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Genetic similarity, human altruism, and group selection

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Abstract: A new theory of attraction and liking based on kin selection suggests that people detect genetic similarity in others in order to give preferential treatment to those who are most similar to themselves. There are many sources of empirical and theoretical support for this view, including (1) the inclusive fitness theory of altruism, (2) kin recognition studies of animals raised apart, (3) assortative mating studies, (4) favoritism in families, (5) selective similarity among friends, and (6) ethnocentrism. Specific tests of the theory show that (1) sexually interacting couples who produce a child are genetically more similar to each other in blood antigens than they are either to sexually interacting couples who fail to produce a child or to randomly paired couples from the same sample; (2) similarity between marriage partners is most marked in the more genetically influenced of sets of anthropometric, cognitive, and personality characteristics; (3) after the death of a child, parental grief intensity is correlated with the child's similarity to the parent; (4) long-term male friendship pairs are more similar to each other in blood antigens than they are to random dyads from the same sample; and (5) similarity among best friends is most marked in the more genetically influenced of sets of attitudinal, personality, and anthropometric characteristics. The mechanisms underlying these findings may constitute a biological substrate of ethnocentrism, enabling group selection to occur.

Keywords: altruism; ethnocentrism; genetics; intelligence; interpersonal attraction; kin selection; mate preference; personality; sociobiology; reproductive strategy

1. Introduction

Resemblance in a variety of demographic, physical, and psychological attributes – including religious affiliation, socioeconomic status, appearance, abilities, attitudes, and personality – has long been considered an important factor in marriage, attraction, friendship, altruism, and group cohesion. Most explanations of the role of similarity in human relationships focus on immediate, environmental effects, for example, their reinforcement value (Byrne 1971). Recent analyses, however, suggest that genetic influences may also be involved. According to Rushton, Russell, and Wells's (1984) "genetic similarity theory," genetic likeness exerts subtle effects on a variety of relationships and has implications for the study of social behavior in small groups and even in large ones, both national and international. The ability to detect genetic similarity may mediate many aspects of interpersonal behavior, including the avoidance of inbreeding and the optimization of mate choice. In this paper, genetic similarity theory will be introduced in connection with altruism. It is proposed that genetically similar people tend to seek one another out and to provide mutually supportive environments such as marriage, friendship, and social groups. This may represent a biological factor underlying ethnocentrism and group selection.

2. The paradox of altruism

Altruism has long posed a serious dilemma for theories of human nature. Most social scientists studying altruism have focused on environmental explanations, although it is known that human differences are influenced by genes too (Rushton, Fulker, Neale, Nias & Eysenck 1986), that altruism is found in many animal species, and that altruism's roots lie deep in evolutionary history (E. O. Wilson 1975). Defined as behavior carried out to benefit others, in extreme form altruism involves self-sacrifice. In humans altruistic behavior ranges from everyday kindnesses, through sharing scarce resources, to giving up one's life to save others (Rushton 1980). In nonhuman animals, altruism includes parental care, warning calls, cooperative defense, rescue behavior, and food sharing; it may also involve self-sacrifice (E. O. Wilson 1975). For example, the poisonous sting of a honeybee is an adaptation against hive robbers. The recurved barbs facing backward from the sharp tip cause the whole sting to be wrenched out of the bee's body, along with some of the bee's vital internal organs. These barbs have been described as instruments of altruistic self-sacrifice (Ridley & Dawkins 1981).

As recognized by Darwin (1871), however, a genetic basis for altruism would represent a paradox for theories of evolution: How could altruism evolve through "survival of the fittest" when, on the face of it, altruistic behavior diminishes personal fitness? If the most altruistic members of a group sacrifice themselves for others, they run the risk of leaving fewer offspring to pass on the very genes that govern the altruistic behavior. Hence altruism would be selected against, and selfishness would be selected for.

The resolution of the paradox of altruism is one of the

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triumphs that led to the new synthesis called sociobiology: By a process known as *kin selection*, individuals maximize their *inclusive fitness* rather than only their individual fitness by increasing the production of successful offspring by both themselves and their genetic relatives (Hamilton 1964; Maynard Smith 1964). According to this view, the unit of analysis for evolutionary selection is not the individual organism but its genes: Genes are what survive and are passed on, and some of the same genes will be found not only in direct offspring but in siblings, cousins, nephews/nieces, and grandchildren. If an animal sacrifices its life for its siblings' offspring, it ensures the survival of common genes because, by common descent, it shares 50% of its genes with each sibling and 25% with each sibling's offspring.

It is accordingly predicted that the percentage of shared genes is an important determinant of the amount of altruism displayed. This is borne out in a number of species. Social ants, for example, are among the most altruistic species so far discovered, and because of a special feature of their reproductive systems, they also turn out to share 75% of their genes with their sisters (E. O. Wilson 1975). Thus, in working for others, and sacrificing their lives if need be, they help to propagate their own genes. Extreme forms of altruism may also occur in clones (e.g., aphids), individuals that are 100% related (Ridley & Dawkins 1981).

Kin selection theory is central to contemporary sociobiological theorizing (Dawkins 1976; E. O. Wilson 1975), although only recently have serious attempts been made to apply it to human relationships (Alexander 1979; 1987; Chagnon 1988; Chagnon & Irons 1979; Daly & Wilson 1988; Freedman 1979; Glassman et al. 1986; Reynolds et al. 1987; van den Berghe 1981; E. O. Wilson 1978). One might expect kin selection theory's emphasis on altruism between relatives to have limited applicability to human beings, whose altruism is frequently directed to nonkin and can usually be explained by such culturally influenced mechanisms as empathy, reciprocity, and social rules.

Adopting the mechanistic viewpoint of the "selfish gene" (Dawkins 1976), Rushton et al. (1984) extended kin selection theory to the human case by applying *genetic similarity theory*. We argued that if a gene can better ensure its own survival by acting so as to bring about the reproduction of family members with whom it shares copies, then it can also do so by bringing about the reproduction of *any* organism in which copies can be found. This would be an alternative way for genes to propagate themselves. Rather than merely protecting kin at the expense of strangers, if organisms could identify genetically similar organisms, they could exhibit altruism toward these "strangers" as well as toward kin. Kin recognition might be just one form of genetic similarity detection.

Much of this had been proposed earlier by others (e.g., Dawkins 1976; 1982; Hamilton 1964; Thiessen & Gregg 1980). Dawkins (1976; 1982), for example, building on the ideas of Hamilton (1964), suggested a thought experiment in which a gene has two effects: It causes individuals who have it (1) to grow a green beard and (2) to behave altruistically toward green-bearded individuals. The green beard serves as a recognition cue for the altruism gene. Altruism could therefore occur without the need for the individuals to be directly related. Similarly, Thiessen and Gregg (1980) suggested that "the flow of altruistic behaviors, the ease of information transfer, and the genetic benefits of positive assortative mating are linked to the degree with which interacting individuals share homologous genes" (p. 111). They reviewed data from both animals and humans to support their contention that "friends of either sex, as well as mates, resemble each other in many ways, suggesting that genetic assortment operates at all levels of social affiliation" (p. 117).

Several researchers have used mathematical models to show that under a variety of conditions, selection may favor genetic mutations that incline organisms to aid other organisms that share copies of genes but are not necessarily kin (Samuelson 1983; but see Hamilton 1987; Glassman et al. 1986; Russell 1987). For example, Russell (1987) showed that if such a mutation occurred, and if the benefit to the recipient was one-and-a-half times as great as the cost to the donor, a gene for directing altruism toward siblings disappears, but a gene for like-gene detection evolves. As discussed in section 10, mathematical models of the coevolution of genes and culture can amplify this process enormously by taking into account that people adopt ideologies and behaviors that benefit large populations.

Another way that sociobiologists have suggested that altruism could evolve is through reciprocity. Here there is no need for genetic relatedness; performing an altruistic act need only lead to an altruistic act in return (Trivers 1971). Genetic similarity and reciprocal altruism may interact: The more genes are shared by organisms, the more readily reciprocal altruism and cooperation should develop because this eliminates the need for strict reciprocity. Axelrod and Hamilton (1981), Rothstein (1980), and Thiessen and Gregg (1980) make the same point. Thiessen and Gregg state that "cooperation among 'nonrelatives' ('reciprocal altruism') may be based in large part on genetic and phenotypic similarity" (p. 133).

3. Detecting genetic similarity

In order to pursue a strategy of directing altruism toward similar genes, the organism must be able to detect genetic similarity in others. There is clearly no such thing as "genetic ESP." For individuals to direct altruism selectively to genetically similar individuals, they must respond to phenotypic cues. The importance of kin recognition mechanisms was noted by Hamilton (1964). Four such mechanisms that have been considered in the literature (Fletcher & Michener 1987; Holmes & Sherman 1983) are discussed in the following sections.

3.1. *Innate feature detectors*

Hamilton (1964) suggests that individuals have "recognition alleles" that control the development of mechanisms allowing them to detect genetic similarity in strangers. Most reviewers have considered the existence of genetic similarity detectors (e.g., the "green beard effect" described earlier) to be improbable. Their existence should not yet be discounted, however, because innate pattern recognition does occur in other areas; several studies will be discussed later that suggest we may indeed have such an ability.

3.2. Phenotype matching

The individual may be genetically guided to learn its own phenotype, or those of its close kin, and then to match new, unfamiliar phenotypes to the template it has learned – for example, Dawkins's (1982) "armpit effect." Individuals that smell (or look or behave) like oneself or one's close kin could be distinguished from those that smell (or look or behave) differently. This mechanism would depend on the existence of a strong correlation between genotype and phenotype.

3.3. Familiarity or association

Preferences may also depend on learning through social interaction. This may be the most common means of kin recognition in nature. Individuals that are reared together are more likely to be kin than nonkin. This may also involve a more general mechanism of short-term preference formation. Zajonc (1980) has shown experimentally that the more one is exposed to a stimulus, the more one prefers it. Based on studies of Japanese quail and of humans, respectively, Bateson (1983) and van den Berghe (1983) have suggested that sexual preferences may be established early in life through an imprinting-like process.

3.4. Location

The fourth kin recognition mechanism depends on a high correlation between an individual's location and kinship. The rule states: "If it's in your nest, it's yours." Where a person is and whom the person encounters can also be based on similar genes – for example, if parents exert discriminatory influence on where and with whom their children interact. Many factors that influence one's locus and contacts, such as intelligence, personality, values, and vocational interests, turn out to have some genetic basis (Loehlin et al. 1988.) Physical proximity has been widely observed to be predictive of friendship formation and spouse choice (Burley 1983).

Currently one can only speculate about the extent to which these four mechanisms operate in humans. They are not mutually exclusive. If there are evolutionary advantages to be gained from the ability to detect genetic similarity, all the mechanisms may be operative. If "stronger" mechanisms (innate feature detectors, phenotype matching) operate, it should be possible to demonstrate that interpersonal relationships are mediated by genetic similarity without the help of learning from familiarity or location. Several animal and human studies are described in the next section that suggest this occurs.

4. Kin recognition in animals

There is dramatic experimental evidence that many animal species recognize genetic similarity. Greenberg (1979) showed that the sweat bee, *Lasioglossum zephyrum*, can discriminate between unfamiliar conspecifics of varying degrees of relatedness. Guard bees of this species block the nest to prevent intruders from entering. In this study bees were first bred for 14 different degrees of genealogical relationship with each other. They were then introduced near nests that contained sisters, aunts, nieces, first cousins, or more distantly related bees. In each case the guard was expected to make a binary decision – either permitting the bee that was introduced to pass or actively preventing it from doing so. There was a strong linear relationship ($r = 0.93$) between the ability to pass the guard bee and the degree of genetic relatedness. The greater the degree of genetic similarity, the greater the proportion of bees that were allowed to enter the hive. The guard bees appear to be able to detect the degree of genetic similarity between themselves and the intruder. In subsequent kin recognition studies, Breed (1983) and Getz and Smith (1983) have shown that the honeybee, *Apis mellifera*, is able to discriminate between full and half sisters raised in neighboring cells.

There is also evidence that the ability to detect genetic similarity exists in various species of plants, tadpoles, birds, rodents, and rhesus monkeys. In studies of the frog *Rana cascadae*, by Blaustein and O'Hara (1981; 1982), tadpoles were separated before hatching and reared in isolation. The individual tadpoles were then placed in a rectangular tank with two end compartments created by plastic mesh. Siblings were placed in one compartment and nonsiblings in the other. The separated tadpoles spent more time at the siblings' end of the tank. Because the tadpoles were separated as embryos and raised in complete isolation, an ability to detect genetic similarity is implicated. Similar findings have been reported for *Bufo americanus* toad tadpoles (Waldman 1982). Kin recognition has been reported in Japanese quail by Bateson (1983), and in Canada geese by Radesater (1976).

Mammals are also able to detect degrees of genetic relatedness (Fletcher & Michener 1987; Holmes & Sherman 1983). For example, Belding's ground squirrels produce litters that contain both sisters and half sisters. Despite the fact that they shared the same womb and inhabit the same nest, full sisters fight less often than half sisters, come to each other's aid more, and are less prone to chase one another out of their home territory. Similar findings have been noted among captive multimale, multifemale groups of rhesus monkeys growing up outdoors in large social troops. Adults of both sexes are promiscuous, but mothers appear to chase *paternal* half siblings away from their infants less often than they do unrelated juveniles, and males (despite promiscuity) appear to "recognize" their own offspring, for they treat them better (Suomi 1982). In the preceding examples, the degree of genetic relatedness was established by blood tests. Walters (1987) has reviewed well-replicated data from several primate species indicating that grooming, alliance formation, cooperative defense, and food sharing occur more readily in kinship groups.

5. Kin recognition in humans

Because language represents a powerful new way to distinguish kin, it is more difficult to demonstrate that humans can recognize kin in a way that parallels kin recognition in nonhuman animals. Some indirect evidence is nonetheless discussed by Wells (1987). One approach is to show that humans have perceptual abilities that enable them to discriminate between kin and nonkin; another is to consider whether there are aspects of human social interactions that reflect differing degrees of kinship.

Humans are capable of learning to distinguish kin from nonkin at an early age. Infants can distinguish their mothers from other women by voice alone at 24 hours of age, know the smell of their mother's breast before they are 6 days of age, and recognize a photograph of their mother when they are 2 weeks old (see Wells 1987, for review). Mothers are also able to identify their infants by smell alone after a single exposure at 6 hours of age, and to recognize their infant's cry within 48 hours of birth.

Human behavior also seems to follow lines of genetic similarity with respect to kin preference. For example, among the Ye'Kwana Indians of South America, the words "brother" and "sister" cover four different categories ranging from individuals who share 50% of their genes (identical by descent) to individuals who share only 12.5% of their genes. Hames (1979) has shown that the amount of time the Ye'Kwana spend interacting with their biological relatives increases with their degree of relatedness, even though their kinship terminology does not reflect this correspondence. Anthropological data also show that in societies where certainty of paternity is relatively low, males direct material resources to their sisters' offspring (to whom their relatedness is certain) rather than to their wives' offspring (Kurland 1979). [See also Hartung: "Matrilineal Inheritance" *BBS* 8 (4) 1985.] An analysis of the contents of 1,000 probated wills revealed that after husbands and wives, kin received about 55% of the total amount bequeathed whereas nonkin received only about 7%; offspring received more than nephews and nieces (Smith et al. 1987).

When the level of genetic similarity within a family is low, the consequences can be serious. Children who are unrelated to a parent are at risk; a disproportionate number of battered babies are stepchildren (Lightcap et al. 1982). Also, unrelated people living together are more likely to kill each other than are related people living together (Daly & Wilson 1988). Converging evidence shows that adoptions are more likely to be successful when the parents perceive the child as similar to themselves (Jaffee & Fanshel 1970).

6. Testing genetic similarity theory

Almost all the studies reviewed so far were carried out to test kin selection theory. However, genetic similarity theory goes further and makes predictions about nonkin and sibling relationships that kin selection theory does not make. Several recent studies have found evidence supporting genetic similarity theory from data on mate choice, sibling favoritism, and same-sex friendships.

6.1. Spouse selection

A well-known phenomenon that is readily explained by genetic similarity theory (but not by most versions of kin selection theory) is positive assortative mating, that is, the tendency of spouses to be nonrandomly paired in the direction of resembling each other in one or more traits more than would be expected by chance. It is widely accepted that there is similarity between human spouses in such characteristics as race, socioeconomic status, physical attractiveness, ethnic background, religion, social attitudes, level of education, family size and structure, IQ, and longevity (Buss 1985; Epstein & Guttman 1984; Thiessen & Gregg 1980). [See Buss: "Sex Differences in Human Mate Preferences" *BBS* 12 (1) 1989]. For example, the median assortative mating coefficient for IQ in one review, averaged over 16 studies of 3,817 pairings, was 0.37 (Bouchard & McGue 1981). Spouse correlations tend to be high for opinions, attitudes, and values (0.40 to 0.70) and low for personality traits and personal habits (0.02 to 0.30). Spouses also resemble each other in a variety of physical features. Rushton, Russell, and Wells (1985) combined anthropometric data from a wide range of studies and found low but positive correlations for more than 60 different measures, including height (.21), weight (.25), hair color (.28), eye color (.21), chest breadth (.20), wrist circumference (.55), and interpupillary breadth (.20). If mating had been random, the correlations between spouses would have been zero.

Less well known is the fact that spouses also resemble each other in socially undesirable characteristics, including aggressiveness, criminality, alcoholism, and psychiatric disorders such as schizophrenia and the affective disorders. Guze et al. (1970) found that both the wives and the sisters of criminals tended to exhibit the same psychopathology. Gershon et al. (1973) reported that both the wives and the relatives of males suffering from affective disorders exhibited a high prevalence of the same problem. Although alternative reasons can be proposed for this finding, such as the consequences of competition for the fittest mates (Burley 1983), it does raise the possibility that the tendency to seek a similar partner may sometimes override considerations such as mate quality and individual fitness.

A study of cross-racial marriages in Hawaii found more similarity in personality test scores among males and females who married across ethnic groups than among those marrying within them (Ahern et al. 1981). The researchers posit that, given the general tendency toward homogamy, couples marrying heterogamously with regard to racial/ethnic group tend to "make up" for this dissimilarity by choosing spouses more similar to themselves in other respects than do persons marrying within their own racial/ethnic group.

One could argue that positive assortative mating has nothing to do with questions about genetic similarity, that it results only from common environmental influences. This view cannot easily account for the incidence of assortative mating for similarity in species ranging from insects to birds to primates, in laboratory as well as in natural settings (Bateson 1983; Fletcher & Michener 1987; Thiessen & Gregg 1980). To have evolved independently in such a wide variety of species, positive assortative mating must confer substantial advantage. Advantages thought to accrue to choosing an "optimal" degree of genetic similarity in human mates include (1) increased marital stability, (2) increased relatedness to offspring, (3) increased within-family altruism, and (4) greater fecundity. Evidence for the first three putative advantages is presented in section 6.2. With respect to the fourth, Thiessen and Gregg (1980) reviewed literature showing positive correlations between number of children and degree of between-spouse similarity in anthropometric variables, intelligence test scores, educational attainment, and family size in the parental generation. Bresler (1970) found that fetal loss increased with the distance

between the birthplaces of parents and with each additional country of birth among great-grandparents. Additional evidence for a relationship between similarity and fecundity is presented in section 6.1.1.

The upper limit on the fitness-enhancing effect of assortative mating for similarity occurs with incest. Too much genetic similarity between mates increases the chances that harmful recessive genes may combine. The negative effects of "inbreeding depression" have been demonstrated in many species, including humans (Jensen 1983; Thiessen & Gregg 1980). As a result, many have hypothesized that the "incest taboo" has an evolutionary basis, possibly mediated through negative imprinting on intimate associates at an early age. [See van den Berghe: "Inbreeding Avoidance" *BBS* 6 (1) 1983.] Optimal fitness, then, may consist in selecting a mate who is genetically similar but not actually a relative. Van den Berghe (1983) speculates that the ideal percentage of relatedness is 12.5% identical by descent, or the same as that between first cousins. Other animal species also avoid inbreeding. For example, several experiments have been carried out with Japanese quail, birds that, although promiscuous, proved particularly sophisticated. They preferred first cousins to third cousins, and both of these relatives to either unrelated birds or siblings, thus avoiding the dangers of too much or too little inbreeding (Bateson 1983).

6.1.1. Blood tests. To directly test the hypothesis that human mating follow lines of genetic similarity, Rushton (1988a) examined blood antigen analyses from nearly 1,000 cases of disputed paternity. Seven polymorphic marker systems – ABO, Rhesus (Rh), MNSs, Kell, Duffy (Fy), Kidd (Jk), and HLA – at 10 loci across 6 chromosomes were examined in a sample limited to people of North European appearance (judged by photographs kept for legal identifications). Such blood group differences provide a biological criterion sufficient to identify more than 95% of true relatedness in situations of paternal dispute (Bryant 1980) and to reliably distinguish even between fraternal twins raised in the same family (Pakstis et al. 1972). They also provide a less precise but still useful estimate of genetic distance among unrelated individuals. The method of calculating genetic similarity is explained in Table 1.

The results (Table 2) showed that the degree of genetic similarity within pairs related to (1) whether the pair was sexually interacting or randomly generated from the same sample and (2) whether the pair produced a child. Sexually interacting couples were found to share about 50% of measured genetic markers, part way between mothers and their offspring, who shared 73%, and randomly paired individuals from the same sample, who shared 43% (all comparisons were significantly different, $p < .001$). In the cases of disputed paternity, genetic similarity predicted whether the male was the true father of the child. Males not excluded from paternity were 52% similar to their partners whereas those excluded were only 44% similar ($p < .001$).

6.1.2. Genetic similarity detection between marriage partners. If people choose each other on the basis of shared genes, it should be possible to demonstrate that interpersonal relationships are influenced more by genetic similarity than by similarity attributable to a similar environment. Positive assortative mating might be expected to occur on the basis of the more heritable rather than the less heritable traits because the more genetically influenced traits reflect the underlying genotype better and provide a more accurate cue for matching. To control for the effects of other variables, this hypothesis must be tested on sets of homogenous traits (e.g., anthropometric versus attitudinal variables; see section 7.2).

The issue of differential heritabilities has not yet been resolved in the behavior genetic literature (Loehlin et al.

Table 1. *Method of calculating degree of genetic similarity*

System	Percentage of similarity
ABO	
If antigens are identical	100
If antigens are different	0
If one subject is A_1 and the other is A_2	85
If one subject is A_1B and the other is A_2B	85
If one subject is A_1, A_2, B and the other O	25
If one subject is A_1B and the other is A_1	50
If one subject is A_1B and the other is A_2	45
Rh	
If antigens are identical in all respects	100
If three antigens are identical	50
If both subjects have antigens c and e	50
If the genotype is possible	45
If antigens are completely different	0
MNSs	
If antigens are identical in all respects	100
If both subjects have either the M or the N	50
If both subjects have either the S or the s	50
If antigens are completely different	0
Kell	
If antigens are identical	100
If one of the two genes is the same in both subjects	50
If antigens are completely different	0
Duffy	
If antigens are identical	100
If one of the two genes is possessed by both subjects	50
If antigens are completely different	0
Kidd	
If antigens are identical	100
If one of the two genes is possessed by both subjects	50
If antigens are completely different	0
HLA[a]	
If both genes are the same	100
If one gene is possessed by both subjects	50
If the gene possessed by one subject is a "split" of the gene possessed by the other subject	95
If the gene possessed by one subject cross-reacts with the gene possessed by the other subject	45
If the genes are completely different	0

[a]A locus, B locus, and C locus

Table 2. *Percentage of genetic similarity in 4 types of human relationships, based on 10 blood loci*

Relationship	Number of pairs	Mean ± SE	Standard deviation	Range	95% confidence interval for mean
Mother-offspring	100	73.37 ± 0.91	9.31	50–88	71.52–75.22
Sexually interacting adults (male is not excluded from paternity)	799	52.02 ± 0.4	11.84	17–90	51.20–52.85
Sexually interacting adults (male is excluded from paternity)	187	44.42 ± 0.9	12.13	15–74	42.67–46.17
Randomly paired individuals	200	43.10 ± 1.0	14.40	11–81	41.09–45.11

1988). One result of our work, however, has been the finding that differential estimates of genetic influence on anthropometric, attitudinal, cognitive, and personality variables are considerably more generalizable, even across distinct ethnic and national groups, than might have been expected (Rushton 1989a). Numerous studies have shown that these estimates do indeed predict the degree of similarity between marriage partners. Several of these correlations are summarized in Table 3. Note that many of the estimates of genetic influence in this table are based on calculations of midparent-offspring regressions using data from intact families; this combines genetic and shared-family environmental influences. The latter source of variance, however, is surprisingly small (Plomin & Daniels 1987) and has not been found to add systematic bias. Nonetheless, it should be borne in mind that genetic influence has often been calculated in this way.

Using a within-subjects design, Russell et al. (1985) examined data from three studies reporting independent estimates of genetic influence and assortative mating and found positive correlations between the two sets of measures ($r = 0.36$, $p < 0.05$, for 36 anthropometric variables; $r = 0.73$, $p < 0.10$, for 5 perceptual judgment variables; and $r = 0.44$, $p < 0.01$, for 11 personality variables). In the case of the personality measures, test-retest reliabilities over a three-year period were available and were not found to influence the results.

Another test of the hypothesis was made by Rushton and Russell (1985) using data on 54 personality traits. It was found that both component and aggregate estimates of genetic influence predicted similarity between spouses ($rs = 0.44$ and 0.55, $ps < 0.001$). Rushton and Russell (1985) reviewed other reports of similar correlations, including Kamin's (1978) calculation of $r = 0.79$ ($p < 0.001$) for 15 cognitive tests and the DeFries et al. (1978) calculation of $r = 0.62$ ($p < 0.001$) for 13 anthropometric variables. Cattell (1982) too had noted that between spouse correlations tended to be lower for the less heritable, more specific cognitive abilities (tests of vocabulary and arithmetic) than for the more heritable general abilities (*g*, from Progressive Matrices). Differential test reliability is unlikely to have been the cause of the findings concerning the anthropometric variables reported by DeFries et al. (1978) or those reported by Russell et al. (1985) because Rushton (1989b, see section 6.3) found that these estimates can be made with very high degrees of precision (e.g., inter-rater reliability > 0.90).

Subsequently, these analyses were extended to include a between-subjects design and the phenomenon was found to be generalizable. Rushton and Nicholson (1988) analyzed data from studies using 15 subtests from the Hawaii Family Study of Cognition (HFSC) and 11 subtests from the Wechsler Adult Intelligence Scale (WAIS); positive correlations were calculated within and between samples. For example, in the HFSC, parent-offspring regressions (corrected for reliability) using data from Americans of European ancestry in Hawaii, Americans of Japanese ancestry in Hawaii, and Koreans in Korea correlated positively with between-spouse similarity scores taken from the same samples and with those taken from two other samples: Americans of mixed ancestry in California, and a group in Colorado. The overall mean, *r*, was 0.38 for the 15 tests. Aggregating across the numerous estimates to form the most reliable composite gave a substantially better prediction of mate similarity from the estimate of genetic influence ($r = 0.74$, $p < .001$). Similar results were found in the WAIS. Three estimates of genetic influence correlated positively with similarities between spouses based on different samples, and in the aggregate they predicted the composite of spouse similarity scores with $r = 0.52$ ($p < 0.05$).

Parenthetically, it is worth noting that partialling out *g* in both the HFSC and the WAIS analyses led to substantially lower correlations between estimates of genetic influence and assortative mating, thus offering support for the view that marital assortment in intelligence occurs primarily with the *g* factor (Cattell 1982; Eaves et al. 1984; Nagoshi & Johnson 1986). The *g* factor tends to be the most heritable component of cognitive performance measures (Vernon 1989). [See also Jensen: "Black-White Difference" *BBS* 8 (2) 1985.]

6.2. *Intrafamilial relationships*

One consequence of genetic similarity between spouses is a concomitant increase of within-family altruism. Several studies have shown that not only the occurrence of relationships but also their degree of happiness and stability can be predicted by the degree of matching of personal attributes (Bentler & Newcomb 1978; Cattell & Nesselroade 1967; Eysenck & Wakefield 1981; Hill et al. 1976; Meyer & Pepper 1977; Terman & Buttenwieser 1935a; 1935b). Because many of the traits on the basis of which spouses choose each other are about 50% heritable (Loehlin et al. 1988), it follows that the matching results

Table 3. *Summary of studies on relation between degree of genetic influence on traits and assortative marriage*

Study	Sample	Test type	Genetic estimate	Correlation with assortment
Kamin (1978)	739 European-American families in Hawaii	15 subtests from Hawaii Family Study of Cognition (HFSC)	Midparent-midchild regression	0.79; $p < 0.001$
DeFries et al. (1978)	73 European-American families in Hawaii	13 anthropometric variables from HFSC	Midparent-midchild regression	0.62; $p < 0.001$
Cattell (1982)	Numerous twin and family studies	Cognitive abilities, specific and general	Multiple abstract variance analysis	Higher on the more heritable traits; magnitudes not reported
Russell et al. (1985)	Asians and North Africans	5 perceptual judgments	Parent-offspring correlation corrected for assortative mating	0.73; $p < 0.10$
	Belgians	36 anthropometric variables	Parent-offspring correlation corrected for assortative mating	0.36; $p < 0.05$
	European-Americans	11 scales from Minnesota Multiphasic Personality Inventory	Midparent-offspring correlation	0.71; $p < 0.01$
Rushton & Russell (1985)	100–669 families in Hawaii (ethnicity not specified)	54 personality scales	Parent-offspring regression	0.44; $p < 0.0001$
			Doubled sibling-sibling correlation	0.46; $p < 0.001$
			Composite of both above	0.55; $p < 0.001$
Rushton & Nicholson (1988)	871 European-American families in Hawaii	15 subtests from HFSC	Midparent-offspring regression	Intragroup, 0.71; $p < 0.01$ Intergroup, 0.43; $p < 0.10$
	311 Japanese-American families in Hawaii	15 subtests from HFSC regression	Midparent-offspring regression	Intragroup, 0.13; ns Intergroup, 0.47; $p < 0.05$
	209 families in Republic of Korea	14 subtests from HFSC	Midparent-offspring regression	Intragroup, 0.53; $p < 0.05$ Intergroup, 0.18; ns
	55 Canadians	11 subtests from Wechsler Adult Intelligence Scale (WAIS)	Midparent-offspring regression	Intragroup, 0.23; ns Intergroup, 0.60; $p < 0.05$
	240 adolescent twins in Kentucky	11 subtests from WAIS	Holsinger's H formula	Intragroup, — Intergroup, 0.68; $p < 0.05$
	120 Minnesota families	4 subtests from WAIS plus total score	Parent-offspring correlation corrected for assortative mating	Intragroup, 0.68; ns Intergroup, 0.64; ns

in genetic similarity. Whereas each trait may add only a tiny amount to the total genetic variance shared by spouses, the cumulative effects could be considerable.

A related prediction can be made about parental care of offspring that differ in similarity. Because kin selection theory emphasizes that all siblings having genes "identical by descent" with a 0.5 coefficient of relationship (Mealey 1985; Trivers 1985), sibling differences have been overlooked as a topic of research. Positive assortative mating for genetically based traits may combine with the vagaries of meiosis, however, to make some children genetically more similar to one parent or sibling than to others. This can be illustrated as follows. If a father gives his child 50% of his genes, 10% of them shared with

the mother, and the mother gives the child 50% of her genes, 20% shares with the father, then the child will be 60% similar to the mother and 70% similar to the father. Although the predictions from kin selection theory are unclear because of its focus on genes that are identical by descent (in which all full siblings share coefficients of 0.5), genetic similarity theory predicts that parents and siblings will favor those who are most similar.

Littlefield and Rushton (1986) attempted to shed light on this hypothesis. In their study of bereavement following the death of a child, it was predicted that the more similar to the parent the child was perceived to be, the greater would be the intensity of that parent's grief experience. (We assume that the perceived similarity with offspring is correlated with genetic similarity, an assumption supported by data from blood tests; see Pakstis et al. 1972). All respondents had to pick which side of the family the child "took after" more, their own or their spouse's. Spouses agreed 74% of the time on this item, and both mothers and fathers, irrespective of the sex of the child, grieved more for the children they perceived as resembling their side of the family more. Other evidence of within-family preferences comes from a review by Segal (1988) of feelings of closeness, cooperation, and altruism in twin pairs. Compared with dizygotic twins, monozygotic twins worked harder for their cotwins on tasks, maintained greater physical proximity, expressed more affection, and suffered greater loss following bereavement.

6.3. A genetic basis for friendship?

Friendships also appear to be formed on the basis of similarity. This assumption holds for similarity as perceived by the friends, and for a variety of objectively measured characteristics, including activities, attitudes, needs, and personality (Berscheid 1985; Thiessen & Gregg 1980). Moreover, in the experimental literature on who likes whom, and why, one of the most influential variables is perceived similarity. Apparent similarity of personality, attitudes, or any of a wide range of beliefs has been found to generate liking in subjects of varying ages and from many different cultures (Berscheid 1985; Byrne 1971). According to the genetic similarity view, there is a genetic basis to friendship and friendship is one of the mechanisms that leads to altruism.

Social psychological studies show that altruism tends to increase with the benefactor's actual or perceived similarity to the beneficiary (Krebs & Miller 1985; Rushton 1980). For example, Stotland (1969) had subjects observe a person who appeared to be receiving electric shocks. When Stotland manipulated the subjects' beliefs about their similarity to that person, perceived similarity was correlated with reported empathy as well as with physiological skin conductance measures of emotional responsiveness. Krebs (1975) has found that apparent similarity not only increases physiological correlates of emotion such as skin conductance, vasoconstriction, and heart rate, but also the willingness to reward the victim. In young children, the frequency of social interactions between friends corresponds closely to the frequency of acts of altruism between them (Strayer et al. 1979).

Data show that the tendency to choose similar individuals as friends is genetically influenced. In a study of delinquency among 530 adolescent twins by Rowe and Osgood (1984), path analysis revealed not only that antisocial behavior was about 50% heritable, but that the correlation of 0.56 between the delinquency of an individual and the delinquency of his friends was mediated genetically, that is, that students genetically disposed to delinquency were also genetically inclined to seek each other out for friendship. In a study of 396 adolescent and young adult siblings from both adoptive and nonadoptive homes, Daniels and Plomin (1985) found that genetic influences were implicated in choice of friends: Biological siblings were more similar to each other in the types of friends they had than were adoptive siblings.

To test further whether friends are more similar to each other genetically than they are to an average person and whether, like spouses, their resemblance is most marked in the more heritable components of shared traits, Rushton (1989b) examined blood types and differential heritability estimates. Their methods paralleled those used in the studies on heterosexual partners described in sections 6.1.1 and 6.1.2.

Seventy-six long-term, nonrelated, nonhomosexual male Caucasian friendship pairs ranging in age from 18 to 57 years were recruited by advertisements from the general community; the friendships had to have existed for at least one year. A control group was formed by randomly pairing individuals from the sample. At the testing session, a 12- to 14-milliliter blood sample was drawn from each person and many variables were measured, including those explicitly chosen because estimates had been calculated of the degree of genetic influence on the various components. For examples, 36 heritabilities were available with respect to 50 social attitude items (see Table 4) from data on 3,810 Australian twin pairs (Martin et al. 1986). For 90 items from the Eysenck Personality Questionnaire, two independent sets of heritability estimates were available for a total of 81 of the items, one set from 3,810 Australian twin pairs (Jardine 1985), and the other set from 627 British twin pairs (Neale et al. 1986). These intercorrelated with $r = 0.44$ ($p < 0.001$) and were aggregated to form a more reliable composite. For 13 anthropometric measures, estimates of genetic influence were available based on midparent offspring regressions from data on 125 families in Belgium (Susanne 1977). Test-retest data were available for the two questionnaire measures and pilot work had shown that the anthropometric measures could be made with very high levels of precision (Rushton 1989b).

6.3.1. Blood tests. The percentage of similarity between the friendship pairs was calculated for the same 10 loci from 7 polymorphic blood systems – ABO, Rhesus (Rh), MNSs, P, Duffy (Fy), Kidd (Jk), and HLA – used in the study of sexually interacting couples. The percentage of similarity for the same measure was also calculated for an equal number of randomly paired individuals from the same sample. The similarities, presented in Table 5, are significantly different from each other ($t(150) = 3.13$, $p < 0.05$). It is unlikely that this outcome is due entirely to stratification effects because within-pair differences in age, education, and occupation did not correlate with the blood similarity scores (mean $r = -0.05$). Thus, friends

Table 4. *Similarity between friends on conservatism items and their relation to heritability estimates (n = 76)*

Item	Heritability estimate	Friendship similarity score	Test-retest reliability	Similarity score corrected for reliability	Similarity score corrected for age, education, and occupation
1. Death penalty	.51	.28	.87	.30	.38
2. Evolution theory	—	.08	.95	.08	.20
3. School uniforms	—	.20	.99	.20	.42
4. Striptease shows	—	.13	.97	.13	.24
5. Sabbath observance	.35	.08	.91	.08	.09
6. Hippies	.27	.03	.97	.03	.15
7. Patriotism	—	.10	.89	.11	.13
8. Modern art	—	.02	.93	.02	.09
9. Self-denial	.28	.08	.79	.09	.12
10. Working mothers	.36	.07	.83	.08	.13
11. Horoscopes	—	.23	.92	.24	.20
12. Birth control	—	.04	−.01	.00	.19
13. Military drill	.40	.10	.96	.10	.22
14. Coeducation	.07	−.05	.74	−.06	−.05
15. Divine law	.22	.25	.82	.28	.20
16. Socialism	.26	.08	.83	.09	.14
17. White superiority	.40	.22	.68	.27	.11
18. Cousin marriage	.35	.04	.89	.04	.24
19. Moral training	.29	.07	.77	.08	.16
20. Suicide	—	.08	.86	.09	.08
21. Chaperones	—	.00	.94	.00	.11
22. Legalized abortion	.32	.13	.96	.13	.29
23. Empire building	—	.02	.85	.02	.05
24. Student pranks	.30	−.02	.88	−.02	.07
25. Licensing law	—	−.20	.85	−.22	−.13
26. Computer music	.26	.02	.91	.02	.16
27. Chastity	—	.00	.76	.00	.13
28. Fluoridation	.34	.08	.86	.09	.04
29. Royalty	.44	.15	.92	.16	.16
30. Women judges	.27	.03	1.00	.03	.08
31. Conventional clothes	.35	.31	.83	.34	.29
32. Teenage drivers	.26	.02	.78	.02	.20
33. Apartheid	.43	.14	.69	.17	.10
34. Nudist camps	.28	.08	.85	.09	−.09
35. Church authority	.29	.08	.86	.09	.21
36. Disarmament	.38	.07	.96	.07	.19
37. Censorship	.41	.03	.81	.03	.10
38. White lies	.35	.06	.76	.07	−.01
39. Caning	.21	.14	.83	.15	.11
40. Mixed marriage	.33	.25	.79	.28	.29
41. Strict rules	.31	.25	.81	.28	.19
42. Jazz	.45	.42	.77	.48	.40
43. Straitjackets	.09	.00	.85	.00	.00
44. Casual living	.29	.18	.63	.23	.55
45. Learning Latin	.26	.03	.97	.03	.10
46. Divorce	.40	.03	.92	.03	.09
47. Inborn conscience	—	.20	.70	.24	−.11
48. Colored immigration	—	.06	.88	.06	.10
49. Bible truth	.25	.30	.95	.31	.47
50. Pajama parties	.08	.08	.91	.08	.24

Table 5. *Percentage of similarity in friends and random pairs, based on 10 blood loci*

Relationship	Number of pairs	Mean	Standard deviation	Range	95% confidence interval for mean
Friendship pairs	76	54.01	12.02	22.20–79.50	51.26–56.76
Random pairs	76	48.17	10.94	22.20–71.70	45.67–50.67

are more similar to each other, genetically, than they are to randomly paired persons from the same sample.

6.3.2. Genetic similarity detection between friends. Examples of varying heritabilities include: 51% for attitude to the death penalty versus 25% for attitude to the truth of the Bible (see Table 4), 41% for having a preference for reading versus 20% for having a preference for many different hobbies (Neale et al. 1986), and 80% for mid-finger length versus 50% for upper arm circumference (Susanne 1977). In the studies summarized in Table 3, estimates of genetic influence in relationships between spouses were based on parent-offspring regressions; however, in the study by Rushton (1989b), described in Table 4, heritabilities calculated from the comparison of monozygotic and dizygotic twins raised together were the measures primarily used. When evaluating these results, it should be kept in mind that the friendship heritabilities were generalized from one sample (e.g., Australian twins) to another (Canadian friends). Behavioral scientists usually consider heritability estimates to be properties of particular populations and not to be highly generalizable (Falconer 1981; cf. Rushton 1989a). This results in a conservative test of the genetic similarity hypothesis because the predicted effect has to be sufficiently generalizable to overcome this problem.

Across the measures, close friends were found to be significantly more similar to each other than to randomly paired individuals from the same sample. Pearson product-moment correlations showed that compared with random pairs, friendship dyads are more similar in age (0.64 vs. −0.10, $p < 0.05$), education (0.42 vs. 0.11, $p < 0.05$), occupational status (0.39 vs. −0.02, $p < 0.05$), conservatism (0.36 vs. −0.02, $p < 0.05$), mutual feelings of altruism and intimacy (0.32 vs. −0.04 and 0.18 vs. −0.08, $ps < 0.05$), 13 anthropometric variables (mean = 0.12 vs. −0.03, ns), 26 personality scale scores (mean = 0.09 vs. 0.00, ns), and 20 personality self-rating scores (mean = 0.08 vs. 0.00, ns). Although these similarities are very small, significantly more are positive than could be expected by chance (13/13 of the anthropometric variables, 18/26 of the personality scale scores, and 15/20 of the personality self-rating scores, all $p < 0.05$, binomial sign test). It should be noted that these relative magnitudes parallel the between-spouse similarities (Buss 1985; Epstein & Guttman 1984; Rushton et al. 1985; Thiessen & Gregg 1980).

As with marriage partners, similarity between friends was most marked among the more genetically influenced of the characteristics. For the 36 conservatism items (see Table 4), the correlation of the estimates of genetic influence and between-friend similarity was 0.40 ($p < 0.01$); this relationship was not altered when corrected for test-retest reliability or when similarity in a composite of age, education, and occupational status was partialled out ($r = .40$, $p < .01$; $r = .32$, $p < .05$, respectively). For the 81 personality items, the correlation 0.20 ($p < .05$) was also not changed by a correction for test-retest reliabiity or socioeconomic similarity. For the 13 anthropometric variables, however, the correlation was not significant ($r = 0.15$). Given the stringent between-sample rather than within-sample test of the hypothesis, it seems reasonable to conclude that friends are choosing each other more on the basis of the genetically influenced components of similarity than on the basis of environmentally influenced components.

7. Discussion

The preliminary evidence presented so far supports the hypothesis that both friends and spouses choose each other partly on the basis of genetic similarity. The blood antigen data clearly show that friendship dyads as well as sexually interacting couples who produce children together are genetically more similar to each other than to random pairs from the same samples. The fact that similarity is greater for the more genetically influenced components of traits than for the environmentally influenced ones suggests that positive assortment is genetically mediated. Objections to these conclusions can certainly be raised, and alternative hypotheses are possible. Different theories, interpretations of data, and underlying mechanisms are discussed in the following sections.

7.1. Theory

It has been objected that genetic similarity theory is implausible and fallacious (for an exchange of views, see Mealey 1985; 1989; Rushton 1989c; Rushton & Russell 1985; see also Trivers 1985, p.423). The main point made by critics is that the overall proportion of genes shared by two individuals is irrelevant unless the genes 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) to follow Hamilton's rules and depend on the statistics of identity by common descent to ensure the presence of altruistic genes in others and (2) to discover some phenotypic character that is very closely linked to or associated with altruism (as in Dawkins's [1976] "green beard" idea,

described in section 2). Critics point out that the existence of such a character is considered unlikely even by the theory's formulators (Hamilton 1964; Dawkins 1976; 1982) because it would be in the interest of unlinked genetic loci to disrupt the altruist locus either by mimicking the phenotypic marker for parasitic purposes or by modifying the marker so the recognition system is foiled. Indeed, if one gene can evolve to produce such a complex phenotypic effect, alleles at other loci might also do so, resulting in an intragenomic "tug of war" as each gene attempts to influence the behavior of its bearer in its own interest (Alexander & Borgia 1978; Dawkins 1982).

These arguments do not altogether refute the theory, however. The mechanisms of gene recognition (if they exist) will be complex, perhaps involving many genes and supergenes on many chromosomes. For example, large groups of genes could become linked and pleiotropic, producing 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; thus aggregation effects would 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 the preceding argument most fully, pointing out that feature detectors, like other phenotypic characters, can be expected to be the product of multiple alleles; therefore, they accurately reflect the overall genome rather than particular parts. He cites hybridization studies of crickets and frogs showing that hybrid females orient preferentially toward 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 preferring like phenotypes would tend to proliferate. If feature detectors exist, they will lead not to *kin* recognition abilities, but to the discrimination of individuals who share appropriate phenotypic traits.

7.2. Interpretation of data

Several questions can be raised about the data sets. It might be suggested that the blood group similarities are due entirely to the effects of social stratification (i.e., finding oneself in the same location because of similar education and social background) rather than preferential assortment. The data do show that such stratification occurs. For example, Rushton (1989b) found friendship dyads to be significantly more similar in age (r = .64), education (r = .42), and occupation (r = .39) – variables for which the random pairs were unrelated. Moreover, investigators such as Beardmore and Karimi-Booshehri (1983) have found that blood groups are stratified by socioeconomic status (SES). In Britain, blood type A is found to occur more frequently in SES 1, the highest group (57% of the time), than in SES 5, the lowest group (41% of the time).

To test the stratification hypothesis, Rushton (1989b) calculated within-pair differences in age, education, and occupation and did not find them to significantly be correlated with friends' blood similarity scores as they should have been if the statification hypothesis was correct. (Nor was it found that such socioeconomic similarity altered the correlation between friendship similarity and the estimates of genetic influence.) It should also be noted that although evidence does show that stratification effects apply at a *single* gene locus (e.g., Beardmore & Karimi-Booshehri 1983), in our study we aggregated across 10 loci using 7 polymorphic marker systems on 6 different chromosomes to assess similarity. As mentioned earlier, such blood group differences provide a greater than 95% confidence rating for inclusion in cases of paternity dispute (Bryant 1980) and distinguish reliably between fraternal twins raised in the same family (Pakstis et al. 1972). On the basis of these preliminary attempts to test the social stratification hypothesis, then, such blood group similarities have not been explained.

With respect to the heritability analyses, one possible artifact could have been the differential reliability of the test items. If some had particularly low reliabilities, they would have reduced the estimate of both genetic influence and between-friend similarity, thus giving rise to a spurious correlation between them. For this reason, item reliabilities were calculated in the majority of the studies and the correlations were computed both with and without correction for item reliability. The estimates of genetic influence were consistently found to predict similarity scores, even across quite disparate samples, and even after controlling for within-pair similarity in age, education, and occupation.

Some confusion may result from a mistaken belief that heritability is being equated with importance and that more assortment should therefore occur in physical features than in social variables because the former are more heritable. We have consistently considered it necessary, however, to examine the relation between similarity scores and degrees of genetic influence within homogeneous data sets rather than comparing across heterogeneous traits, for two reasons. First, this presumably holds more constant the (unknown) number of genes involved (hair texture may be highly heritable but may only involve one or two genes; a behavioral item may be less heritable but may involve more genes), and the theory predicts that overall similarity is what matters. Second, since sequential filtering may be involved in the formation of interpersonal relationships, it may be best to make the comparisons at the same level.

Some readers may doubt whether variance in measures can legitimately be apportioned into estimates of genetic and environmental influence. Such uncertainties are common in both the evolutionary and social sciences. Increasingly powerful behavioral genetic techniques are available, however, for testing hypotheses about development (Eaves et al. 1978; Plomin 1986). Twin and adoption studies converge in showing moderate to substantial effects of genetic influence on the transmission of both socially undesirable traits such as crime, obesity, and schizophrenia, and more conventional characteristics such as vocational interests and value systems (Loehlin et al. 1988). In fact, Martin et al. (1986), in their study on the heritability of social attitudes, felt confident enough about the reliability and validity of their measuring instrument, theoretical model, estimation techniques, and

sample sizes (3,810 pairs of adult Australian twins plus a secondary analysis of 825 pairs of British twins) to predict between-person correlations in conservatism scores in other relationships if their model was accurate: 0.00 between foster parent and adult foster child; 0.52 between parents and children; and 0.62 for separated monozygotic twins. Recently, a study of 44 monozygotic twins reared apart has confirmed the last prediction of Martin et al., showing an intraclass correlation of 0.53 on a scale measuring traditional moral and family values (Tellegen et al. 1988).

7.3. Mechanisms

Section 3 included a discussion of four different mechanisms by which similarity detection can come about: innate feature detectors, phenotypic matching, familiarity or association, and location. The strongest version of genetic similarity theory suggests that individuals detect genetic similarity in the absence of previous familiarity or other proximal mechanisms. The weaker versions predict that organisms will tend to direct altruistic behavior toward similar individuals when the similarity is detected by means other than genetic recognition. One such means would be phenotype matching based on familiarity with self or kin. Location (section 3.4) may be an additional means. This is likely to be based on similar genes because intelligence, socioeconomic status, values, and vocational interests have been shown to be genetically linked (Loehlin et al. 1988). If there are evolutionary advantages to be derived from the ability to benefit individuals who are genetically similar to oneself, many mechanisms may be involved.

Although the data reported here are only correlational, their pattern allows us to eliminate certain alternative hypotheses as implausible. Reverse causality, for example, whereby mutual interaction within the partnership *causes* the similarity, cannot be occurring because the genes that cause the blood types and individual traits clearly preceded the onset of the relationship. Moreover, several studies have shown that human assortative mating for similarity on the more genetically based items can be predicted by measures obtained early in or even prior to the marriage and that the degree of resemblance between spouses does not change over time (see Russell et al. 1985, for review).

Hypotheses that "third variables" are causes of both similar genes and social assortment are also unlikely to be valid because they should predict zero correlations between heritabilities and within-trait similarity. Consider the hypothesis that people vary genetically only in the dispositions that cause them to enter different professions, geographical areas, or subcultures within a stratified society, but that thereafter they associate randomly. Associates might thus be expected to be genetically similar compared with individuals chosen at random from the global population, but only as an "artifact" of genes for location. This would fail to explain why similarity is more pronounced in the more genetically influenced components of anthropometric, attitudinal, personality, and cognitive variables. It is unlikely that the patterning of the item heritabilities will be relevant to the individual's location. Moreover, contrary to the stratification hypothesis, Rushton (1989b) showed that the degree of dyadic difference in variables such as level of education was uncorrelated with the similarity scores on the blood tests or with heritability estimates.

The validity of purely environmental theories of the similarity attraction hypothesis also seems unlikely. The widespread social psychological view that people choose similar friends and spouses because of past histories of socialization and enculturation that enable them to reinforce each other more effectively (Byrne 1971) seems to imply a prediction *opposite* from the one made by genetic similarity theory: There should be a negative, not a positive, correlation between item heritabilities and degree of similarity because the more environmentally modifiable an item, the greater the degree to which it can be shared by people as a result of common experiences. This differential prediction is disconfirmed.

Finally, other, very different hypotheses about human relationship formation can be ruled out. The views that "opposites attract" and that people assort on the basis of complementarity rather than similarity are not supported by the data. Whereas such effects may still occur in individual cases or for particular traits, no evidence for them was found in the present review. This is not to deny the anomalies that exist. For example, Johnson (1984) has shown that about 50% of all civilian marriages in Hawaii are cross-ethnic today. Although data indicate that in such marriages couples appear to "make up" for ethnic dissimilarity by choosing greater similarity in other areas (Ahern et al. 1981), this finding does indeed appear to contradict these views. However, it is also clear that human mate choice depends on many more variables than similarity [see Buss: "Human Mate Preferences" *BBS* 12 (1) 1989].

8. Distal-proximal levels of explanation

Some readers may be reluctant to accept the hypothesis that there is a genetic component in the "similarity attraction" link, arguing that we like those who are similar to us for cognitive reasons (such as the validation of our view of the world). Others will point to the experimental evidence that familiarity and social learning also mediate relationships (Berscheid 1985; Byrne 1971). There may also be a reluctance to accept the proposition that cognition, choice, and learning are partly derivatives of genetics. A preference for proximal explanations, however, should not rule out more distal factors. Consider Figure 1 in which explanations are depicted as varying in a time dimension; the figure shows that there need be no conflict between levels. Evolutionary biologists do not find the heritability of traits problematic, trait theorists can accept the view that dispositions are modified by later learning, and learning theorists can accept that the products of early experiences interact with subsequent circumstances to produce emotional arousal and cognition.

8.1. Epigenetic rules in social development

Cultural practices and social learning play such an important role in human social behavior that the concept of an "epigenetic rule" (defined as a program, complete with self-correcting feedback system, whereby individual de-

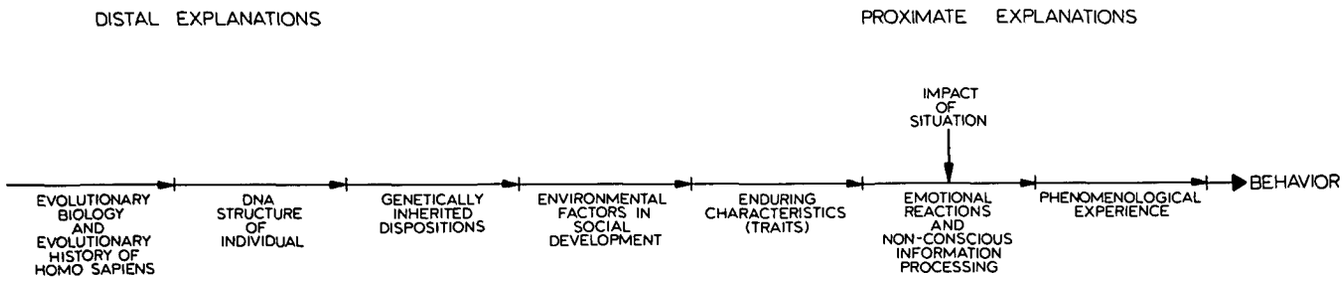

Figure 1. The distal-proximal dimension and levels of explanation of social behavior. When explanations move from distal to proximal, controversy does not ensue, whereas the converse is not always true. (*Source:* Rushton 1988b.)

velopment is guided in one direction rather than another) may help us understand how social influences are genetically channeled (Lumsden & Wilson 1981). Epigenetic factors are most apparent in embryology, where the physical development from fertilized egg to neonate follows a consistent course in normal environments. Channeled development is also illustrated by findings from behavior genetics (Bouchard 1984; Plomin & Daniels 1987; Rushton 1988b). Identical twins show a high degree of concordance in age of onset of puberty, timing of first sexual experience, and menopause. Genetic timing mechanisms also affect cognitive development, as shown in a large sample of twins who were studied from 3 months to 15 years of age; the synchronies between lags and spurts in mental development were found to average about 0.90 for identical twins, but only about 0.50 for fraternal twins (R. S. Wilson 1983).

Psychological development is also guided by epigenetic rules, from sensory filtering through perception to feature evaluation and decision-making (Lumsden & Wilson 1981). For example, whereas the brain perceives variation in luminance along a continuum, it divides hue into categories, using language to do so (Harnad 1987). Many social scientists used to believe that the division into red, green, and so forth were arbitrary, but linguistic and cross-cultural studies have shown that they are in fact closely tied to the inborn physiology of color perception. Epigenetic rules governing more complex social behavior have also been identified. Targeted endpoints appear to underlie the evolutionary function of smiling, attachment, and separation responses in infants (Freedman 1974). [See also Lamb et al: "Security of Infantile Attachment" BBS 7 (1) 1984.] Similar interpretations can be advanced for the life-cycle stages documented to occur in ego development, morality, and psychosocial functioning (Alexander 1987).

Other behavior genetic evidence also provides support for the role of epigenetic rules in social development. For example, whereas small fluctuations in one or two molecules might affect ontogeny, studies show that siblings raised apart for many years in complex environments grow to be significantly similar to each other in a variety of traits; their degree of similarity is predicted by the number of genes they share (Tellegen et al. 1988). It has also been found that the environmental factors influencing development are not shared but are unique to each sibling; that is, the important environmental variance turns out to be *within* a family, not *between* families. Such factors as social class, family religion, parental values, and child-rearing styles are *not* found to have a common effect on siblings (see Plomin & Daniels 1987). This is true even of traits such as altruism and aggression, which parents are expected to socialize heavily (Rushton, Fulker, Neale, Nias & Eysenck 1986).

These data suggest the existence of genetically based stabilizing systems that channel development in such a way that, within the constraints allowed by the total spectrum of cultural alternatives, people create environments maximally compatible with their genotypes (Lumsden & Wilson 1981; Plomin & Daniels 1987; Scarr & McCartney 1983; Rushton, Littlefield & Lumsden 1986). Thus, even within the same rearing environments, genetically different siblings are biased to learn different items of information because they have different sets of epigenetic rules channeling their common environment in individual ways.

In an analysis of the effects of television, for example, Rowe and Herstand (1986) found that although same-sex siblings resemble one another in their exposure to violent programs, it is the more aggressive sibling who (1) identifies more with aggressive characters, and (2) views the consequences of the aggression as positive. Within-family studies of delinquents have found that both intelligence and temperament distinguish delinquent siblings from those who are not delinquent (Hirschi & Hindelang 1977; Rowe 1986). It is not difficult to imagine how intellectually and temperamentally different siblings might seek out different social environments.

Of all the decisions people make that affect their environment, choosing friends and spouses may be one of the most important. Thus, epigenetic rules, by influencing preferences, may prove useful in ordering the hypothetical levels in Figure 1, for any distal "purpose" of the genes must necessarily be mediated by proximal mechanisms.

9. Ethnocentrism and ideology

The potential effects of epigenetic rules on behavior and society may go well beyond ontogeny. Through cognitive phenotypes and group action, altruistic inclinations may find their expression in charities and hospitals, creative and instructional dispositions can give rise to academies of learning, martial tendencies to institutes of war, and delinquent tendencies to social disorder. The idea that genes have such extended effects beyond the body in which they reside, biasing individuals toward the production of particular cultural systems, is a central focus of current thinking in sociobiology (Boyd & Richerson 1985;

Dawkins 1982; Lopreato 1984; Lumsden & Wilson 1981; Ruse 1986). [See also multiple book review of Lumsden & Wilson in *BBS* 5 (1) 1982.]

The implications of the finding that people moderate their behavior as a function of genetic similarity may be far-reaching. They may suggest a biological basis for ethnocentrism, for example. Despite enormous variance within populations, it can be expected that two individuals within an ethnic group will, on average, be more similar to each other genetically than two individuals from different ethnic groups. According to genetic similarity theory, people can be expected to favor their own group over others. Ethnic conflict and rivalry is one of the great themes of historical and contemporary society (Horowitz 1985; Reynolds et al. 1987; Rushton 1986; van den Berghe 1981). Local ethnic favoritism is also displayed by group members who prefer to congregate in the same area and to associate with each other in clubs and organizations. Many studies have found that people are more likely to help members of their own race or country than they are to help members of other races or foreigners, and that antagonism between classes and nations may be greater when a racial element is involved (see Cunningham 1981, for review).

Traditionally, political scientists and historians have seldom considered intergroup conflict from an evolutionary standpoint. That fear and mistrust of strangers may have biological origins, however, is supported by evidence that animals show fear of and hostility toward strangers, even when no injury has ever been received. For example, direct analogies have been drawn between the way monkeys and apes resent and repel intruding strangers of the same species and the way children attack another child who is perceived as being an outsider (Gruter & Masters 1986; Hebb & Thompson 1968). Many influential social psychologists have pondered whether the transmission of xenophobia could be partly genetic. W. J. McGuire (1969) wrote that "it appears possible for specific attitudes of hostility to be transmitted genetically in such a way that hostility is directed towards strangers of one's own species to a greater extent than towards familiars of one's own species or towards members of other species. It would not be impossible for xenophobia to be a partially innate attitude in the human" (p. 265).

Theorists from Darwin and Spencer to Allport and Freud and now Alexander, Campbell, and E. O. Wilson have considered in-group/out-group discrimination to have roots deep in psychobiology. (For a historical review, see van der Dennen 1987.) Recent developmental psychological studies have found that even very young children show clear and often quite rigid disdain for children whose ethnic and racial heritages differ from their own, even in the apparent absence of experiential and socialization effects (Aboud 1988).

Despite this background, many sociobiologists are equivocal about whether there are fitness implications for ethnic or national preference (Dawkins 1981; Lopreato 1984; Reynolds et al. 1987; Trivers 1985; van den Berghe 1981; E. O. Wilson 1978). Dawkins (1981) has written: "The equating of 'kin-ship', in the sense of kin-selection, with 'ties of race' appears to result from an interesting variant of what I have called the fifth misunderstanding of kin-selection" (p. 528). Trivers (1985) notes: "Indeed, for large political groups like the United States of America, degrees of relatedness between virtually all members are nearly zero" (p. 135). Many sociobiologists, in an effort to condemn racism, may inadvertently have minimized the theoretical possibility of a biological underpinning to ethnic, national, and racial favoritism (see, for example, almost all contributors to Reynolds et al. 1987). As Hamilton (1987) has remarked, in the context of why kin discrimination, even among animals, is not more readily expected: "in civilized cultures, nepotism has become an embarrassment" (p. 426; see also Alexander 1987, p. 192).

Many of those who have considered nationalist and patriotic sentiment from a sociobiological perspective have emphasized its apparent irrationality. Johnson (1986) formulated a theory of patriotism in which socialization and conditioning engage kin-recognition systems so that people behave altruistically toward in-group members, as though they were genetically more similar than they actually are. In Johnson's analysis, patriotism may often be an ideology propagated by the ruling class to induce the ruled to behave contrary to their own genetic interests, while increasing the fitness of the elite. He noted that patriotism is built by referring to the homeland as the "motherland" or "fatherland," and that bonds between people are strengthened by referring to them as "brothers and sisters."

According to genetic similarity theory, patriotism may often be more than just "manipulated" altruism working to the individual's genetic detriment. It may be an epigenetically guided strategy by which genes replicate copies of themselves more effectively. The developmental processes that Johnson (1986) and others have outlined undoubtedly occur (Rushton 1980), as do other forms of manipulated altruism (Dawkins 1982). However, if these were sufficient to explain the human propensity to feel strong moral obligation toward society, patriotism would remain an anomaly for evolutionary biology. From the standpoint of optimization, one might ask whether evolutionarily stable ethical systems would survive very long if they consistently led to reductions in the inclusive fitness of those believing in them (Alexander 1987; Ruse 1986; E. O. Wilson 1978). [See also Vining: "Social versus Reproductive Success" *BBS* 9 (1) 1986.]

If epigenetic rules do incline people toward constructing (Findlay & Lumsden 1988) and learning those ideologies which generally increase their fitness, then patriotic nationalism, religious zealotry, class conflict, and other forms of ideological commitment (even "international socialism") can be seen as genetically influenced cultural choices that individuals make that, in turn, influence the replication of their genes. Religious, political, and other ideological battles may become as heated as they do partly because of implications for fitness; some genotypes may thrive more in one ideological culture than another. In this view, Karl Marx did not take the argument far enough: Ideology serves more than economic interest; it also serves genetic purpose.

Two sets of falsifiable propositions follow from this interpretation. First, individual differences in ideological preference are partly heritable. Second, ideological belief increases genetic fitness. There is evidence to support both propositions. With respect to the heritability of differences in ideological preference, it has generally been assumed that political attitudes are mostly determined by the environment; however, both twin and

adoption studies reveal moderate heritabilities of specific social and political attitudes (Table 4), as well as of stylistic tendencies such as authoritarianism and the voicing of extreme views (Eaves & Eysenck 1974; Martin et al. 1986; Scarr & Weinberg 1981; Tellegen et al. 1988).

Obvious examples of ideologies that might increase genetic fitness are religious beliefs that regulate dietary habits, sexual practices, marital custom, infant care, and child rearing (Lumsden & Wilson 1981; Reynolds & Tanner 1983). Amerindian tribes that cooked maize with alkali had higher population densities and more complex social organizations than tribes that did not, partly because cooking with alkali releases the most nutritious parts of the cereal, enabling more people to grow to reproductive maturity (Katz et al. 1974). The Amerindians did not know the biochemical reasons for the benefits of alkali cooking, but their cultural beliefs had evolved for good reason, enabling them to replicate their genes more effectively than would otherwise have been the case.

By the way of objection, it could be argued that although some religious ideologies confer direct benefits on the extended family, ideologies like patriotism decrease fitness (hence most analyses of patriotism would ultimately rest entirely on social manipulation). Genetic similarity theory may provide a firmer basis for an evolutionary understanding of patriotism, for benefited genes do not have to be only those residing in kin. Members of ethnic groups, for example, often share the same ideologies, and many political differences are genetic in origin. One possible test of genetic similarity theory in this context is to calculate degrees of genetic similarity among ideologues in order to examine whether ideological "conservatives" are more homogenous than the same ideology's "liberals." Preserving the "purity" of an ideology might be an attempt to preserve the "purity" of the gene pool. Now that numerous measures of genetic distance are becoming available (Jeffreys et al. 1985; Jorde 1985; Stringer & Andrews 1988) this line of research is becoming feasible.

10. Group selection

Humans have obviously been selected to live in groups, and the line of argument presented so far may have implications for determining whether group selection occurs among humans. Although the idea of group selection, defined as "selection that operates on two or more members of a lineage group as a unit" (E. O. Wilson 1975, p. 585), and as "the differential reproduction of groups, often imagined to favor traits that are individually disadvantageous but evolve because they benefit the larger groups" (Trivers 1985, p. 456), was popular with Darwin, Spencer, and others, it is not currently thought to play a major role in evolution. Hamilton's (1964) theory of inclusive fitness, for example, is regarded as an extension of individual selection, not group selection (Dawkins 1976; 1982). Indeed, in recent times group selection has "rivaled Lamarkianism as the most thoroughly repudiated idea in evolutionary theory," as D. S. Wilson put it (1983, p. 159). Mathematical models (reviewed in D. S. Wilson 1983) show that group selection could override individual selection only under extreme conditions such as small intergroup migration rate, small group size, and large differences in fitness among groups.

In the recent past it was Wynne-Edwards (1962) who brought the altruism issue to theoretical center stage. He suggested that whole groups of animals collectively refrain from overbreeding when the density of the population becomes too great – even to the point of directly killing their offspring if necessary. Such self-restraint, he argued, protects the animals' resource base and gives them an advantage over groups that do not practice restraint and become extinct as a result of their profligacy. This extreme form of the group selection claim was immediately disputed by other biologists. A great deal of argument and data was subsequently against the idea (Williams 1966). There did not seem to exist a mechanism (other than favoring kin) by which altruistic individuals could leave more genes than selfish individuals who cheated.

One compromise position was offered by E. O. Wilson (1975), who suggested that although genes are the units of replication, their selection could take place through competition at both the individual and the group levels; for some purposes these can be viewed as opposite ends of a continuum of nested, ever enlarging sets of socially interacting individuals. Kin selection is thus seen as intermediate between individual and group selection. Genetic similarity theory, according to which genes maximize their replication by benefiting *any* organism in which their copies are to be found, may provide a mechanism by which group selection can be enhanced.

Among humans, the possibility of conferring benefits on genetically similar individuals has been greatly increased by culture. Through language, law, religious imagery, and patriotic nationalism, all replete with kin terminology, ideological commitment enormously extends altruistic behavior. Groups made up of people who are genetically predisposed toward such moral behaviors as honesty, trust, temperence, willingness to share, loyalty, and self-sacrifice will have a distinct genetic advantage over groups that did not. In addition, if strong socialization pressure, including "mutual monitoring" and "moralistic aggression," is used to shape behavior and values *within* the group, a mechanism is provided for controlling, and even removing, the genes of cheaters (Campbell 1983; Rushton 1980). Several recent analyses suggest that evolution under culturally driven group selection, including migration, war, and genocide, may account for a substantial amount of change in human gene frequencies (Alexander 1987; Ammerman & Cavalli-Sforza 1984; Chagnon 1988; Melotti 1987; D. S. Wilson 1983).

Findlay, Lumsden, and Hansell (in press) have provided a mathematical group selection model in which genetic and cultural parameters are allowed to interact; this goes considerably beyond the previous group selection theories, which examined altruism only as a function of genetic information. The logic of Findlay et al. (in press) is as follows. Since humans have evolved to be "trend watchers," the most common phenotype is likely to be adopted, irrespective of its effect on fitness or on the genetic contribution to altruism. This means that relative to the pure genetic case (on which all traditional group selection models are based), the correlation between genotypic and phenotypic values is reduced. The inten-

sity of individual selection against altruistic genotypes is consequently reduced. On the other hand, because of group structure and frequency-dependent learning, "selfish" groups become more selfish and "altruistic" groups become more altruistic, thereby increasing the variance among groups. Since variance is what drives group selection, the end result is an increase in the efficacy of group selection in biocultural systems compared with purely genetic ones.

Given also that human populations differ, genetically, in the mechanisms underlying their behavior (Rushton 1988c), group selection may be expected to have additional effects on human evolution. Genetic similarity theory, together with the Findlay-Lumsden view, suggests that there should be a correlation between an individual's genotype and the particular trend that is watched. As reviewed earlier, social learning is biased by individualized epigenetic rules. Social psychological studies of cultural transmission make clear that people pick up trends more readily from role models who are both similar and of high status (Bandura 1986). If different ethnic groups learn from different trend-setters, the group selection models of Findlay et al. (in preparation) may have further implications for fitness. In the evolutionary past, for example, those groups that adopted an optimum degree of ethnocentric ideology may have replicated their genes more successfully than those that did not.

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A methodological critique of the evidence for genetic similarity detection

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In this commentary I argue that evidence crucial to Rushton's hypothesis is statistically flawed. I will describe three sources of error: spurious index correlations, inadequate definition of "traits," and invalid significance testing for correlations calculated on nonindependent samples.

A critical assumption of the genetic similarity hypothesis, which differentiates it from alternative phenotypic models of preference formation, is that somehow people "know" the heritabilities of traits and prefer other people who are similar to them in traits with high heritabilities. Thus, the one block of evidence in Rushton's target article that cannot be explained by alternative phenotypic models is the reported correlation between heritability of "traits" and degree of assortment (in couples or between pairs of friends) of those traits. Unfortunately, there are severe problems with the way Rushton calculates the magnitude of r and tests the significance of these correlations.

First, the r values reported in Tables 3 and 4 are spuriously large and positive because both heritability and intrapair correlation are ratios with a common variable denominator. McNemar (1969) demonstrated that correlations between such ratios can be as high as 0.5 *even when the numerators are completely uncorrelated.* This applies to the correlations between heritability and assortment in the following way: The heritability of a trait is defined as the ratio s^2_A/s^2_T, where s^2_T is the total variance in the population for that trait and s^2_A is the variance in the population attributable to additive genetic effects. The estimated product-moment correlation coefficient r_{XY} for a trait is defined as $s_{XY}/s_X s_Y$, where s_{XY} is the covariance between X and Y for the trait, and s_X and s_Y are the standard deviations of the X and Y samples, respectively. When s_X and s_Y are about equal (a safe assumption for pairs of friends, and a likely one for couples), their product will be close to the population variance for the trait, s^2_T, because both approximate s_T, and the ratios will indeed share a common variable denominator. Under these conditions, even if all the pairwise correlations between the numerators and the common variable denominator are zero, there will still be a positive correlation between the ratios. It is a function of the coefficients of variation respectively of the numerators and of the common denominator; if these are all equal, this correlation coefficient is 0.5 (McNemar 1969, p. 181).

This fact completely alters the interpretation of Tables 3 and 4. The positive correlation coefficients that appear there result (at least in part) for the spurious component arising from definitions of the correlation coefficient and heritability. They do *not* imply that the numerators of those ratios (the additive genetic variance and intrapair covariance of the trait) are correlated, and they cannot be construed as evidence supporting the genetic similarity hypothesis. To present these data correctly, Rushton should reanalyze each correlation according to McNemar's (1969) formulas; only the component not attributable to the common variable denominator is relevant to his hypothesis.

The second problem with Rushton's analysis in Tables 3 and 4 is that the significance tests are biased toward rejection of the null hypothesis because the sampled "traits" are not independent. This bias stems ultimately from his failure to define the population of traits relevant to the genetic similarity hypothesis. In failing to give such a definition, he has violated two important assumptions of correlation analysis: The population of traits should be sampled randomly or systematically, and the sampled traits for each analysis should be independent. These violations are so serious that significance testing is probably invalid for the correlations in Tables 3 and 4, even if they are corrected for the spurious index component.

Rushton's "traits" range from subscales of tests, to items on a questionnaire, to arbitrary physical or performance measurements. The most serious errors occur in the latter two categories. Consider, for example, Table 4, in which Rushton correlates heritability and intrapair correlations for 36 items on a questionnaire and uses 36 as his sample size for the significance test. The authors of the conservatism questionnaire (Martin et al. 1986) state that the questionnaire can be reduced to only two factors, tough-mindedness and political left/right. Thus, the 36 items are really just strongly intercorrelated measures of (at most) two "traits"; the sample size should be 2. The same objections apply to other arbitrary collections of "traits" re-

ported in Table 3, such as "36 anthropometric variables" (Susanne 1977), "54 personality scales" (Rushton & Russell 1985), and "5 perceptual judgments" (Russell et al. 1985).

Although the analysis of collections of subscales is less seriously flawed, correlations between subscales of a given test are rarely 0, so a correction of the degrees of freedom is also needed for each case (if significance testing is at all valid for such nonrandom samples).

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Why help friends when you can help sisters and brothers?

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There are two parts to this commentary. In the first I question the relevance or adequacy of two of the three lines of evidence cited by Rushton to support the application of genetic similarity theory to altruism. In the second I argue that the theory is not a logical extension of kin selection, and therefore cannot account for evidence suggesting that human friends are genetically similar.

The three sources of evidence Rushton cites to support the view that human altruism can be explained by genetic similarity theory are spouse selection, intrafamilial relationships, and friendship. It is likely that spouse selection is based on a different selective advantage, however, involving a balance between the costs of inbreeding and the costs of outbreeding (Bateson 1980; 1982; van den Berghe 1983). Although Rushton acknowledges that possibility, he does not recognize that such an explanation is quite different from the one he advocates, namely, selection for altruistic behavior directed toward individuals showing signs of genetic relatedness. Although the distinction has clearly been made in papers on kin recognition in animals (e.g., Waldman 1987, p. 160), the two explanations have also been conflated in other papers by Rushton and his colleagues (Rushton 1988a; Rushton & Nicholson 1988; Russell, Wells & Rushton 1985).

The evidence cited by Rushton for intrafamilial relationships is restricted to two studies. One was of perceived closeness in monozygotic and dizygotic twins. The other (Littlefield & Rushton 1986) sought to test a number of predictions about differences in the severity of grief following the death of a child, mainly according to the degree of genetic relatedness of the grieving person to the deceased. As indicated in a reply (Archer 1988), this study had a number of methodological weaknesses, such as the use of a single-item rating scale to measure grief severity, low inter-rater reliabilities, and an invalid analysis of variance. All three criticisms apply to the finding (highlighted in the target article) that grief is more severe when the child is seen as resembling the bereaved relative's side of the family; the 74% agreement mentioned in the target article (sect. 6.2, para. 3) is not high considering that there would be 50% agreement by chance. A statistic that corrects for chance, such as Cohen's kappa (Cohen 1960) is more appropriate than percentage agreement in this case.

Rushton's third source of evidence concerns similarity amongst friends. Research in this area always encounters the problem of deciding on an appropriate control group; there are also a number of other hypotheses that may explain some of the evidence cited. Nevertheless, this line of evidence provides more convincing support for the application of genetic similarity theory to altruism than do the other two. I would argue that this evidence cannot supply the correct explanation, however, because the theory is *not* a logical extension of kin selection, as Rushton claims.

In a reply to a paper containing arguments very similar to those in Rushton's target article, Mealey (1985) argued that genetic similarity theory adds little that is new to "kin selection," and that what is new is logically flawed. As Rushton acknowledges, Hamilton (1964), Dawkins (1976), and others recognized that kin selection does not simply predict responding altruistically to close kin and nonaltruistically to others. It is the *degree* of relatedness that is important – or, more precisely, the probability that two individuals share an "altruistic gene" (Mealey 1985); this is associated with the degree of relatedness, however (Dawkins 1979; Rushton & Russell 1985).

If the degree of relatedness is viewed as a continuum, and it is realized that "there are no definite lines to be drawn between family and nonfamily" (Dawkins 1976, pp. 101–2), does this inevitably mean that kin selection is equivalent to genetic similarity theory? Not according to Mealey (1985), who argued that there is a distinction between phenotypic similarity due to relatedness, which is correlated with the probability of sharing an altruism gene, and phenotypic similarity not due to relatedness, which is uncorrelated with the probability of sharing an altruistic gene. Mealey argued that the latter is likely to be selected out. It is possible, however, for individuals to possess a proximate mechanism that causes them to behave altruistically toward those who physically resemble them; by doing so they would indirectly be aiding their kin (Dawkins 1976, p. 107). If the number of "mistakes" is very small, such a mechanism may not be selected out, and it could form the basis of some aspects of friendship choice. But this assumption is not the same as "genetic similarity theory."

Since the degree of relatedness does fall on a continuum, one prediction from both kin selection and genetic similarity theory is that close relatives will be helped to a greater extent than will distant relatives or unrelated individuals. Any altruistic tendency toward second or third cousins, for example, will be greatly diluted compared with that directed toward sibs because the probability that such individuals share an altruistic gene will be much smaller. It will be even smaller still, as Mealey pointed out, with unrelated individuals who are genetically similar: As relatedness decreases, so do the likely benefits from helping each individual; and the total cost of helping enough individuals to enhance fitness to the same extent as would be achieved from helping close kin becomes greater. Hence it should generally be more advantageous to help close kin such as brothers or sisters than to help distantly related or unrelated individuals. This conclusion is consistent with analyses of human altruistic behavior, such as the often cited study of axe fights among the Yanamamo of Venezuela (Chagnon & Bugos 1979), who take risks to aid kin rather than nonkin. Here the data can be accounted for by the desire to help *close* kin, but not by making distinctions between distant kin (Hinde 1987, pp. 168–69), as genetic similarity theory would predict.

Another consideration is competition. When the benefits of obtaining resources, or the costs of not obtaining them, are high, even closely related individuals show pronounced competition; for example, the fratricide among several species of eagle (Stinson 1979). Thus competition, as well as altruistic behavior, influences fitness, even that of close kin. Distantly related or unrelated individuals would be expected to take such competitive routes to enhancing fitness in preference to altruistic ones under a wide range of circumstances.

In conclusion, I view genetic similarity theory as unlikely to operate widely in animals as an explanation of altruism owing to (1) the greater advantage to be gained from helping close kin; (2) the unlikely chance that distantly related or unrelated individuals will share an altruistic gene; and (3) the tendency to compete with individuals who are not close kin.

A firmer basis for a functional approach to friendship would,

in my opinion, be obtained by considering the increased advantages that arise from forming alliances with other individuals who possess specific characteristics.

On distinguishing evolved adaptation from epiphenomena

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Rushton has made a real contribution through his imaginative and wide-ranging efforts to assess empirically the ways in which various human social phenomena are nonrandomly patterned with respect to the genetic relatedness of the parties. His interpretations of those patterns are often debatable, however. Here, I offer alternatives to Rushton's explanations of two specific phenomena, followed by two more general criticisms.

In his abstract, Rushton claims that sexually interacting couples who produce a child together are more alike genetically than those who do not. The basis for this claim seems to be the finding (section 6.1.1) that within a sample of cases of disputed paternity, men not excluded from paternity had significantly greater genetic concordance with their partners (the children's mothers) than did those who were excluded. Unfortunately, the probability of exclusion in the event of misattributed paternity is itself influenced by the genetic concordance of the mother with the misidentified sire, so that the observed difference might be expected even if fertility and genetic similarity were unrelated. A more complex null model is necessary.

In Table 3, Rushton shows that the degree of positive assortative mating on a given trait tends to be correlated with that trait's heritability. He takes this as evidence that the more heritable traits are more heavily weighted as mate choice criteria because they "reflect the underlying genotype better and provide a more accurate cue for matching." The same correlation would arise, however, if individuals acquired mate choice criterion templates from exposure to their parents (or other relatives) and combined criteria in a way that did not weigh the more heritable traits more heavily. If men marry the images of their mothers, for example, then men will most resemble their wives in those traits in which they themselves most resemble their mothers, hence the most heritable traits. In either case, mate choice strategies might reflect a specific adaptation for avoiding excessive outbreeding, but Rushton's account would seem to require a distinct calibration by natural selection of the relevance of each mate choice criterion according to its characteristic level of heritability over evolutionary time, whereas the "parental templating" process envisioned here would operate more parsimoniously and generally. These alternatives can be distinguished empirically, by determining whether the correlation between strength of assortment and heritability disappears when one assesses the similarity of Ego's mate to Ego's parents rather than to Ego.

Turning to more general criticisms, I question the rationale for suggesting that phenomena like "ethnocentrism" and "patriotism" can be understood as evolved "strategies." This argument seems to ignore the probable characteristics of the environments in which the human psyche evolved – where people of other ethnicities (let alone nationalities) were essentially never encountered, and where the selection process whose microstructure was sufficiently fine to shape adaptations was surely that of differential survival and reproductive success within local populations. No one doubts that "group selection" in the sense of differential survival and expansion of subpopulations *occurs*. The unpopularity of the concept derives from skepticism that group selection could sculpt complex adaptation out of random variation when opposed by the much more numerous selective events taking place between individuals, and I cannot see how Rushton's arguments make a *potent* group selection any more plausible. Whether "patriotism" actually contributes to the longevity of "gene pools" is neither here nor there with respect to its status as a putative evolved adaptation. There is a potentially important difference between interpreting ethnocentrism, patriotism, and class conflict as epiphenomenal to evolved nepotistic strategies and calling them strategies in their own right.

Finally, the claim that "genetic similarity theory" constitutes a more general theory than "kin selection" warrants criticism. In section 7.1, Rushton claims that "if like appearance is positively correlated with like genes, any mutation toward preferring like phenotypes would tend to proliferate"; he tries to distinguish this process from "kin selection" by suggesting that such proliferation will occur regardless of kinship. But it will not. The hypothetical mutation will benefit itself (hence proliferate) only if the phenotypic (and genetic) similarity being assessed is predictive of concordance at the mutated locus controlling the behavioral preference, and such a concordance is to be expected only because of kinship. Rushton cites criticisms of his theory and evidently feels he has refuted them, but he neither refutes nor acknowledges their central point: that there is no reason to expect an estimate of "genetic similarity," based on no matter how many loci, to be predictive of similarity at other loci except by virtue of kinship.

Thus, I must concur with the critics Rushton cites that his claims for genetic similarity theory represent a fallacy, and I have a hypothesis about its origins. I think that the fault lies with the catch phrase "kin selection," which has engendered two widespread and unnecessary confusions." The more pervasive confusion is to misunderstand "kin selection," which properly refers to an evolutionary process, as referring instead to an evolved product of that process, namely, selectivity in one's social behavior in relation to kinship. Rushton promotes just such a misunderstanding when he introduces the phrase in section 2: "By a process known as *kin selection* individuals maximize . . . ." The second common confusion about "kin selection" derives from the phrase's parallelism with "individual" selection and "group" selection, which obscures its lack of semantic parallelism. These two concepts refer to the differential survival and reproductive success of discrete "individuals" and "groups," but there are no analogous discrete and differentially successful "kins" in "kin selection." The confusion engendered here is that Hamilton's theory is widely misconstrued as identifying a "level of selection" intermediate between individuals and groups. An implicit conception of this sort may explain Rushton's apparent equation of the "kin" in "kin selection theory" with close "family members," and his contrasting of "kin" with "strangers," as if these orthogonal concepts were opposites (section 2). For the sake of clear communication, it is time to abandon the unfortunate phrase "kin selection."

Genetic similarity theory needs more development

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Rushton raises some important questions concerning the evolutionary mechanisms influencing not only human beings but all other higher organisms. We need to be absolutely clear, however, about just what is involved. The theory of kin selection is unusual as sociobiological theories go because it is extremely limited in scope. It defines the conditions under which altruistic behaviour can evolve when organisms have no information at all

about the identity of those individuals who are carriers of the same altruistic gene. Hamilton's rule (Hamilton 1964) states, quite simply, that all other things being equal, relatives are more likely to be carriers than nonrelatives. The theory cannot be used to explain the evolution of any trait other than altruistic behaviour. It cannot, for example, be used to explain the evolution of assortative mating for similar traits. What Rushton's genetic similarity theory offers us is hence something rather different. For that reason, it deserves serious consideration, even though as it stands, it simply will not do.

The core of the problem with genetic similarity theory is the underlying genetic mechanisms it assumes. Insofar as we understand the processes of inheritance, it seems that genetic linkage cannot be counted on to entrain many characters together through successive generations. This is why evolutionary questions have to be phrased in terms of selection for specific genes. Unless and until someone can determine the processes involved in such a linkage at the molecular level, we are obliged to assume that it does not occur. For this reason, Hamilton's theory of kin selection (and indeed the whole concept of inclusive fitness) is phrased very specifically, not in terms of the proportion of shared genes (contrary to Rushton's statement), but in terms of the probability of sharing a given gene by descent from a common ancestor (see Hamilton 1964). The question of correlated genes simply does not arise.

The one unsatisfactory feature of the theory of kin selection is, of course, the underlying assumption that individuals have no information (other than degree of relatedness) on which to base a decision as to whether a potential recipient of their altruism actually possesses the same gene for altruism. The arguments for the evolution of a mechanism for identifying carriers with greater certainty are just as plausible as those for the evolution of "assessor" genes in animal conflicts (see Parker & Rubenstein 1981). The difference lies only in the fact that we are not, in the present case, considering single genes acting in isolation. The challenge for genetic similarity theory, then, is to show how the genetics could actually arise and work within a Mendelian framework.

One way of doing so would be to show that the presence of other inherited traits in any given individual must inevitably be a better (even if still approximate) indicator of the presence of the crucial gene for altruism than mere knowledge of the degree of relatedness. Alternatively, and perhaps more plausibly, the question might be phrased in terms of whether the use of trait similarity provides a better estimate of relatedness (and hence of the probability of sharing the gene for altruism) than any other possible index (e.g., spatial proximity, membership of the same family group).

In this context, it seems to me that, impressive as Rushton's amassed evidence for genetic similarity in human dyads is, it cannot unequivocally demonstrate that genetic similarity theory is at work. Too many alternative explanations can be adduced in each case. Thus, evidence for assortative mating by personality or by physical traits may simply reflect the mechanical demands of meshing in relationships if reproduction is to occur successfully: people of radically different size or incompatible personalities may be unable to mate with sufficient regularity to ensure conception. That identical genes are propagated as a result may be an incidental property arising out of the overriding need to perpetuate one's own genes rather than a result of selection at any higher level.

One final comment concerns Rushton's reference to E. O. Wilson's use of the term *group selection*. It is clear that what Wilson (1975) had in mind here was not group selection (as conventionally defined) but rather a form of kin selection (see Maynard Smith 1982). Group selection *sensu stricto* requires the extinction of whole groups at the same time and can work only if such extinctions occur at relatively high rates. Such conditions are implausible among vertebrates in general and demonstrably do not occur among human populations in particular. Great care should be taken not to confuse these two classes of evolutionary mechanism.

Altruism, nativism, chauvinism, racism, schism, and jizzum

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Genetic similarity detection. Genes, even when equipped with an organism possessing numerous sensory channels of marvelous subtlety, augmented by a pattern recognizer with a virtually limitless memory and virtually instant access time, are nonetheless *not* clairvoyant, as Rushton recognizes. Therefore I simply do not believe they can tell blood types without going through the ordinary procedures of drawing some blood and dropping in typed antigens. If there is indeed assortment by blood type beyond chance, there must be more plausible explanations. Rushton's view is that there are visible (or olfactory, or behavioral, or other conventionally detectable) markers reliably indicating blood type; he should divulge what these markers are, both to increase the credibility of his thesis and to perform a useful service for diagnostics. I make a point of this because it is the only trait Rushton mentions that I can believe is genetically determined.

The ants. Most ants, like most social bees, haven't got a prayer of reproducing anyway; they are sterile. Their social arrangement is necessary to and dependent upon this oddity, and is not all that generalizable.

"It is known." Much as I respect E. O. Wilson's (1975) initial exciting and creative work, I do not think anything controversial about the determination of human behavior by genes can be claimed to be "known" on his authority. A sophomore philosophy student would not get by with this ploy. A lot of what is to be proven is assumed in this casual clause. [See *BBS* multiple book review of Lumsden & Wilson's "Genes, Mind and Culture" *BBS* 5(1) 1982.]

The definition of altruism. Is altruism "defined," as Rushton says, "as behavior carried out to benefit others"? If so, then it becomes unverifiable, at least for the inarticulate and perhaps less introspective species (and probably for articulate species accomplished in self-deception). Is it instead behavior that *does* benefit others, without any discernible benefit to the individual exhibiting it? And shall we, or shall we not, limit altruism to behavior benefiting others in the same species? Would it be reasonable to suppose that the carelessness of the deer, just when the wolf is hungry, is a case of altruism? (Does anyone remember shmoos?) Need behavior be useful immediately, or can it be altruistic if it, say, tends to modify the beneficiary's behavior in the long run in a manner that is likely to enhance his survival and the spreading of his genes? These are not unnecessarily nitpicky questions; on them turns the possibility and the means of delimiting the thesis we are trying to test, and how to tell whether it is likely to be true or false. [See also Logue: "Research on Self Control" *BBS* 11 (4) 1988.]

What do altruistic genes want? There is in my mind also some doubt: Are altruism genes supposed to advertise their presence? Is a creature with an altruism gene more likely to help another creature with an altruism gene than to help any other creature, however similar? If so, then altruism genes are selfish, which may be just the ticket for solving the problem of how they could be perpetuated; but it makes them not particularly altruistic (although they cause altruistic acts on the part of the animal possessing them). On the other hand, Rushton talks about resemblances of a far more general sort, and if kin selection is an issue, then the notion of altruism should be restricted to animals sharing more genes than just the altruism gene; otherwise altruistic bears will be giving their lives for altruistic chipmunks.

What do altruistic bear genes want – to foster selfish bear genes, or to foster altruistic chipmunk genes? (Alter all, a chipmunk could do a bear a favor some time.)

"Not actually a relative." What is "genetically similar but not actually a relative"? The more I reflect on that one the loster I become. Either the creatures share genes or they do not. If they are members of the same kingdom, they must share genes; the question is, then, what's a relative?

Mates' resemblance on socially undesirable characteristics. Since Rushton's examples are all human, it is fair to mention the cruel truth (credited to the penetrating acumen of Guze et al. 1970) that losers generally have to settle for losers, since the mates they prefer can do better for themselves. Moreover, many undesirable behavioral patterns mentioned by Rushton often do not manifest themselves until too late, unless it is through one of these unspecified markers of Rushton's. Although tendency toward alcoholism, like one toward diabetes and cancer and a thousand other ills, may be genetically transmitted, I strongly doubt that any man chooses a mate (1) because he knows she has one of the afflictions, or (2) because he is like herself in having a marked genetic predisposition to one of them – even unconsciously. And even if, straining credulity, this were so, then how would it increase the prosperity of this pair's genes? The child of two diabetics, or of two depressives, or two alcoholics, is not a good candidate for father of his country. Recent news suggests that the child of even one psychotic has precious little future. Are genes selfish to the point of damn foolishness?

What happens anyway when there is a dispute among genes, in which one faction prefers one mate (friend, someone to rescue) and another prefers another? I imagine that the normal genes trapped in an individual with some pathological genes have a real problem; I'd like to know how they solve it. Might makes right? Majority rule?

Genes are well advised to advertise the presence among them of an altruism gene, in case it evokes altruism from others with the same gene; but they are better off without such a dangerous partner, and better off still if they advertise falsely. Perhaps, as Rushton appears to suggest, the altruism gene clings fiercely to a normal gene that marks itself, and thus can get noticed by the altruism gene in another organism in which the gene providentially knows that the normal gene's marker is also a marker for the altruism gene. Then, the animal either of them is in can be rescued by the animal the other is in, and thereby altruism genes pass on to progeny. An altruism gene would almost certainly have to be such a parasite; otherwise the sucker would quickly get tossed off the bus.

The people who would dearly like to know the detectable markers for such tendencies as alcoholism are doctors and the potentially afflicted, so those with the tendencies can be treated; and everybody in the mate market, so those individuals can be avoided. Rushton should reveal this valuable information.

Perceived similarity to victims of experiments. If this perception is based on known genes, Rushton should say so. Kindness to someone because he looked or acted like you, but had no more genes in common with you than anybody else, not only would be a waste of time but, insofar as it put you (and therefore your family jewels) at risk, would be a Bad Thing. There is the effect of the fake green beard at work. In short, perceived similarity is not good enough. It has to be the real thing – gene copies. Appearances are deceiving. Most of the time that doesn't matter; but when your genes, eager to live on, are involved, it probably does.

The heritability of death penalty views and birth control. Birth control, one is tempted to say, is definitely not heritable. But such lame attempts at levity are eclipsed by the exquisite humor of Table 4. Of course, *You* know and I know (and courtesy demands that I assume Rushton knows) that "heritable" does not mean genetically transmitted; but what if the less reputable newspapers got a hold of this? They wouldn't recognize it as a tarted-up version of the commonplace observation that children tend to accept their parents' social and political mind-set, which is often shared by the members of their community anyway. No, if it is a slow week for celebrity disasters and diets, we are going to see Table 4 as scientific evidence that bigotry, a dislike of modern art, and a fondness for military drill are in the blood. It wouldn't be the first time this dirty little word trick was played.

Twin studies. Did somebody really find 3,810 pairs of Australian identical twins reared apart? (I assume reared apart; surely that person would not offer reared-together ones to support a hypothesis about genetic determination of social attitudes.) Seven thousand, six hundred and twenty people must be a substantial fraction of the population of Australia (which is, last I heard, about the same size as that of New York State), and the existence of so many separated twins bespeaks a very great number of unseparated twins (unless Australians are a stranger lot than one had supposed). By the way, are twins that common? (About British twin studies: That well is waiting for the EPA.)

"Shown to be genetically linked." "[I]ntelligence, socioeconomic status, values, and vocational interests have been shown to be genetically linked . . . ." I accepted "heritable" as deceptive but technically correct, but I draw the line at "genetically linked." Nobody can possibly have *shown* that these traits are biologically genetically transmitted. Despite all those Australian twins, there haven't been enough generations, controlled crossings, and rearing of genetically identical individuals in experimental settings (controlled except for the ideological, social, or attitudinal variables in question) to *show* anything of the sort. It's hard enough to show reliably, without cheating, that sweet pea color and fruit fly curly wings are genetically determined traits. Human generations are long, humans' offspring are few, and humans generally object to being told with whom to mate, not to mention having their kids removed for experimental rearing.

Into the wild wild yonder. My admiration for the silliness of Table 4 pales before awe at the inspired irresponsibility of sections 7–10. The "genetic influence on the transmission of . . . vocational interests and value systems" reminded me of nothing so much as Steve Martin in *The Jerk*, wherein a white child is reared in a black family and never told he is white. (He assumes he will get darker when he gets older.) He is distressed that he can't snap his fingers in time to the radio as his brothers and sisters do; then one day he accidentally tunes in to some Muzak. His face lights up and he starts swaying in perfect time to the music.

The film, of course, is meant to be funny.

Conclusion. There is little, if anything, that I have said here that Rushton has not already heard. This is evident from his Discussion (sect. 7). He simply dismisses most of it, however, while adducing the weakest arguments to support – nay, not even to support, merely to be compatible with – his thesis. In the face of so little genuine willingness on his part to engage in a real, coherent argument, I have difficulty organizing a coherent rebuttal. What's to rebut?

In response to many of the difficulties (superfluously) mentioned here regarding the plausibility of individual gene markers, Rushton abandons them, and instead conjectures that the genes in a big happy puppy pile look out for each other in order that all will get reproduced at once; this provides for selection of puppy piles but not of individual genes. Witness: "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: [each for itself? each for all? each for one?]; 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." And, "If like appearance is positively correlated with like genes, any mutation toward preferring like phenotypes would tend to proliferate." Species would tend toward increasingly static uni-

formity, rather than the increasing variety they seem to exhibit now, which supplies the material for natural selection to act upon.

I have no problem with the existence of apparently altruistic behavior, in humans or in any other beast. And I recognize that any behavior has to be compatible with genetics (and physics and chemistry and . . .); but this does not make altruistic behavior the expression of a gene. Nothing Rushton says, that I can believe, makes altruistic behavior, or religious fundamentalism, or even silliness the expression of a gene.

What could Rushton say to make his case? (1) He could define "altruism" or "altruistic behavior" clearly, so its occurrence can be recognized. (2) He could tell us what some of the markers for the traits in question are. The marker for genes coding red hair is red hair. The markers for genes coding for male sex are well-known primary and secondary sex characteristics. These genes obey relatively determined rules of combination and expression, and any dummy can recognize their markers. What is such a marker for blood type? What, aside from altruistic behavior itself, is the marker for the gene coding for altruism? What dummy can recognize it? (3) He could produce some hard evidence that the assortative traits, which are supposed to show that there are noticeable markers for genes like intelligence, alcoholism, and a fondness for straitjackets, are expressions of real genes on real DNA strands obeying real genetic rules. If it is true, evidence supporting it is theoretically possible to find. How one is to obtain it in a lifetime, and legally, is not my problem. If anyone does, I am willing to be convinced.

Familiality, xenophobia, and group selection

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The data compiled by Rushton are indeed impressive, and one hopes they will stimulate the additional research necessary to further test his hypothesis. That attraction toward similarity has a genetic component seems highly probable. It also fits the general predictions of sociobiological theory, which has never ruled out group selection but has considered it a special and rarely occurring case of kin selection. The more closely we look at man, the more evident it becomes that traits that came about by individual selection have become characteristic of the group and therefore have repercussions for fitness at the group level under conditions of intergroup competition.

In Rushton's target article he mentions the issue of how a gene for altruism arose and spread throughout a population. I do not see this as the problem it seems to represent for most sociobiologists. With the evolution of maternal care, the disposition to be friendly or altruistic and the corresponding behaviors came into the world in the form of caretaking behavior along with the motivation to be altruistic in response to young and infantile appeals that triggered caretaking responses. For example, many of the behavioral patterns we observe during courtship are derived from parental behavior. Maternal caretaking behavior was the turning point in the evolution of vertebrate social behavior, which up to that time had been based on dominance and submission (Eibl-Eibesfeldt 1972; 1989).

Familial caretaking behavior could easily be spread to close kin through kin selection. Hand in hand with this may have evolved the ability to distinguish other genetically similar conspecifics and the tendency to form bonds with them, thus first bringing in- and out-group thinking into the world. The extension of altruistic behavior beyond close kin, however, must have been accompanied by the development of the ability to calculate the costs and benefits of relationships of mutual reciprocity and to regulate altruism toward distant kin accordingly.

Xenophobia, man's fear of his conspecifics, was certainly a trait that facilitated the inclination to distinguish between in-group and out-group members, and there is little doubt that xenophobia is a partly innate trait in human beings. It manifests itself in all cultures we have so far studied (Eibl-Eibesfeldt 1979; 1989), although this fear is tempered by personal acquaintance. Xenophobia, like altruism, appears to have its roots in the mother- (parent-) child bond, which is defended by both the parents and the child against any intrusion from outside the family. Feinman's (1980) experiments on xenophobia in infants indicate that fear of strangers is greater for individuals who are dissimilar to the parents than for similar individuals.

Group demarcation on the basis of similarity is often greatly enhanced through cultural means. Erikson (1966) pointed to man's inclination toward in-group demarcation, which eventually leads to what he aptly calls "cultural pseudospeciation," the starting point for biological subspeciation. Man's indoctrinability, also a partly biologically based trait, is the vehicle by which further cultural demarcations are transmitted (Eibl-Eibesfeldt 1982). Ethnic ideological similarity can override genetic similarity as has been well documented by many studies of intragroup warfare. However, it would be interesting to see whether reconciliation is more easily achieved in such cases of biological similarity.

Group selection, which may only have been a strong force in the past 20,000 years or so of human evolution, has brought with it advantages and disadvantages. Its main advantage is, of course, increasing human diversity and the potential to adapt to a wide range of conditions, as well as the innovations resulting from competition. Its disadvantages are ethnocentricity, ethnocide, and the arsenal of weapons developed in the service of intergroup competition. To find a way to preserve the positive benefits of group selection while ridding ourselves of its negative aspects is an imposing task. To accomplish this more knowledge about and open intellectual discussion of even the most "sensitive" issues such as ethnocentricity and racism are essential. In this respect, Rushton's paper is certainly an important contribution.

Testing one of Rushton's predictions

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The wide scope of Rushton's target article precludes a detailed analysis. He points out that "two sets of falsifiable propositions follow from this interpretation. First, individual differences in ideological preference are partly heritable. Second, ideological belief increases genetic fitness" (sect. 9, para. 9). It is difficult to test the second of these predictions; the first, however, has received strong support from a series of recently reported studies (Eaves et al. 1989) of two separate populations of twins, a London sample of 825 pairs and an Australian sample of 3,810 pairs. The two different measures used were the Eysenck Public Opinion Inventory (Eysenck 1954) and the Wilson-Paterson Conservatism Scale.

Table 1 lists the ten items of the Eysenck scale with the highest components of additive genetic variation or V_A. All the heritabilities are above .50, thus clearly supporting Rushton's first prediction. Slightly lower heritabilities were found for the two major social attitude factors, conservatism/radicalism and tough-mindedness/tender-mindedness. For the Wilson-Paterson Scale the five major factors and their heritabilities (in parentheses), were as follows: authoritarianism (.51), religion (.41), socialism (.49), prejudice (.59), permissiveness (.63).

High values were also found for assortative mating. Table 2 gives the correlations for five components of assortative mating

Table 1. *Items in Eysenck Public Opinion Inventory with highest components of additive genetic variation*

Item no.		V_A
29	Sex crimes, such as rape and attacks on children, deserve more than mere imprisonment; such criminals ought to be flogged or worse.	.63
12	Men and women have the right to find out whether they are sexually suited before marriage.	.59
14	The average man can live a good enough life without religion.	.56
18	The death penalty is barbaric and should continue to be abolished.	.56
10	Crimes of violence should be punished by flogging.	.55
21	Birth control, except when recommended by a doctor, should be made illegal.	.54
33	The Church should attempt to increase its influence on the life of the nation.	.54
39	Only by going back to religion can civilization hope to survive.	.54
25	We should believe without question all we are taught by the Church.	.53
47	Our treatment of criminals is too harsh; we should try to cure them, not punish them.	.51

between husbands and wives; the average coefficient of assortative mating is about .50. Similar values were found for conservatism/radicalism and tough-mindedness/tender-mindedness.

The correlation between spouses' social attitudes is highest for identical traits, and much lower for cross-traits, suggesting that assortative mating operates on a trait-by-trait basis. The degree of assortative mating for attitudes is so high that its genetic consequences could account for all the additional resemblance between twins that our earlier analyses (Eaves & Eysenck 1974) had ascribed to the "family environment." When we allow for the joint effects of genes, cultural inheritance, and assortative mating in our model for family resemblance in conservatism, for example, estimates of the cultural parameter do not differ significantly from zero. This result does not agree with our initial intuition that cultural attitudes derived from parents are major determinants of family resemblance in attitude.

The analyses of the data in Eaves et al. (1989) are of course much more detailed than the preceding discussion. They fully support Rushton's first prediction, to a degree that we would not have expected. Common sense certainly would not suggest a high degree of heritability of social attitudes, and most texts in social psychology assume without argument a null contribution of nonenvironmental factors to individual differences in social attitudes. It is the discovery of such unexpected and counterintuitive results that makes theories derived from sociobiology so interesting and important.

Table 2. *Correlation coefficients of spouses' assortative mating on five factors of social attitides*

	RL	A	S	PJ	PR
RL	.52	−.03	−.05	.03	−.33
A	−.11	.56	−.11	.29	.19
S	.07	−.11	.54	.02	.09
PJ	−.04	.30	.10	.35	.16
PR	.30	.20	.18	.16	.52

Key: RL = Religion; A = Authoritarianism; S = Socialism; PJ = Prejudice; PR = Permissiveness.

Biocultural versus biological systems: Implications for genetic similarity theory

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There is much to ponder in Rushton's target article. My comments will be limited to several small but (I think) important points. With regard to testing genetic similarity theory, it seems to me that what is required is a prediction (or better still, a set of predictions) that would allow investigators to distinguish among theories based on genetic similarity and those based on competing explanations. For example, the fact that humans tend to mate assortatively for various characteristics is certainly consistent with genetic similarity theory (at least on one level), but it is equally consistent with other explanations. So how can we distinguish among them? What we need is a theory that generates specific predictions about the behavior of individuals in particular circumstances, predictions that are different from those based on competing theories. Testing these predictions would give us a clearer idea of whether we're on the right track.

A further comment on assortative mating. Genetic similarity theory predicts that individuals *choose* spouses on the basis of perceived genetic similarity. This is *not* equivalent to the prediction that there will be positive correlations between spouses with respect to various characteristics. Several investigators (including myself) have pointed out that one generally cannot infer individual behavior from population statistics. Resemblance between spouses in terms of race, social attitudes, and religion may simply reflect the fact that in the environments where mate selection occurs, there are more individuals who are similar than dissimilar, so that within the pool of available mates, individuals do not exercise choice. In this scenario, assortative mating at the population level results not from *preference* (i.e., choice) but rather from the *prevalence* of various mates when and where pairing occurs. To document the former, we must set up choice experiments in which subjects are presented with a set of potential mates (presumably members of the opposite sex) and asked to evaluate each in terms of perceived suitability as a spouse.

Finally, a note about gene culture theory and in particular the influence of natural selection in biocultural systems. In a nutshell, gene culture theory asserts that the evolution of behavior depends on both biological and cultural factors whose influences may not be strictly additive. This lack of independence can arise from several sources. The biological fitness of a genotype may depend on various aspects of the cultural environment. Alternatively, cognitive integration of various aspects of the cultural environment may depend on the genotype. Irrespective of the exact nature of the interaction, however, the end result is that we cannot in general predict the evolutionary fate of a trait on the basis of its effect on biological fitness or cultural fitness *alone*. This implies that we should be rather wary of applying the results of standard evolutionary theory directly to the biocultural case. (This is not to say of course, that standard evolutionary theory cannot generate good predictions for biocultural systems, only that it need not.) For example, it can be shown that in evolutionary games where strategies are subject to both biological and cultural transmission, strategies satisfying

the standard evolutionarily stable strategy conditions (for purely biological games) are generally *not* evolutionarily stable (Findlay et al. 1989; in press). Hence predictions based on standard evolutionarily stable strategy theory are not likely to be very accurate when applied to biocultural systems except under restricted conditions.

This is where human sociobiologists often run into trouble, and Rushton is no exception. He writes that "from the standpoint of optimization one might ask whether evolutionarily stable ethical systems would survive very long if they consistently led to reductions in the inclusive fitness of those believing in them." The implication is (I suspect) that they *wouldn't* survive very long, because the tendency of natural selection to move in the direction of increasing fitness would ultimately cause such systems to become extinct. This argument may be true of biological systems, but it is not generally true of biocultural systems, because such systems need not evolve in the direction of increasing biological fitness. In biocultural evolutionary games, stable strategies are those with a high degree of biocultural fitness, not necessarily a high degree of biological fitness. So it is very possible that ethical systems that confer reduced fitness will persist indefinitely.

Uncompelling theory, uncompelling data

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The scientific community may be compelled to take notice of a theory when the theory (1) proves to be deductively contained within a well-corroborated theory and thus represents new derivations of accepted knowledge (Hamilton's conceptual treatment of inclusive fitness meets this criterion; Kitcher 1985); or (2) yields well-confirmed predictions that are unlikely to obtain under plausible alternate conceptions of the world. Does genetic similarity theory meet these criteria? With respect to the first criterion, it comes up short. As others (e.g., Mealey 1985) have argued, there is a link missing between genetic similarity theory and the accepted corpus of evolutionary biology. Rushton's statement, "If like appearance is positively correlated with like genes, any mutation toward preferring like phenotypes would tend to proliferate," is, quite simply, not an unqualified truth; it is probably true only with respect to genes that are correlated, or linked, with the traits in which the other individual is similar. The link missing between genetic similarity theory and evolutionary biology is a specification of the process whereby linkage can occur. Rushton recognizes this shortcoming and addresses it by claiming that linkage would naturally be expected to underlie any complex function, of which detection of genetic similarity is an instance – a response that merely begs the question. Genes do not become linked simply because they interact to produce a complex trait. Various phenomena (e.g., assortative mating, epistatic effects on fitness) lead to linkage. We have yet to hear what phenomena could lead to the sort of linkage that would render genetic similarity theory possible.

How does genetic similarity theory rate with respect to the second criterion? Rushton discusses five tests of theory. With regard to several tests, genetic similarity theory does not uniquely account for the data. Friends may be similar on relatively heritable traits merely because genotypes systematically seek environments to which they are adapted (Scarr & McCartney 1983), and individuals in the same environment tend to become friends. That blood markers of friends are more similar than those of randomly paired persons may, in light of the linkage disequilibrium that probably exists between blood markers and other genes (Carter-Saltzman & Scarr-Salapatek 1975), tell us only that some positive assortment in friendship exists – nothing new. Finally, the correlation between parental grief following the death of a child and perceived similarity of the child to the grieving parent has a number of obvious alternate explanations (e.g., selection for kinship recognition on the basis of phenotypic similarity; Wells 1987).

Two tests remain – tests that Rushton has emphasized in his previous writings as being particularly accurate. Both concern assortative mating. He claims that (1) assortative mating is most pronounced for traits with relatively high heritability; (2) sexually interacting couples who produce a child share blood antigens to a greater extent than sexually interacting couples who do not produce a child or randomly paired couples. From a theoretical standpoint, these findings are potentially quite interesting.

One question to ask about test one is whether the claimed effect truly exists, a question that most of the analyses Rushton presents can't answer. In eight of the thirteen studies presented in Table 3, the estimator of genetic variance involved a parent-offspring (P-O) correlation, an estimator that assumes random mating. The presence of positive assortative mating biases estimates positively and thus introduces a very problematic confound: The *error* component of the genetic estimates correlates with the *true* component of assortative mating estimates. In eight studies, then, the reported effects may reflect no more than this confound. Of the five remaining studies, two yielded significant effects – hardly strong support for genetic similarity theory.

Let us suppose that covariation between assortative mating and genetic variance *does* exist, as predicted. Are we really without alternative explanations? Not at all. An obvious explanation is a rather elementary truth of population genetics: Assortative mating increases additive genetic variance. By assuming a simple additive genetic model and many contributing loci (as Rushton does), one can partial out the effect of assortative mating on heritability (Crow & Felsenstein 1968). In one study Rushton cites (Susanne 1977), partialled estimates were even provided. Their correlation with assortative mating across 36 anthropometric variables was 0.19, ns, attenuated from the figure of 0.36 that Rushton cites.

A theoretically more interesting scenario could produce the hypothesized finding as well. Suppose that within a monogamously mating population there is widespread genetic variation in nose length, which has little to do with fitness. A mutant allele that predisposes mating with long-nosed others enters the population, and cross-trait assortative mating occurs between those with long noses and those with this gene. Over generations, genetic covariance develops between the gene and genes for long noses; those who carry the gene predisposing mating with long-nosed individuals tend to have long noses themselves. De facto, assortative mating for nose length occurs. This scenario could occur with respect to any trait with substantial additive genetic variance and no correlation with fitness. It cannot occur, however, with respect to traits with no additive genetic variance. Assortative mating could thereby accrue specifically for traits of high heritability – the same outcome that Rushton predicts. This scenario produces the outcome, however, in the absence of selection for altruism toward similar others. I emphasize that this scenario does not justify some of the more far-reaching and undoubtedly controversial derivations of genetic similarity theory, such as selection for ethnocentrism.

With regard to the second test (concerning the difference between the blood type similarity of sexually interacting couples who produce a child and those who do not), we must again ask whether the hypothesized effect is real. Ascertainment of paternity relied upon a fallible procedure of blood group matching that, as it turns out, is less likely to correctly identify the nonpaternity of men who are genetically similar to the mother than the nonpaternity of men who are not genetically similar to

the mother. Thus ascertainment bias alone could have produced Rushton's finding.

Nonetheless, it would not be surprising if at least some of the reported difference reflected a true effect. Indeed, an association between Rh and ABO maternal-fetal incompatibility and spontaneous abortion has been independently documented (see, for instance, Vogel & Motulsky 1979). But how strongly does this evidence weigh in favor of genetic similarity theory? In light of additional considerations, perhaps not much. Substantial evidence indicates the *opposite* relation for HLA antigens: An association between HLA *sharing* between mother and fetus and spontaneous abortion clearly exists; thus couples who are similar with regard to HLA loci have relative difficulty producing a child (for a review, see Thomas et al. 1985). So some findings are consistent with genetic similarity theory, others are not. From a Popperian perspective, those that are inconsistent should weigh more heavily. It may be argued, with some merit, that the HLA system is not representative of the full genome and thus findings concerning it may represent anomalies. However, the same argument could be made about the nonrepresentativeness of blood antigens. After all, given that they were originally identified precisely because immune attacks against them are strong and reliable, they would hardly be expected to be representative in this regard.

A theory that is not deductively derivable and does not explain a known fact for which there is no plausible alternative explanation would seem to be a theory without much going for it. If genetic similarity theory is to avoid the fate of being just such a theory, a more compelling conceptual analysis or more compelling data must be forthcoming.

Genetics versus economics as the basis for friendships and other preferences

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Sociobiology presents itself as a legitimate branch of evolutionary biology. There is no reason why this ideal cannot be realized, but all too often sociobiologists have failed to apply the Darwinian canons of evidence. In particular, we need clearly formulated hypotheses that are not teleological, that generate predictions that cannot be otherwise explained, and that are preferable to reasonable alternatives.

Rushton's most sweeping claim, that "people detect genetic similarity in others and give preferential treatment to those who are most similar to themselves," is undesirably teleological because it presupposes that a certain kind of causal nexus underlies the phenomena. Because there is a correlation between preferences and genetic similarity, it is inferred that the things preferred are preferred because they are genetically similar (not just similar *simpliciter*), and this is taken to justify selection at the level of the gene rather than the level of the organism. The conclusions do not follow and other interpretations might be drawn.

Rushton's definition of altruism is misleading, for it includes acts like providing shelter for one's castrated slave. The real issue is whether people prefer to make social and reproductive alliances with organisms like themselves because, although making such alliances reduces their organismal fitness, doing so is nonetheless favored by selection at some other level. The question we should address is whether we can find a perhaps more parsimonious explanation, in terms of an organism having greater reproductive success than its conspecifics.

Why should we prefer companions who are more or less like ourselves? An economic possibility is that such persons may be better resources than those who are different. We rely upon those around us for various kinds of assistance, and providing for our own and others' needs is more efficiently done if we know what those needs are. People who speak different languages obviously encounter difficulties, as suggested by the Tower of Babel myth. Although they can be overcome by learning, the cost in terms of time and effort is high. In spite of the fact that relatives tend to speak the same tongue, there are no genes for any particular language. But features of temperament evidently do tend to follow the Mendelian rules to a certain extent. We can learn to understand persons of different temperament, but empathizing with those of similar temperament is nonetheless easier. Hence it would be perfectly reasonable for friendships to form between persons who are aggressive or intellectual, irrespective of the genetic basis. Of course, there are reasons for preferring those who are different, such as to produce a unit that benefits from the combination of different talents. Schools of fishes and flocks of birds tend to be monospecific – but there are exceptions.

There is good reason to believe that the similarities to ourselves that we value in others will tend to occur together in the same organism. This is partly because of common descent and common environment, and partly because of pleiotropy and its nongenetic analogues. Traits, whether genotypic or phenotypic, are not atomic. Hence any similarity of others to ourselves, whether or not it is valuable in and of itself, may be used as an indicator, or token, of other similarities. Therefore, the mere fact that some such preferences have a genetic basis does not provide compelling evidence as to whether we are selecting genotypes or phenotypes, what they are, or the adaptive significance of any given trait.

Organisms do indeed have preferences with respect to mate choice and resource allocation. There are obvious advantages to mating only with conspecifics. Recognizing one's offspring and favoring them can obviously contribute to reproductive success. Although these are both straightforward instances of selection at the level of the individual organism, familial selection for some other preferences is a reasonable extrapolation.

Explaining such matters in terms of inclusive fitness seems considerably less plausible when we consider our preferences for organisms outside our own gene pool. To some extent the animals we choose as pets tend to be our close relatives. Nonetheless, dogs are more popular than monkeys as pets.

We can easily imagine how, as Rushton suggests, a preference for similars might be conducive to group selection, albeit only to the extent that such behavior might lead to the formation of groups that are in fact supraorganismal individuals (such as families) and therefore can be selected (see Ghiselin 1981). But it is another thing to claim that genes are selected as classes of similars. Dawkins's (1976) use of the term "gene" turns out to be highly equivocal; some of his "selfish genes" turn out to be selfish chromosomal deletions (Ghiselin 1987). It therefore seems to me that all such arguments need to be spelled out with respect to the precise mechanisms involved.

Genetic similarity between friends and lovers: Is an evolutionary view warranted?

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> One would be in less danger
> From the wiles of the stranger
> If one's own kin and kith
> Were more fun to be with.
>
> Ogden Nash, *Family Court*

Perhaps we are in less danger from strangers than we might think. In the target article, Rushton suggests that human beings

have evolved an ability to detect genetic similarity in others in order to give preferential treatment to those most similar to themselves. He reasons that "if organisms could identify genetically similar organisms, they could exhibit altruism toward these 'strangers' as well as toward 'kin'." Thus, kin recognition might simply be one form of genetic similarity detection. Genetic similarity theory is proposed as an extension of kin selection theory (Hamilton 1964). However, its calculus is not nearly so well worked out as that of its predecessor.

The heart of kin selection theory is Hamilton's rule, which states that animals are selected to be altruistic when $rb - c > 0$, where b and c are the benefit and cost to the receiver and performer, respectively, of altruistic acts and r is the coefficient of relatedness. The many recent studies of kin recognition in animals (reviewed in Fletcher & Michener 1987) are important to the topic of kin selection because they have provided evidence of abilities not only to discriminate kin from nonkin, but in many instances, to discriminate among relatives differing with respect to r. In sections 4 and 5 of his article, Rushton reviews some of these studies, describing them as "test of 'kin selection' theory." Most of the studies cited, however, do not qualify as attempts to test kin selection theory because no measures of the costs and benefits of acts were made, nor were there assessments of whether the actions were consistent with Hamilton's rule. Genetic similarity theory apparently has nothing equivalent to Hamilton's rule to serve as a guiding principle predicting when altruism should occur between unrelated individuals.

Rushton argues that should mutations arise that have the fortuitous pleiotropic effects of producing an identifiable phenotype (label) and directing preferential treatment to others with that same label, altruism toward nonkin could evolve. This line of reasoning follows ideas expressed by Hamilton (1964, p. 25) and Dawkins (1976, p. 96, the "green beard effect"). Rushton expands the scenario by imagining gene complexes producing both feature detectors and altruistic behavior that lead to the discrimination of individuals who share "appropriate" phenotypic traits. We are never told what phenotypic traits might, either on theoretical or empirical grounds, be deemed appropriate. Are these labels the products of "outlaw" genes (Alexander & Borgia 1978; Dawkins 1982) promoting their own survival at the expense of the genome? In such case the adaptiveness of those phenotypes for the organism is irrelevant and *any* label produced by genes fortuitously linked to the complex would be attractive. This nonadaptationist interpretation is apparently not the one preferred by Rushton, however, since in the case of spouse selection he posits, and attempts to provide evidence for, advantages conferred upon genetically similar mates. Thus the adaptive significance of the phenotypes involved in the discrimination of genetically similar individuals, and of the phenomenon itself, is important. Rushton suggests a number of advantages to having a genetically similar mate, including marital stability. How some of the traits reported to be similar, such as alcoholism and aggressiveness, contribute to the stability of relationships is unclear. Another suggested benefit is increased relatedness to offspring. I am not sure why increased relatedness per se should be an advantage, unless selecting a mate with a similar genome ensures that adaptation to a local environment and coadaptation of the genes will not be disrupted in the offspring. No direct evidence is offered in support of this conclusion, however. An additional putative advantage, fecundity, would seem to be a difficult one to evaluate in the age of birth control.

In support of genetic similarity theory, Rushton reviews his own research and that of others showing greater than expected correlations (few of which are impressive in terms of variance accounted for) between various human phenotypes and presumably, but not necessarily in all cases, genotypes of unrelated social partners (spouses, friends). He acknowledges, but dismisses, an alternative explanation that common environmental influences, rather than genetically based *active* discrimination, are responsible for the similarities observed. Rushton argues that the environmental view cannot easily account for the observation that assortative mating is common in nonhuman species. To have evolved independently so often, he contends, assortative mating must confer substantial advantage. This may or may not be true, but its validity should be determined on the basis of empirical evidence that is in most examples lacking. Furthermore, the literature is not always clear on the advantages of assortative mating in animals, or on the mechanisms involved in its occurrence. For example, Cooke and Davies (1983) have shown assortative mating for color in snow geese (*Anser caerulescens*) but could not demonstrate a selective advantage (in terms of surviving young) to making the "correct" choice in terms of plumage color. Oyster catchers (*Haematopus ostralegus*) mate assortatively on the basis of two very different learned behavioral feeding specializations (Norton-Griffiths 1969). The young birds take up to two years to become proficient feeders and it appears important that they not have conflicting parental models. Young reared by foster parents learn the feeding strategy of their foster parents. Thus in some species there are clear advantages to assortative mating, but the relevant phenotype is an acquired one. It is important to note that in the case of oyster catchers, and no doubt in many species known to mate assortatively, it is not known whether mate selection is active, or whether assortative mating is simply the consequence of a greater likelihood of encountering individuals of similar phenotype. Rushton provides no evidence for active selection of a partner in any of the studies cited in support of genetic similarity theory. Data incompatible with a simple environmental explanation of some similarities among friends (blood groups) are apparently forthcoming (Rushton 1989b). The reliance on ex post facto designs renders the evidence for genetic similarity theory equivocal. It would be interesting to examine the question experimentally by testing whether subjects required to select preferred potential partners from a sample are genetically more similar to those chosen than to others available.

Mate selection: The wrong control group

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In order for Rushton's genetic similarity theory to work, he needs to demonstrate that (1) the average degree of relatedness between mates (or friends, or whoever is favoured) is higher than it is in the population from which the choice of mates is made, and (2) this choice is made on the grounds of the degree of genetic similarity, not relatedness. It is important that both of these conditions be fulfilled.

Although we are unaware of any economic pressures in Western society to marry kin, and there certainly are specific legal and religious prohibitions against marrying very close kin, the degree of relatedness between mates in most Western societies is typically less than or about equal to 0.002 (May 1979). If we have done our sums correctly, this is slightly more than that of fourth cousins. The question is: What is this degree of relatedness in mates due to? Assuming that it is not due to kin selection (which Rushton must also assume), it can be due, as Rushton argues, to the attraction of similar genotypes, or alternatively, to the way human society is fractionated. The problem in telling these apart is one of finding the correct control group.

Rushton made a comparison between mates in the study of blood groups (1988a). The samples were taken from cases of disputed paternity and compared with randomly generated

pairs from the population (of blood samples) on seven polymorphic marker systems. He found that the degree of genetic similarity between sexually interacting pairs (52.02% ± 0.4) was higher than the degree of genetic similarity in the random sample (43.10% ± 1.0).

We do not know what they get up to in North America, but in Scotland we do not breed at random. Rushton's sample was limited to people of North European appearance, judging by photographs. But there is a great deal of evidence that mate selection in humans is limited by a large number of factors besides race and ethnicity; it is also limited by social class, educational background, religion, and political affiliation, for example. The most important determinant of whom we marry, however, appears to be proximity; 76% of the individuals studied by Kennedy (1943) contracted marriages within a distance of 20 blocks from their home. Since the social group is so important in determining choice of sexual partner, the correct control group is one that is drawn from this group. Given the small difference between the genetic similarity of the sexually interacting couples and that of the random sample couples, even a slight change in the genetic variance could remove the difference altogether.

How could a study be done in which the control group was better than merely a random sample of the whole population, including controls for other factors known to be important? As a first approximation, we suggest that Rushton's work be repeated in a community where a high proportion of peoples' friends and potential spouses is found within a circumscribed geographic area. A random selection of people from this circumscribed community would be the control group.

Unfortunately, finding such communities has become more difficult, although as recently as 1974, Udry found proximity still overwhelmingly important in the choice of marriage partner in the United States. Choice of friends and spouses would be even more constrained in a country lacking urbanisation and still based on village communities; such a community would be a better site than Canada for a test of Rushton's theory. Of course, even in such a village community, social constraints might reduce the number of possible relationships from that assumed by randomness, and it would be more feasible to control for these constraints in a circumscribed community. We at least would accept that as a fair test of Rushton's theory.

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Green beard theory

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The argument of Rushton's target article is vague and confused on two rather important issues: the logic of competition and cooperation, and the relationship between genetic and social factors in human behaviour. The confusion begins with his idea of altruism. Rushton defines it as "behavior carried out to benefit others;" he says that in humans it "ranges from everyday kindnesses, through sharing scarce resources, to giving up one's life to save others." But the "paradox of altruism" would not be paradoxical at all for evolutionary biology unless altruism inherently involved "behaviour that increased another entity's welfare *at the expense* of one's own" (my italics; see Dawkins 1976, p. 4; Hamilton 1963, p. 354). I have pointed out (Hallpike 1984) that kindness is frequently not altruistic in this sense because it need not involve any significant decrease in the welfare of the benefactor (e.g., lending one's lawnmower to a neighbour, or telling someone the way to an address). Even when some diminution of individual welfare does occur, as in the sharing of scarce resources, there are institutions of reciprocity to ensure that short-term sacrifices by one individual are spread among the whole group in the long term, as in meat sharing by hunters. In human reciprocity, therefore, there is often no clear distinction between altruism and selfishness because altruism often proves to be enlightened self-interest. [See also Caporael et al: "Selfishness Examined" *BBS* 12 (4) 1989.]

Rushton's examples of marriage and friendship illustrate my point. In marriage both partners help one another and may therefore suffer short-term losses in individual welfare, but the long-term pattern of self-sacrifice normally increases the personal fitness of both parties, as well as their inclusive fitness through reproduction. In the case of friendships, Rushton indeed provides no evidence at all that these have any effect on the personal fitness of those involved, or even that significant self-sacrifice is involved. The most that his data on marriage and friendship choices could establish is that, other things being equal, people prefer to consort with those whom they perceive as similar to themselves in a variety of ways (not a very surprising statement!). This, however, has nothing to do with the relative success of different genotypes as the result of *competition* in the struggle for life.

Employers cannot afford the luxury of choosing employees because of their genetic similarity rather than their personal qualifications, yet one's job clearly has much more effect than one's friendships on personal fitness. Again, it may be that there is greater overall genetic similarity within such groups as policemen, lawyers, and farmers than between such groups, or compared with a random sample of the population. But the fitness of the members of these groups is not affected by their *genetic similarity* to other policemen or lawyers or farmers but by such factors as income and conditions of work.

With regard to the relationship between genetic and social factors, Rushton advances the causal argument that "people detect genetic similarity in others *in order to* [my italics] give preferential treatment to those who are most similar to themselves" (Abstract). The clear implication is that social cooperation is the result of genetically based resemblances between individuals, which in turn are the result of a strategy by genes to replicate themselves. An obvious alternative explanation is that the requirements of social cooperation produce the genetic similarities. How can genetic similarity be detected? Rushton maintains that humans at least have the innate ability to distinguish kin by purely perceptual means, but his evidence does not bear examination.

For example, the recognition of infants by mothers (and vice versa) is not *kin* recognition at all but merely discrimination between individuals already known to one another. To demonstrate kin recognition, it would be necessary to remove infants from their mothers at birth and substitute the infants of other mothers and then to see whether either infants or mothers could detect that something was wrong. Anthropological data do *not* show that the avunculate is produced by low paternity confidence, a theory I have already refuted in some detail (Hallpike 1984, pp. 137–42). Although it is true that stepchildren are more likely to be battered than children living with their natural parents, we should expect either on the basis of kin selection theory or of genetic similarity theory that adopted children, who are related to neither parent, are in the highest risk category for battering, but this is not the case (e.g., Costin & Rapp 1983). The suggestion that because kin received 55% and nonkin received only 7% of the estates in 1,000 probated wills this is therefore evidence either for kin selection theory or genetic similarity theory could only convince someone already determined to believe in them.

Genetic similarity theory would be an interesting challenge to sociological explanations of cooperative behaviour if *specific* traits had a high predictive value: if, for example, members of a particular political party had a distinctive armpit odour or green

beards. Rushton, however, gives no evidence for the influence of such specific traits, emphasising overall genetic similarity instead. But if individuals were attracted by overall genetic similarity per se, then it would surely follow that in choosing spouses or friends the genes for anthropometric traits would be at least equal in importance to the genes for inheritable personality traits and social attitudes, since *all* genes are supposed to be "trying" to maximize their frequency. But the data on friendship choices show that conservatism, for example, is far more significant than either the anthropometric or the personality traits, while the marriage data show that opinions, attitudes, and values are far more important than the anthropometric traits, with personality traits coming in between. Rushton also concedes that in the choice of spouses the social factors of ethnicity, class, religion, and level of education are the most important, and that in friendship choices the social factors of age, education, and occupational status are the most important. These results do not seem to give much support to genetic similarity theory, but are more or less what would have been predicted by common sense.

Testing genetic similarity: Out of control

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The problem with good scholars is that they can muster evidence for any plausible argument. For example, if the prevailing assumption was that people choose their friends according to genetic similarity, and a minority of dissidents suspected that factors as farfetched as cultural background and sheer proximity play a significant role, good scholars could gather much evidence that would be "compatible with" the heretical view. But the distinction between evidence that is compatible with a hypothesis and evidence that tests a hypothesis is the distinction that separates scholarship from science. Unfortunately, except for the data on blood relatedness between friends (section 6.3.1, Table 5), the evidence gathered by Rushton does not actually test his hypothesis. That is, if instead of being in the observed direction, the reported evidence had no direction or had an opposite direction, in addition to having a low probability of being brought to the reader's attention, it would not falsify Rushton's hypothesis.

The evidence that could falsify the hypothesis strikes me as coming close to doing just that, or, at best, failing to leave the hypothesis unscathed. The first problem is the weakness of the result (as distinct from the weakness of the statistical inference attending the result, which is considerable in this case because a t-test is used on data that are clearly not ratio or interval or even bona fide percentage measures). As Rushton notes, there are inbreeding constraints on how closely related sexually reproducing friends ought to be. No such constraints exist for nonreproducing friends, yet such friends are less related to each other than are mates, according to Rushton's measure.

The second problem is Rushton's measure. The appropriate control (nonfriend) genetic relatedness should be randomly selected from some set of reasonably likely potential friends. Unless we are going to consider pen pals, a minimal requirement would be people from a common neighborhood. (The question is not whether pairs randomly chosen from within a homogeneous neighborhood are more related than pairs chosen from across such neighborhoods, but whether pairs randomly chosen from within a neighborhood are less genetically similar than pairs of friends chosen from within a neighborhood.) In short, the realization that people are slightly more related to long-term friends than they are to persons whose only common attribute is having responded to the same advertisement does not suggest a causal link between friendship and relatedness. Indeed, I am surprised that a factor like culturally induced ethnic prejudice did not cause a greater than observed difference in similarity between friends and people who answered the same ad. Perhaps this is due to a relative ethnic homogeneity in the study's focal area. If this investigation had been conducted in New York City, where great genetic distances are traversed over short linear distances, the disparity between control relatedness and friend relatedness would probably have quadrupled, but that would not make the evidence stronger for Rushton's hypothesis because the control would still be inappropriate.

The importance of this objection can be tested by recalculating the control similarity for Rushton's friendship data. The average of each individual's average similarity to *all* nonfriends should be compared with the average friend-pair similarity by means of an appropriate concordant/discordant nonparametric analysis. The friend/nonfriend disparity in similarity using this procedure should then be compared with the disparity using Rushton's already chosen set of 76 nonfriend pairs. If utilizing all of the information decreases the disparity (compared with utilizing only one random sample for control), then the only evidence presented that could potentially falsify the hypothesis should be disregarded as the artifact of an artificially exaggerated measure of genetic dissimilarity among controls. I hope that Rushton will prepare such an analysis for his reply.

The fact that correcting the correlation between friends for heritable traits by various stratification factors does not diminish those correlations is not relevant to the preceding argument because Rushton's entire "genetic similarity detection" section pertains only to his proposed mechanism of effect and is not evidence in support of the central hypothesis. That is, although it is true that "if people choose each other on the basis of shared genes, it should be possible to demonstrate that interpersonal relationships are influenced more by genetic similarity than by similarity attributable to a similar environment," it does not follow that people who are similar in heritable traits are genetically similar *unless the trait's variances are determined by a small number of alleles at a few loci.* For traits like height and intelligence, even if h^2 were 1, there is little or no evidence that similar heights or IQs are determined by the same alleles at the same loci (see Hartung 1984; 1985a; 1985b). This can be demonstrated by a comparison of all winning or losing poker hands from an evening's play, which reveals that there is very little card-for-card identity between winning hands or losing hands, and that the winning hands from some rounds of play would have lost other rounds of play (just as a genome that does well in one environment may not do well in another). Suffice it to say that although being A_1B in the ABO system may make a person 85% identical *at these loci* to someone who is A_2B *at those loci,* having an IQ of 100 does not imply any percentage of genetic identity with other people who have IQs of 100.

Both Rushton's hypothesis and the noncrucial evidence (the scholarly evidence) in its favor deserve due regard. The biological foundations of friendship are as policy-neutral as are the biological foundations of racism, and I have never been keen on the distinction between genes that are alike as a result of convergent evolution and genes that are identical by descent. If Rushton's hypothesis is correct, that truth is well worth knowing, but the evidence presented here only suggests an intriguing hypothesis that might be valid and should be properly tested.

It will not do simply to note that "it is unlikely that this outcome [referring to Rushton's Table 5] is due entirely to stratification effects because within-pair differences in age, education, and occupation did not correlate with the blood similarity scores," because, regardless of the mean r obtained, this invites a type II error (false negative) into an analysis that has negligible statistical power and does not match controls for other known (and therefore for some unknown) confounding variables. A proper test would compare the genetic similarity of

friend pairs with the genetic similarity of nonfriend pairs who are matched for relevant variables. I would not be surprised to find that nonfriend pairs who match in great and appropriate detail could be culled by comparing individuals with their most disliked personal enemy. I would also not be surprised to find that best enemies are as genetically similar as best friends.

Recognising kin = Recognising genetic similarity

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For Rushton's theory of genetic similarity to function, individuals must be able to recognise genetic similarity in their conspecifics. Such a process requires the existence of both a perceivable cue and the ability to classify this cue as belonging to genetically similar individuals.

There is no doubt that many animal species (see Hepper 1986), including humans (Hepper, 1988), possess cues that are determined by their genes. It should be noted, however, that cues reflecting genetic similarity can also be produced by environmental factors, such as common diet, communal pool of microorganisms, and labelling (Hepper 1986). Irrespective of whether the cues are genetic or environmental in origin, much information exists indicating that individuals possess perceivable cues that reflect their genetic makeup.

The major problem faced by individuals is how to classify a particular cue correctly as belonging to a genetically similar individual. The "strong" version of Rushton's theory is that individuals are genetically preprogrammed to do so. Although it is theoretically possible for such a system to exist (Hepper 1985), the demonstration of its existence is virtually impossible; to date there is little evidence to suggest that such a system exists (but see Hepper 1986).

Exclusion of a classificational mechanism programmed by genes requires that individuals learn who is genetically similar. They will be completely nonselective about this process; that is, they will not say, "Someone is genetically similar to me, therefore I shall learn from that person" but will learn from the most salient individual in the environment. For the recognition of kin, strategies have evolved to ensure that individuals learn only from kin (Hepper 1986; Hepper, in press). Thus, individuals may commence learning in the womb, a time when it is unlikely that nonkin will be encountered. An individual may learn from oneself; or individual learning may be confined to a sensitive period when only related individuals are likely to be present. However learning is achieved, individuals are ensured of learning from genetically related individuals.

If learning to recognise genetic similarity is to be achieved other than through learning to recognise kin, it has to be restricted to times when only unrelated genetically similar individuals will be encountered. It is difficult to envision the environmental contingencies that would permit this to happen. Thus any recognition of genetic similarity will be based on the ability to recognise kin.

There can be little doubt that individuals ought to be able to generalise from their ability to recognise kin to recognising genetic similarity. Indeed, since recognition is based on perceptible cues and not on the whole genome, any individual bearing similar cues will be responded to as kin. Following the green beard analogy, once an individual has learned that a green beard signifies kinship, any other individual possessing this cue will be responded to as kin, irrespective of other genes that do not influence the cue.

The question of whether individuals will then recognise genetically similar individuals who are not kin will be determined by the accuracy of the cue in delimiting different kinship groups in the population. Many cases of kin recognition appear to be based on cues that allow fine discriminations of kinship (e.g., Yamazaki et al. 1982); it is unlikely that unrelated individuals will exhibit an identical cue. Some individuals in the population, however, will possess a cue similar to that denoting kinship. The extent to which overlapping cues for recognising kin exist in a population remains to be determined, this will determine the amount of genetic similarity between unrelated individuals.

Given that some individuals will exhibit similarities in their cues and will thus resemble kin to a certain extent, cases of genetic similarity attraction may be founded on an optimal preference for kinship. Individuals prefer those who are sufficiently similar to themselves (or to their representation of kinship) to avoid the consequences of outbreeding but sufficiently dissimilar to avoid inbreeding, that is, individuals will demonstrate optimal outbreeding (Bateson 1983a). Given the absence of mechanisms for recognising genetic similarity other than those for recognising kinship, and given a preference for optimally different kin, it is possible that genetic similarity attraction is based entirely on the ability to recognise kin, and that its occurrence is determined by the overlapping of kin recognition cues between unrelated individuals.

"Total perceived value" as the basis of assortative mating in humans

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As Rushton notes, there is no such thing as "genetic ESP." The means by which individuals detect genetic similarity in others must therefore depend on perceivable phenotypic cues. Thus one of the main tasks of genetic similarity theory is to provide a clear and comprehensive explanation of why important human affiliations result in a much greater than chance degree of genetic similarity between the affiliated members who are not kin by common descent. The relationship between spouses is generally the closest relationship between nonkin, but many of the characteristics for which there is positive assortative mating are probably also the basis of other nonkinship affiliations, such as close friendships. Hence statements about assortative mating are to some degree generalizable to other nonkinship affiliations.

Assortative mating for many characteristics, physical and psychological, results in genetic similarity to the extent that the characteristics are heritable. Rushton argues that the degree of positive assortative mating for various psychological traits, at least, is directly related to their heritability, probably because these traits are perceived both as the more important aspects of personality and as being less subject to circumstantial influence.

There is another aspect of assortative mating, however, that Rushton does not consider, but that would augment genetic similarity between mates (and between friends). This phenomenon can be termed *cross-assortative mating*, or the marital correlation between phenotypically and genotypically different traits, particularly when the marital correlation (i.e., the coefficient of assortative mating for either or both of the separate traits) is lower than (or not higher than) the marital correlation between the two traits. Marriage partners may accept some degree of trade-off of one valued trait for another; that is, in assortative matings equally valued traits may be equivalent, and unequally valued traits will have different "exchange" values. This seems to be the case for the traits of intelligence and physical stature, for example, probably because above-average status in these traits is deemed desirable in Western culture.

Women of the same social class of origin tend to marry up or down in socioeconomic status (as indexed by husband's occupational status, correlated about 0.65 with husband's IQ), in accordance with the women's height; taller women have the advantage (Schreider 1964; Tanner 1969); and height contributes to women's social mobility independently of the population correlation between height and IQ. The genetic effect of cross-assortative mating for two independently heritable (i.e., non-pleiotropic) genetic traits is to bring about a genetic correlation between the traits in the offspring generation, due to the common assortment of the independently segregating alleles that affect each trait – which is manifested in the population as a *between*-families, but not a *within* families, correlation component (Jensen 1980). The population correlation (of about 0.20) between height and IQ, for example, appears to be attributable exclusively to the between-families component of the correlation (Laycock & Caylor 1964), as would be expected in the case of common assortment of independently segregating alleles. Assuming that such common assortment occurs for a large number of genetically different traits, the result will be various constellations of genetically and phenotypically correlated traits in the population; such constellation will strengthen the basis for assortative and cross-assortative mating, and even some genetic traits that are not directly perceived as a basis for assortative mating will be drawn into the overall genetic resemblance between partners.

I propose that assortative mating should not be thought of strictly in terms of selection and assortment according to the degree of resemblance in each of a number of specific traits, but as the comparison by each partner of the subjective estimate of his or her own total perceived value (a subjectively weighted sum of perceived assets and liabilities reflecting individual, familial, and cultural values) with that of the other. This would result in both direct assortative and cross-assortative mating for many traits and constellations of traits, inevitably resulting in a quite marked genetic similarity between mates. Virtually all studies of assortative mating report marital correlations only for single traits (mostly in the range of 0.10 to 0.30), with mental abilities and general intelligence being the highest (0.3 to 0.5) (Jensen 1978). But many traits have assortative mating coefficients above 0.10, so a more telling index of such mating would be a canonical correlation between mates, that is, the correlation between an optimally weighted sum of all the trait variables measured for each partner. Such a canonical correlation between mates might be as high as 0.7 or 0.8, or even higher, depending on the number of traits considered.

Because of the correlation between casually perceivable traits and some "invisible" genetic traits, such as certain blood antigens, assortative and cross-assortative mating increase genetic similarity in the "invisible" traits as well. It has been found, for example, that 16 blood groups predicted IQ with a multiple correlation of about 0.60, separately in white and black samples (Osborne & Suddick 1971). Assortative mating for IQ, therefore, would inevitably result in greater than chance similarity between mates in terms of blood group genes and would perpetuate a genetic correlation between IQ and blood groups in subsequent generations through the common assortment of genes affecting both variables. This association between IQ and blood groups alone might be sufficient to explain Rushton's observation that spouses and friends are more similar in blood groups than are randomly paired individuals. The correlation between certain perceptible psychological traits and "invisible" physical traits probably reflects the past history of stratification of the population by national origin, ethnicity, and social class. A few such correlations, such as between IQ and myopia, appear to be related more directly by pleiotropy; that is, the same gene(s) affect two phenotypically distinct traits, and show up *within* as well as *between* families, unlike correlated traits that merely reflect common assortment of segregating alleles through assortative mating or population stratification and are detected only in the *between*-families component of the population correlation (Cohn et al. 1988).

Finally, the chief benefit of assortative and cross-assortative mating, from an evolutionary standpoint, hardly emphasized by Rushton, is that both types of mating increase genetic variance in the population (Jensen 1978), and genetic variance is the sine qua non for biological evolution and environmental adaptation. In the interest of environmental adaptation, many "selfish" genes survive far beyond the species that have harbored and transmitted them; the number of species living today represent hardly more than 1% of all the species that have ever existed. Species struggle to survive by every means at their disposal, but individual genes seem to be the only truly long-term survivors.

Altruism, Darwinism, and the gift of Josiah Wedgewood

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But for Josiah Wedgewood's altruism toward Charles Darwin, we might not be having the present discussion of genetic similarity and prosocial behavior. Wedgewood gave Darwin and his bride a lifelong grant that freed young Charles to pursue an otherwise impractical career as a naturalist. Wedgewood's wedding gift is a poetic fit with Rushton's neo-Darwinian arguments about nepotistic altruism and genetic self-affinity. Darwin's bride was his cousin Emma Wedgewood – Uncle Josiah's daughter.

Although research on the unfair cognitive power of vivid cases warns me against letting such an appropriate vignette stand in the way of criticizing Rushton's data, I must admit a reluctance to differ with the overall argument. My own examination of the social psychological literature forces agreement with the premise that people are superabundantly attracted to, and generous toward, those of a similar stripe (cf. Kenrick & Trost 1987). The exceptions, such as aging men's preference for increasingly younger females, still fit a general Darwinian model (Kenrick & Keefe 1989; Kenrick & Trost 1989). It also seems almost tautological that any genetic mechanisms favoring such "ethnonarcissism" would have been stamped in by natural selection. But I have some specific points of disagreement nevertheless. Rushton seems unduly glib about the fact that anthropometric measures do not predict attraction as strongly as the less reliable personality measures, and his claim that personality traits may be linked to relatively greater numbers of genes seems stretched. Rather than quibble with the methodological aspects of Rushton's article, however, I will emphasize three theoretical issues.

1. Rushton may be prematurely committed to a particular type of mechanism. Recent data suggest that *people are not so much attracted to similar others, as they are repulsed by those who are dissimilar*. Rosenbaum (1986) exposed some subjects to targets who made statements in agreement with their own opinions, and others to targets who made statements in disagreement. In comparison to control subjects who had no information about a target, similarity led to little increase in attraction. However, dissimilarity did lead to increased disliking for the target person. Could Rosenbaum's "repulsion hypothesis" explain Rushton's data? Given the notion of selfish genes, it makes as much sense to imagine alleles that are out to get the competition as alleles that are out to boost the home team. And since our ancestors lived in small groups of closely related hunter-gatherers, a few simple exclusionary detectors would have been more efficient than the abundant similarity detectors that Rushton's theory necessitates. On a proximate

level, such a mechanism could operate at several stages of courtship. Perhaps there is aversion toward others who look or act markedly different from those we were raised around, which would block formation of relationships at the starting gate. Or perhaps there is easy annoyance with others whose personal preferences, attitudes, and interests are markedly at variance with those we are accustomed to, and this blocks a relationship's progress. Finally, there may be continuing distrust of minor incongruities in the personal habits even of those we allow close to us. Indeed, friendships and marriages often break up over a few differences of opinion that loom large against a massive background of similarities.

For the most part, a dissimilarity-repulsion model leads to predictions compatible with Rushton's perspective. The advantage of a repulsion mechanism is that it could ride on the preadapted coattails of other mechanisms designed to detect outsiders, who are always a threat. A rejection mechanism has the efficiency of Strong Inference (Platt 1964) – positive information allows a wide range of ambiguous possibilities, but negative information deals a one-shot death blow. Shepher's (1971) classic findings indicate that the kin recognition mechanisms of children raised in the kibbutz can easily be led astray (see also van den Berghe 1983). Those data argue against the existence of finely tuned similarity detectors and favor the existence of simple and blunt aversion mechanisms.

2. Hawaiian cross-marriages suggest that the *"ethnonarcissism" mechanism is not strongly impervious to experience.* In the evolutionary environment of our ancestors, it probably did not need to be. Before the development of modern urban culture and democratic/socialistic governments, human elders probably did little preaching about the virtues of tolerance toward outsiders. Christianity began to develop in the rapidly overpopulating Middle East only 2,000 years ago, and may have been a cultural adaptation to the novel experience of living among closely allied nonkin. An analogous explanation is often offered for the widespread adoption of birth control devices during this century, which have not been around long enough to have been a strong force in natural selection. In the same way, our ancestors' inclusive fitness may rarely have been challenged by cultural pressures against ethnocentrism. In addition, there is the possibility of a profitable genetic trade-off in being flexible about attraction and mating with members of outgroups. If you grow up in a neighborhood with a prominent alien clan, mating with a member of the other group could increase the survivability of your children, who would share characteristics of both groups. If this second point is correct, incidentally, any aversion mechanism would be expected to operate only at the initial filtering level. Repeated exposure to outgroup members under pleasant circumstances might lead to an acquired taste for their specific features, much as an initial aversion for spicy foreign foods can be replaced by an acquired craving.

3. Our own program of research on the socialization of altruism (Cialdini et al. 1981) suggests that *even encultured altruism is ultimately consistent with a "selfish gene" model.* Altruistic behavior seems like pure self-punishment to a very young child, but after several years of socialization against selfishness and toward charity, acts of public kindness increasingly function as a means of obtaining adult reward (Kenrick et al. 1979). After a few more years of socialization, altruism seems to function as a conditioned self-reinforcer (Baumann et al. 1981). Thus, even the effects of socialization on altruism are compatible with Rushton's general viewpoint; there is no need for a sociobiological theorist to be defensive about the flexibility of the mechanism. It is not that socialization overrides selfishness, as suggested in Campbell's (1975) paper on ethnocentrism and altruism. It is just that society makes us an offer that the totality of our selfish genes cannot refuse.

Not genes: Behaviour

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In this target article Rushton attempts to demonstrate that a variety of social psychological phenomena, such as friendship, choice of spouse, altruism, and even xenophobia, are strongly influenced by genetic similarity. In a word, it would appear that we prefer those most like ourselves.

In his demonstration Rushton cites a huge amount of evidence. His interpretation of this evidence and his failure to cite conflicting evidence are the subject of this commentary. In section 3.3, he argues that "the more one is exposed to a stimulus, the more one prefers it." In addition, he claims that sexual preferences may be established early in life through an imprinting-like process. Neither of these arguments can be supported. The first may have been advanced by Zajonc (1980) but it has little generality. For example, the smell of petrol may at first be attractive but one rapidly tires of it; the same is true for music, and literature. With regard to the second argument, sexual potency is often restored by a new partner. It is true that sexual preferences may be established early in life, but only in the sense of taboos, as Rushton admits, using the same evidence to make this case in section 6.1. This double use of the evidence suggests that the arguments can go either way, as has been demonstrated in sociobiology generally (see Kline 1988).

In section 3.4 Rushton argues that the high correlation between an individual's location and kinship shows the importance of genetic similarity because physical proximity has widely been observed to be predictive of friendship formation and success in meeting a potential spouse. This is social psychology at its silliest. It is impossible to be a friend of someone with whom there has been no contact; even the members of the jet set have their physical limits. This cannot be used as evidence for the role of genetic similarity.

Kin preference is held to have a genetic basis because (excluding husbands and wives) in 1,000 wills kin received 55% of the total bequeathed, whereas nonkin received only 7% and offspring received more than nephews and nieces. In our society parents are responsible for their children and may have little contact with nephews and nieces. An explanation of kin preference does not require the notion of genetic similarity. Similarity, the finding that a high proportion of battered children are stepchildren reflects the social circumstances of stepchildren, for example, the problems of single parents, who are attempting to form new relationships. The finding that unrelated people living together are more likely than related people to murder each other must reflect the fact that the majority of domestic murders are of husband or wife, that it is the tensions of marriage rather than the nonidentity of genes that create the murderous rage.

The arguments in section 6.1 that the correlation of spouses' IQs reflects genetic similarity preference are not powerful. So much is influenced by level of IQ – interests and attitudes, for example – that disparate IQs in marriage are likely to be a source of difficulty. If partners are to enjoy the same plays and other forms of entertainment, such selection is simply sensible. Correlation of IQs also affects other known predicting variables, such as location (there are not many low IQs at MIT) and education.

The argument that inbreeding in marriage (section 6.1) offers the optimal solution suggests that marriages between cousins should be more common than they are. Thus among human beings other factors have overcome this tendency, if it was ever present. This is, however, precisely the point. Humans' biological imperatives are overruled by culture. That is why genetic similarity is unlikely to be a powerful determining factor for human beings.

There seem to be other confusions in the argument about genetic similarities, for example, in section 6.3.1: It is shown that friends are more similar genetically to each other than are random pairs. However, if genetic similarity is measured in terms of IQ, skin colour, and occupation, this result is explicable in terms of their effects, as I discussed in the section on IQ. It is not a sufficient counter to this claim that blood similarity scores do not correlate with the within-pair differences, which are bound to be less reliable.

For all these reasons the evidence cited by Rushton is far from compelling. True, there is a possible explanation in terms of genetic similarity running through all the evidence. However, on examination it is not, in most cases, the most simple or most likely explanation.

In section 8.1 (para. 5), inadvertently, I believe, Rushton destroys most of his thesis. "It is not difficult to imagine," he writes, "how intellectually and temperamentally different siblings" [yet those sharing 50% of the genetic material] "might seek out different social environments." This is the counter to all the claims that genetic similarity leads to friendship or marriage. [See also Plomin et al.: "Why Are Children From the Same Family So Different?" *BBS* 10 (1) 1987.] Rushton implies that intelligent introverts simply prefer to spend time with intelligent introverts. That these variables are genetically determined is neither here nor there. The argument in this thesis is not unlike the claim that cannibalism is genetically bred out because men prefer the taste of cow (genetically remote) to the taste of chimpanzee, whereas the appetite for vegetables implies a curvilinear relationship.

Detecting genetic similarity without detecting genetic similarity

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Animals sometimes extend preferential treatment to those who are similar to them genetically. That is a fact of nature, not a theory. A theory predicts when, shows how, and explains why. The mechanisms usually invoked to explain preferential treatment involve kin recognition and kin selection. The problem Rushton faces in his provocative cross-disciplinary pursuit of a new theory is that the closer he stays to kin selection, the less original his position, but the farther he strays, the more implausible it becomes.

The logic of kin selection is tricky and much misunderstood (see Dawkins 1979). Consider, for example, the evolution of altruism. Hamilton (1964) showed how self-sacrificial altruism could evolve when $k < 1/r$, where k = the ratio of recipient benefit to altruist cost and r = the coefficient of genetic relatedness. Note that in Hamilton's formula, all individuals who share genes by descent – that is, share a value of r greater than zero – are kin. Rushton seems to view kinship in a different way, as a qualitatively distinct demarcation. As emphasized by Dawkins (1979) and Mealey (1985), r does not refer to the *proportion* of genes shared by two individuals (which has been estimated as more than 98% for humans and chimpanzees); and it is not significant theoretically as a measure of the proportion of genes two individuals share *by descent*. Rather, it is important because it provides an estimate of the *probability* that two individuals share the *altruism-inducing* gene *by descent*. The evolution of the altruism gene, not any other, is in question.

Although it might seem that a necessary condition for extending preferential treatment to kin is the ability to detect genetic similarity, this is not the case. Genetic programs like "help all animals who live in your colony" and "help all animals who are familiar to you" will mediate the evolution of altruism through kin selection if kin are more likely than nonkin to meet these conditions. The human capacity for altruism may have evolved through kin selection in this manner (our ancestors lived in relatively small groups of extended families) and altruism may now be evoked "mistakenly" by nonrelatives who possess the characteristics that elicited altruism toward kin in the past (Krebs 1987). This does not explain why individuals show preferences toward genetically similar strangers from different locations, however.

Kin selection is not particularly efficient – the probability of a "hit" (sacrificing oneself for someone who shares a gene for altruism) is only as high as the coefficient of relatedness. It would be more efficient for an altruism-inducing gene to produce a phenotypic characteristic (such as a green beard) *and* the inclination to help those who possess that characteristic (and therefore the gene). Genetic similarity theory appears to advance the assumption that not only one, but many such genes have accomplished this feat. The problem is that this assumption is implausible, both genetically and logically. Dawkins, who popularized the possibility as the "green beard effect," calls it "a kind of academic biological joke" (Dawkins 1986, p. 206). The probability of a mutation mediating these effects is extremely low. A gene would have to "recognize" its actual DNA, because other DNA sequences could produce the same phenotype. Even if such a mutation did occur, it would be an "outlaw" (Alexander 1979; Dawkins 1982), opposed by genes at other loci. Finally, such a gene would be so effective that it would flood the population, producing a species in which all individuals possessed the "green beard" and behaved altruistically toward everyone else. This may characterize some social insects, but, sadly for some, it is not the human case.

Rushton and Russell (1985) rebut the implausibility of recognition alleles by arguing that (1) if one gene cannot mediate the process, many genes might, and (2) humans *must* detect genetic similarity, because the traits on which they assortatively mate tend to be highly heritable. Of course, many genes are involved in complex discriminations, but that doesn't alter the logic of the argument. If linkages of genes survive meiotic division, they are, in effect, equivalent to one gene, and would be opposed by the rest of the genome. If linkages divide during meiosis, the combinations producing altruism-evoking "green beards" without the altruism-giving counterpart would prevail over all other combinations. If Rushton insists on basing his theory on recognition alleles, he must explain how they could overcome these obstacles.

Why insist on mechanisms able to detect genetic similarity, when another, perfectly plausible mechanism – phenotype matching – can account for all the findings outlined by Rushton? In phenotype matching, individuals inspect some aspect of their phenotype or the phenotype of their kin, and prefer others who possess the same or similar phenotypes. Most of the researchers cited by Rushton interpreted their results in terms of phenotype matching, not allele recognition (although, as Blaustein 1983 points out, the two mechanisms are difficult to distinguish empirically). Phenotype matching differs from allele recognition in two main ways: (1) the gene (or linkage) directing the matching program is different from the gene producing the phenotypic characteristics (the matching gene does not recognize *itself* directly, and it may mediate a preference for many different phenotypes, such as red beards, white beards, and black beards, not just one, such as a green beard); and (2) phenotype matching is a mechanism for recognizing kin, and thus is propagated through kin selection. The matching gene is not opposed by genes at other loci because the other genes have the same chance as it does of being represented in the kin displaying the phenotype. The statement "If like appearance is positively correlated with like genes, any mutation toward preference for like phenotype would tend to proliferate" (Rushton & Russell 1985, p. 581) is correct only if like-appearing

individuals are more likely to carry the gene or genes mediating the *preference* for like phenotype (i.e., the matching gene) – a probability represented in their coefficient of relatedness.

Consistent with Rushton's data, but contrary to his interpretation of them, individuals would be expected to match on highly heritable characteristics if those characteristics provided the most reliable cue to kinship. Many species match on odor, which supplies a highly reliable cue to kinship. (Police dogs appear to be able to distinguish between all individuals except identical twins; Kalums 1955.) Converging with psychoanalytic theory, humans might match for altruism on their own appearance – a kind of genetic narcissism – and they might match for mate choice Oedipally on the appearance of their same-sex parent. ("I want a girl just like the girl who married dear old Dad".) It is not unreasonable to expect humans to match on complex combinations of characteristics, both inherited (aspects of physical appearance) and acquired (nationality), genes may program the human brain to perform any number of spectacular feats.

Mechanisms mediating mate preference would be expected to differ from mechanisms mediating altruism. (Space does not permit developing this point.) The matching rule for mating does *not* direct individuals to prefer others who are *most* genetically similar to themselves, otherwise they would court their parents and siblings; indeed, it instructs them to avoid rather than to prefer high levels of genetic similarity (incest). Most of the evidence Rushton adduces is correlational in nature; it is to be hoped that he and others will direct future attention to uncovering the causal mechanisms underlying the observed relationships.

In summary, it seems that the vagaries of genetic transmission and the laws of evolution block the most direct route that self-seeking genes might take to replicate themselves through others – the route on which the "strong" version of genetic similarity theory is based. Genes cannot detect copies of themselves in others directly, but they can mediate the ability to "detect" how closely others are related by descent, and thereby the probability that others possess a matching gene directing preferences for those who are similar to them. It is in this sense that individuals detect genetic similarity without detecting genetic similarity.

Phenotypic matching, human altruism, and mate preference

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Although it is not made entirely clear in the target article, Rushton et al. (1984, p. 189) earlier distinguished between "strong" and "weak" versions of genetic similarity theory. "The 'strong' version of the theory implies that individuals possess the ability to recognise genetic similarity in the absence of previous familiarity or other proximal mechanisms" (the "green beard effect," Dawkins 1976); "The 'weak' version . . . implies that the ability to detect genetic similarity is acquired through a process of exposure to appropriate stimuli."

These definitions apparently tie strong genetic similarity theory to the hypothesised mechanism of innate feature detectors. Weak genetic similarity theory could operate via the proximate mechanisms of phenotype matching, familiarity or association, or location.

Kin selection theorists have of course long postulated that mechanisms such as these four would be responsible for preferential altruism directed towards kin. So what is new about Rushton's approach? There appear to be four elements.

1. He argues for a strong version of genetic similarity theory, contrary to most previous commentators who have rejected the likelihood of innate feature detectors.

2. He argues that if the theory operates via phenotype matching, then we can expect an outcome not predicted by conventional kin selection theory: discrimination amongst others of the same genealogical class, on the basis of phenotypic (and hence, supposedly, genotypic) similarity.

3. He argues that phenotypic matching will apply not only to altruistic behaviour but also to mating preference.

4. He applies these arguments to human behaviour.

We offer brief comments on each of these points.

1. Innate feature detectors remain unlikely. As Waldman (1987, p. 170) puts it, "the hypothesis that any form of behavioral discrimination could be due to the operation of a single-locus recognition system lacks empirical support." *None* of Rushton's empirical data support any such mechanism in humans. Rushton then adopts phenotypic matching as his preferred mechanism, citing the reasonable point made by Waldman that recognition must occur via feature detectors that are likely to be the product of (and hence to respond to the phenotypic effects of) multiple alleles. In taking this position, Rushton no longer has to pin his theory to the green beard effect, but it consequently becomes less original.

2. The mechanism of phenotypic matching has regularly been proposed as a mechanism of kin selection when (as is often the case) neither familiarity nor location seem to be a primary determinant of altruism. Phenotypic matching is a basic element of kin selection theory, and it is not immediately clear what point is served by calling it "genetic similarity theory." However, Rushton has correctly drawn attention to a rather neglected point: If phenotypic matching is an important mechanism for altruism, then we may expect individuals to discriminate amongst others in ways apart from their genealogical category. First, they may discriminate between individuals within the same category; second, they may favour similar individuals with whom they share no apparent pedigree ties.

Some questions may be raised about the logic of the latter point regarding unrelated similar individuals. Grafen (1985) has argued that the extension of favouritism to nonrelatives is highly unlikely, since they will not share sufficient genetic similarity to the altruist to justify discrimination. Grafen's calculations are admittedly based on a nonstructured population – that is, on negligible amounts of assortative mating and no incidental genetic stratification. However, it seems important for Rushton to provide a more convincing rationale for the existence of sufficient degrees of overall genetic similarity amongst nonrelatives than that "aggregation effects would be expected." He needs to argue either that molecular identity (i.e., genes shared by virtue of common descent) is not the homology we require for the inclusive fitness model, or that the population does in fact show evidence of trait clustering. The point is that one cannot simply state without some justification that "kin selection might be just one form of genetic similarity detection," since the value of common descent is precisely that it negates the cost of altruism to the genome.

With regard to Rushton's empirical data, virtually any theory of friendship would predict some similarity amongst friends in interests and beliefs. What is novel is the linkage of friendship to genotypic characters. Such a finding requires a strong replication study to rule out the possibility of artifact, particularly given the small number of loci looked at. We plan to examine such data further on the basis of the more powerful technique of genetic fingerprinting (Jeffreys 1987).

Rushton's point that phenotypic matching is expected to mediate discrimination within the family is also important. Previously, deviations from the belief that altruism is apportioned by genealogical category have been related only to such measures as reproductive value, which refer to differing fitness levels for the individual concerned. Here, the most relevant set of Rushton's data are on parent's grief at the death of a child

(taking grief as a proxy for attachment and hence altruism). Given that Scarr and Grajeck (1982) and Pakstis et al. (1972) have demonstrated that perceived similarity corresponds to measured genetic similarity, we feel that this study offers fairly strong evidence that a phenotypic matching mechanism promotes within-category discrimination. In a study in progress of three-generation family relationships (Leek & Smith 1988), we have found some evidence confirming this, in that variance in perceived altruism within a family correlates with perceived phenotypic personality similarity, and to a lesser extent with physical similarity. This is true of both parent-to-offspring relationships and grandparent-grandchild relationships. We are currently testing the further hypothesis that the observed variance relates to genotype by using antigen analysis with a subset of these families.

3. Rushton also suggests that a phenotypic matching mechanism can be applied to mating preferences. One would expect phenotypic matching to be used differently from the way it is used in altruism per se. With altruism, the correspondence with degree of phenotypic matching would be expected to be monotonic; with mating preference it would perhaps be expected to be curvilinear, with both extreme similarity and extreme dissimilarity avoided (although see Darlington 1960). This could result from the interaction of a phenotypic matching mechanism with a familiarity mechanism (Bateson 1983a).

4. If humans use phenotypic matching mechanisms, in choosing either recipients of altruism or mates, then it will be of further interest to unravel which aspects of the phenotype are most relevant in each case. Is it some very general estimate of overall genetic similarity, aggregating over a large number of indexes, or are some phenotypic characteristics (e.g., physical appearance, or temperament) or particular significance? Are the same phenotypic matching mechanisms used in different situations? There exists a large body of relevant psychological literature (Bentler & Newcomb 1976; Buss 1985; Hill et al. 1976), but comparing the theory with other findings will be complicated because humans are influenced by social and cultural beliefs. These beliefs may cause wide variation with respect to any canalised processes of the phenotypic matching hypothesised to be characteristic of the human species.

Balanced polymorphism for ethnocentric and nonethnocentric alleles

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Racial and ethnic conflict is occurring throughout the world – between blacks and whites in the United States, South Africa, and Britain; Basques and Spaniards in Spain; and Irish and British in Northern Ireland. These conflicts have defied explanation by the disciplines of sociology, psychology, and economics. Rushton's genetic similarity theory represents a major advance in the understanding of the cause of these conflicts.

One phenomenon to which genetic similarity theory might plausibly be applied is the effort by ethnocentric groups to promote their own language. In Ireland there is a powerful lobby for the establishment of Gaelic as the official language of Eire. Although virtually no one actually speaks Gaelic, successive governments have passed legislation to appease the Gaelic lobby; for example, all children are required to learn the language at school; street names are written in both Gaelic and English; a number of radio and television programs are broadcast in Gaelic; all public employees have to pass an examination in Gaelic; and professors at the University College of Galway are required to deliver their lectures in Gaelic, if called upon to do so. All these things are also happening in Wales with regard to Welsh; here again, hardly anyone actually speaks the language or has any wish to do so.

The importance attached by ethnocentrists to the promotion of these minor and virtually extinct languages is an illustration of the apparent irrationability of ethnocentrism, since it seems obvious that children would be much better off learning one of the major world languages rather than Gaelic or Welsh. Nevertheless, the phenomenon can be understood in terms of genetic similarity theory as an attempt to promote ethnic group inbreeding. When group members speak different languages and can no longer communicate with each other, breeding between them must necessarily be considerably reduced. Most (although perhaps not all) individuals prefer to be able to talk to their mating partners. That this curious and otherwise inexplicable phenomenon can be explained by genetic similarity theory illustrates the explanatory power and range of the theory.* [*See Editorial Note following this commentary.*]

Ethnocentrism is undoubtedly a powerful and widespread emotion and one for which I believe Rushton has provided a convincing explanation in sociobiological terms. Nevertheless, it is clear that by no means are all humans ethnocentric. Many humans display no animosity to other races or ethnic groups and are quite happy to mate with and marry into other groups. How are these individual differences in ethnocentrism to be explained?

The most promising line of explanation is balanced polymorphism. In balanced polymorphism, there are two alternative alleles, each of which confers some selective advantage and both of which therefore survive. In the case of ethnocentrism, the selective advantage to a group of having some nonethnocentric members might be that some of them would outbreed with a member of another group, thus introducing new and possibly advantageous alleles into the group. If the new allele was advantageous, it would spread throughout the group into which it was introduced. For instance, when groups of early *Homo sapiens* migrated from Africa to Europe, they developed white skins. This conferred the selective advantage of being able to absorb vitamin D from sunlight. There must have been mutants for white skin that spread from one breeding group to another through outbreeding. If all members of a group had been militantly ethnocentric, the outbreeding would not have occurred and the advantage of acquiring the new allele would not have been gained. Maximum selection advantage would therefore accrue when many members of a group possessed the ethnocentrism allele, in accordance with Rushton's thesis, but some would not.

EDITORIAL NOTE

*The foregoing two sentences, if they were not intended as a joke, illustrate the degree to which uncritical biological determinism can blind us to the obvious. Surely even an astrological account of our doings and destiny would have more "explanatory power and range" than an account like this one. On the other hand, this sounds as if it would apply quite well to birdsong. Perhaps the difference between the two cases might have something to do with the fact that human languages have not only form but content, and that that content too may play some small role in our doings and destiny.

If "birds of a feather. . . ," why do "opposites attract"?

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Rushton presents evidence that genetic similarity significantly influences the selection of mates and friends, but he also admits that some humans tend to choose dissimilar partners. These

Table 1. *Dating practices of five ethnic groups in Hawaii (in percent)*

	Date mostly or only own group	Indifferent ("50/50")	Date mostly or only other groups
Males (n = 70)	33	39	29
Females (n = 154)	55	34	11
Total (n = 224)	48	36	17

**Source:* Recomputed from Johnson & Ogasawara 1988, Table 2. Ethnic composition: Caucasian (14 males, 27 females); Chinese (7 males, 14 females); Filipino (6 males, 9 females); Hawaiian (9 males, 21 females); and Japanese (34 males, 83 females). In all groups except the Japanese (where the frequency of exclusive within-group dating was similar for both genders), only females reported restricting dating entirely to their own group. On the tendency for females to be more risk-averse than males, see Masters, in press.

exceptions deserve examination, especially since elsewhere Rushton has shown important variations in reproductive strategies that are associated with mate choice (Rushton & Bogaert 1987). In a recent study of dating in Hawaii, less than half of the sample restricted partner choice primarily to their own ethnic group – and the frequency of selecting partners from another group varied substantially depending on the individual's gender (Table 1) as well as ethnicity and income (Johnson & Ogasawara 1988).

Have we learned anything if "birds of a feather" often "flock together," but sometimes "opposites attract"? To understand the mate selection process further, it is important to focus on the ethnology of bonding and its relation to reproductive strategy. As a characteristic of either individual personalities or social groups, the preference for similar partners seems to be a *variable* associated with *K*-selective reproductive strategies, not a species-specific constant. If viewed in this way, plausible mechanisms can explain Rushton's findings without postulation of innate feature detectors like Dawkins's hypothetical gene for a "green beard."

Although Rushton points out that "according to genetic similarity theory, people can be expected to favor their own group over others," this preference need not be constant, either within or between social groups. Particularly in a species like our own, individual strategies of social behavior are likely to be mixtures of cooperation and competition (Alexander 1979; Frank 1988; Masters 1989). Personality differences can therefore be understood as an evolved repertoire of traits leading individuals to respond to a given situation or task in different ways.

Cloninger (1986; 1987) has suggested a classification of human personality based on three dimensions, each of which appears to be normally distributed in human populations: "harm avoidance" (vs. risk taking), "novelty seeking" (vs. stereotypy), and "reward dependence" (vs. social independence or task persistence). Although they are defined in terms of observed personality assessments, the three dimensions can be linked to distinct neurotransmitter systems (based, respectively, on serotonin, dopamine, and norepinephrin). [See also Zuckerman "Sensation Seeking" *BBS* 7(3) 1984.]

Cloninger's theory explains personality types and affective disorders on the basis of the individual's functioning with regard to each of the three underlying dimensions. For example, a passive-dependent personality is likely to be high in harm avoidance, low in novelty seeking, and high in reward dependence (Cloninger 1987, p. 581). Although genes probably influence the baseline activity of each neurotransmitter system, thus creating heritable predispositions to affective disorder and other social traits, individual experience through ontogeny modulates the underlying neurotransmitter functions and behavioral repertoire (Ginsburg 1988). As a result, both genetic variation and individual experience can contribute to observed differences in personality and social behavior (Hoffman 1981; Rushton, Littlefield & Lumsden 1986). [See also Plomin et al.: "Why Are Children From the Same Family So Different?" *BBS* 10(1) 1987.]

Neurotransmitter systems are a plausible organic substrate of individual differences in behavioral strategy. Thus serotonin, implicated in the harm avoidance vs. risk-taking dimension (Cloninger 1986; 1987), is associated with impulse control, perhaps because it is an inhibitory neurotransmitter in sensory pathways and an excitatory one in the motor cortex. Individuals with low levels of serotonin are likely to exhibit impulsiveness, depression, aggressiveness, or suicidal tendencies (Stanley & Mann 1987; Linoilla et al. 1983; Wurtman & Wurtman 1989), whereas high levels of serotonin are associated with dominant social status (McGuire & Raleigh 1986). [See also Soubrié: "Reconciling the Role of Central Serotonin Neurons in Human and Animal Behavior" *BBS* 9(2) 1986.]

Neurotransmitter levels and turnover are influenced by the environment as well as heredity. Serotonergic functions, for example, are sensitive to many environmental factors, including carbohydrate intake, exposure to light, and social interaction (Wurtman & Wurtman 1989). The enhanced level of serotonin specifically associated with dominance depends on the leader's status: The mediating variable is the sight of submissive behavioral cues rather than physiological traits or genetic predisposition (Raleigh & McGuire 1986).

At the behavioral level, one proximate mechanism in personality seems to be the perception of and response to nonverbal social cues. Mothers' facial displays elicit latencies and response patterns in infants at 4 months that remain stable at 7½ months (Izard et al. 1987); despite many differences in development in the first three years, individual stability in social response is often observed (Kagan 1988; Bernstein 1988; Montagner et al. 1988). Among adults, similar individual differences are evident in responses to seeing known leaders on television. Although viewers reliably decode the nature of a leader's nonverbal displays (Masters et al. 1986; Sullivan & Masters 1988), there is an individual bias or sensitivity in perceiving hedonic or agonic cues across excerpts showing different displays or leaders, after controlling for the viewer's political opinions, the social context, or the media condition (Carlotti 1988).

These individual differences in perceiving identical stimuli are of direct significance in bonding. Not only is the perception of positive or reassuring traits in another highly correlated with positive affect (Sullivan & Masters 1988), but the ratio of perceived hedonic to agonic cues is a significant predictor of emotional response after controlling for display type, partisanship, and other variables. In much the same way, young children differ in the ratio of hedonic to agonic behaviors, and this ratio is strongly predictive of social bonding (Montagner et al. 1988).

Cloninger's three personality dimensions, by influencing the perception and display of nonverbal social cues, can channel bonding behavior in three ways. First, individuals differ in the extent to which they take risks; highly harm avoiding personalities are more likely to seek similar mates in order to minimize the chances of deception and loss. Second, individuals also differ in their tendency to seek novelty – and high novelty seekers should be more likely to bond with partners differing from themselves in personality or social traits. Third, insofar as

individuals differ in sociability or reward dependence, those high on the "reward dependent" trait are likely to seek reassuring cues from others and to bond more readily to those who give them frequently.

It is tempting to hypothesize that optimum bonding occurs between pairs that are similar in two of these three personality dimensions, but there is only impressionistic evidence for this prediction. Since we know that individuals vary in personality, however, such traits can be used to explain differences in the frequency of within- or between-group bonding. Since women are more likely to be risk-averse than men (Masters, in press), for instance, this approach suggests that they are also more likely to seek similar partners (see Table 1).

Superimposed on this general pattern, moreover, are probably variations of risk taking associated with social status or culture. In other species, environments with abundant and stable resource flows are associated with *K*-selective strategies, whereas scarcity and unpredictable ecological settings often elicit *r*-selective strategies (Barash 1977; Wilson 1975). Because such differences are also found in human populations (Dickemann 1979; Masters 1984; Rushton & Bogaert 1987), similarity in mate choice could also vary across cultures. This would explain, for example, the fact that interethnic dating is more frequent among poorer ethnic groups in Hawaii (Johnson & Ogasawara 1988).

Differences in the extent of phenotypic (or genotypic) matching in pair bonds thus seem quite explicable. For those with little to lose, whether because they have "novelty-seeking" and "risk-taking" personalities or because their socioeconomic status predisposes them to an *r*-selective strategy of reproduction, "opposites attract." For harm-avoiding or novelty-averse individuals, particularly those living in environments characterized by *K*-selective strategies of mating, bonding is more likely among those who are similar.

This addition to Rushton's hypothesis specifies proximate mechanisms that could mediate the observed genetic as well as phenotypic similarities among mates or friends. Even more important, it explains variations in this phenomenon, making it possible to predict when individuals or members of groups are more or less likely to bond to members of outgroups. Unless such hypotheses are formulated and tested, genetic similarity theory can hardly go beyond the folk wisdom that "opposites attract" but "birds of a feather flock together."

How important are distal genetic factors in human assortative mating?

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Undoubtedly many of the commentaries on Rushton's target article will focus on whether any of the findings concerning kin recognition, altruism, and mate selection in lower animals can be extrapolated to assortative mating in humans. Since one cannot (or should not, at any rate) manipulate human mating systems, Rushton has had to build his case for the operation of genetic similarity theory in humans by piecing together several diverse elements of the human behavior genetic literature. In my commentary I consider elements of this literature that I feel are contradictory or missing and thus fail to support genetic similarity theory.

Empirical support for genetic similarity theory is derived from the significant correlations between the degree of similarity between spouses (or friends) and the degree of heritability across a set of traits. Rushton is aware of the dangers of generalizing assortative mating coefficients and heritabilities from different samples, but he argues that these parameters are in fact highly generalizable (Rushton 1989a). For intelligence, at least, this assumption is questionable. In an earlier *BBS* commentary, Johnson & Nagoshi (1987) noted that over the years, estimated heritabilities and common environmental influences on intelligence have declined in Western samples. Egyptian and Korean families have been found to have considerably higher between-spouse and parent-offspring resemblances than Western samples (Abdel-Rahim et al. 1988), and data from a sample of Egyptian twins suggest that heritabilities for cognitive abilities may be lower, although influences of common family environment may be greater in Egypt (Abdel-Rahim et al. 1989). This same pattern of lower heritabilities and more similar environment factors has also been found in studies of African-American twin pairs in the United States (Willerman 1979, pp. 440–44). Clearly, cultural and historical factors can have important influences on the expression of genetic effects.

Rushton also makes the common but nevertheless wrong assumption that if a trait is highly heritable, then it is not easily modifiable by the environment. Investigators of methods of quantitative genetics (e.g., Falconer 1981) are very careful to state that heritability estimates apply only to the populations they are derived from, in part because the degree of heritability of a trait in one population tells little about the degree of genetic and environmental influence on the trait that would be found in a different population and environment. Nagoshi et al. (1984) found that in a large sample of Caucasian and Japanese-American families from Hawaii, those cognitive tests which yielded the highest parent-offspring and sibling resemblances also showed the greatest environmentally mediated mean changes across generations, that is, more heritable cognitive abilities were also the most affected by environmental influences.

The influence of human mating systems on genetic transmission can also be considerably more complicated than Rushton has taken into account in his mechanisms for detecting genetic similarity. The cross-cultural difference in degree of between-spouse resemblance noted earlier between Western samples and Egyptian and Korean samples is the result of a profound difference in the mating systems in the two sets of countries. At the time the spouses in the Egyptian and Korean samples were marrying, almost all marriages were arranged by matchmakers (Abdel-Rahim et al. 1988). Although similarities between potential spouses in personality, ability, and physique were important in the decisions of the matchmakers, social class was clearly the overriding priority. The practice of arranged marriages nevertheless probably had the effect of increasing the genetic similarity of spouses, but this mechanism has no counterpart in nonhuman species.

Recent analyses of Japanese- and Chinese-American sibling pairs and their spouses in Hawaii (Nagoshi et al. 1987; Nagoshi et al. 1989) indicated that between-spouse resemblances in educational attainment, verbal ability, and the use of a uniquely Hawaiian dialect of English were partly or entirely due to social homogamy rather than to spouses' active attempt to seek similar phenotypes in their mates. Social homogamy is the result of spouses having met and married within the same social stratum; between-spouse correlations are the result of phenotypic variability across strata (Heath & Eaves 1985). This social homogamy mechanism, similar to Rushton's proposal of location as a means of producing genetic similarity between spouses, is undoubtedly important in accounting for the high frequency of across-ethnic group marriages in Hawaii (Johnson 1984). Given the variability in genotypes within any given social stratum, however, assortment due to social homogamy will result in less genetic similarity between spouses than would be the case for active phenotypic assortment.

Assuming that assortative mating and heritability coefficients are generalizable, the patterns of correlations between degree of assortment and degree of heritability may still be somewhat contradictory to those resulting from application of genetic similarity theory. Rushton argues against correlating between-spouse resemblances and heritabilities across domains of traits,

as this correlation may be confounded by the number of genes determining traits in different behavioral domains and by "sequential filtering" of traits. Yet, if one attempted such a correlation, one could, for example, take the high between-spouse correlations for opinions, attitudes, and values and the lower between-spouse correlation for intelligence and find a negative correlation with the lower heritabilities for attitudes relative to those for IQ. Traits in these behavioral domains are probably polygenetically determined, so it would be hard to argue that this result is due to differences in the number of genes being assorted for. In addition, if the most important traits selected for in spouses are those pertaining to attitudes, and attitudes are uncorrelated with some other highly heritable traits, such as anthropometric characters, then homogamous assortment for the former set of traits may reduce assortment for the latter set.

The lack of correlation between domains of heritable traits in fact raises another problem for genetic similarity theory. A history of homogamous assortment for traits in two domains would have the effect of producing genetic correlations (i.e., shared genetic influences) between the two domains as a result of genetic linkage which should in turn result in phenotypic correlations. If one result of assortment for genetic similarity were indeed the maintenance of "altruism genes" in the population, then one would expect to find significant genetic and phenotypic correlations between measures of altruism and other domains of heritable traits. Empirical support for the existence of such genetic correlations is currently lacking in the human behavior genetic literature.

The intent of the preceding discussion is to make the point that what Rushton calls more "proximate" causes of human behavior can have enormous influences in mediating the more distal genetic causes. This not only makes generalizing genetic findings from nonhuman species to humans problematic, but also creates doubts as to the relative importance of genetic similarity in human mate selection.

When is similarity genetic?

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When is similarity genetic? Monozygotic twins I know have the same genes and they look so similar that it takes time to tell them apart. Dizygotic twins are no more similar genetically than ordinary brothers and sisters, and their phenotypes can differ considerably. The phenotypic differences are just as genetic as the similarities; if we argue that the similarity arises from the probability that they share 50% of their genes with each other, then by definition they differ with respect to the other 50%. For people less closely related than sibs, the extent of genetic difference will be greater as their kinship weakens, until a point is reached when the genetic similarity of the least related pair in a population is vanishingly small. The coefficient of relatedness of the population as a whole will be somewhere between that of the siblings and that of the least related pair. If a custom such as consanguineous marriage prevails in a society, as it does in all societies where either cross- or parallel cousin marriage is preferred, then marriage partners will tend to have more genes in common with each other than with randomly picked individuals in the population. The reason will be the social institution regulating the choice of marriage partner, not individual choice. Indeed, a recent study (Bittles et al. 1988) found that 33.07% of 65,492 marriages in Karnataka, India, from 1980 to 1985, were consanguineous, and many of them were arranged by the parents, not the spouses.

Clearly, the basis of such parentally arranged cousin marriages cannot be solely the genetic recognition of similarity by the spouses themselves. From my own questioning of Indians in India, it appears that spouses may have a lot to do with choice of marriage partner. They might be responsive to similarities. However, parents seek to marry their offspring upward or at least on an equal level in the caste hierarchy. Evidence for this can be found every week in the Sunday issues of the *Bombay Times* and other major Indian newspapers.

What has genetics to do with this? In India, the caste system lays down stringent rules about who may and may not interact. To interact in the wrong way with a person from a lower caste or subcaste is to cause pollution, which can be removed only by ritual means. As a result, there are genetic differences between the castes. But like marries like, and the system is perpetuated. Thus marriage patterns have a lot to do with the distribution of genes in populations; in fact, they go a long way toward determining them.

But Rushton wants us to believe that the process works the other way around, that we are genetically programmed to detect similarity in others, to be attracted to them as friends and as marriage partners. Can there be any truth in this? I think there may be a little, but not anywhere near as much as Rushton suggests.

There are strong theoretical grounds for arguing that individuals who share higher than average numbers of genes should cooperate with each other when resources permit. Genes for cooperation should out-replicate genes for noncooperation in related individuals, assuming that they provide survival advantages. Likewise, genes for mating with cousins should out-replicate genes for mating with less-related individuals.

The problem with simple application of genetic similarity theory is the wording "genes for." This conventional phrasing arises from ethology and sociobiology and has progressively more meaning as we move down the animal scale to viruses, and progressively less as we move up it towards man. With man it may have no meaning at all. Interpreting "genes for" too literally leads to the Lumsden-Wilson theory of gene-driven epigenetic rules of culture. [See *BBS* multiple book review, *BBS* 5(1) 1982.] The alternative offered by Plotkin and Odling-Smee (1981) allows for behaviour to be organized at any of four levels: genes, epigenesis, learning, and culture, or between any combination of them.

Almost all of Rushton's evidence for genetic similarity as a basis for assortative mating can be explained as learning similarity or cultural similarity. He repeatedly demonstrates the correlation of mate selection with psychometric variables; for example, the "g" factor in intelligence. Quite apart from the debate about how heritable that factor is (and the other debate about whether it exists) [See Jensen: "Spearman's Hypothesis" *BBS* 8(2) 1985.], it is certainly multifactorially inherited. Now say that for the sake of argument we give "g" quite a high heritability, something like 50%; then that heritability relates to the flow of genes from parents to offspring. This tells us nothing about the similarity of genes between potential mates or spouses. Two highly intelligent people who fall for each other may have inherited different sets of "intelligence genes" from their parents. They are in fact similar because they share a phenotypic trait based on different genes in each of them. This objection can be raised to all of Rushton's data where multifactorial inheritance is involved.

Where multifactorial inheritance is not involved, Rushton's claims are strongest and so it is the blood group data that require most scrutiny. The data from the cases of disputed paternity are interesting but fail to validate Rushton's claims. What they show is that "males not excluded from paternity were 52% similar to their partners whereas those excluded were only 44% similar ($p < .001$)." But the women were presumably interacting sexually with *both* of the men, both were preferred mates at some time, and the only thing the results show is that the genetically closer couples were more fertile. Although this is interesting, it is not

germane to Rushton's argument, which concerns mate *selection*.

The only other single-gene data provided relate to friendships. Rushton's Table 5 indicates that actual genetic similarity as measured by blood groups was greater in 76 friendship pairs than it was in randomly selected pairs, and that this was not a result of social stratification. This result is as convincing as the associated probability ($p < 0.05$). Presumably it implies that the demonstrated genetic similarity underlies a wider genetic similarity, which has been in part at least responsible for the pairs' becoming friends. This is interesting and awaits validation from other studies.

I agree with Rushton that something remains to be explained. Ethnocentrism, far from being only a sociogenic or psychogenic phenomenon, is coming to be seen as rooted in biology (Reynolds et al. 1987; Shaw & Wong 1989). It may well be based on a tendency to prefer people who are perceived as similar to oneself. But in humans, it is not only behaviour but perception that is at one or two removes from genetic control. The similarity and difference we perceive are deeply imbued with cultural meaning. These perceptions have to be properly understood before we can have a satisfactory theory of mate selection in humans. On the theoretical grounds supplied by sociobiology, genetic similarity theory ought to be right, and it does seem to work for some species. But it may not work for all species, and the human evidence is not yet convincing. Anthropometric factors rarely show a between-spouse correlation of more than 0.25, and often there is no correlation at all (Coleman 1977). Correlations of such factors as height and age can be adequately accounted for by social factors in the majority of cases, although for height an ultimate sociobiological origin is possible. Mascie-Taylor (1987) found that for height and weight of the parents of children in the U.K. National Child Development Survey, the effects of social class, region, years of education, and age on the observed assortative mating were very small; assortative mating for height declined by only 0.015 and for weight by zero. Between-spouse correlations for life-style characteristics are highly significant (Harrison & Palmer 1981) but are unlikely to be based on genetic similarity.

When does natural selection favour assortative mating?

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I wish to make four points about Rushton's evidence for, and theory of, assortative mating. The first two points are theoretical. Natural selection will favour assortative mating under much the same conditions as it favours inbreeding or self-fertilization in hermaphrodites (Maynard Smith 1978, pp. 125, 139). Let us consider a gene for assortative mating, M_A, with an allele for random mating, M_R. When an M_A individual mates with another M_A individual, they influence each other's reproductive success. The condition for the spread of the M_A gene is that the total number of M_A genes propagated by the two individuals must exceed the total number of M_R genes that would be propagated by an equivalent two individuals. The crucial factor that determines whether the condition is met is whether the M_A individuals can breed normally with other individuals in the population. If they can, the assortative mating is an extra mating and the gene will spread; if they give up as many reproductive opportunities as they gain, the gene is neutral.

In the case of inbreeding, a female can be selected to "give" her brother a mating, if her brother retains his average reproductive opportunities elsewhere. His reproductive success is then increased, and because (in the absence of inbreeding depression) the female's reproduction is not altered, the total fitness of the incest gene is higher than that of its allele. The assumption of this argument, that the male retains his average reproductive possibilities elsewhere, requires that there be no paternal care. The argument will therefore not apply to humans. If males care for the offspring, then when a mutant male mates with his sister, he will not also be mating with other females.

In assortative mating in humans, because the assortatively mating individual will not simultaneously pair with other individuals, the M_A gene has no advantage. Suppose each individual produces n offspring. The selection is then between a pair of M_A individuals that produce n M_AM_A offspring, having $2n$ M_A genes, and two M_R individuals that produce n M_R- offspring through the male and another n M_R- offspring through the female, which again adds up to $2n$ M_R genes. The total number of genes propagated is the same. For the M_A gene to be favoured, it must produce *both* a normal number of M_A- offspring *and* the extra M_AM_A offspring as a result of assortative mating. Merely increasing the relatedness to one's own offspring is not favoured if it is exactly compensated, in terms of gene numbers, by the loss of other offspring. I therefore doubt whether Rushton's theory can be applied to humans.

My second theoretical point is that "genetic similarity" is likely to lead to selection for assortative mating only between kin. The M_A gene has to have a higher probability of being in a genetically more similar spouse; there must be (as Rushton appreciates) a genetic correlation between genes in general and the M locus. For kin, the gene loci are correlated; genetically more similar individuals are more likely to have the M_A gene. But for nonkin different loci will usually be randomly associated. Associations between the M locus and other loci would require high degrees of linkage disequilibrium, for which we have neither evidence nor argument (Maynard Smith 1978; Hedrick et al. 1978). Genetic similarity theory can apply for kin, but for nonkin the conditions are as implausible for assortative mating as they are for altruism (Grafen 1985). (By the way, Hamilton's "inclusive fitness" is not the same as "kin selection"; Hamilton 1975 explicitly differentiated the two; inclusive fitness is calculated for relatedness whether or not it is due to kinship.)

Now for the evidence. My first query concerns the blood data. Pairs were more similar in cases in which the male could not be excluded as the father. But surely this has to be true! The offspring will have the mother's genes anyhow; therefore if the male is more similar to the mother, it will be more difficult to exclude his paternity by this technique. Suppose males and females paired at random by blood group. In some pairs by chance blood would be more similar than in other pairs. Suppose the male fathers a constant proportion of the female's offspring, independently of the pair's blood. If we then apply the blood test to try to exclude the male as the father, it must automatically be more difficult in the pairs in which the male is more similar to the female, simply because the offspring have their mother's genes. How has this been taken into account?

Finally, I have a question about the association between higher heritability and higher degree of assortative mating. One simple explanation would be regional (Grafen, in press). Suppose that height is nonheritable. Then if tall individuals live in one place, and short ones in another, and we measure "heritability" and assortative mating for the whole area, we find that both are positive; if height is randomly distributed in space, we find neither heritability nor assortative mating. I also have a more complex explanation. In theory, assortative mating does not have much effect in the measurement of heritability, but that is true only if there is no gene-environment correlation. In quantitative genetics, of course, genes can be randomized across environments, and the theoretical result applies. But in humans environmental as well as genetic effects will be inherited. If there is gene-environment correlation, the offspring-midparent regression will reveal a spuriously high heritability,

because the environmental effects will appear to be "inherited" by the offspring (as indeed they are, but not through genes!). Does the mating system effect the variance due to gene-environment interaction, the V_{GE} term (the term is Falconer's 1981)? The question is whether an individual mates with other individuals that have the full range of environmental effects. In random mating, it will, and $V_{GE} = 0$. But in assortative mating, an individual with a high genetic and a high environmental effect will tend to mate with individuals that also have high genetic and environmental effects, and conversely for individuals with low genetic and environmental effects. The gene-environment correlation builds up: $V_{GE} > 0$. If not allowed for, this effect will spuriously inflate estimates of heritability in assortative mating and will generate an artifactual relation between the degree of assortative mating and the degree of heritability. I'm not arguing here that the "heritability" is really environmental, or that assortative mating automatically generates heritability; I'm concerned with a subtler effect of assortative mating on V_{GE}. How has this been taken account?

Why birds of a feather flock together: Genetic similarity?

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The social sciences provide ample evidence that like prefer like. Such preferences are shown in friendships and marriages; both spouses and friends are more alike in many traits than individuals picked at random. Ethnic group antagonisms remain a persistent human problem; as Martin Luther King concluded, each generation must negotiate anew its interethnic group relationships.

How could a genetically determined preference for genetically similar individuals evolve? Kin selection mechanisms can produce a preference for genetically similar kin – but the degrees of genetic relatedness among kin are much greater than they are among nonrelatives, whose genetic interests will diverge. Typical kin selection mechanisms are unlikely to explain the evolution of favoritism toward strangers.

Genetic similarity theory, however, may be supported by a hypothesis advanced by Alexander (1987) to explain altruism and indirect reciprocity in human groups. Alexander proposes that intergroup competition (e.g., raids, warfare) in humans led to selective pressures favoring groups showing a high level of intragroup cooperation and altruism. Because group members typically share more genetic relatedness than outsiders, this kind of selective mechanism may have favored reproductively those individuals who directed altruism toward individuals who were genetically like themselves but not necessarily kin (especially if genetically dissimilar individuals were to abandon one group for a rival group closer genetically to themselves).

Nonetheless, a picture of evolutionarily selective forces is hard to draw. A preference for (genetic) similarity can conflict with other choices that may maximize fitness. For example, why not choose a mate with the best, fitness-maximizing traits, rather than one who is most similar (e.g., the most physically attractive, the brightest, the not overly shy). Could not unattractive, not very bright, shy individuals have greater reproductive success with a mate possessing heritable traits unlike their own? Mate matching may be the result of "slim pickings" among remaining potential partners, after those individuals with the more desirable traits have already paired. The apparent similarity of "poor" quality mates, then, could be a byproduct of a process in which everyone prefers mates with the most desirable qualities. Moreover, it may be in mothers' reproductive interest to deceive men about the paternity of illegitimate children. This selection pressure would tend to degrade heritable mechanisms that detect genetic similarity.

Because we lack a record of human evolutionary history, it is impossible to know how the competition among different selective forces has played out. For this reason, direct investigations testing Rushton's genetic similarity theory are required. For this purpose, behavioral genetic research designs may be helpful.

One aspect of behavioral genetic research was used in the target article – comparing spouses' and friends' trait resemblances with the same traits' heritabilities. This test of the theory, however, presents several difficulties. Heritability estimates tend to be unstable from one study to another. We need more replication, for example, of the heritabilities of single-attitude items before we can be confident that the rank order of item heritabilities is stable. More important, however, is the lack of relationship between heritability and the number of contributing genes. A trait influenced by few genes could have a high heritability; one influenced by many genes, a low heritability. Would not genetic similarity theory predict greater assortment on the latter kind of trait? If so, what is needed is not measures of heritability but of total genome involvement in a trait. Such measures do not exist. An alternative that may be practical would be better measures of genetic similarity between individuals, based on advances in molecular genetics.

Behavioral genetic designs offer other means of testing genetic similarity theory. For instance, the theory can be tested by cross-correlating the traits of the *adult friends* of monozygotic (*MZ*) and dizygotic (*DZ*) twins, or by calculating these friends' genetic similarities. If each twin possesses a mechanism for preferring genetic similarity in friends, then greater genetic alikeness should be found for the friends of *MZ* twins than for those of *DZ* twins because of the different degrees of genetic relatedness among twin types (r_g *MZ* = 1.0; r_g *DZ* = 0.5). A similar method can be applied to twins' spouses.

In individuals, a trait may correlate to some extent with the same trait measured in a friend. With a behavioral genetic design this "assortment" correlation can be analyzed for its genetic and environmental components. According to genetic similarity theory, a genetic component should be a part of "assortment" correlations.

In the target article Rushton mentions several possible mechanisms through which a preference for genetically similar individuals may be achieved: innate feature detectors, phenotype matching, familiarity, and location. Of these mechanisms, phenotype matching seems particularly suited to explain the close emotional relationships that develop between *MZ* twins, even when raised apart. Such twins look and behave much alike – this may be the basis for their strong mutual attraction and mutual altruism. Phenotype matching detects easily 100% genetic identity.

Nonetheless, not all *MZ* twins form close friendships: In some pairs the individuals actively dislike one another. Such a *range* of responses suggests that the preference for phenotypic similarity may itself be variable. If competing selective forces have influenced the phenotypic similarity detection mechanism, then variation in its expression may be heritable and genetically additive, which can be investigated with behavioral genetic research designs. Scarr (1981) reports that the personality trait of authoritarianism is heritable. Although this trait would seem to be a candidate for intolerance by anyone different, authoritarianism scores are confounded with intellectual abilities (Scarr et al. 1981, p. 408). Other approaches to assessing this preference should be investigated. Young children display a preference for similar others (Sigelman et al. 1986). Behavioral genetic investigation of a variation in childhood preferences may indicate whether a preference for phenotypic similarity is genetically based.

In sum, genetic similarity may explain some part of variation in mate and friendship choice, if such effects are real, they are

one part of a system of competing influences, not the whole story. Whether such a preference for genetically similar strangers has evolved during human prehistory is difficult to establish. Behavioral genetic research designs, however, may be able to verify or to disprove some important predictions of genetic similarity theory.

How not to explain psychological phenomena

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At the outset I must admit to a considerable degree of discomfort in addressing this target article. When I was preparing this commentary (early February 1989), Professor Rushton's name suddenly began to appear in the headlines of my morning paper and to be mentioned in radio and television newscasts. Rushton's recent presentation at the American Association for the Advancement of Science conference was reported by the wire services and elicited a great deal of strident commentary, at least in Canada (e.g., Strauss 1989). Much of this commentary focused on Rushton's contention that there are racial differences in intelligence. That, of course, is not the focus of the present target article; the question of race is only hinted at in section 9. The reason for my unease is that the debate over Rushton's work has now left the confines of academia and an entirely different criterion is being applied to assess the validity of this material by the mass media, namely, its newsworthiness. In particular, journalists have been quick to quote eminent geneticists who have denounced the racial theory of intelligence but, owing to the nature of media presentations, the issues have not been dealt with seriously or at length. The publication of this paper in *BBS* along with critical commentaries should go some way toward clarifying certain issues raised by Rushton's work.

Notwithstanding the public brouhaha, I should like to focus on the implications of Rushton's paper for psychology. In particular, I believe that the author has seriously misconstrued what a psychological explanation is and this in turn has serious implications for his functional analysis of altruism.

Section 8 contains a brief digression that, on the face of it, deals with the problem of explanation. According to Rushton, readers' reluctance to accept a genetic component of the "similarity-attraction" link may be due to the preference for more proximal levels of explanation such as those provided by cognitive or social learning models. Such preferences do not rule out distal explanations of the sort provided by Rushton; they are all compatible if we view them in a time dimension, which the author does in Figure 1. In a cryptic note in the figure caption, Rushton claims that "when explanations move from distal to proximal, controversy does not ensue, whereas the converse is not always true."

How are we to interpret this notion of explanation in view of Rushton's own theorizing? He has chosen to explain altruism via the proximal to distal route, but what possible role does a proximal explanation have in genetic similarity theory? Is a proximal explanation superseded by a distal explanation? Is a distal explanation a stronger or more scientifically respectable explanation? Although Rushton does not address such questions in his article, there are some indications that he has certain preferences.

Rushton's statements about the data he presents indicate only that genetics may *influence* such decisions as choice of friends, or that "both friends and spouses choose each other *partly* on the basis of genetic similarity" (emphasis added). Nevertheless, Rushton claims that "purely environmental theories" of similarity attraction are untenable. Purely environmental theories include, according to Rushton, an examination of histories of socialization and enculturation. Whatever might be meant by a "purely" environmental theory, the processes of socialization and enculturation do seem to be more than environmental. Only some limited set of behavioral views (now largely historical) would support a notion of strict environmental determinism. Rushton appears here to do what he specifically claims he is not doing, namely, to oppose a proximal with a distal explanation. But his proximal explanation is the proverbial straw man.

Rushton's argument that the social psychological view actually predicts a negative correlation between item heritabilities and degree of similarity betrays a peculiar misunderstanding of a social psychological explanation. I am aware of nothing in any social psychological view of altruism that predicts a relationship between heritabilities and similarity. In making this claim Rushton asks social psychological theories to make predictions that they are not capable of making. Again, it betrays a penchant on Rushton's part to see biological and psychological views not as complementary, but as opposing.

The notion of epigenetic rules in social development (section 8.1) leads Rushton to claim that "people create environments maximally compatible with their genotypes." In support of this argument he cites, as one example, research on the effects of television and within-family studies of delinquents. Aggressive siblings identify with aggressive characters and do not view the consequences of aggression negatively; nondelinquent siblings can be distinguished from delinquents by intelligence and temperament. Rushton concludes that siblings that differ in temperament and intellectual variables will seek out different social environments."

But note that these findings are amenable to numerous explanations (e.g., cognitive, social learning). Environmental preferences do not need epigenetic rules as an explanatory device. Rushton's claim that such rules are useful in ordering hypothetical levels in Figure 1 is unwarranted. He states that "any distal 'purpose' of the genes must necessarily be mediated by proximal mechanisms." What is the nature of this mediation? There are no answers.

The claim that proximal mechanisms are included in the analysis is quickly belied in section 9. In the space of a few paragraphs the potential effects of epigenetic rules are said to account for such things as charities and hospitals, academies of learning, institutes of war, delinquent tendencies leading to social disorder, ethnocentrism, xenophobia, patriotism, and political ideologies.

One issue that confuses at the outset revolves around the use of single terms to group vast ranges of animal behavior and human action in a single category. The construction of a hospital or other charitable institution is simply not the same as the honey bee's loss of its sting no matter how complex the sociobiological theory used to tie the two together. Altruism is not a natural category of analysis or a single event in nature but a social construct imposed on a variety of semantically related, but not identical, phenomena.

The major problem here stems from Rushton's failure to distinguish between functions (that is, survival value) and causation, or mechanisms. It was Tinbergen (1963) who applied these distinctions to explanations of animal behavior (but see Klama 1988 for a discussion of their applicability to sociobiology). In short, to have explained how something has come about is not to have explained what that something is. To ask questions about the reproductive success stemming from a set of behaviors is not the same as asking questions about the mechanisms by which those behaviors become manifest in current practices.

One very important implication of the distinction between causation and survival value is that whereas all characteristics of organisms may be said to have a cause, this is not true of survival value. Grief is a case in point. Rushton argues that grief intensity is proportional to the degree that parents perceive their de-

ceased child as similar to them. Yet bereavement may not provide a selective advantage to the bereaved. This point has already been made by Archer (1988) in relation to Rushton's work; the reader may follow the arguments there. My point is simply that there is no evidence currently available that supports the argument that grief does, or does not, increase inclusive fitness (cf. Parkes 1972). Rushton merely assumes that all of the human behaviors, human institutions, and contemporary social phenomena he addresses in his article must provide some advantage to fitness. In most cases, and grief is but one, this assumption is nothing more than a bald assertion.

In this respect the conclusions about aggression drawn by Klama (1988) can be applied with equal force to the problem of altruism. If we are not dealing with a unitary natural event, then arguing that altruism is distally caused but proximally mediated is not sensible. Nor can we locate altruism in some set of genes. Rather we must qualify our discussion of altruism by referring to specific animal populations and specific environments.

This argument can be illustrated by considering whether building hospitals and giving to charity are intelligible acts. Whatever the merits of a functional explanation of animal and human behavior, one of its criteria is not intelligibility. As Robinson (1985) has argued, we do not impose the criterion of intelligibility on causal relations of this sort. The structure of DNA could have been other than what it is, and had this been the case we should not have been surprised. But the act of building a hospital must be at once goal-directed and tied to past intelligible actions and beliefs. This is frequently referred to as the distinction between causes and reasons; that debate need not detain us here, however. The point is, actions are not intelligible as purely natural events. No amount of sociobiological tinkering will allow us to explain the founding of, say, St. Joseph's Hospital in Rushton's London, Ontario. At some point he has to appeal to the structure of women's religious orders in the Roman Catholic Church and the state of health care in Canada in the late nineteenth century. At most, genetic theories provide permissive conditions (Robinson 1985), but they do not give us an explanation (scientific or otherwise) of what had occurred among persons and social structures. Without some account of agency or intentionality, human acts of altruism are unintelligible.

At some level Rushton appears to recognize the problem. How else could we explain the statement acknowledging that "because language represents a powerful new way to distinguish kin, it is more difficult to demonstrate that humans can recognize kin in a way that parallels kin recognition in nonhuman animals." In what way can we say that language distinguishes kin? Modern languages have a vocabulary that distinguishes kin from nonkin, but presumably that is not what is meant here. Vocabulary is meaningless without our recognition that the sense we attach to such words requires us to be intelligible and intelligent creatures. It is not language that distinguishes our kin for us; we do. And we do so as human agents.

I am not saying that we should expect agency to figure in a functional analysis; that would be inappropriate. Questions of fitness do not focus on the intentions of human persons. The point is that the lack of a clear understanding of proximal and distal explanations evident in Rushton's article leads him to force each and every instance of "altruism" into his Procrustean bed of genetic similarity. The result is a psychology that necessarily distorts its subject matter to fit a theory that is itself shot through with untested and untestable assumptions.

Finally, genetic similarity theory is not compatible with standard accounts of human history. If organisms can identify other genetically similar organisms, then they may behave as altruistically towards them as they would towards their kin. Thus Rushton claims that human phenomena such as patriotism can be explained by genetic similarity theory because the genes of the group, and not necessarily those of kin, are benefited. If this is the case, then Rushton must explain the rather late arrival of patriotism in human history. Certainly the concept of the nation-state as something for which one can feel anything approaching loyalty is a modern, that is, postmedieval, phenomenon. Furthermore, how does genetic similarity theory account for the bloodshed, documented in human history, peoples fighting groups that were genetically similar in some periods, but not others? Greek city-states fought each other as a matter of course, but it would be considered aberrant if Athens were to take up arms against its neighbors today. Only in recent centuries have internally consolidated nation-states eliminated internecine battles among their populations. As Weber (1956) observed some time ago, only modern nation-states retain the legitimate right and monopoly to use force. In order for genetic similarity theory to account for this it would have to argue that the tendency to identify and exhibit altruism towards genetically similar nonkin is a very recent and unstable one in human evolution, or one that had been masked by other tendencies throughout most of human history. For a functional explanation, that would be most unusual indeed.

Kin selection, genic selection, and information-dependent strategies

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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

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Heritable phenotypes and ethnicity

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For years, Rushton has been prodding me to go beyond my 1981 statement on the biological basis for ethnocentrism (van den Berghe 1981). His suggestion in the target article that fear of treading on the ideological minefield of race and racism has made intellectual cowards of us all was perhaps the necessary catalyst for the present response.

Basically, Rushton suggests that genetic narcissism – a kind of generalized "green beard effect" (*sensu* Dawkins 1976, p. 96) – is an important proximate mechanism of sociality. He claims that, although genetic similarity selection would often replicate the effects of a hypothesized kin selection, it is both a wider, more general explanation *and* one that yields a more accurate model of cooperative behavior than kin selection alone. I must confess I was initially swayed by Dawkins's argument against the plausibility of a green beard effect spreading: The genome would be invaded by mimics or spoilers. Now it seems to me (though I would be hard put to demonstrate why) that genetic similarity is equally plausible with a somewhat narrower nepotism as a sociality mechanism.

Let us apply this model to racial and ethnic relations. In 1981, I suggested that ethnocentrism and racism were explainable as cases of extended nepotism. One of the main objections to this view is that the relatedness of fellow ethnics is too distant (and often too fictive) for such a greatly diluted nepotism to produce the strong (and often situationally variable) effects associated with ethnocentrism and racism. I also attempted to account for the choice of *markers* of group membership, cultural and physical, leading to the formation of ethnic as distinguished from "racial" groups. Whatever markers predict genetic relatedness best (i.e., most reliably, easily, quickly, and cheaply) in a given situation, I suggested, are likely to be used by flexibly opportunistic, selfish maximizers like humans. In most historical situations of neighboring and relatively stable groups, cultural markers (notably of speech) are more reliable than physical markers (where within-group variance is often greater than between-group variance). After large-scale, long-distance migration across physical phenotype gradients, such features as skin color, hair texture, and stature can become efficient markers of group membership, and often do.

My 1981 formulation suggested a rather complex and flexible mechanism of ethnic group recognition and marking. The rule would, in effect, be: Pick any criterion that correlates highly with nature membership in the group (such as linguistic accent, since it is difficult for a postpubertal newcomer to fake).

There are at least two alternative epigenetic rules that could explain ethnocentrism and racism. The first is a rule of undiscriminating narcissism: Pick "your kind of people," that is, people who both look and behave like you, even if the likeness can easily be faked (e.g., grooming or clothing style). The second is, of course, genetic similarity, which is genetically discriminating narcissism: Pick people who are like you in traits with high heritability.

The empirical question is: Which of these three hypothetical rules best fits the evidence? As with most human behavior, a clear answer is difficult to find because of both poor data and confounding factors. For instance, assortative mating is confounded by female hypergyny and male promiscuity, which can produce a great deal of anisogamy (e.g., between masters and slaves across "racial" barriers). Let us, however, try.

There is some evidence of sociality based on undiscriminating narcissism (e.g., based on cultural fads such as style of music, hairdo, or clothing), but these associations tend to be ephemeral and not to crystallize into stable affiliations such as those characterizing both ethnicity and race. Precisely because these forms of likeness can be so easily assumed, they are unlikely to become the main markers of ethnicity. Even relatively open and assimilative ethnic groups police their ethnic boundaries against invasion by strangers.

Of the other two rules, which explains ethnic affiliation better? I think the answer may well be that both do in combination. Ethnicity has both a primordial and an instrumental dimension. It is both rooted in descent and manipulated for gain. Thus, learned, acquired cultural traits with low heritability can be and often are used as ethnic markers. The French government's attempt to create a superethny of *francophonie* and the Spanish counterpart, *hispanidad*, are examples. Of course, not all such manipulative creations are successful. Some criteria seem to have more staying power than others, and the ones with high heritability appear to have an edge. When the chips are down, Frantz Fanon, Aimé Césaire, and Léopold Senghor are not really French, even though they wrote "better" French than 99% of "real" Frenchmen are capable of producing. They have the "wrong" genes and do not conform to what Hoetink (1967) called the "somatic norm image" of a

Frenchman. Hoetink suggested that members of an ethnic group carry a mental image of what a fellow ethnic should look like and exclude phenotypically discrepant individuals.

Thus, the ethnic labels "Canadian," "English," and "American" evoke in most members of these groups a "Caucasoid" somatic norm image. Phenotypically discrepant individuals with, say, African or Asian ancestry continue to be hyphenated members of these groups, despite the fact that in some cases their claims to ancestral nativity in the society are more valid than those of members whose physical phenotype conforms to the somatic norm image. Blacks are an obvious case in point. On the average their ancestors have been in the United States much longer than those of whites. The converse is also true: The claims of long-term white residents in Africa to be Africans have also met considerable resistance, both in Africa and in other nations.

However – and the qualification is a crucial one – the dominance of heritable physical markers over learned, cultural ones in defining ethnic membership will appear only in situations where intergroup variance in these physical markers is greater than intragroup variance. Such situations, which typically result from recent, large-scale, long-distance migration, have become common in the last few centuries but are still far from universal. Scandinavians, for example, will be hard put to distinguish Norwegians from Swedes by heritable phenotypes. Even in these situations, genetic similarity may still be the basis of preferential behavior, but it cannot become a workable marker of ethnicity or race. Thus, among both Norwegians and Swedes, left-handers might be mutually attracted, but left-handedness could only become an ethnic marker in populations in which the distribution of the phenotype is significantly associated with preexisting groups.

The evidence tends to show that whenever intergroup variation in heritable phenotypes is greater than intragroup variation, such phenotypes are used as group membership markers. Covariation between genetically heritable and nonheritable markers (or, better, between markers of widely ranging heritability) empirically confounds the picture and leads to complex systems of multiple markers. However, there is much observational evidence that, when put to a discriminating test, the more heritable markers tend to "win" over the less heritable ones in situations where heritable markers significantly correlate with ethnicity. Given half a chance, racism (in the sense of discriminatory behavior based on heritable phenotypes) will develop. Conditions are ripest for racism when hitherto biologically isolated populations enter into large-scale contacts (e.g., through conquest or immigration), yet maintain barriers of endogamy.

Unfortunately, as in many aspects of human behavior, most evidence is anecdotal, and more rigorous tests of genetic similarity preference are needed. The field of ethnic relations is particularly suitable to such hypothesis testing because the range of empirical situations is sufficiently wide to allow control of key variables.

The role of genes in genetic similarity detection

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Resistance to sociobiological analyses, especially in explanations of human behaviour, has been fierce and extreme (e.g., Lewontin et al. 1984). Much of the blame must go to sociobiologists themselves, in that careless and overspeculative claims invite serious misunderstandings about what the core theories and concepts actually imply. Given the provocative nature of some aspects of Rushton's thesis, it is a pity that he is sometimes less than rigorous. For example, in not defining very explicitly the sense of altruism that makes its evolution appear paradoxical; his biological use of the term also differs from how it is used in other disciplines. The significance of "identity by descent," in references to proportions of genes shared by kin, might also have been highlighted more in an attempt to clarify the still pervasive misunderstandings of kin selection discussed by Dawkins (1979).

Although the basic idea is admittedly not new, Rushton's claim that genetic similarity detection underlies but extends beyond kin selection is – if sustainable empirically – another important sociobiological contribution to the explanatory power of evolutionary theory. It is important, however, not to appear to be overselling a promising theory. Kitcher (1985) [see also *BBS* multiple book review, BBS 10(1) (1987] is one of the fairest and the most trenchant critics of premature and speculative "pop" sociobiologizing; he insists that a convincing Darwinian history requires very thorough analyses before a trait can be attributed to fitness-optimizing selection for facilitating alleles. Evolutionary origins, functional adaptedness, proximate causation, and so on must each be approached with the aim of identifying and discriminating empirically between rival explanations at each level. In general, Rushton appears to be alert to such requirements, even if genetic similarity theory cannot yet claim to be comprehensively elaborated and fully supported for our own species (but see Archer 1988).

Lewontin et al. (1984, p. 235), viewing sociobiology through their own ideological spectacles, find that the "central assertion . . .is that all aspects of human culture and behaviour . . . are coded in the genes and have been moulded by natural selection." If genetic similarity theory is not to be taken as another piece of ammunition by such critics of genetic determinism and adaptationist assumptions, it needs to be framed so as not to exaggerate its dependence upon strong or implausible assumptions about the roles of genes themselves. For those of us introduced to the theories of Hamilton and Trivers by the "Bristol school" of evolutionary thought (e.g., Crook 1980), it has been evident that given "some kind of feedback from the evolutionary goals of behaviour to the actual behavioural decision process, . . . it is possible for behaviour to be geared to evolutionary ends without being genetically programmed in any meaningful sense" (Dunbar 1987, p. 54). Thus for kin altruism to be selected, what matters is that "genes for altruism" make a relevant developmental difference to an organism's phenotypic distribution of social aid amongst others. Reliable transmission of nepotistic traits between generations is what counts. Particularly for advanced primates like ourselves, the elaboration of very simple discriminative mechanisms by experiential learning during socialization could suffice. Such amplification of minimal direct and specific gene effects would also allow for the evident flexibility of adult human altruism, through which we can become capable of transcending fitness constraints on our social behaviour (Vine 1983).

Rushton does not explicitly espouse more rigid and specific genotype-phenotype linkages, and indeed the epigenetic rule is a step away from simplistic genetic determinism. Although he acknowledges several proximate means by which humans might achieve functionally adequate genetic similarity detection, he seems to lean towards the "strong" version of the theory (Rushton et al. 1984), which asserts that we innately discriminate and prefer those persons who show signs of genotypic similarity to ourselves. Insofar as innate feature detectors rely upon "green beard" forms of genetic organization, they remain theoretically improbable and lacking is unequivocal evidential support (Dawkins 1982). Dawkins's "armpit effect" mechanisms can achieve genetic similarity detection by following rules that depend on exposure learning; the "weak" version of the theory appears thus far to be more plausible.

The evidence cited by Rushton leaves little doubt that genetic similarity detection does occur between related animals; but whether we can rule out at least a minimal amount of learning from any experiment seems doubtful. Data for humans is bound to be ambiguous on this point. Nevertheless, the results on assortative mating, sibling favouritism, and same-sex friendship certainly give support for the operation of some form of genetic similarity detection in a number of cases – and in directions with plausible, if not typically demonstrated, fitness benefits. Yet an incautious, if understandable, enthusiasm for the power of genetic similarity theory appears to lead Rushton to cite as supportive too much data that could have other explanations. Thus, assortative mating explanations of between-spouse correlations in attributes such as criminality, alcoholism, and affective disorders will scarcely be parsimonious unless there is good evidence for prepairing similarities. At one point Rushton even comes close to making the fallacious claim that because "assortative mating for similarity of the more genetically based" measures fits the theory, this strengthens his interpretation for other measures that correlate appreciably.

I remain sceptical about assumptions of substantial genetic similarity in cases where any similarity between partners in relationships is psychological, rather than physical. And for within-family similarities, the uncertainties, ambiguities, and even arbitrary decisions about how to deal with a number of theoretically distinguishable sources of variance when estimating heritability are notorious. Measures and circumstances of comparison rarely justify confident inferences about any one source in isolation. Gene-environment covariance can arise simply because individuals are treated the same because of similar physical attributes as well as similarity on the trait in question. Both are "potential sources of artificial inflation of h^2 estimates based on either r_{MZA} or kinship procedures," (Taylor 1980, p. 70), as are covariations such as those that result from individuals' active attempts to shape their own environmental conditions in line with prior gene-based dispositions. Some of Rushton's coworkers, such as Eysenck, are committed to approaches that yield very high estimates of direct genotypic influences on phenotypic traits of a psychological kind.

Finally, I remain uneasy about hypothesizing any very specific and rigid genetic determination of ethnocentric preferences. The caution that Rushton detects amongst contributors to Reynolds et al. (1987) is surely wise so long as predominantly cultural explanations fit the facts (Vine 1987). Rushton's claim that manipulated altruism renders patriotic fervour "an anomaly for evolutionary biology" appears to be based upon an excess of genetic determinism and faith in invariant fitness optimization. Although I acknowledge Rushton's open-minded alertness to the objections, I feel that here, and when he flirts with revamped group selection notions, he risks discrediting the fundamental insights of genetic similarity theory.

Science or prejudice?

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Why BBS? Rushton invites commentary on his speculations by a wide spectrum of academics, yet he seems to pay no attention whatsoever to previous objections to sociobiology in the pages of this journal or elsewhere. *Vaulting Ambition*, by Kitcher (1985), the subject of peer commentary in *BBS* (1987), provided an incisive critique of Wilsonian sociobiology, and most of his points apply directly to Rushton's article. Yet Rushton proceeds as though those words had never been written. His article is presented as a development *within* a narrowly conceived sociobiology, and he cites the works of like-minded colleagues whom he considers authorities on everything from marriage and child abuse to patriotism and war.

Where's the model? Much is vague and informal in this article. Rushton talks about gene frequencies, differential heritabilities, and relative strengths of conflicting tendencies but never does he commit himself to a quantitative model that would allow a decisive evaluation of his ideas. Population genetics is supposed to be a precise discipline, yet we find no equations, no path diagrams or anything of the sort in this article. Is Rushton hoping the *BBS* commentators will do the mathematical thinking for him? Much more is required before this can be regarded as a scientific hypothesis.

When does the rigor begin? Sloppiness abounds. Phenotypes are said to be "the product of multiple alleles" (section 7.1), though the context implies Rushton really means multiple genetic *loci*. The meaning and consequences of multiple allelism at a locus and multiple loci heredity are quite different. He argues that mechanisms for both detecting and producing features of the organism are "genetically coupled" (section 7.1), which implies genetic linkage, when he apparently means pleiotropic gene action or genetic correlation. Again, the disparate effects of linkage and pleiotropy in a model hardly make them synonyms. Rushton claims that "it is advantageous for a single gene to work for copies of itself" (section 7.1), having earlier claimed (section 6.1) that too much genetic similarity is bad and leads to inbreeding. He imbues the little gene with a narcissistic affinity for its own kind, but instructs it not to do what comes naturally if this would contradict the theory. If such a contradiction were somehow integrated into a comprehensive and nonlinear quantitative model, one might think of it as part of science and call it dialectic; but lacking this, the conflicting claims appear to be equivocation.

Are the genes communist? Rushton maintains that "genes maximize their replication by benefiting *any* organism in which their copies are to be found" (section 10) and claims that this property is broadly representative of "the overall genome" (section 7.1). It just so happens that techniques of molecular biology indicate there are from 60,000 to 200,000 structural genes in the human cell nucleus coding for distinct proteins, but only 2,208 loci have been validated, many of which are known from very few individual cases and cannot be considered polymorphic (McKusick 1988). Considerably less than 10% of the loci in the human genome are polymorphic at the level of polypeptide gene products. What humans have in common genetically vastly exceeds their differences; hence genetic similarity theory would seem to require that those genes at fixed loci should do their utmost to guarantee the propagation of all people, regardless of ancestry, and that, being most numerous, they should prevail over their vacillating and sparse neighbors. Rushton's views about patriotism, xenophobia, and war simply do not follow from his premises.

Polygenic favoritism in the family? In section 6.2 Rushton uses a fictitious example as the basis to estimate that a child could have 60% genetic similarity to its mother and 70% to its father. This is absurd. First, over 90% of the loci will be fixed in the population. Second, at loci with many alleles in the population, the parents will usually not have any alleles in common. If the two parents have four different alleles, the child will always have the same 50% genetic similarity with both parents. Third, genetic similarity to the mother and to the father at a locus can differ only when the parents have two or perhaps three alleles among them. If there are two alleles in the population, then three genotypes (AA, Aa, and aa) and nine mating combinations of male and female are possible. In five of these combinations, including Aa × Aa, the genetic similarity of a child to it mom and dad must be identical. If the frequencies of the alleles are p and q, and assuming the population is in Hardy-Weinberg equilibrium, a little algebra reveals that the expected genetic similarity of a child to its mom is $100(1 - pq)$ and that the variance is

$100^2\ pq\ (\frac{1}{2} - pq)$ for the one locus. If there are N such independent loci in linkage equilibrium, the expected mean genetic similarity across the N loci is also $100\ (1 - pq)$ and the 95% confidence interval for the genetic similarity to mom is

$$100\ (1 - pq) \pm 1.96\ (100)\ \sqrt{pq\ (\tfrac{1}{2} - pq)/N}.$$

Likewise, the 95% confidence interval for the *difference* between genetic similarities to mom and dad is

$$0 \pm 1.96\ (100)\ \sqrt{pq\ (p^2 + q^2)/N}.$$

If the alleles are equally prevalent ($p = q$, the maximum variance case) and there are 5,000 such loci, the interval for genetic similarity to mom is from 74.931% to 75.069%. If there are about 100,000 structural genes in the human genome, of which 90% are fixed for one allele, 5% are highly polymorphic, such that the parents usually have four alleles, and 5% have two equally frequent alleles, the 95% confidence interval for overall genetic similarity of a child to its mom is 96.247 to 96.253, and the interval is $0 \pm 0.049\%$ for the *difference* in genetic similarities to mom and to dad. Rushton's figures of 60% for mom and 70% for dad would be plausible only if the mating were between a man and something much more remotely related than a chimpanzee (Jones 1986). He simply conjures up these numbers, rather than deriving them from established facts.

Conclusion or sentiment? The premises in Rushton's article are incompletely specified and major components of the nascent model are grossly at variance with facts. His article provides yet another example of how "Neo-Darwinian sociobiology can be used to give pseudo-scientific support to what are actually mere prejudices" (Saunders 1988). Rushton's argument for a genetic cause of ethnic conflict and xenophobia (section 9) must be regarded as an asseveration of personal belief rather than a tentative scientific conclusion. [See Wahlsten: "Insensitivity of the analysis of variance to heredity-environment interaction" *BBS* 13 (1) 1990.]

Sociobiology, sociology, and pseudoevolutionary reasoning

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Genetic models provide the key to resolving many of the apparent paradoxes of social evolution. By delineating the genetic equivalence of collateral and descendent relatives with respect to natural selection, Hamilton (1964) revolutionized biological perspectives on sociality and launched the field of sociobiology (Wilson 1975). Witnessing the transformation of Hamilton's ideas and their application to the study of human societies is at once heartening and disquieting. Rushton's thesis, that a preference to interact with genetically similar individuals serves as the common thread weaving together diverse aspects of human sociality, is particularly troubling. Although "genetic similarity theory" considers sociological questions in terms of genetic models, it does so in the absence of any rigorous evolutionary framework.

Sociality entails both costs and benefits (Alexander 1974). If the potential gains (e.g., from cooperative feeding or defense) are greater than the associated risks incurred (e.g., from spread of disease or from interference behaviors), social systems should flourish; otherwise they should break down. With inclusive fitness (Hamilton 1964) as the currency with which to evaluate these costs and benefits, the conditions under which sociality can evolve become more favorable. Gains no longer need to exceed the risks computed for each and every individual, because fitness effects on the relatives of these individuals are also considered (see Grafen 1982; 1985; Hughes 1988). Individuals may be selected to act "unselfishly" or even "altruistically" if their relatives thereby benefit. Kin selection can promote the evolution of group living, which in turn makes possible increased opportunities for reciprocity (Axelrod & Hamilton 1981).

These arguments seem to imply that if sociality is advantageous, then the formation of social groups or alliances with close genetic relatives is even better. But this is not invariably true. Genetic similarity may lead to altruism, but it can also translate into greater overlap in resource utilization patterns. Under these conditions, competition among close kin will be intensified compared with that among more distantly related individuals (see Waldman 1988). Genetic similarity can thus have deleterious as well as beneficial social consequences. Indeed, simply because of their proximity in social groups, kin are often principal competitors (Armitage, in press). Field studies have frequently revealed that close kin compete most severely with one another, even killing each other's offspring (e.g., Hoogland 1985).

The simple expectation that individuals who choose genetically similar mates should somehow do better than those who mate randomly betrays confusion about the biological meaning of altruism. By mating with a close relative and therefore restricting its breeding opportunities (in a monogamous society), individuals effectively deprive both themselves and their mates of additional collateral relatives. An incestuous individual thus leaves exactly as many copies of its genes as an outbreeding individual (Dawkins 1979). Mating with genetically similar individuals, if it resulted in superior offspring, might still be selected. But the preponderance of available evidence on the viability of offspring of inbred and outbred matings suggests that inbreeding, not outbreeding, incurs heavy genetic costs (Charlesworth & Charlesworth 1987). Only when the costs of inbreeding avoidance (dispersal or kin recognition) exceed these genetic costs should inbreeding be evolutionarily favored (Waser et al. 1986). Breeding with nonrelatives might conceivably disrupt coadapted gene complexes, in which case mating with an individual of some "optimal" intermediate degree of relatedness would be advantageous (Shields 1982; Bateson 1983a). Nonetheless, data supporting this idea are meager (Barnard & Fitzsimons, 1989; Bateson 1982; Waser & Price 1983); indeed they are virtually nonexistent for natural animal populations.

Compatibility between partners may well be an important component of reproductive success (Rowley 1983). Compatibility among males may similarly enhance the effectiveness of their alliances ("friendships"). In either case, however, (1) the benefit is more likely to be somatic (result in some material gain for the young or for the group members) than genetic, and (2) compatibility need not be based on genetic similarity even if it is correlated with it. Much of the support mustered for genetic similarity theory relies on estimates of heritability. Yet these data are suspect with regard to the theory, because more often than not, they reflect the extent to which traits are transmitted from parents to offspring, whether genetically, culturally, or environmentally, rather than the extent to which individuals' phenotypes are determined by genes transmitted from their parents.

Evidence that monozygotic twins are more similar than dizygotic twins in their views on the death penalty, white superiority, and water fluoridation (Martin et al. 1986) hardly demonstrates that these traits are genetically determined. That genetically identical individuals should develop similar personae is not surprising. Convergence in the personal views of monozygotic twins might be fostered by the treatment they receive from their parents, teachers, and peers; by the manner in which their phenotypic resemblance influences their interactions with one another; and quite possibly by some similarity in temperament that may indeed be genetically influenced. Beyond this, any claim that the development or acquisition of opinions such as those presented in Rushton's Table 4 is genet-

ically specified – or that variation in these traits is overwhelmingly attributable to additive genetic effects (Martin et al. 1986) – is preposterous!

Genetic similarity theory purports to go beyond kin selection in predicting that individuals who share alleles should act altruistically toward one another regardless of common ancestry. Biologists have largely ignored this revelation because the idea can be found in Hamilton's (1964) original paper (see pp. 24–25). A valid theoretical distinction exists here, but is it important in the real world? Perhaps, but only in special circumstances; for example, if discrimination were based on a limited number of genes. Sea squirts act in an altruistic fashion toward neighbors with whom they share a histocompatibility allele. Whether the allele is inherited from a common ancestor is unimportant, as is the overall genetic relationship between the individuals (Grosberg & Quinn 1986). In contrast, if comparisons of large numbers of genetically determined traits serve as the basis for discrimination, as argued in Rushton's article, only individuals descended from common ancestors (although possibly distantly removed) are likely to be sufficiently similar to elicit recognition (see Grafen 1985). The favorable treatment of individuals based on their overall genetic similarity leads neither to "selfish gene" selection nor to group selection, but merely to ordinary kin selection.

Despite these problems, I can accept Rushton's general premise that genetically similar individuals assort preferentially, at least in some contexts. The genetic data are intriguing, if rather difficult to evaluate, and I cannot disagree with conclusions such as "at the very least . . . the hypothesis that friends choose each other partly on the basis of genetic similarity warrants further investigation" (Rushton 1989b). My reluctance to regard genetic similarity theory as a useful tool for making behavioral predictions stems not from any disinclination to examine ultimate, as opposed to proximate, causes of behavior. Rather, the failure of the theory to make any compelling evolutionary statement simply leaves me bewildered by its conclusions.

Problems with the altruism hypothesis

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Although I am intrigued with the concept of directing altruism toward genetically similar others, I am also bothered by major shortcomings in the theory and empirical work that Rushton reviews. The following comments are intended not to reject the idea, but to focus the attention that the idea deserves.

(1) The relationship between the genes that allow recognition and the genes that cause altruistic behavior (Dawkins's "green beard" effect) is a major unsolved problem. The beauty of kin selection is that all genes identical by descent are correlated with each other, so that a similar appearance serves to identify a similar propensity to behave altruistically. That correlation disappears for unrelated individuals. Rushton does little to resolve the problem in section 7.1, and it remains a fertile area for theoretical work. I have only two observations to offer. First, genetic similarity theory remains interesting even if it is subsumed by kin selection. Second, the assumption that the genes coding for altruism cannot themselves be recognized seems unwarranted. If only altruists behave altruistically, then why rely on a similar face or a similar smell when the behavior itself can be observed and recognized? In this sense genetic similarity theory seems to merge with evolutionary game theory. Should the tit-for-tat strategy, which dictates that one cooperate with cooperators and defect with defectors, be regarded as an example of behaving altruistically toward others with shared genes? I suspect that many theorists would resist this comparison, but why?

(2) The idea of ethnic altruism in its simple form is almost certainly fallacious. Consider two ethnic groups that differ in the frequency of an allele that causes individuals to behave altruistically toward all members of their own group. By definition, in each group this allele is selected against. Even if the more altruistic group exterminates the less altruistic group, the global increase in altruism is only transitory and selection within the successful group will ultimately run its course. Group selection requires the continuous generation of groups that vary in the expression of altruism, which is difficult to imagine at the scale of whole ethnic groups.

Rushton's treatment of ethnocentrism and ideology is rather vague, and at times he seems merely to argue for an adaptive basis. Genetic similarity theory is a specific hypothesis, however, that can be rejected without rejecting adaptive explanations in general.

(3) Almost all of the empirical evidence shows that associates are genetically similar to each other, relative to a control group, but does not address the question of whether they associate *in order to* behave altruistically. In the case of marriage partners, Rushton himself provides a much stronger advantage in the form of increased personal fitness. More generally, genetic similarity theory is only one of several ways to explain genetic similarity among associates. First there is the problem of an appropriate control. Recruiting associates for an experiment and forming a control group by randomly re-pairing them is inappropriate if the subjects originally chose their associates from genetically different populations, as Rushton acknowledges. This problem could be solved by measuring the genetic similarity of associations that develop in groups that originally comprised strangers. Second, psychologists have long known that like attracts like at the phenotypic level, which translates into genetic similarity if the human phenotype has a genetic basis. Genetic similarity theory – that the attraction evolves to benefit the genes of self in others – must therefore contend with more traditional psychological explanations, many of which can be translated into evolutionary arguments.

The idea that interactions between individuals are nonrandom has numerous implications for evolutionary biology and the human sciences. Genetic similarity theory is one hypothesis about how and why such interactions evolved, something that is best studied in this wider context.

Author's Response

Similarity and ethnicity mediate human relationships, but why?

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Not one of the 33 commentaries seems to deny the importance of similarity in mediating human behavior; nor would they be expected to do so given the ubiquity of the phenomenon. Yet Nature could have organized things differently; the adage that opposites attract could have described the prevalent phenomenon, and complete randomness might have been a less energetically expensive determiner of our choice of social partners.

Why, then, do people prefer in general to be with others similar to themselves?

In the target article I proposed an extension to kin selection and inclusive fitness theory in which the idea of "genetic similarity" between individuals was substituted for "relatedness" as the more general and appropriate concept: By benefiting people who share genes not identical-by-almost-immediate-descent, in addition to "kin," genes can replicate themselves more readily. Most of the commentators' remarks are oriented toward two main categories of concern: (1) theory and (2) data.

1. Theory

It was gratifying that so many commentators (e.g., **Dunbar, Eibl-Eibesfeldt, Eysenck, Findlay, Hartung, Hepper, Jensen, Kenrick, Krebs, Leek & Smith, Lynn, Masters, Reynolds, Rowe, van den Berghe, and Vine**) found that aspects of the theory looked promising and deserved serious attention. This does not mean, of course, that they were uncritical about other aspects. Still others found the general premise intriguing, if troubling (**Waldman, Wilson**). Perhaps the most frequently voiced issues concerned the status of the various mechanisms involved in detecting genetic similarity in nonkin – evolutionary, genetic, and psychological mechanisms.

Evolutionary mechanisms. Several commentators stressed how unlikely they thought it was that natural selection could bring about a preference for similarity except by virtue of relatedness (**Archer, Daly, Gangestad, Ridley, Tooby & Cosmides, Wahlsten, Waldman,** and **Wilson**). Ethnic favoritism in particular seemed to be a problem for many because group microstructure would have selected for success in local populations where the greatest advantage would have obtained by helping very close kin even over distant kin, and where intragroup competition would have been a strong tendency. The probability of ever encountering people of very different genetic structure during most of the Pleistocene was doubted.

The solution to the problem may lie in other remarks made by these critics. As **Archer** writes, citing Dawkins (1976), there are no definite lines to be drawn between family and nonfamily. Or, as **Tooby & Cosmides** and **Waldman** emphasize, genetic similarity has arisen because of shared ancestry, however recent or distantly removed. **Economos** may have put it most succinctly. After pointing out that all members of the same kingdom must share genes she asks "then, what's a relative?" Far too many confusions have arisen as a result of the catchphrase "kin selection" and, as **Daly** suggests, perhaps it is time to abandon it.

Most of the formulations of kin selection theory have focused on alleles identical-by-almost-immediate-descent and not on more distant ancestry. With each generation the genetic relationships are alleged to become 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 possible 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, emphasis in original). However, 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. Elizabeth II may well be more similar to William the Conqueror than she is to the average person.

Other commentators made important remarks about genetic similarity theory in the context of the evolution of altruism and kin recognition. **Dunbar** states that kin selection is extremely limited in scope, being even unable to explain assortative mating. For him it only specifies the conditions under which altruistic behavior can evolve when there is no information at all about the identity of the other interactants, and so genetic similarity theory may really have something useful to offer. For **Eibl-Eibesfeldt** (and **Kenrick**) there is not the same problem conceptualizing the spread of altruism genes that exercises most sociobiologists and he emphasizes the continuity with maternal behavior, courtship, and general social bonding. Of importance for my thesis is Eibl-Eibesfeldt's (and **Rowe**'s) discussion of group selection; they argue that for at least the last 20,000 years group selection based on intergroup competition has been a strong force, bringing into the world in- and out-group thinking and the partially innate trait of xenophobia. This necessitates the development of the capacity to calculate considerably more distant degrees of kinship than those typically discussed. As **Hepper** makes clear, individuals should be able to generalize from their ability to recognize kin to recognizing genetic similarity in unrelated people. **Reynolds** and **van den Berghe** also find the evolution of a biological basis for ethnic nepotism very plausible.

Although no one seems to really doubt that group selection can occur, its importance is differentially perceived. **Daly** deemphasizes it and refers to nepotistic strategies as epiphenomena. **Dunbar** defines it *sensu strictu* to almost rule it out of consideration but doesn't discuss its use in the less strict form I defined in section 10. **Findlay** correctly points out that more sophisticated selective mechanisms than are generally considered may be possible in structured biocultural systems, although I was surprised that he didn't elaborate on my use of his and Lumsden's group-selection model. **Ghiselin** *seems* to agree that preferences for similars might be conducive to group selection, but his is a highly qualified statement. **Gangestad, Hallpike, Vine,** and **Wahlsten** are the most critical of my emphasis on the relation between ethnocentrism and group selection, with Wahlsten almost implying that I seek only to rationalize my own bigotry! This clearly remains a controversial issue.

The vagueness of my terminology and specification of variables was commented on. **Wahlsten** takes me to task for providing only qualitative ideas and demands a quantitative formulation before taking me seriously, and **Gouzoules** observes that there is nothing in genetic similarity theory that matches the elegance of Hamilton's Rule. At least five commentators (**Economos, Ghiselin, Hallpike, Vine,** and **Waldman**) state that key concepts such as altruism are poorly defined. Such indeterminacy precludes powerful evolutionary models. I have to agree that there is much scope for improvement here, but I also note that sophisticated modeling is of most value when testing theoretical ideas embedded in established data.

Genetic mechanisms. Many commentators imply that until it is possible to specify the processes occurring at the molecular level, the postulation of innate templates for detecting genetic similarity must be considered speculative (**Dunbar, Gangestad, Ghiselin, Gouzoules, Krebs, Leek & Smith, Ridley, Tooby & Cosmides, Wahlsten, Waldman,** and **Wilson**). I was especially disappointed in reading Waldman's commentary because I had interpreted his 1987 paper as providing support for this "strong" version of genetic similarity detection (section 7.1). It seemed to me that if innate feature detectors were the product of multiple alleles they would accurately reflect the overall genome rather than particular parts. Animals have all sorts of innate preferences; why not ones for others who are similar? Like food preferences, social predilections can be highly individualized. Innate feature detectors, canalized learning, and idiosyncratic experiences may all have a role to play in ontogeny. We shall have to wait for more hybridization and other selective breeding studies in animals where control can be exerted over the rearing conditions to see whether innate similarity detectors exist. **Kenrick** hypothesizes that a few exclusionary detectors would be more efficient than the abundant similarity detectors he believes would be necessary. Certainly an absence of defects in social partners might make for smoother interaction, but on the other hand it seems that the number of potential ways someone could be dissimilar far exceed the number of ways one could be similar.

Eysenck, Nagoshi, and **Rowe** want behavioral genetic designs brought more fully to bear on human studies. In fact these might help to differentiate detection systems. One useful design involves the comparison of monozygotic and dizygotic twins reared together. Although critics have argued that the twin method is invalid, detailed empirical work demonstrates the critiques to be of limited importance (Scarr & Carter-Saltzman 1979). In a twin study the total phenotypic variance can be partitioned into the following three sources: V(G), additive genetic effects; V(CE), common environmental influences that affect both twins equally; and V(SE), specific environmental influences that affect each twin individually. Thus, the total phenotypic variance is partitioned as V(G) + V(CE) + V(SE). Now, if phenotype matching based on parents or other close relatives is important, then lower G and higher CE effects might be expected in a twin study of social preference; if innate feature detectors carry most of the burden for recognition, however, then G might be higher and CE effects might be small or nonexistent. Imprinting on self rather than kin should lead to more of the variance being accounted for by SE.

As **Daly, Krebs,** and **Leek & Smith** in particular point out, it is unnecessary to insist on genetically constructed feature detectors to carry all the burden. Phenotype matching, whereby the feature detectors are built up through experience with self and others, might account for the data, especially if the more heritable components of traits are the most reliably occurring indicators of kinship. **Tooby & Cosmides** suggest that phenotype matching plays a role in human coalitional psychology and interethnic hostility, but if I read them correctly they actually make the differential prediction that nongenetic phenotypic traits such as linguistic patterns and other potentially alterable group membership "badges" may prove better predictors of relatedness than more heritable markers. This would not fit my finding that assortment is greatest on the more heritable items; it also seems to be at odds with the argument of **Reynolds** and **van den Berghe** that ethnicity has a "primordial" dimension.

Psychological mechanisms. What is provided by **Jensen** is, in effect, a new model for the psychology of social assortment based on the notion of "total perceived value." Because of previous assortative mating, traits become genetically correlated, either through the common assortment of the independently segregating alleles that affect each trait, or through pleiotropy. The concept of assortative mating on single traits, as it is usually discussed, is therefore misleading, because aggregation effects will make partner similarity as high as .7 or .8 as measured by a canonical correlation. Jensen proposes that people subjectively match themselves with their partners on their total perceived values, a subjectively weighted sum of perceived assets and liabilities reflecting both genetic and cultural values. This thesis could generate interesting new lines of research.

Kenrick, as mentioned, considers the avoidance of defects to be as important as the attraction of similarity and cites Rosenbaum's (1986) data that whereas variations in a target person's similarity do little to change initial attraction, escalating amounts of dissimilarity lead to increased disliking. Kenrick also emphasizes the way in which cultural factors affect the psychology of altruism, citing his own research program on the socialization of altruism. Thus the direction of altruism to particular public targets becomes internalized through several stages – first to avoid parental punishment, then as a means of gaining public rewards, and finally as an internal self-reward system. **Stam** also emphasizes the importance of agency in goal-directed behavior but objects to my use of the distal-proximal continuum (section 8) for understanding levels of explanation – apparently because it reduces agency to a genetic imperative: In his view no amount of sociobiological tinkering will allow for an explanation of the building of St. Joseph's Hospital here in London, Ontario. I am uncertain how much we are actually in disagreement, because I fully acknowledge the independence of effects at each stage of the distal-proximal continuum as well as the importance of agency and the human mind, whereas he accepts that genetic theories provide permissive conditions. Littlefield and Lumsden (1986) have discussed how genes, mind, and culture may interact in producing altruistically motivated health care facilities.

2. Data

Much commentary focuses on analysis and discussion of the data sets provided – of blood antigen analyses, within-family favoritism, differential assortment on genetic traits, and ethnocentrism.

Blood antigen analyses. Commentators varied in how convincing they found the blood tests as indications that social assortment followed lines of genetic similarity. For **Economos** the blood antigens were the only data I pre-

sented that she could accept as genetic but she expressed skepticism as to how blood similarities could have come about given that genes are not clairvoyant. **Jensen** provides one answer with the notion of cross-assortative mating in which marital correlations between different traits also pull in those that are not directly perceivable. Jensen suggests that the association between IQ and blood groups is sufficiently high to explain the observation that spouses and friends are more similar in blood groups than are randomly paired individuals. **Findlay, Gangestad, Graves & Byrne, Hallpike, Hartung, Kline, Reynolds, Tooby & Cosmides,** and **Wilson** accept the data but only as weak evidence, either because the levels of statistical significance are low or because the pattern of causality is unclear; for example, it was suggested that, despite the statistical controls I used, ethnicity and social stratification could have accounted for the similarity of blood type between social interactants in which case the reported observations are an effect, not a cause. **Ghiselin** is the most critical of this use of correlational data, referring to my causal postulates as "undesirably teleological."

Several commentators are concerned about strengthening the controls used in these studies. With respect to the data from the sexual interactants, **Daly, Gangestad,** and **Ridley** wonder whether dependencies in gene frequencies between mother and offspring or mother and sexual partner may have differentially affected the capacity to exclude paternity. I should have mentioned in the target article that although because of financial limitations my own study could only examine markers at 10 loci, the paternity exclusion carried out by Serological Services for court cases was an absolute one, using protein analyses where necessary (Rushton 1988a). With respect to the data from friends, **Hartung** suggests that rather than comparing the similarity of friendship dyads with one other person chosen at random a more adequate control group would be the averaged similarity across all 76 nonfriend pairs. I should have mentioned in the target article that for all the psychological data gathered a control group was constructed by randomly selecting *five* nonfriends and averaging their scores (Rushton 1989b), a procedure that should have allowed adequate testing of the hypothesis.

Some commentators came up with imaginative ideas for future studies. **Findlay** and **Gouzoules** independently suggest the use of experimental choice situations where subjects are presented with a set of potential mates and asked to evaluate each in terms of their perceived suitability. **Graves & Byrne** suggest locating the studies in a more circumscribed community such as a Scottish village, which would be genetically homogeneous and where other features could be more readily statistically controlled. **Leek & Smith** report their plans to examine the genetic basis of social assortment using the powerful new technique of genetic fingerprinting. **Wilson** suggests that measures of genetic similarity be taken as associations develop in groups that were originally formed from strangers.

Within-family favoritism. In this section I reviewed data to show: (a) The more closely matched spouses are, the happier and more stable is their marriage. (b) The more related siblings are genetically, the closer they feel to each other, at least when one compares monozygotic (MZ) and dizygotic (DZ) twins. (c) Following the death of a child, parental grief intensity is correlated with the child's similarity to the parent. Very little commentary focused on either (a) or (b), although **Rowe** points out that phenotype matching accounts for why MZ twins raised apart typically form very close emotional relationships when they eventually meet. Rowe also poses the interesting question of why it is that some pairs come to actively dislike one another. It would be interesting in this respect to look at variations in genetic similarity among DZ twins to see whether they predict degrees of liking.

Several commentators remark on the study of parental bereavement by Littlefield and Rushton (1986). **Archer** and **Stam** (and **Vine**) independently cite Archer's (1988) critique of this study, pointing to both methodological and theoretical issues, but they do not refer to the rejoinder by Littlefield and Rushton (1989), in which we show that our methodology is sound enough to support our conclusions. We rebut the mistaken notion that every feature of living organisms has immediate adaptive advantage, that is, contributes directly to fitness. We were sympathetic to Archer's (1988) contention that bereavement is part of a syndrome of responses to separation from a loved one, which includes preoccupation, searching, and anger, and which in the majority of cases may be adaptive as it helps to reunite the individual with the lost person.

Wahlsten's discussion of my derivation of the family favoritism hypothesis seems decidedly uncivil in tone. Wahlsten asserts that the figures I gave to illustrate the differences in parent-child similarity arising from assortative mating are so absurd that they wouldn't occur even if parental dissimilarity were such that dad was a human and mom was a chimpanzee! He ponderously derives, from algebraic equations and Hardy-Weinberg equilibria, that the actual differences would be considerably closer together – assuming that all the assumptions he starts with hold. If we change these assumptions to suggest, for example, that the important differences between people lie in a few regulator genes and not the whole genome, the differentials can loom large again. Nor is it difficult to make the point that parents are differentially related to their children: Consider 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 or Aa. In the first case, the offspring is 100% similar to the father and 50% similar to the mother; in the second, 50% similar to the father and 100% similar to the mother. Wahlsten seems to have missed the important point that parents are not equally "related" to their children and that differential degrees of similarity may affect family preferences.

Both **Gangestad** and **Leek & Smith** accept the findings of the bereavement study and attribute the results to phenotype matching, but whereas Gangestad concludes that the results are uncompelling, Leek & Smith find them to be important: Leek & Smith also report that they found confirmatory results in an ongoing study of three generations of family relationships in which perceived altruism correlates with perceived similarity in personality and physical appearance. We look forward to the publication of their results.

Differential assortment of genetic traits. The data on differential assortment provide much of the empirical substance of the target article and 15 commentators have something to say about them. **Eysenck** makes an important contribution by presenting data from his new book (Eaves, Eysenck & Martin 1989) in which model-fitting techniques are used to reanalyze data on personality and social attitudes from both cultural and genetic perspectives, taking genetic/environment interactions into account. His calculation of both high heritabilities and high assortative mating coefficients for social attitudes supports my conclusions. Also supportive is his observation that the highest coefficients of assortment occur for identical traits, with lower ones being observed for cross traits, suggesting that assortment operates on a trait-by-trait basis (although recall **Jensen**'s commentary discussed earlier). It is unfortunate that Eysenck does not present the item-level correlations between the estimates of heritability and assortment to test the genetic similarity theory prediction directly; relating them at the level of the factor, as in Eysenck's Table 2, unfortunately for me, does *not* support the prediction.

For many readers, the most surprising of **Eysenck**'s conclusions will be that the sort of cultural factors that operate between families (such as the occupation and socioeconomic status of parents) account for very little (if any) variance in family resemblance in social attitudes, a discovery that runs counter to prevailing theories of personality development. Yet the observation that the environmental factors that influence development are those that are specific to each sibling, rather than common, is robust, having been replicated using samples of four different types: twins reared together, twins reared apart, adoptive parents and their offspring, and adoptive siblings. The evidence reveals that whereas genetic influences have an important role to play, the common family environment alone has little apparent effect. That siblings raised apart for many years grow to be significantly similar to each other and that their degree of similarity is predicted by the number of genes they share implies the presence of genetically based stabilizing systems that channel development. Such systems operate within families and make siblings different from one another (see Plomin & Daniels 1987).

Rowe suggests that behavior genetic designs could be used to test genetic similarity theory explicitly. For example, both the spouses and the best friends of MZ twins should be more similar to each other than the spouses and best friends of DZ twins. Also, assortment itself should be partly heritable and there should be heritable differences between individuals in the degree to which they positively socially assort, that is, some people will be more inclined to choose similarity in partners than others. Rowe suggests that intolerance of differences in close social partners shows up in early childhood and may be related to the partly heritable personality trait of authoritarianism, which has also been linked to low IQ.

Lynn and **Masters** also consider whether there are heritable differences in the tendency to assort for similarity. Lynn focuses directly on the tendency toward ethnocentrism by hypothesizing a balanced polymorphism in society with some people inclined to in-group chauvinism and others more attracted to out-group differences. Masters suggests the types that might be involved: People who prefer ethnocentric similarity are hypothesized to be *K*-strategists, reproductively speaking, with an emphasis on very high amounts of parental care, a strategy he suggests may be mediated by harm-avoidant personalities and high levels of serotonin. People preferring opposites are hypothesized to be *r*-strategists, emphasizing direct mating effort, a strategy that may be mediated by novelty-seeking personalities and lower levels of serotonin. It is interesting to note that whereas Master's conjecture about *K*-reproductive strategies suggests that high IQ will be associated with ethnonarcissism, **Rowe**'s conjecture about authoritarianism suggests that it will be low IQ that is so associated.

I find a great deal of merit in the hypotheses of **Lynn, Masters,** and **Rowe** and have been thinking along similar lines (Rushton 1985; 1988c). It was my suggestion that racial differences may exist in *r*/*K* reproductive strategies, with Oriental populations being the most *K*, that led to the public brouhaha referred to by **Stam.** I also have research in progress that will examine what (if any) heritable characteristics relate to positive social assortment. I have gathered questionnaire data not only from MZ and DZ twins but also from their best friends and their spouses. Thus this research will test the hypothesis that the tendency to positively assort socially is a phenomenon on which there are heritable differences and that the spouses and friends of MZ twins are more similar to each other than are the spouses and friends of DZ twins.

Not all commentators, however, are happy with behavioral genetic designs. Even **Nagoshi** and **Rowe,** themselves behavior geneticists, take issue with my use of heritability estimates to "probe the genes" and predict degrees of assortative mating. They reiterate the received view that genetic estimates are not very stable across different samples, and Nagoshi, while acknowledging my review paper on the topic (Rushton 1989a), discusses his own work, which shows cross-cultural and cross-generational changes in the magnitudes of estimated heritabilities, common environmental influences, parent-offspring resemblances and assortative mating coefficients on intelligence test scores. Clearly, cultural and historical factors can have important influences on the expression of genetic effects. The question, however, is whether there is a sufficient genetic residual to be predictive. The data I present in Tables 3 and 4 of the target article, in which genetic influences are calculated from one sample and assortative mating is calculated from another, constitute a conservative test of the genetic similarity hypothesis because the observed relationship has to be sufficiently robust to overcome variance changes due to environmental perturbations. Moreover, with intelligence subtests I have shown that genetically based inbreeding depression scores calculated in Japan on the WISC in the 1950s predict the magnitude of black-white difference scores on the WISC-R in the 1970s (Rushton 1989a). Such results show that estimates of genetic influence are more robust across populations, languages, time periods, and measurement specifics than has been considered to date.

To test **Nagoshi**'s conjectures that (a) cross-cultural factors limit the operation of genetic similarity detectors in mate choice and that (b) overly homogenous items may

have artifactually inflated the reported results, I calculated a correlation between the spousal similarity scores and estimates of genetic influence across 12 tests of intelligence and 11 of personality administered to the 57 Egyptian families discussed by Nagoshi (Abdel-Rahim et al. 1988). Although the between-spouse and between-sibling correlations were observed to be consistently higher than in the American families, which the authors attributed to the effects of arranged marriages and social stratification, I was nonetheless able to use the mid-parent-offspring regressions to predict spousal similarities across the 23 traits $r = 0.69$ ($p < 0.001$). These Egyptian data thus join those reported in Table 3 of the target article (which includes data from arranged marriages in Korea) to support the view that genetic similarity operates as a basis for mate choice and, more generally, that universal laws operate to govern who people prefer as mates (see also Buss 1989).

The above notwithstanding, a widespread mistrust exists over the legitimacy of calculating heritabilities. I referred to this in the target article (section 7.2) and it is amply confirmed by commentators **Economos, Vine,** and **Waldman,** who are especially exercised about "genetic determinism" when it comes to such social attitudes as ethnocentrism. Waldman goes so far as to say that the high heritabilities found by Martin et al. (1986) and reported in Table 4 of the target article are "preposterous." These data, based on twins reared together, are clear and have been replicated with other designs including twins raised apart (e.g., by Tellegen et al. 1988), using increasingly sophisticated methodologies, as shown in the articles cited by **Eysenck.** Some confusion, however, may arise from not being able to conceptualize mechanisms. Clearly, genes do not cause behavior directly. They code for enzymes which, under the influence of the environment, lay down tracts in the brains and nervous systems of individuals, thus differentially affecting people's minds and the choices they make about behavioral alternatives. With regard to ethnocentrism, for example, as suggested by **Masters,** some people may inherit serotonin levels disposing them to novelty-avoiding and other tendencies making up a constellation of attributes associated with *r*/*K* reproductive strategies. There are many plausible routes from genes to behavior; collectively, these routes may be referred to as epigenetic rules (see section 8.1).

A few of the commentators attempt to dismiss the empirical relationships found between the estimates of genetic influence and those of social assortment by invoking a variety of artifacts. Thus **Anderson** claims that the *r* values are spuriously inflated because the same terms enter into the denominators of both the heritability estimate and the intrapair correlation; this is simply incorrect, manifestly so in the cases where the genetic estimates are calculated in one sample and the intrapair correlations in another. She also suggests that because the various test items used are often intercorrelated, significance testing is inappropriate. The items are hardly isomorphic, however, and even if the significance levels *were* to be corrected downward, the repeated conceptual replication of the observation across different traits and samples is not to be dismissed. Such converging validities also go some way toward answering the criticisms of **Gangestad, Ridley, Vine,** and **Waldman,** which are directed at particular ways of calculating genetic influence estimates. Many of these apprehensions are in any case quite misplaced (Plomin et al. 1980).

More challenging than the appeals to statistical artifact are the suggestions by **Dunbar, Ghiselin, Kline, Hallpike,** and **Ridley** that although the positive relationships between assortment coefficients and estimates of genetic influence do exist, they are epiphenomena resulting from (a) expenditure savings in relationship meshing, (b) the economic benefits of interaction (Ghiselin's notion that humans prefer dogs rather than monkeys as pets), and (c) the regional distribution of traits in space. But if the replication of identical genes in others is also aided, natural selection might be expected to operate on any behavior bringing it about. Consider the advantages of assortative mating from this perspective, which Ridley apparently found problematic: If meshing in marital relationships leads partners to stay together longer and to more readily sacrifice for each other, more offspring will be raised to reproductive maturity and inclusive fitness will thereby be increased.

Ethnocentrism. The question **Lynn** raises is for me fascinating: Why do people remain as irrationally attached as they do to languages, even almost dead ones such as Gaelic and Welsh? Debates over the English/French divide here in Canada attest to the fervor with which the issue can be joined. Lynn speculates that one function of language barriers is to promote inbreeding among fellow ethnics. The EDITORIAL NOTE suggests that this hypothesis represents a case of uncritical biological determinism. Yet the mapping of linguistic on genetic trees has been found to occur to a degree that would never have been predicted even a short time ago. Cavalli-Sforza et al. (1988) have grouped gene frequencies from 42 populations into a phylogenetic tree based on genetic distances and related it to a taxonomy of 17 linguistic phyla. Despite the apparent volatility of language and its capacity to be imposed by conquerors at will, considerable parallelism between genetic and linguistic evolution has been found. Hence it does not even seem unreasonable to wonder whether there are brain structural differences between populations making the acquisition of one language type easier than another (compare the ideographics of the Mongoloids, the phonetics of the Caucasoids, and the clicks and implosions of the Khoisan). It may be inappropriate to dismiss too readily speculations on the relationship between varieties of linguistic expression and genetic similarity.

Eibl-Eibesfeldt, Kenrick, Lynn, Reynolds, and **van den Berghe** believe that ethnocentric perceptions and ethnic relationships provide suitable areas for hypothesis testing, partly because the range of empirical situations is sufficiently wide to control key variables. I agree with these suggestions; examining genetic similarity within an ethnic context could not only help make some predictions clearer but it could also help rule out some criticisms of the theory at the outset. Thus **Hallpike** claims that employers do not use genetic similarity in choosing employees and that membership in political parties does not covary with particular traits, while **Kline** suggests that location only works as an artifactual cause and not as a consequence of social assortment. The ethnic data are clearly against them (e.g., Glazer & Moynihan 1970). I

was surprised that **Masters,** a professor of government, did not bring his discipline's expertise to bear here but instead wrote of personality differences and *r/K* reproductive strategies; perhaps, though, that is an indication of the degree of cross-fertilizing influence evolutionary theory has had on the social sciences.

3. Conclusion

Target articles that examine how genes bias the development of complex human social behavior in one direction over alternatives evoke particularly strong reactions; for some readers they seem to threaten not only the way we conceptualize human nature but also what we think is right and what we think is wrong. Studies of altruism and political attitudes in twins estimate that about 50% of the variance is associated with direct genetic inheritance, virtually 0% with the twin's common family environment, and the remainder with each twin's specific environment. Studies of human marriages and friendships show that people choose each other on the basis of similarity, assorting on the most genetically influenced of a set of homogenous attributes. These data imply a genetic canalization of social influences so that, within the constraints allowed by the total spectrum of cultural alternatives, people create environments maximally compatible with their genotypes.

Darwin's theory of evolution tells us that the ultimate reason for behavior, like morphology, is to enhance inclusive fitness. One job for behavioral scientists is to discover the proximal mechanisms by which this occurs. **Findlay** states that high biological fitness is not necessarily to be found in biocultural systems such as human societies and cites the case of how ethical systems might carry on indefinitely even though they reduce the fitness of believers; this seems to miss the essential point, however. The *particular* genes acquiring the fitness-reducing culturgen would diminish relative to those acquiring the fitness-enhancing culturgen. For example, for women in the United States, childlessness exceeds 10 percent only among the highly educated and the nonreligious (Jacobson et al. 1988). Assuming that (a) some genetic variance underlies each of these tendencies (Tellegen et al. 1988), and (b) the tendencies are causal, my point is made.

As Eysenck (1980) observed in an earlier *BBS* commentary, it is time for human sociobiology to stand firmly on both legs: evolutionary theory *and* behavioral genetics. I followed this up, also in a *BBS* commentary, proposing that genetically based individual differences should "become a crucible for theory construction . . . so that the formulation of hypotheses should lead to an immediate individual-difference test" (Rushton & Russell 1984, p. 741). It is striking, however, that even avowed human sociobiologists soft-pedal this notion. For example, Daly and Wilson (1988) go so far as to state at the outset of their book *Homicide* that to attribute a propensity to violence to certain types of individual differences is not a theory but a "facile disparagement (revealing) more about the prejudices of their proponents than about the causes of violence" (p. 1). But it is not every husband who, when faced with his wife's infidelity, becomes angry enough to murder her. Not all people have the traits to achieve headman status among the Yanomamo.

Too many studies of twins and adoptees have been conducted for the genetic contribution to individual differences in aggressiveness, intelligence, law-abidingness, sexuality, and – yes, probably – preference for similarity in social partners, to be legitimately ignored. The notion of the "species typical individual" still too often prevails. Surely it is now time for genetic variance, the first postulate of Darwinian theory, to be more formally incorporated into theorizing about human behavior in both the evolutionary and the social sciences. By implication, it would then more readily be seen to follow that variance in human behavior, including such complex social endeavors as mate choice, selective friendship, and in-group patriotism, has a genetic basis.

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Letters a and r appearing before authors' initials refer to target article and response respectively.

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