Kin selection, genic selection, and information-dependent strategies

John Tooby[a] and Leda Cosmides[b]

[a]*Department of Anthropology, Harvard University, Peabody Museum, Cambridge, MA 02138 and* [b]*Department of Psychology, Stanford University, Stanford, CA 94305*

Although Rushton explores some interesting phenomena in his target article, the theoretical framework he uses to integrate them suffers from a series of defects. These include (1) the failure to understand fully the theory of kin selection (see, e.g., Dawkins 1979; Mealey 1985), (2) the failure to distinguish the operation of kin selection as a selection pressure from the operation of adaptations that evolved in response to kin selection (e.g., phenotype matching), and (3) the failure to distinguish circumstances reliably present during human evolutionary history to which we can have evolved adaptations (e.g., encounters with near and distant kin) from recently emerged circumstances to which we cannot have evolved adaptations (e.g., encounters with those of other races).

Kin selection theory explores how natural selection shapes genetically inherited traits that simultaneously influence the reproduction of the bearer of the trait and the reproduction of other individuals who share the gene(s) underlying the trait (Hamilton 1964; Williams & Williams 1957; Williams 1966). Rushton proposes an extension of kin selection theory in which the idea of "genetic similarity" between individuals is substituted for relatedness as the more general and appropriate concept.

Analyzed at the level of the individual, there is no single standard of fitness, such as inclusive fitness, that definitively characterizes what the evolutionary process maximizes because the genome contains subsets of genes whose fitnesses cannot all be simultaneously maximized (Cosmides & Tooby 1981; Dawkins 1982); since selection operates at the genic rather than at the individual level, the nature of kin selection and inclusive fitness must be addressed at the genic level (Cosmides & Tooby 1981; Dawkins 1982). Moreover, the question of kin selection is a game-theoretic one concerning which a phenotypic strategy of reproductive trade-offs between bearer and recipient will maximally propagate a gene coding for that strategy; the optimal strategy will depend (in part) on the information available to be used by the strategy. Flaws appear in the intuitive notion of "genetic similarity" when it is scrutinized in this way. At the genic level there is no genetic similarity: There is either identity, nonidentity, or some information reliably indicating the probability that another individual contains and will propagate a

replica. In the *absence* of constraints on information on strategy implementation, a gene would be selected to promote the reproduction of its replicas, regardless of which individuals they were in. However, situations in which such constraints are absent are vanishingly rare; "green beard" selection (Dawkins 1976) in the real world is limited to aposematic coloration, in which predators from other species, through foraging, incidentally solve for the "green beard" genes the otherwise insurmountable problems of (1) reliable identification of replicas, (2) the linkage between the genes used for identification and the genes for conferring benefits, (3) mimicry, and (4) the implementation of altruistic consequences on the "green beard" genes in other individuals.

Leaving aside such exceptional and stringent circumstances, any trait with social consequences will typically involve many genes from many loci. Hence the question is: What kin selection principles govern the evolution of adaptations that are polygenic and information-limited? In particular, the question that Rushton addresses concerns the significance of "genetic similarity," measured across loci, as hypothetically distinguished from genetic relationships that arise due to common ancestry. Rushton's discussion of "genetic similarity" theory in fact raises two distinct questions: (1) Does genetic similarity operate as an evolutionary principle *independent of common ancestry*? (2) Can and does a phenotype-matching process that samples heritable phenotypic markers (in order to modulate altruism or mating) operate in humans?

The answer to the first question is straightforward: Genetic similarity does not arise independently from relatedness in the real world because of the size of the genome (e.g., Bachmann 1972) and the free recombination it displays when genotypes of nonrelated (genetically distant) individuals are compared. Although one might, as a thought experiment, imagine random assortment by chance creating individuals who are very similar genetically, given the estimated 100,000 to 200,000 freely recombining genes present in the human genome, the probability that a Pleistocene human would during his lifetime encounter a nonrelative who was substantially more "genetically similar" than the local population average was negligible. Nor would it matter if he did. No plausible mechanism can assay genetic "similarity" across all loci in the genome; the most that can be imagined is a mechanism that monitors a restricted subset of the genotype, comparing a limited number of heritable phenotypic markers between individuals. Assuming that such a mechanism detected "genetic similarity" in the sense of such shared markers between two nonrelatives, this would still provide no basis for the evolution of altruism between them because, in the absence of common ancestry, the existence of "genetic similarity" at some loci predicts nothing about the identity of alleles at other loci. Because tracking genetic markers provides no information relevant to whether an unlinked gene is present in a nonrelative, an independently assorting gene cannot use such information to pursue an altruistic strategy toward nonrelatives. Rushton's invocation of hypothesized linked genes and supergenes cannot save "genetic similarity theory" as an evolutionary principle because sex and recombination interpose so many recombination events between individuals who are genetically distant enough to qualify as "nonrelatives" that few or no linked genes are likely to remain (in fact, the dissociation of linked genes throughout the genome is probably the function of sex; see, e.g., Tooby 1982; Seger & Hamilton 1988).

In contrast, kinship (common ancestry) does create what amounts to linkage – probabilistic associations between alleles across loci. In the presence of common ancestry, sampling genetic similarity (i.e. recognizable heritable phenotypic markers) at distributed loci becomes a useful predictor of the presence or absence of genetic identity at other loci and hence provides information on which to base a strategy for the regulation of altruistic acts. Because kinship creates these probabilistic associations across loci, it creates circumstances in which polygenic adaptations regulating altruistic acts toward kin can evolve. Thus, although the answer to question (1) is no, genetic similarity theory is not sustainable as an extension of kin selection theory, the answer to question (2) is yes, the monitoring of "genetic similarity" (i.e., phenotype matching) could have evolved via traditional kin selection in humans as an adaptation for assessing relatedness between kin in order to regulate kin-relevant behavioral strategies such as altruism and mating. Kinship in this sense refers to genetic similarity that has arisen because of shared ancestry, however recent or far back, and however aggregated from many small components, as it commonly is in a local population (particularly in species with a rich population structure).

Hence only those parts of genetic similarity theory that are consistent with the standard concept of phenotype matching as a kin-recognition mechanism remain (e.g., Waldman 1982). Given that kin selection creates the selection pressures involved, what can be made of the phenomena that Rushton weaves together under the rubric of "genetic similarity theory"? It is certainly possible that phenotype-matching systems supplement other kin recognition systems, thus influencing mating, friendship, and altruism in humans, and the data on assortative mating and affiliation based on quantitative characters are interesting and suggestive. (The functions of assortative mating and "assortative affiliation," however, are not entirely clear, and are certainly not explained by genetic similarity theory as a selective principle.) Given paternity uncertainty and the imperfect reliability of other cues (such as location, identification of sexual contacts, association with mother) available under Pleistocene circumstances, information supplied by heritable phenotypic markers could help in reconstructing the local pattern of kinship; it would be an important advance in our knowledge to trace out the properties of such a mechanism.

However, Rushton's blood group data only bear tangentially on these issues and other explanations seem sounder. For example, similarity of blood group antigens, after excluding close relatives, predicts with modest reliability the more diffusely aggregated common ancestry arising out of common derivation from the same ancestral population (see, e.g., Mourant et al. 1976). Even after migration to the New World, immigrants tended to live near others from their ancestral locality. (Those living on the same street in North America were often from the same small village in Europe; Sowell 1981; Whyte 1955). This practice was so pronounced and widespread that 50 years after such mass immigration ended, 50% of southern Europeans would have had to be relocated to achieve a random distribution (Glazer 1975). Thus, similarity of blood group antigens is likely to reflect common ethnicity and, more specifically, similarity of ancestral population derivation, which is associated with present residential clustering and cultural background. This could explain Rushton's data: It is not surprising to find that people befriend more often or have more reproductively successful marriages with those of similar cultural and residential backgrounds, although phenotype matching (on quantitative characters) may reinforce such tendencies. According to this view, similarity of blood group antigens is a consequence, not a cause, of the affiliative patterns he reports.

Finally, it is important to bear in mind that our complex innate psychological mechanisms evolved during the Pleistocene and were created by histories of selection (see Daly & Wilson 1988). Modern phenomena such as friction between people of different "races" and wars between nation-states, cannot be adaptations to modern circumstances, but rather reflect the misfiring of Pleistocene adaptations under modern circumstances. In fact, nonrelatives from one's own "race" are only slightly more genetically similar than nonrelatives from a different "race" (Lewontin 1982); this modest difference could not have led to any behavioral adaptations, because in the Pleistocene, humans would not commonly have encountered people from different "races." Instead, competition could only

have been between neighboring groups; typically, intergroup conflict would have reflected cooperation with nearer kin against more distant kin. Although in such small-group conflicts the relatedness of many of the participants in the same coalition must have been very low, the influence of an individual's decisions on coalition formation, coalition fissioning or exclusion, and coalitional aggression, when summed over the members of the two groups, would often have aggregated into substantial inclusive fitness effects. This would have promoted the evolution of specialized mechanisms governing human coalitional psychology (Tooby & Cosmides 1988) without recourse to the group selection that Rushton favors.

It is certainly possible that phenotype-matching processes play some role in human coalitional psychology, but this role should be limited by how useful such markers would have been as providers of information about the best inclusive fitness strategy for making coalitional decisions during the Pleistocene. Markers do not seem particularly well suited to this task. They are useful in tracing close kinship links (e.g., who is the father?); but the more distant the relationship tracked, the more likely it is that noisy fluctations in background levels will render the markers erroneous sources of information, particularly in the small local populations characteristic of Pleistocene life. (For example, a Swiss may, by chance, look more like the residents of another Swiss village than he does his own second cousins; he is, however, still likely to resemble his parents and siblings to a recognizable degree.) Nongenetic phenotypic traits that are passed from parents to offspring (such as linguistic patterns or cultural practices) but that decay substantially across several generations may prove to be better trackers and predictors of relatedness among (say) sets of third- or fourth-degree kin than the distribution of genetic markers in relatively homogeneous local populations. Irwin's work (in press) on accent as a badge of group membership adds weight to such a view. Although the mechanism of phenotype matching, misfiring maladaptively under modern circumstances, may contribute to tendencies toward interethnic hostility, it certainly does not swamp other factors. For example, immigrants originally from neighboring villages in Italy were prevented from working together in the United States because of the serious violence that would erupt; yet these same individuals lived peacefully among Chinese immigrants (Sowell 1981). In sum, we believe Rushton's interesting empirical results could be pursued more productively and framed more illuminatingly if freed from the distorting influence of genetic similarity theory.

NOTE

*Please address all correspondence to Leda Cosmides.

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