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Abstract.—Natural selection at all levels requires heritable phenotypic variation among units. At the group level, variation is often increased by reproduction coupled with limited dispersal, which forms the basis of kin selection and traditional group selection models. Assortative interactions are another possible mechanism for creating variation among groups that has received less attention. We present a series of models in which altruism is a continuously varying trait and individuals are free to choose their associates, based on information that is acquired through experience, observation, or cultural transmission. Assortative interactions can generate highly nonrandom variation among groups, favoring the evolution of altruism and other group-level adaptations among genealogically unrelated individuals. Altruism can evolve even when the initial phenotypic variation in altruism is not heritable, a form of genetic assimilation. The importance of assortative interactions depends in part on cognitive abilities that allow the phenotypes of social partners to be assessed. The minimal cognitive prerequisites are likely to be found in many species. The most cognitively sophisticated species, such as humans, might be highly group selected despite low genealogical relatedness among interacting individuals.

Although group selection was once a heretical concept, it has now become common for evolutionary biologists to think about natural selection as a process that operates on a nested hierarchy of units (e.g., Price 1970, 1972; Hamilton 1975; Wade 1985; Frank 1986; Buss 1987; Boyd and Richerson 1990; Dugatkin 1990; Queller 1991; Goodnight et al. 1992; Peck 1992; Aviles 1993; Werren and Beukeboom 1993; Bull 1994; Maynard Smith and Szarthmary 1995; Seeley 1995; reviewed in Wilson and Sober 1994). One of the consequences of the hierarchical view is a unification of previously discrepant theories. Kin selection and evolutionary game theory, which were initially developed as alternatives to group selection, are now seen as special cases of natural selection acting in potentially different directions within and between groups—kin groups in the case of kin selection and groups of size N in the case of N-person game theory.

In all group selection models, the balance between levels of selection is influenced by the partitioning of variation within and among groups. For example, consider an asexual population composed of two types, A and B, distributed into groups of size N. If each group has exactly the same proportion of A and B

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types, there is no variation among groups, and there can be no among-group selection. If each group is composed of pure A or pure B, there is no variation within groups, and there can be no within-group selection. Any mechanism that increases heritable phenotypic variation among groups therefore tends to increase the relative importance of group selection.

The most commonly studied mechanism for increasing variation among groups is reproduction coupled with limited dispersal. If each group is founded by a small number of individuals (N_1) and then grows to a larger size (N_2) without mixing with other groups, the variation among groups will be greater than if the groups were colonized directly by N_2 individuals. This is the reasoning behind traditional group selection models, in which isolated demes are founded by small numbers of individuals (reviewed in Wade 1978). It is also the reasoning behind kin selection models, in which sibling groups, for example, are "founded" by $N = 2$ individuals (the parents).

Another possible mechanism for increasing variation among groups is assortative interactions. Consider an asexual population of two types, A and B, who interact in groups of size N. If individuals can distinguish between types and can choose their associates, the distribution of A and B types into groups may well be nonrandom. In particular, if A types are altruists and B types are freeloaders, then altruists might band together, leaving the freeloaders to associate with each other by default.

Even though the scenario outlined here sounds plausible, few theoretical models have explored the possibility and implications of assortative interactions in group-structured populations (Hamilton 1971; Boorman and Levitt 1973, 1980; Wilson 1977; Fagen 1980; Eshel and Cavalli-Sforza 1982; Michod and Sanderson 1985; Toro and Silio 1986; Frank 1988; Peck 1993). Most game theory models assume that individuals interact at random. The tit-for-tat strategy (Axelrod and Hamilton 1981), for example, can change the way an individual behaves toward a defecting partner, but it cannot abandon the association to search for a new partner. Hamilton (1971, 1975), Fagan (1980), Eshel and Cavalli-Sforza (1982), Michod and Sanderson (1985), and Toro and Silio (1986) appreciated the implications of assortative interactions for the evolution of altruism, but their specific models were based on an analogy with inbreeding coefficients, in which a fraction (m) of individuals interact with partners of their own type while the remainder ($1 - m$) interact at random. The specific mechanisms that might allow a degree of assortative interactions were not investigated.

Boorman and Levitt (1973, 1980), Eshel and Cavalli-Sforza (1982), Frank (1988), and Peck (1993) modeled partner choice in a population consisting of choosy altruists and freeloaders, demonstrating a variety of stable and unstable equilibria. Depending on the parameter values, a population of choosy altruists can resist invasion by freeloaders or coexist with freeloaders in a stable equilibrium. In all cases, however, the choosy altruists could not invade a population of freeloaders (see also Charlesworth 1979). This problem, which we shall call the problem of origination, is easy to understand. At a low frequency, choosy altruists must interact with many freeloaders before they find another altruists with which to form a stable relationship. A threshold frequency (= unstable equilibrium) must be surpassed for choosy altruists to evolve to a stable equilibrium or fixation.

How severe is the problem of origination? The main purpose of this article is to show that it is an artifact of the simplifying assumption that strategies exist as a small number of discrete types that are introduced into the population at mutation frequency. When altruism is modeled as a quantitative trait, the problem of origination disappears because it is always possible for individuals that are more altruistic than average to find similar individuals with whom to interact (see also Wilson 1979). As a result, assortative interactions may provide a second important mechanism, along with reproduction and limited dispersal, for increasing phenotypic and genetic variation among groups.

First we will develop a series of models to show how assortative interactions increase variation among groups when altruism is modeled as a quantitative trait. Then we will extend a quantitative genetic model by Boyd and Richerson (1980) to compare assortative interactions versus kin selection as mechanisms for the evolution of altruism. Finally, we will discuss the implications of assortative interactions for the genetic assimilation of altruism and the possibility of group selection among genealogically unrelated individuals. See Charlesworth (1990), Christiansen (1991), Iwasa et al. (1991), Taper and Case (1992), Abrams et al. (1993), and Taylor (in press) for other models that relate quantitative genetics to game theory.

ASSORTATIVE INTERACTIONS AND VARIATION AMONG GROUPS WHEN ALTRUISM IS A QUANTITATIVE TRAIT

Consider a metapopulation of individuals that exist in groups of size N. The absolute fitness of an individual is determined both by its own phenotype and the composition of its group. We will begin with a model in which each individual (i) has a value of a quantitative trait (x_i) that increases the fitness of everyone in the group (including itself) by an amount $B(x_i)$, at a personal cost of $-C(x_i)$. For simplicity, let $B(x_i) = bx_i$ and $C(x_i) = cx_i$. All members of the group will benefit from this behavior, but the individual bears the cost. The absolute fitness of an individual (i) in a given group is therefore

$$W_i = (b \Sigma x_j) - cx_i. \tag{1}$$

The j subscript sums across all N individuals in the group. The benefit term is the same for all members of the group, while the cost term reflects only the individual's value of x, so individuals with the *lowest* value of x_i always have the *highest* relative fitness within groups. At the same time, groups of individuals with higher summed values of x contribute more to the gene pool than groups with lower summed values of x. Thus, there is a conflict between levels of selection with respect to x. To make this example biologically plausible, imagine that x is a behavior that modifies the physical habitat, promoting the growth of a resource that can be used by everyone in the group. An individual that performs this behavior will have a lower fitness than "freeloaders" within the same group, but groups of individuals who perform the behavior will have a higher fitness than groups that do not.

The balance between levels of selection depends on how the individuals are distributed into groups. Let us initially assume that the groups are randomly formed and that $\bar{x}$ is the mean value of x_i in the metapopulation. Individuals with a value of x_i experience a group that consists of themselves plus $N - 1$ neighbors whose average value is $\bar{x}$, which leads to a summed benefit of $b(x_i + [N - 1]\bar{x})$. The average fitness of individuals with phenotype x_i in the metapopulation is therefore

$$\begin{aligned} W_i &= b[x_i + (N - 1)\bar{x}] - cx_i \\ &= b\bar{x}(N - 1) + x_i(b - c) . \end{aligned} \tag{2}$$

Since the $b\bar{x}(N - 1)$ term is a constant for all values of i, natural selection favors high values of x_i when $b - c > 0$ in randomly structured groups. Before proceeding, it is important to clarify the difference between equations (1) and (2). Equation (1) gives the fitness of x_i within a single group, whereas equation (2) gives the fitness of x_i, averaged across all groups in the metapopulation, when groups are randomly formed. Some authors have interpreted equation (2) as the "individual fitness" of i, which implies that it does not invoke group selection (e.g., Grafen [1984]; Nunney [1985]). However, multilevel selection theory defines individual selection in terms of relative fitness within groups, which is given by equation (1). Group selection becomes a component of natural selection when there is *any* variation among groups, including random variation. Equation (2) therefore gives the particular balance between levels of selection that pertains when variation among groups is random. As we shall see, the balance shifts when different assumptions are made about variation among groups. (For a more detailed discussion of this issue, see Wilson 1990; Wilson and Sober 1994.)

Against this background, we can now consider the effect of assortative interactions on variation among groups. We begin by making a number of extreme assumptions that will later be examined in more detail: all individuals know the x values of all other individuals in the metapopulation, all individuals form into groups of size N, all individuals attempt to choose associates that maximize the summed x value of their group, and membership in a group requires the consent of all parties. Given these assumptions, the N individuals with the highest x values will form one group, the N individuals with the next highest x values will form another group, and so on, until the N individuals with the lowest x values will form a group, not because they want to but because they have been rejected by all the others. More generally, individuals will be rejected by others with higher x values and will themselves reject others with lower x values, leaving those with similar x values as associates. If the metapopulation is infinitely large, then all members of a given group will have the same value of x, leading to an average absolute fitness of

$$\begin{aligned} W_i &= bNx_i - cx_i \\ &= x_i(bN - c) . \end{aligned} \tag{3}$$

High values of x will evolve whenever $bN - c > 0$ (i.e., whenever the benefit to the entire group exceeds the cost to the individual). This is total altruism. The four assumptions listed earlier are admittedly extreme, but it is worth noting that the most extreme form of assortative interactions among genealogically unrelated individuals produces a population structure that is equivalent to clonal reproduction in kin selection models ($r = 1$), in which there is no genetic variation within groups and maximum genetic variation among groups.

We will now examine and relax some of the extreme assumptions of the model. The assumption of omniscience (all individuals know the x values of all other individuals in the metapopulation) can be relaxed in a number of ways. For example, individuals might know the x values of only a few other individuals in the metapopulation. To explore this possibility, we ran simulations in which the metapopulation is very large but randomly broken up into patches of size P. Individuals learn the x values of everyone in their patch and form into groups of size N, as outlined earlier. Thus, if $P = 4$ and $N = 2$, then groups are formed by randomly drawing four individuals from the metapopulation (the patch) and allowing the two highest values to form one group and the two lowest values to form a second group. This procedure will increase genetic variation among groups but will not entirely eliminate variation within groups. The question is how the partitioning of variation within and among groups will change as we vary P relative to N.

Figure 1 shows the results of simulations in which the frequency distribution of x_i in the metapopulation is normally distributed (fig. 1*A*). The other graphs show the group phenotype (Σx_j) experienced by the average individual with a phenotypic value of x_i. In other words, it is the regression of group phenotype on individual phenotype that serves as a measure of r in kin selection models (Price 1970, 1972; Hamilton 1975; Frank 1995). When individuals are randomly distributed into groups, this relationship is given by equation (2) and is represented by the line with the shallow slope. We will call this the line of random sorting. When individuals are maximally sorted into groups, the relationship is given by equation (3) and is represented by the line with the steep slope, which we will call the line of maximum sorting. Figure 1*B* shows the relationship when group size is $N = 2$ and patch size is varied from $P = 2$ (*squares*) to $P = 16$ (*diamonds*). Figure 1*C* shows the relationship when group size is $N = 10$ and patch size is varied from $P = 10$ (*squares*) to $P = 80$ (*diamonds*). When $P = N$, the groups are random samples of the metapopulation, and the results of the simulation correspond to the line of random sorting, as expected. As P is increased relative to N, the relationship converges quite rapidly on the line of maximum sorting. Also, the relationship between individual and group phenotype is nonlinear, with the middle of the distribution converging on the line of maximum sorting faster than the ends of the distribution.

Another way to relax the assumption of omniscience is to assume that individuals initially form groups at random, after which sorting occurs among groups in a pairwise fashion. To explore this possibility, we ran simulations in which the metapopulation consists of 500 pairs of individuals, randomly drawn from the phenotypic distribution shown in figure 1*A*. Pairs were then assumed to encounter each other at random and to resort into new pairs consisting of the two most

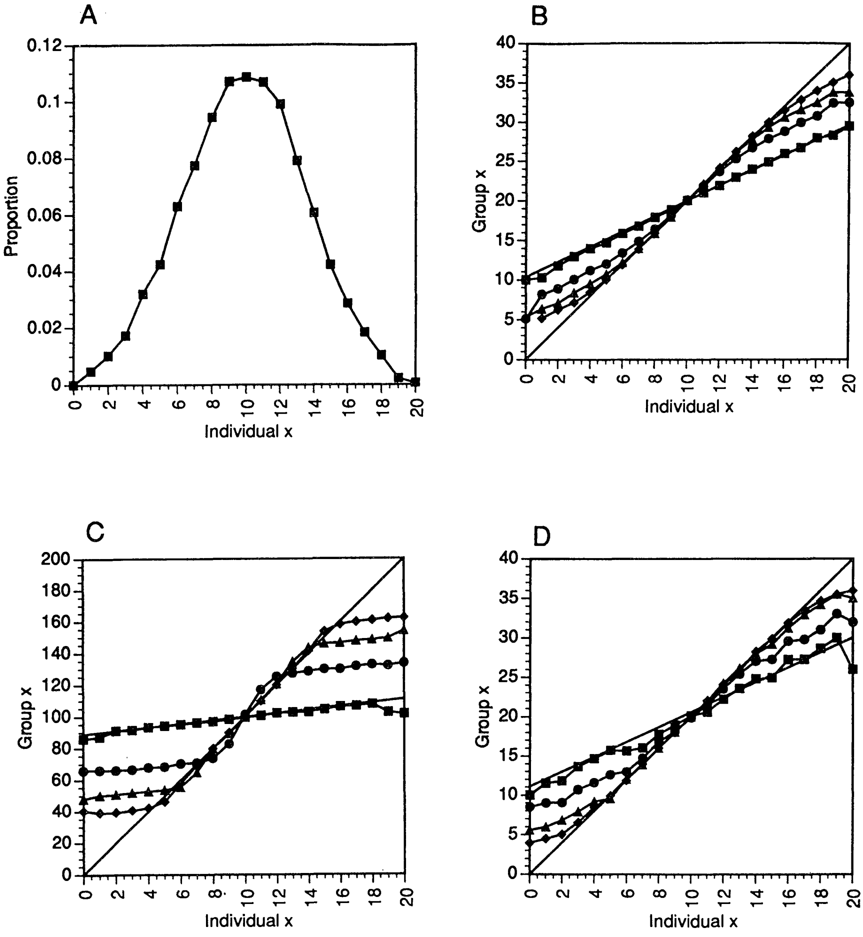

FIG. 1.—*A*, A population's phenotypic distribution showing effects of assortative interactions. *B–D*, The group phenotype (*Y*-axis) experienced by the average individual phenotype (*X*-axis). The group phenotype is the sum of the individual phenotypes in the group. In each graph, the line with the shallower slope represents random sorting (eq. [2]), and the line with the steeper slope represents maximum sorting (eq. [3]). In figure 1*B* and *C*, groups of size N are formed by assortative interactions in patches of size P that are random samples of the global population. In figure 1*B*, $N = 2$ and $P = 2$ (*squares*), 4 (*circles*), 8 (*triangles*), and 16 (*diamonds*). In figure 1*C*, $N = 10$ and $P = 10$ (*squares*), 20 (*circles*), 40 (*triangles*), and 80 (*diamonds*). In figure 1*D*, groups of size $N = 2$ are initially formed at random and then merge in a pairwise fashion during which assortative interactions occur. The number of merging cycles is $C = 0$ (*squares*), 1 (*circles*), 5 (*triangles*), and 10 (*diamonds*). In all three graphs, increasing the degree of assortative interactions causes the relationship between individual phenotype and group phenotype to converge on the line of maximum sorting, and the convergence is faster for the middle of the phenotypic distribution than for the ends.

altruistic and two least altruistic individuals. This model assumes that individuals know the values of only $2N - 1$ individuals at any one time, which is probably well within the cognitive ability of many species. Figure 1*D* shows the amount of sorting that occurs after one, five, and 10 cycles of 500 pairwise sorting events (a cycle consists of an average of one sorting event per group). Once again, there is a rapid convergence on the line of maximum sorting, with the middle of the phenotypic distribution converging faster than the ends. To summarize, assortative interactions can create considerable variation among groups, even when individuals select their associates from among a small pool of known individuals.

Our second assumption, that all individuals form into groups of size N, is a common simplifying assumption of game theory and other metapopulation models. Unlike the first assumption, it does not obviously bias the results in favor of altruism. It could be relaxed by making group size variable or allowing individuals to exist in a solitary state (Miller 1967; Orbell and Dawes 1993; Dawes and Orbell 1995). It is unlikely that these more complicated assumptions would alter the basic advantage of assortative interactions, so we will not consider them here.

Our third and fourth assumptions are that all individuals attempt to choose altruistic associates and that membership in a group requires the consent of all parties. Together, these assumptions cause individuals to associate with others that are similar to themselves. The evolution of assortative interactions is often studied in the context of speciation. Given two incipient species, A and B, whose hybrid progeny are relatively unfit, a gene that increases the probability of mating with A will be beneficial only when it occurs in an A individual. Linkage disequilibrium is required for A to prefer A and B to prefer B, which makes models of assortative mating problematic (e.g., Felsenstein 1981). However, this problem does not exist in our model because all individuals are assumed to prefer the most altruistic individuals in the population. Starting with a randomly interacting population, a mutant gene that increases the probability of interacting with an altruist will be favored, no matter what individual the gene occurs within. Linkage disequilibrium is not required, which makes the evolution of preference for altruists straightforward. Of course, preferring altruists does not result in assortative interactions unless altruists can exclude freeloaders from their groups. Little is known about either preference for associates or free choice in most species, so it is currently impossible to assess the generality of these assumptions (Dugatkin and Wilson 1993; Dugatkin and Sih 1995). However, they clearly apply to enough species and situations to justify pursuing the model.

Another potential problem with choosing altruists has been labeled "the greenbeard effect" by Dawkins (1976). Starting with a population in which it is easy to distinguish altruists from freeloaders, what prevents the evolution of freeloaders who mimic the appearance of altruists? This is a special case of a more general problem concerning the evolution of honest communication, but it is not insurmountable (Zahavi 1987; Frank 1988; Grafen 1990; Johnstone and Grafen 1993). For example, the signal for altruism can be costly or can be based on the actual act of altruism, which is impossible to mimic. Minimally, we can suppose that it is impossible to assess social partners before interacting with them but that choosy altruists can change partners after the first act of defection, in the same way that the tit-for-tat strategy changes its behavior after the first act of defection. Later we will review evidence that altruistic tendencies are readily observable, which enables individuals to choose associates even without prior interactions.

To summarize, if altruism is modeled as a quantitative trait, then assortative interactions provide a mechanism for creating above-random genetic variation among groups that is distinct from genealogical relatedness. Our next task is to compare the two mechanisms in a quantitative genetic model.

KIN SELECTION VERSUS ASSORTATIVE INTERACTIONS AS MECHANISMS FOR THE EVOLUTION OF ALTRUISM

We will extend a quantitative genetic model of kin selection by Boyd and Richerson (1980) that allows us to compare genealogical relatedness with assortative interactions as mechanisms for the evolution of altruism. Boyd and Richerson assumed that the absolute fitness of an individual is a product of two functions ($W_i = f(x_i)g(\Sigma x_j/N)$), one that depends on the phenotypic value of the individual ($f(x_i)$) and another that depends on the mean phenotypic value of the group ($g(\Sigma x_j/N)$). The individual component of fitness, $f(x_i)$, is maximized at a value of x_1 and declines at both higher and lower values as a normal distribution with a standard deviation of s_1. The group component of fitness, $g(\Sigma x_j/N)$, is maximized at a value of x_2 and declines at both higher and lower values as a normal distribution with a standard deviation of s_2. Within-group selection favors the individual optimum (x_1), because the group component does not alter relative fitness. Group selection does not favor the phenotypic value that maximizes the group component of fitness (x_2), because the individual component of fitness would cause the productivity of such groups to be low. Instead, group selection favors the value of x_i that maximizes the product of the two functions (W_i) when all members of the group are phenotypically identical. We chose parameter values such that within-group selection favors a phenotypic value of 75 and between-group selection favors a phenotypic value of 100 ($x_1 = 75$, $x_2 = 125$, $s_1 = s_2 = 400$).

Following Slatkin (1972) and Cavalli-Sforza and Feldman (1976), Boyd and Richerson (1980) do not explicitly model a multilocus genetic system but rather assume that the phenotypic value of an offspring is a sum of the midparental value and a normally distributed error term. Thus, starting with a global population in which the phenotypic distribution of x is normally distributed with a mean of 75 (the individual selection optimum) and a variance of V, our computer simulation version of their kin selection model consists of the following steps: (1) individuals mate randomly, and (2) groups of size N are formed from the progeny of mated pairs (full siblings). The phenotype of each offspring is normally distributed with a mean of the midparental value and a variance of $0.5V$. A variance of $0.5V$ within sibling groups creates a variance of V in the total population. (3) Fitness is calculated on the basis of individual and group phenotype, as described earlier. (4) Individuals disperse from the sibling groups after selection to comprise the new global population with a new mean and variance. Total phenotypic change is influenced by both selection within groups and the differential contribution of

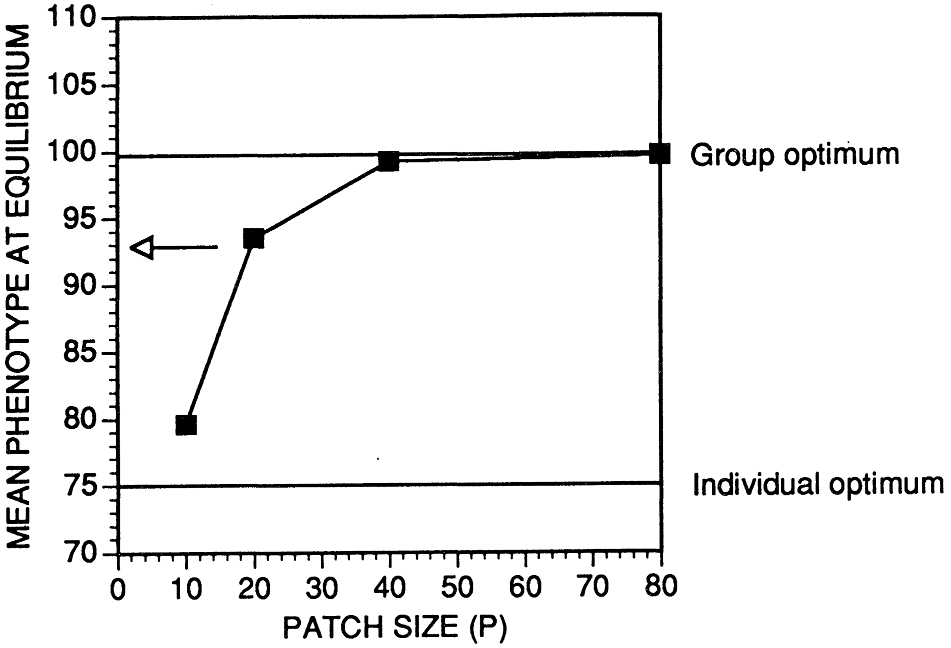

FIG. 2.—The effect of assortative interactions on the balance between levels of selection in a quantitative genetic model. Relative fitness within groups is maximized at a phenotypic value of $x = 75$, and the fitness of groups is maximized at a phenotypic value of $x = 100$. Group size is $N = 10$, and groups are formed by assortative interactions in patches of size P that are random samples of the global population, as in figure 1*C*. When $P = 10$, variation among groups is random, and the mean of the phenotypic distribution at equilibrium shifts slightly away from the individual optimum toward the group optimum. This represents a balance between levels of selection in which group selection is a significant but relatively minor force. Assortative interactions change the partitioning of variation within and among groups, shifting the balance in favor of group selection. For comparison, the arrow gives the balance between levels of selection in a kin selection model with interactions among full siblings.

groups to the global population. (5) Steps 1–4 are repeated until the global phenotypic distribution reaches an equilibrium.

Our simulation model of assortative interactions was identical except for step 2. Rather than forming groups of full siblings, patches of size P were drawn randomly from the global population, ranked with respect to their x values, and sorted into groups of size N, as described in the previous section. The members of these groups are genealogically unrelated to each other, but the sorting process acts as a mechanism that creates above-random genetic variation among groups.

Figure 2 shows the results of simulations in which $N = 10$ and P was varied from 10 to 80, as in figure 1*C*. The results of the kin selection version are shown by the arrow for comparison. For each run, the initial mean for the global population was $x = 75$, which corresponds to the optimum favored by within-group selection. When $P = 10$, groups are formed randomly from the global population.

As mentioned earlier, even random variation introduces a component of group selection into the model, which shifts the equilibrium phenotypic distribution to a mean value of 79.8. Assortative interactions reduce variation within groups and increase variation between groups, which shifts the balance further in favor of group selection. When $P = 20$, the degree of altruism that evolves is comparable to kin selection among full siblings (93.1 vs. 92.4). When $P = 40$, the mean of the equilibrium phenotypic distribution virtually converges on the group optimum of 100. These computer simulation results have recently been confirmed by an analytical model that was stimulated by our study (Taylor, in press).

To summarize, a process that involves repeatedly sampling $2N$ genealogically unrelated individuals, ranking them and splitting them into two groups at the midpoint, creates as much variation among groups as a process that involves repeatedly selecting two unrelated individuals (the parents) and creating groups of size N from their progeny. A process that involves repeatedly selecting $4N$ unrelated individuals, ranking them and splitting them into four groups, creates so much variation among groups that the conditions for the evolution of altruism are almost indistinguishable from clonal reproduction ($r = 1$). Thus, the model suggests that assortative interactions might compare quite favorably with genealogical relatedness as a generator of above-random variation among groups.

It is important to stress that our simulations began with a worst-case scenario, in which the global phenotypic distribution was centered on the within-group optimum ($x = 75$). Nevertheless, an initially low *average* degree of altruism is irrelevant to the evolution of altruism by assortative interactions. What is important is the *variation,* which allows above-average individuals to interact preferentially with each other. Genetic variation is likely to be maintained by a balance between mutation and selection, even when a polygenic trait is subject to stabilizing selection (Lande 1976; Turelli 1988). Thus, the initial conditions required by our model are eminently plausible.

It is even possible that altruism can evolve by assortative interactions when the initial phenotypic variation is not heritable. To see this, consider a population in which there are initially no genes for altruism. Nevertheless, individuals still vary in their altruistic tendencies, because of purely environmental factors. Despite the fact that altruism does not have a genetic basis, a genetic preference for *associating* with altruists might still evolve, and altruists will still preferentially interact with each other if free choice is possible. Against the background of this new population structure, a gene for altruism, which could not have evolved in the initial population structure, might now be able to spread. This would be an interesting form of genetic assimilation, made possible by assortative interactions.

DISCUSSION

Ever since the introduction of Hamilton's (1963, 1964) inclusive fitness theory, the evolution of altruism has appeared to depend primarily on genealogical relatedness. Maynard Smith (1964) relabeled inclusive fitness theory "kin selection" to distinguish it from group selection. Wilson (1975, p. 3) regarded the evolution of altruism as the central theoretical problem of sociobiology and offered the following solution: "The answer is kinship: if the genes causing the altruism are shared by two organisms because of common descent, and if the altruistic act by one organism increases the joint contribution of these genes to the next generation, the propensity to altruism will spread through the gene pool."

Evolutionary game theory was developed to explain cooperation among nonrelatives, but interactions are usually assumed to occur at random. The few game theory models that do include assortative interactions either assume that a certain fraction of individuals interact with their own type (similar to the way that inbreeding is modeled) without specifying how this happens or encounter the problem of origination. In addition, these models are sometimes presented in a way that does not make their relationship to either inclusive fitness theory or group selection theory clear.

Ironically, Hamilton (1971, 1975) was among the first to clearly see that genealogical relatedness and assortative interactions are alternative mechanisms for accomplishing exactly the same thing; above-random variation among groups. Using Price's (1970, 1972) covariance formulas for gene frequency change in structured populations, Hamilton (1975) showed that inclusive fitness theory involves within-group selection against altruism and between-group selection for altruism. The effect of genealogical relatedness is to increase variation among groups, creating a positive regression between the phenotype of an individual and the phenotype of the group that it inhabits. The coefficient of relatedness should be interpreted not as the probability of sharing genes identical by descent but as the slope of the regression line. Any mechanism that increases variation among groups (i.e., increases the slope of the regression) can favor the evolution of altruism. According to Hamilton (1975, p. 141), "it obviously makes no difference if altruists settle with altruists because they are related . . . or because they recognize fellow altruists as such, or settle together because of some pleiotropic effect of the gene on habitat preference." Unfortunately, Hamilton's reformulation of his own theory did not have the same impact as the original version. A citation analysis of journals included in the *Science Citation Index* shows that Hamilton (1975) was cited only four times in 1994, compared with 115 citations of Hamilton (1964). For many evolutionary biologists, genealogical relatedness remains the one and only explanation for the evolution of altruism.

Our model suggests that assortative interactions may qualify as another mechanism for increasing genetic variation among groups, rivaling and even surpassing genealogical relatedness for at least some species. Three basic assumptions are required for the model to work. First, altruism must be a quantitative character with a mean and a variance rather than a discrete trait that enters the population at a very low frequency. This appears to be a safe assumption, since modeling behaviors as discrete traits is more of a mathematical convenience than a biological reality. Second, individuals must know the phenotypes of at least some other individuals in the population, by experience, observation, or the cultural transmission of information. Third, an element of free choice must exist in social interactions, which allows the most altruistic individuals in the population to associate and forces the less altruistic individuals to interact with each other by default.

The second and third assumptions require a certain amount of cognitive sophistication that may only exist in some species. Little is known about either preferences or free choice in most species, but it is likely that the cognitive prerequisites for assortative interactions will often be satisfied. For example, predator inspection in fish is a behavior that probably results in a benefit (information about the predator) that is shared by all fish in the vicinity at a cost (risk of predation) to the individual inspector (reviewed in Dugatkin and Godin 1992). In a study with guppies (*Poecilia reticulata*), it was shown (Dugatkin and Alfieri 1991*b*) that the propensity to inspect predators is a continuously varying trait, satisfying one of the basic assumptions of our model. Furthermore, guppies inspect each other at the same time that they inspect the predator. If three guppies are exposed to a predator and one guppy is later given a choice between its two partners for future interactions, it tends to prefer the one that went closest to the predator. It is possible that this preference is not based on the inspection behavior per se but on other characters that correlate with predator inspection. This possibility was tested by repeating the experiment and allowing a fourth guppy, which did not observe the predator inspection, to choose among two of the fish. Choice was random, which demonstrates that preference in the original experiment was based on observing the actual act of predator inspection (Dugatkin and Alfieri 1991*a*).

These experiments show that guppies are highly sensitive to the behaviors of their conspecifics and can attempt to choose future associates based on past interactions. Furthermore, it is easy to discern a partner's behavioral tendencies when the actual behaviors can be observed. Comparable cognitive abilities have been demonstrated in bluegill sunfish (*Lepomis macrochiris*) (Dugatkin and Wilson 1992). However, it has not yet been demonstrated that preference results in assortative interactions. In the aforementioned experiment (Dugatkin and Alfieri 1991*a*), guppies preferred to associate with inspectors regardless of their own propensity to inspect. As expected from our model, everyone loves an altruist, and an extra step is required to show that inspectors can form their own groups and avoid the company of noninspectors. This experiment can easily be performed by measuring the inspection behavior of genealogically unrelated individuals and then allowing them to form groups in a naturalistic environment. If the groups are nonrandom with respect to predator inspection behavior, then this will provide strong evidence that above-random variation among groups can be created by assortative interactions in a lower vertebrate rather than genealogical relatedness.

At the opposite end of the cognitive spectrum, humans have a fantastic ability to acquire information about conspecifics based on personal interaction, direct observations, and cultural transmission. This information is used largely to seek out trustworthy individuals and avoid cheaters in social interactions. According to Cosmides and Tooby (1992), cheater detection is a specialized cognitive adaptation in humans that often overrides logical reasoning ability. The ability to detect cheaters is likely to select for the ability of cheaters to avoid detection (Trivers 1971), but deception is unlikely to be completely successful (Frank 1988; Wilson et al. 1996). Even if initial intentions can be disguised, behavioral tendencies can be reliably discerned from the actual behaviors, whenever the history of past interactions is known. Unlike the large anonymous societies of today, human evolution took place in small groups whose members had extensive opportunities to observe and talk about each other. Information about a single act of defection can quickly spread through a social network and spoil a person's reputation, with grave consequences for future social interactions (e.g., Boehm 1993; MacDonald 1994; Sober and Wilson 1997).

The consequences of these human cognitive abilities may help resolve a longstanding paradox about human social behavior. On the one hand, at least some human social organizations appear elaborately organized at the group level, rivaling the eusocial insects and colonial organisms. On the other hand, human social groups do not have the high degree of genealogical relatedness that characterize other ultrasocial species. If ultrasociality requires genealogical relatedness, then its appearance in human groups must be an illusion, a coincidental by-product of individual self-interest. That is how human social behavior is often interpreted (e.g., Alexander 1987). It is also common to suppose that human altruism evolved in an ancestral environment when members of groups were more highly related genealogically and that altruism is maladaptive when expressed toward nonrelatives in modern groups (e.g., Ruse 1986). All of these ideas are predicated on the assumption that the evolution of altruism requires genealogical relatedness. Our model suggests a different possibility: that humans are a highly group-selected species but that the variation among human groups comes from assortative interactions rather than genealogical relatedness.

Assortative interactions can help solve a number of problems that appear enigmatic from other perspectives. For example, helping among nonrelatives is often thought to require reciprocity, such that benefits are provided only to individuals that can be expected to return benefits in the future. This prediction is violated by many forms of helping in humans (and perhaps other species) that are based on meeting a need rather than on maximizing the probability of return benefits. When genealogical relatedness and assortative interactions are viewed as alternative ways of generating variation among groups, reciprocity is no more required among altruists that have become segregated by assortative interactions than it is among genealogical relatives. It is enough for an individual to know that he or she is in a group of trustworthy individuals without requiring that specific acts of altruism be reciprocated.

In most game theory models, individuals who employ conditionally altruistic strategies, such as tit-for-tat, change their *behavior* in response to defection. In our model, individuals change their *partners* in response to defection. In reality, individuals probably employ both strategies, selecting their social partners whenever possible and changing their behavior toward undesirable social partners that they cannot avoid. Also, we might expect individuals to be facultatively altruistic in situations that allow assortative interactions and more exploitative in situations that do not. Additional models are required to explore these more complicated social strategies, but assortative interactions are likely to remain a potent force for the evolution of altruism, facultative or otherwise.

For many years, genealogical relatedness was the guiding light for biologists interested in the evolution of altruism. It has become apparent more recently that variation among groups is the essential ingredient, which can be accomplished by more than one mechanism. In addition to genealogical relatedness, assortative interactions provide another plausible mechanism whose prerequisites and consequences are largely unexplored. These and other mechanisms should be studied as part of the larger conceptual framework, which is natural selection as a process that operates on a nested hierarchy of units.

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