Does Genetic Similarity Theory Go Beyond Kin Selection?
A Response to Mealey

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Contrary to Mealey's view, it is shown that offspring can be more similar to one parent than to another. Focusing on genes identical by descent can obscure a more general principle. If a gene can ensure its own survival by acting so as to bring about the reproduction of family members with whom it shares copies, then it should also be able to do so by benefiting any organism in which copies of itself are to be detected. If so, an explanation is provided for assortative mating, within-family favoritism, selective friendship, and ethnocentrism. Discussion is provided of how genetic similarity detection may occur.

While Mealey (1989) accepts the claim that "children may resemble their parents more than 0.50 through assortative mating" (p. 309), she nonetheless asserts that a) "the proportion of genes shared between parents and children because they are identical by descent, is always .50 with no variance" (p. 309, emphasis in original) and b) it "is impossible" for the vagaries of meiosis to make a child more similar to one parent than to the other. Consider, however, the case of a homozygotic AA male breeding with a heterozygotic Aa female. Under the Mendelian laws of segregation and independent assortment, and assuming no dominance, the offspring has an equal chance of being AA as Aa. In the first, the offspring is 100% similar to the father and 50% similar to the mother; in the latter, the offspring is 50% similar to the father and 100% similar to the mother. Mealey’s argument appears to be refuted. Her other points do not really contradict what has been written previously.

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Resolution of the apparent disagreement may depend upon whether the focus is on alleles identical by almost immediate descent, or on overall genetic similarity. This was the point of departure for the genetic similarity extension to kin-selection theory, and Mealey’s commentary is welcome for raising the distinction to scrutiny. The original statement of genetic-similarity theory postulated that if a gene could ensure its own survival by bringing about the reproduction of any organism in which copies of itself were to be found, then kin-selection theory could be incorporated under a more general principle (Rushton et al., 1984). Rather than merely protecting kin at the expense of strangers, it was suggested that organisms have a tendency to detect other genetically similar organisms and to exhibit altruistic behavior toward these “strangers,” as well as toward its own relatives. In order to pursue this general strategy, it must, in effect, be able to detect copies of its genes in other organisms.

Objections on the grounds of “implausibility” and “fallacy” were raised almost immediately (for an exchange of views, see Mealey [1985] and Rushton and Russell [1985] see also Trivers [1985, p. 423]). The main point made by critics is that the overall proportion of genes shared with another individual is irrelevant unless they are linked to a “gene for altruism,” and such a link is unlikely to remain across generations because genes assort independently. Two ways to avoid this problem are: 1) follow Hamilton’s rules and depend on the statistics of identity by common descent to ensure the presence of altruistic genes in others, or 2) discover some phenotypic character that is very closely linked to or associated with altruism (e.g., Dawkins’ 1976 “green beard” idea). Critics then point out that the latter is considered unlikely even by its formulators (Hamilton 1964; Dawkins 1976, 1982) since it would be in the interest of unlinked loci to disrupt the altruist locus either by deception through mimicking the phenotypic marker for their own parasitic purposes, or modifying the marker so that the recognition system is foiled. Indeed, if one gene can evolve to produce such a complex phenotypic effect, alleles at other loci might also, resulting in an intragenomic “tug of war” as each gene attempts to influence the behavior of its bearer in its own interest (Alexander and Borgia 1978; Dawkins 1982).

These arguments do not refute the theory. The mechanisms will be complex, perhaps involving many genes and supergenes on many chromosomes. For example, large groups of genes could become linked and pleiotropic to produce both feature detectors and altruistic behavior. Moreover, if it is advantageous for a single gene to work for copies of itself, it should be advantageous for all genes to do the same, and thus aggregation effects are to be expected. This makes it reasonable to talk of overall genetic similarity and not to distinguish between the proportion of shared genes and the probability of a shared altruism gene. Waldman (1987) has developed this argument most fully, pointing out that feature detectors, like other phenotypic characters, can be expected to be the product of multiple alleles and thus reflect accurately the overall genome rather than particular parts. He cites
hybridization studies showing that in crickets and frogs, hybrid females orient preferentially towards vocalizations produced by hybrid males; this suggests that the mechanisms responsible for their detection and production are genetically coupled.

The strong version of genetic-similarity theory thus suggests that some phenotypes are inherently more attractive to organisms than others. The evolutionary origin of such a mechanism could be simple: If like appearance is positively correlated with like genes, any mutation toward preference for like phenotype would tend to proliferate. If feature detectors exist, they will not lead to *kin* recognition abilities, but to the discrimination of individuals who share appropriate phenotypic traits. This, of course, is the essence of genetic-similarity theory.

Genes identical by descent are postulated by evolutionists only as a mathematical convenience; it is obvious that siblings share vastly more than 50% of their genes. (On some metrics humans are said to share 98% of their genes with chimpanzees.) While the identical-by-descent postulate has led to much useful attention being focused on relatedness from a selfish gene perspective it can also mislead. Ambiguities and complexities require continual clarification (Dawkins 1982). Even experts get it wrong. For example, while Mealey (1989) concurs with Barash (1982) that relatedness between siblings varies, she disagrees with him on whether relatedness between grandparents and grandchildren “is exactly 0.25” (Barash, p. 71) or “averages, but varies, around .25” (Mealey, p. 309). With each generation the problem becomes greater, and the relationship weaker. Dawkins (1976) goes so far as to say that although Britain’s Queen Elizabeth II is a direct descendant of William the Conqueror (1066), “it is quite probable that she bears not a single one of the old king’s genes. We should not seek immortality in reproduction . . . the *collection* of genes which is anyone of us . . . will be forgotten in three generations” (p. 214, italics in original).

Through assortative mating, and other cultural practices, the selfish gene’s capacity to replicate itself in combination with those clusters of other genes with which it works well, may be extended for hundreds of generations, not three. For example, even after being scattered around the world for two millennia, Jewish populations have been found to remain—to a significant degree—genetically distinctive. Analyses show that Jews from Iraq have more in common from a genetic viewpoint with Jews in Poland than either group has with the non-Jews among whom they have lived for centuries (Karlin et al. 1982; Meyers 1985). Elizabeth II may well be more similar to William the Conqueror than she is to an average person.

New data collected to test genetic similarity theory may require the modification of existing orthodoxy. For example, empirical studies have shown that nonkin (both friends and spouses) assort on the basis of overall genetic similarity. Supportive data has been provided from both blood antigen analyses and from observations showing that partner resemblance is most marked on the more genetically influenced components of similarity.
rather than on the more environmentally influenced components, across a variety of anthropometric, cognitive, personality, and attitudinal characteristics (Rushton 1988; Rushton and Chan 1988; Rushton and Nicholson 1988; Rushton and Russell 1985; Russell et al. 1985). Contrary to Mealey's argument, while Hamiltonian "inclusive fitness" is more encompassing than "kin-selection," it is unclear that these findings would have been predicted from current formulations.

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REFERENCES


