numbers or probabilities of action alleles in actors and recipients, as in the forms of the collective fitness rule (equations 1–5), to counting the recipient copies from the perspective of the actor by a measure of relatedness. This is the essential difference in the accounting procedures used in calculating collective and inclusive fitnesses.

Substituting from (11) into (7) gives Hamilton's rule for the selection of altruism:

$$\frac{b_t}{c_a} > \frac{1}{r_{at}} \tag{12}$$

For a single act of selfishness, similarly substituting from (11) into (8) gives

$$\frac{b_a}{c_t} > r_{at} \tag{13}$$

An inclusive fitness rule for social acts can be obtained by dividing the collective fitness rule (5) throughout by g_a and substituting r_{at} for g_a/g_t. A social act is selected if

$$e_a + \sum_t e_t r_{at} > 0 \tag{14}$$

For multiple acts that combine additively, similarly dividing (6) throughout by g_a and substituting r_{at} shows the acts are selected if

$$\sum_j \left(e_{aj} + \sum_t e_{tj} r_{atj} \right) > 0 \tag{15}$$

This long set of derivations does more than prove the basic rules for the selection of social acts among relatives. It shows the conditions under which relatedness calculated as identity by descent gives, *by itself*, the appropriate weighting for effects on the participants.

In particular, relatedness must be the only factor affecting the relative number of copies of an action allele in actors and recipients. It also confirms that obtaining fitness advantages by using inclusive fitness or collective fitness measures are equivalent procedures. A calculation of identity by descent through a pathway connecting an actor to a recipient has two parts. The probability that genes have descended identically depends on the probability that the action allele in the actor came from a common ancestor, $P(\mathrm{A})$, and the probability that the allele in that ancestor also went to the recipient, $P(\mathrm{T})$. The calculation of relatedness as identity by descent combines the two components by using the actor as the starting point and working backwards in time to the ancestor and then forwards to the recipient. A calculation of collective fitness incorporates the same probabilities as probabilities of descent from the common ancestor. Both types of computation accurately record the distribution of action alleles to actors and recipients when these are controlled solely by kinship. Collective fitness and inclusive fitness then provide alternative means of weighting fitness effects on actors and recipients. The equivalence of inclusive fitness and collective fitness formulation is parallel to that shown for inclusive and neighbour-modulated fitness by Abugov and Michod (1981).

Calculations of collective fitness continue to provide an accurate account of natural selection in situations where the distribution of alleles from a common ancestor is not determined solely by kinship, as we will see later. But first we must look more closely at the limitations of inclusive fitness models that use the IBD method of calculating relatedness.

IDENTITY BY DESCENT FORMULATIONS

The preceding derivation of the rules of kin selection identifies a number of assumptions underlying the use of identity by descent measures of genetic relatedness.

1. The action alleles operate autonomously. An autonomous gene model, in turn, assumes that the effects of the action allele are constant whenever they are present. The fitness effects on the recipients are constant only if relatedness is dosage- and frequency-independent (see below). When the effects on all interactants are constant, the selection interests of the action allele and the individuals expressing it coincide and selection can also be described as acting on individual phenotypes.
2. All copies of the action allele in actors and recipients are inherited from a common ancestor independently of each other and randomly. This further assumes that: (a) the probability that an action allele is transmitted from a common ancestor to a recipient is independent of the probability that it is transmitted to the actor; (b) if more than one action allele is transmitted from an ancestor to the actor (as in endosperm) or a recipient, the multiple copies are inherited independently; (c) the roles of actors and recipients are determined separately (this is not so for mutual cooperation); and (d) meiosis is unbiased. Only if these four assumptions are met are the probabilities $P(\mathrm{A})$ and $P(\mathrm{T})$ fixed for any pedigree as

$$P(\mathrm{A}) = (1/2)^{n\mathrm{a}} \text{ and } P(\mathrm{T}) = (1/2)^{n\mathrm{t}}$$

3. The calculation of fixed probabilities also assumes that selection is weak, as Hamilton (1964) recognized in his pioneering exposition of kin selection. The operation of natural selection causes the alleles to be transmitted at different rates.

4. There are no other factors altering the transmission of genes between generations, such as non-random mating, genetic drift or population substructure.
5. The inequalities are readily extended to multiple acts only if the costs and benefits combine additively; otherwise, the analysis is more complicated (Queller, 1985, discusses the treatment of synergistic effects).

Much of the attractiveness of kin selection explanations comes from their simplicity. This stems in part from the property that genetic relatedness provides the only weighting required to combine costs and benefits into a selection model. In the identity by descent representation of relatedness, relatedness depends only on the pedigree connecting the participants. It is calculated by a simple, intuitively obvious method. This form of relatedness means just one thing, kinship – how one individual is genealogically connected to another. The elegant simplicity of IBD formulations of kin selection is only possible if all the above assumptions apply.

In the generation since the concept of kin selection was expounded by Hamilton, considerable attention has been paid to departures from some of the above assumptions, especially those due to inbreeding and other genetical effects such as parent-specific gene expression (Michod, 1979; Grafen, 1985; Queller, 1989) and the additivity of multiple acts (reviewed in Queller, 1989). On the other hand, the assumption that all copies of action alleles in actors and recipients are inherited independently and randomly has received little attention.

When the probability of genes being passed from an ancestor to each of its descendants in a particular genealogy depends solely on Mendel's Law of Segregation, as in the derivation of equations (12)–(15), the alleles of all loci are transmitted with identical probabilities. The probability that an action allele in an actor is also present in a recipient then coincides with the *proportion of the actor's genes* that are identical by descent in the recipient. In these circumstances, the action allele and shared genes representations of relatedness give numerically identical results. But they are not biologically interchangeable. An actor with an action allele helps or harms a relative in part because the relative has a certain probability of possessing the action allele, not because they share a particular fraction of their genes. This fraction is irrelevant to the selection of the act, which depends entirely on the alleles of action loci controlling the behaviour. Nevertheless, many loosely worded expositions of kin selection have relied on the shared genes interpretation of kinship.

FREQUENCY-INDEPENDENCE OF KIN SELECTION

The simplicity of kin selection depends on the fortunate property that the conditions for selection of a social act are independent of the dosage and frequency of the action allele. These features have been demonstrated formally by Hamilton (1964), Grafen (1985) and others, and are particularly clearly presented by Trivers (1985). They can be appreciated from a simple verbal argument. The inclusive fitness representation of the selection of social acts is based on the assumption of an autonomous action allele. An autonomous allele that controls a social act may be associated in the genotype of the actor with a second copy of the action allele or with a copy of the null allele. The frequencies of the two types of allele partners will be the frequencies of the alleles in the population. Similarly, the frequencies of alleles (other than those that are identical by descent) in recipients will be

the population frequencies. Hence the effects of the social act will be experienced equally by the randomly associated action alleles and null alleles in the actor and recipients, except for the copies inherited from the common ancestor. The net effects of a social act are confined to the autonomous action allele and copies of the same allele that are identical by descent in related recipients. Thus selection is dosage- and frequency-independent. The frequency-independence of kin selection is essentially a corollary of the autonomous gene assumption.

Derivations of the basic rules of social behaviour that use the IBD representation of relatedness,

$$r = \sum_{c} (\tfrac{1}{2})^{n}$$

rule out the operation of any frequency-dependent evolutionary factors *in the calculation of relatedness*. The formula for r assumes that it is determined by nothing other than the pedigree connecting an actor and recipient. IBD representations of kin selection are usually frequency-independent simply because there is nothing in the relatedness measures or the effects to cause frequency-dependence. There is, however, no reason why IBD kin selection arguments cannot incorporate frequency-dependent *phenotypic effects*.

In fact, it is not difficult to introduce frequency-dependence into the costs or benefits of kin selection models. For instance, the cost to an actor of aiding relatives might depend on how frequently the act is performed (and hence on the allele and phenotype frequencies) if predators learn to recognize or anticipate an altruistic act. Similarly, the benefit to a recipient would depend on how frequently it has experienced the act if it learns to respond more rapidly or appropriately. Terms for phenotype frequency could readily be added to the effects on fitness in models of kin selection. Such frequency-dependent fitness effects, like differences in reproductive value, have largely been ignored in quantitative kin selection models.

THE DISTRIBUTION OF ALLELES TO ACTORS AND RECIPIENTS

A calculation of relatedness as identity by descent starts at the actor and goes backwards in time to an ancestor, then forwards to a recipient. For these computations, $P(\mathrm{A})$ is interpreted in a backward direction as the probability that an action allele in an *actor* came from a particular ancestor, and $P(\mathrm{T})$ is the forward probability that the same ancestor gave the allele to a recipient.

When additional factors other than the pedigree connecting actors and recipients influence the distribution of an action allele, calculations of identity by descent do not give accurate estimates of the relative numbers of action alleles in actors and recipients. In these situations, the probabilities that an ancestor transmitted an action allele to an actor or to a recipient can still be calculated quite simply, however. Then $P(\mathrm{A})$ is interpreted in a forward direction as the probability that an action allele in a particular *ancestor* transmitted the allele to the actor. When factors other than their pedigree affect the distribution of alleles to interactants, the simplest method of analysing selection is to use the collective fitness rule directly. For this purpose, the numbers of alleles distributed to actors and recipients are calculated directly from first principles, incorporating any relevant factors into the counts. Then the numbers can be inserted into one of the forms of the collective fitness rule for acts among relatives (5 or 6).

The two equivalent procedures, the collective fitness rules and the inclusive fitness rules, both use identity by descent formulations of relatedness. Collective fitness calculations are applicable to situations involving any factor that affects the distribution of action alleles. Consequently, they can be applied to phenomena such as offspring competing equally for parental resources, endosperm strategies and mutual cooperation. In these situations, the distribution of alleles to actors and recipients is not random and the genetic correlation between actors and recipients is not caused by their descent from a common ancestor, so identity by descent cannot be used to weight the effects on actors and recipients.

When the relative numbers of copies of action allele in actors and recipients is frequency-dependent or dosage-dependent, a single model of an autonomous gene no longer suffices to describe the direction of selection at all allele frequencies. In these circumstances, the most complete method of analysis is to use a full genetic model. Such models calculate 'neighbour-modulated' fitnesses that attribute actor and recipient effects directly to all genotypes. Hamilton (1964) showed that the use of neighbour-modulated fitness gives the same results as inclusive fitness calculations. Models employing neighbour-modulated fitness are likely to be involved, however, as Maynard Smith (1981) demonstrated with a worked example.

Fortunately, analyses of the operation of selection on social behaviour, including situations where there are frequency-dependent factors, can be greatly simplified by the use of ESS models of the collective fitness of rare alleles. The general consequences of frequency-dependence can be uncovered by examining events when the action allele is rare and when it is prevalent (the null allele is rare). These situations give conditions for the invasion and fixation of the action allele, respectively, but they do not provide a full description of the course of selection.

The utility of inclusive fitness and collective fitness representations of social acts stems from the simplifications of the processes of selection that they permit. Both representations allow attention to be confined to the participants involved in a social act, without having to trace the transmission of competing alleles in the entire population. Apart from the benefits and costs to actors and recipients, the descriptions require only a measure of relatedness (for inclusive fitness calculations) or the actual distribution of alleles to the participants (for collective fitness calculations).

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