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What is wrong with absolute individual fitness?

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One of the most basic facts about evolution is that fitness is a relative concept. It does not matter how well an organism survives and reproduces, only that it does so better than other organisms bearing alternative traits. Nevertheless, many evolutionary arguments are framed in terms of absolute individual fitness. The absolute fitness criterion (AFC) can be justified in terms of relative fitness only given certain assumptions that are frequently violated in nature. In particular, interactions must occur in groups that are randomly formed and phenotypic variation among groups must be tightly coupled to genetic variation. Complicating the genotype–phenotype relationship can cause phenotypic variation among groups to become nonrandom, even when the groups are randomly formed, favoring traits that do not maximize absolute individual fitness. Complex genotype–phenotype relationships and complex population structures require explicit models of evolutionary change based on relative fitness differences within and among groups.

Consider an organism with two behavioral options, A or B, which would increase its fitness by two or one units respectively. Which behavior is likely to evolve by natural selection? Many ecologists, animal behaviorists and evolutionary biologists use this decision-making algorithm to reason about the outcome of natural selection and would confidently predict that A evolves because it maximally increases the absolute fitness of the individual. Optimization models employ the same reasoning more formally. Nevertheless, it is universally accepted that natural selection is based on relative fitness and not absolute fitness *per se*. Given certain assumptions, the absolute fitness criterion (AFC) is equivalent to the relative fitness criterion (RFC) and correctly predicts the outcome of natural selection. However, these assumptions are seldom made explicit and can frequently be violated in natural populations. The purpose of this article is to clarify the assumptions and show how they have obscured important possibilities for the evolution of traits that deviate from absolute individual fitness in the direction of both spite and altruism.

Maximizing absolute individual fitness is equivalent to maximizing relative fitness for traits that affect only the fitness of the actor, but what about traits that also affect other members of the population? For example, suppose that behavior A provides a public good that increases the fitness of everyone in one's group (including oneself) by two units at a private cost of one unit, whereas behavior B involves doing nothing. Behavior A increases the absolute fitness of the actor (one versus zero) but decreases its relative fitness within its group (one versus two). Nevertheless, the AFC is often justified even in this case, based on the following logic: if groups are formed at random from a large population, then the other members of one's group are a random sample and any effect on them, positive or negative, will have no effect on global evolutionary change. This leaves only the effect of the individual on itself as a meaningful evolutionary force [1,2].

This argument works only for a large population that is subdivided into randomly formed groups (i.e. for two alternative traits, the variance expected from the binomial distribution). As variation among groups becomes less than random and ultimately declines to zero, relative fitness within groups becomes the appropriate criterion and behavior A will not evolve in spite of increasing the absolute fitness of the actor. As variation among groups becomes greater than random, ultimately resulting in groups that are either pure A or pure B, the fitness of the whole group becomes the appropriate criterion. Not only will behavior A evolve, but so also will altruistic behaviors that decrease the absolute fitness of the actor. The below-random case has received little attention, in part because it is difficult to think of mechanisms that generate below-random variation among groups. The above-random case has received much attention because variation among groups is frequently above random, such as when the groups comprise genetic relatives. In these cases, it is well recognized that absolute individual fitness must be supplemented by an additional term reflecting one's effect on the fitness of others, weighted by the coefficient of relatedness (r) or other index of above-random grouping [3–5].

What is not well recognized is that the AFC frequently breaks down even in randomly formed groups. To see why, it is important to distinguish between genetic variation and phenotypic variation. These are tightly linked in the simplest evolutionary models because genes are assumed to encode phenotypes directly. For example, in a typical altruism model, the altruistic behavior is encoded by an altruistic allele and the only way for a group to be behaviorally uniform is for it to be genetically uniform. Complicating the genotype–phenotype relationship opens a Pandora's box of possibilities, in which phenotypic

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variation among groups can become highly above or below random, even though the groups themselves are randomly formed. In these cases, the AFC can fail to predict the outcome of evolution, much as when genetic variation is nonrandom.

Below-random phenotypic variation

An example of below-random phenotypic variation in randomly formed groups is shown in Box 1, based on a model of food sharing in hunter–gatherer societies [6–8]. Hunting in such societies is a good example of a public good provided at private expense because it is a costly activity and hunters are usually obliged to share their catch. A person in a group with x hunters who cares only about his own absolute fitness might reason this way: 'If I don't hunt, I will get my share of the food provided by x hunters. If I do hunt, I will get my share of the food provided by $x+1$ hunters (the other hunters plus myself). I will do whatever gives me the larger amount'. Surprisingly, this decision based on the AFC fails to predict the outcome of natural selection in randomly formed groups when individuals are allowed to vary in their propensity to hunt based on the number of other hunters in the group. Examining relative fitnesses within and among groups can help us see why. Within each group, hunters are less fit than free-riding 'scroungers'. The only way to counterbalance the relative fitness disadvantage of hunting within groups is for there to be a relative fitness advantage of hunting at the group level; that is, for groups to vary in the number of hunters and for those with more hunters to contribute differentially to the total gene pool. Relative fitness in the total population is a combination of these two opposing forces. When phenotypic variation is random, hunters have the highest relative fitness in the total population when the AFC is satisfied. However, when individuals vary in their propensity to hunt based on the number of other hunters in the group, they tend to compensate for each other, producing below-random phenotypic variation among groups, even though the groups are randomly formed. As a result, the among-group component of natural selection (favoring hunters) becomes weak compared to the within-group component (favoring scroungers) and the equilibrium frequency of hunters becomes much less than predicted on the basis of the AFC.

Above-random phenotypic variation

An example of above-random phenotypic variation in randomly formed groups is shown in Box 2, based on the two strategies 'cooperate (C)' and 'defect (D)' of the prisoner's dilemma model [9]. Imagine that individuals are randomly distributed into groups of size N, where they randomly interact for a period of time before dispersing back into the total population. Selection within each group favors D but groups contribute to the total gene pool in

Box 1. Below-random phenotypic variation among randomly formed groups

Blurton Jones [6,7] used the AFC to claim that hunting in hunter–gatherer societies evolves on the basis of self-interest, even though it is a costly activity and the benefits are shared with all members of the group, including 'scroungers' who don't hunt. A numerical example of his model is shown in Table I. Groups of size $N = 10$ comprise hunters who provide meat and scroungers who receive a share without providing. The units are in grams, which are assumed to be proportional to fitness. Hunting requires being absent from the group 25% of the time, which accounts for the hunter's lower share of the total catch. Hunters have a relative fitness disadvantage within each group (compare across columns within each row) but groups with more hunters contribute more to the total gene pool than groups with fewer hunters (compare across rows). The diagonal lines represent the reasoning of a self-interested individual deciding whether to be a scrounger in a group of x hunters or a hunter in a group of $x+1$ hunters. Becoming a hunter yields a higher payoff if there are less than four hunters in the group.

Wilson [8] examined the same model in a computer simulation that explicitly accounted for evolutionary change within and among randomly formed groups. The two unconditional strategies 'always hunt' and 'always scrounge' resulted in a polymorphism with ~40% hunters, as predicted by the AFC. However, when conditional strategies of the form 'hunt if there are less than x other hunters in your group' were included, the equilibrium frequency of hunters declined to ~1.5 per group. The reason is that the conditional strategies tend to compensate for each other, creating below-random phenotypic variation among the randomly formed groups. Table II shows an example in which two groups vary genetically and in their average threshold value of x but become phenotypically identical when all of the thresholds are satisfied. Note that the $x = 5$ strategy adopts the hunter phenotype in group 1 and the scrounger phenotype in group 2. Because variation among groups is required to balance the selective disadvantage of hunting within each group, the compensation effect shifts the balance in favor of within-group selection.

Table I. The tolerated theft model of hunting as a self-interested activity

Number of hunters	Scrounger's share	Hunter's share	Total catch
1	205	154	2000
2	421	316	4000
3	649	487	6000
4	889	667	8000
5	1143	857	10 000
6	1412	1059	12 000
7	1697	1273	14 000
8	2000	1500	16 000
9	2323	1742	18 000
10	–	2000	20 000

Table II. Two groups that vary genetically but not phenotypically

	Group 1			Group 2		
Type (x)	No. types	No. scroungers	No. hunters	No. types	No. scroungers	No. hunters
0	3	3	–	0	–	–
1	0	–	–	1	1	–
2	0	–	–	1	1	–
3	2	2	–	1	1	–
4	0	–	–	0	–	–
5	1	–	1	2	2	–
6	1	–	1	1	–	1
7	1	–	1	1	–	1
8	2	–	2	0	–	–
9	0	–	–	2	–	2
10	0	–	–	1	–	1

Box 2. Above-random phenotypic variation among randomly formed groups

Behaviors are socially acquired in humans and many other species, creating the potential for phenotypic variation to become nonrandom in randomly formed groups. However, the rules that govern social transmission might still be genetically influenced. Wilson and Kniffin [9] considered a model involving two acquired behaviors, the 'cooperate' (C) and 'defect' (D) strategies of the prisoner's dilemma model, and four genetically encoded transmission rules: R1 (retain your behavior unless influenced by another transmission rule), R2 (converge upon a single behavior chosen at random from the original composition of the group), R3 (biased toward C), and R4 (biased toward D). The degree to which R1 is influenced by the other transmission rules is a variable of the model. Figure I shows an example of a single group initiated by 10 individuals bearing behaviors acquired from previous interactions (6 C, 4 D) and their genetically encoded transmissions rules (8 R1, 2 R2). First the R2's converge upon one of the behaviors (D for the upper branch, C for the lower branch). Then each R2 'converts' a single R1 individual. The important point is that the R2 transmission rule increases phenotypic variation among groups, favoring the evolution of cooperation. The model shows that genetic transmission rules such as R2 and R3, which promote behavioral uniformity within groups and are biased toward cooperation, can plausibly evolve in competition with other transmission rules in randomly formed groups and even more so when genetic variation among groups is above-random (as in kin groups).

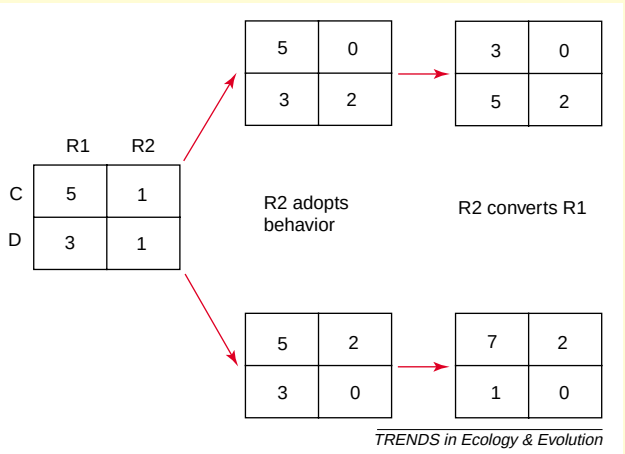

Figure I.

direct proportion to their frequency of C. Given the payoffs that define the prisoner's dilemma model, above-random phenotypic variation among groups is required for C to evolve, requiring above-random genetic variation if the strategies are encoded directly by genes. However, what happens if the strategies are socially acquired by genetically encoded social transmission rules? Individuals enter a given group bearing a behavior acquired from previous interactions and their genetically encoded transmission rules. The behavioral composition of the group changes on the basis of the transmission rules and selection takes place on the basis of the newly created phenotypic variation within and among groups. These models are still fully genetic but complicate the relationship between the genotype (the social transmission rule) and the phenotype (the behavioral strategy, which is determined by the initial behavioral composition of the group and the interactions among the transmission rules). As shown in Box 2, transmission rules that create behavioral uniformity within groups and that are biased toward the cooperative strategy can plausibly evolve in randomly formed groups.

How do these two examples escape the argument that effects on others in randomly formed groups are irrelevant to global evolutionary change? This argument assumes that the behavior of one individual does not prompt responses by other individuals. The decision-making individual in the first example should have reasoned as follows: 'If I don't hunt, I will get my share of the food provided by x hunters. If I do hunt, I might get my share of the food provided by $x + 1$ hunters. On the other hand, one of the x hunters might stop hunting based on my decision to hunt, leaving me a hunter in a group of x hunters'. Similarly, the decision to become a cooperator in the prisoner's dilemma model can be a wise choice if it prompts others to make the same decision.

Beyond sophisticated behaviors

Although these two examples involve relatively sophisticated behavioral interactions, the basic point is more general and applies to any complex genotype–phenotype relationship, behavioral or otherwise. To see this, choose any given species and imagine forming several groups at random from a large population, allowing members of the group to interact for a period of time, and then measuring any given phenotypic trait of the individuals (e.g. growth rate) or the groups (e.g. size or age composition). How often will phenotypic variation be random in these randomly formed groups? This need not be a thought experiment because most experiments require the establishment of randomly formed replicate groups within each treatment. Whenever phenotypic variation among replicates becomes non-random, something has happened on the basis of interactions within the groups. I invite readers to consult their own data to see how often this is the case.

In addition to this inadvertent information, several studies have deliberately created groups at random, measured phenotypic variation within and among groups, and artificially selected individuals within groups and groups within the total population to determine heritability [10–13]. Not only is phenotypic variation among groups usually greater than random, but the traits are often more heritable at the group level than at the individual level, as described in Box 3. These results make sense in terms of complex genotype–phenotype relationships, which not only increase phenotypic variation among groups but also contribute to the non-additive (and therefore non-heritable) component of variation at the individual level more than the group level [11].

The assumption that natural selection maximizes the absolute fitness of individuals is pleasing in its simplicity. Informally, it enables us to think of selection as like an individual making a decision based only on its own fitness. Formally, it enables us to employ powerful techniques such as optimization theory. Unfortunately, these methods do not explicitly keep track of evolutionary change in multigroup populations, so their validity depends upon several simplifying assumptions. The assumption of randomly formed groups is well known, but the assumption of strict genetic determinism linking phenotypic variation to

Box 3. Phenotypic variation and heritability in laboratory experiments

Phenotypic variation within and among groups and their evolutionary consequences can easily be studied in the laboratory. In an early experiment, Goodnight [10] created groups of the cress *Arabidopsis thaliana* (initially at random) and measured the phenotypic trait of leaf area at both the individual and group levels. Phenotypic variation among groups was considerable, even when the groups were randomly formed. When the progeny of the largest plants within each group were used to form the next generation of groups, the trait was not heritable (i.e. did not respond to selection). However, when the progeny from the largest groups were used to form the next generation of groups, the trait was heritable. These results can be explained by the fact that leaf area was not an individual trait but rather a reflection of interactions among individuals in a group. For example, a plant can become largest in its group by suppressing its neighbors. Selecting such individuals does not increase and can even decrease average size (negative response to selection). When whole groups are selected, the interactions are favored in addition to the individual traits, resulting in a positive response to selection. By the same token, phenotypic variation in leaf area within and among randomly formed groups is expected to be random only if leaf area is an individual trait, a statistical assumption that is violated when leaf area is influenced by interactions within groups. See [11–13] for reviews of similar experiments on a variety of organisms and traits.

genetic variation is less well appreciated. No one defends this assumption as realistic but only as a convenient simplification that hopefully does not influence the validity of the results. Yet, when relaxing the assumption alters something as important as phenotypic variation within and among groups, the validity of the results are very much in doubt. It is disturbing that all of the core evolutionary models of social behavior, and therefore our intuition based upon these models, rest upon such an uncertain foundation.

Is there anything to replace the pleasing simplicity of the AFC? Evolution in multi-group populations can be doubly complex: first, the dynamics of each group can be complex, as I have emphasized here. Second, the multi-group population structure can be complex, far more than the randomly formed transient groups considered here [14–18]. There is no substitute for explicit models of evolutionary change in multi-group populations to understand these complexities, but models that partition selection into within- and among-group components have a pleasing simplicity of their own. Focusing first on the dynamics of single groups allows that level of complexity to be understood in its own right before adding the complexity of population structure. In addition, these models address one of the most fundamental and misunderstood issues in evolutionary biology. For decades, students of evolution have been taught that within-group selection is almost invariably stronger than among-group selection [19]. The only way to evaluate this claim is by making a series of relative fitness comparisons, within and among the relevant levels of multi-group populations. Once these comparisons are made explicit, the importance of among-group selection becomes clear and mechanisms that create above-random phenotypic variation in randomly formed groups offer a new field of possibilities for the relative fitness of groups to influence global evolutionary change.

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