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Personality and genetic similarity theory[†]

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This article advances two theoretical schemas. The first concerns nomothetic human personality traits. We provide evidence that personality traits (a) exist, (b) are longitudinally stable, (c) can be assessed by several converging indices, (d) are inherited, and (e) have adaptive significance. We provide a review of some data on the inheritance of individual differences in activity level, aggression, altruism, chronogenetics, criminality, dominance, emotionality, intelligence, locus of control, political attitudes, sexuality, sociability, values, and vocational interests. We also suggest ways in which personality traits could arise in accordance with established genetic principles.

The second half of the paper presents genetic similarity theory (GST). Going beyond kin selection, GST states that a gene may ensure its own survival by acting so as to bring about the reproduction of any organism in which copies of itself are to be found. An organism may have a tendency to exhibit favoritism toward genetically similar strangers as well as toward its own relatives. We order several data sets with this theory including (a) kin recognition studies in animals, (b) assortative mating, (c) intrafamilial relations, (d) human friendship and altruism, and (e) ethnic nepotism. We discuss a strong and weak version of GST and offer some predictions for future research.

This paper is divided into two main sections. The first of these discusses the role of genetic variability and individual differences in evolutionary theory. It is argued that the topic of genetically based differences in human personality is much neglected in sociobiology and is of major importance. The second section provides a theoretical extension to sociobiology. What we refer to as genetic similarity theory states that a gene may ensure its own survival by acting so as to bring about the reproduction of any organism in which copies of itself are to be found. A useful means of pursuing this general strategy is for genes, in effect, to be able to detect copies in other organisms.

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Personality traits

While the existence of human morphological traits has been well accepted by evolutionary biologists, the parallel existence of traits of social behavior has not. Yet, as we hope to demonstrate the existence of genetically based individual differences in personality and social behavior is well established and of direct relevance to sociobiology.

Most of the work in sociobiology has focused on between-species differences in social behavior or on universals in human behavior. Yet the theory of evolution requires that there be genetic differences within species. Indeed, the first premise of Darwin's theory is that individuals of the same species differ and that their characteristics are inherited by their offspring. The second premise is that some individuals produce more offspring that grow to reproductive maturity than others. This differential success results in some characteristics increasing in frequency and others decreasing, in the next generation. This is the defining feature of evolution and natural selection is the process by which evolution occurs. To date, sociobiologists have not seriously addressed the implications of genetic variability within *Homo sapiens*. There is, however, a growing body of research from the behavior genetic and psychometric traditions of psychology which is of direct relevance to sociobiological theorizing. This is the study of genetically based individual differences in personality and social behavior. In this section we will attempt to demonstrate that consistent patterns of individual differences in behavior (i.e. personality traits) (a) exist, (b) are longitudinally stable, (c) can be measured in several ways, (d) are inherited, and (e) have adaptive significance.

The existence of stable individual differences in behavior

The sociobiological perspective is quite compatible with the traditional trait approach to personality. This approach consists of a search for general laws in which consistent patterns of individual differences in behavior play a central role. For several decades, however, there have been two opposing viewpoints on the question of whether human behavior is consistent across situations. The classic study of this problem is the enormous 'character education enquiry' (Hartshorne & May, 1928; Hartshorne *et al.*, 1929; Hartshorne *et al.*, 1930). These investigators gave 11,000 elementary and high school students some 33 different behavioral tests of altruism, self-control, and honesty in home, classroom, church, play, and athletic contexts. Concurrently, ratings of the children's reputations with teachers and classmates were obtained. Scores on the various tests were correlated to discover whether behavior is specific to situations or consistent across them. This study will be discussed in some detail for it is the largest study of the question ever undertaken, it raises most of the major points of interest, and it has been seriously misinterpreted by many investigators, as noted by Eysenck (1970), and Rushton (1980).

We first consider the results based on the measures of altruism. Any one behavioral test of altruism correlated, on the average, only 0.20 with any other test. But when the five behavioral measures were aggregated into a battery, they correlated 0.61 with the measures of the child's altruistic reputation among his or her teachers and classmates. Furthermore, the teachers' and peers' perceptions of the students' altruism were in close agreement ($r = 0.80$). Similar results were obtained for the measures of honesty and self-control. Any one behavioral test correlated, on average, only 0.20 with any other test. If, however, the measures were aggregated into batteries, then much higher relationships were found with other combined behavioral measures, with teachers' ratings and with the children's moral knowledge scores. Often, these correlations were of the order of 0.50 to 0.60. Thus, depending on whether the focus is on the relationship between individual measures or on the relationship between averaged groups of behaviors, the notions of situational specificity and situational consistency are both supported.

Hartshorne and May (1928–1930) focused on the smaller correlations. Consequently they argued for a doctrine of specificity. Their conclusions and data have often been cited in the literature as support for situational specificity (e.g., Mischel, 1968). It is now known, however, that correlations between only two indices of a trait are often unrepresentative and create a misleading impression. A more accurate picture is obtained by using the principle of aggregation and examining the predictability achieved by using a number of measures (Rushton *et al.*, 1983). Correlations of 0.50 and 0.60 based on aggregated measures support the view that there is cross-situational consistency in altruistic and honest behavior. The argument presented for the existence of moral traits also applies, to the existence of other personality traits (e.g., Epstein, 1979; 1980; Eysenck, 1970; 1981).

The longitudinal stability of personality

The question we raised earlier concerning the degree of cross-situational consistency becomes a question about longitudinal consistency when the time dimension is introduced. To what extent, over both time and situation, does a person's behavior stem from enduring traits of character? Block's (1971; 1981) exemplary longitudinal studies demonstrate the coherence of personality over several decades. Data were first obtained on about 170 individuals when the subjects were in junior high school. Further data were gathered when the subjects were in their late teens, in their mid-30s, and in their mid-40s. Clinical psychologists studied individual dossiers and rated personality using the Q-sort procedure—a set of descriptive statements such as 'Is anxious' which can be sorted into piles that indicate how representative the statement is of the subject (Block, 1961). No psychologist rated the same subject at more than one time period. The assessments by the different raters (usually three for each dossier) were found to agree with each other to a significant degree, and they were averaged to form an overall description of that subject at that age.

Block (1971; 1981) found a degree of personality stability across the ages tested. Even the simple correlations between Q-sort items over the 30 years between adolescence and the mid-40s provided evidence for stability. For example: for the male sample, 'genuinely values intellectual or cognitive matters' 0.58, 'is self-defeating' 0.46, 'has a high aspiration level' 0.45; for the female sample, 'is an interesting, arresting person' 0.44, 'esthetically reactive' 0.41, and 'is cheerful' 0.36. When the whole range of variables for each individual was correlated over thirty years, the mean correlation was 0.31. These are lower bound estimates, uncorrected for the inevitable presence of unreliability of measurement. Even more substantial relationships occur when typologies are created (Block, 1971; 1981). These results therefore demonstrate that when personality is measured adequately, longitudinal stability is found.

Assessing personality

There are various ways of assessing personality traits. Among the most common are: self-report questionnaires, self-ratings, ratings made by others, naturalistic observations, and performance on experimental tasks. Many studies have demonstrated that these

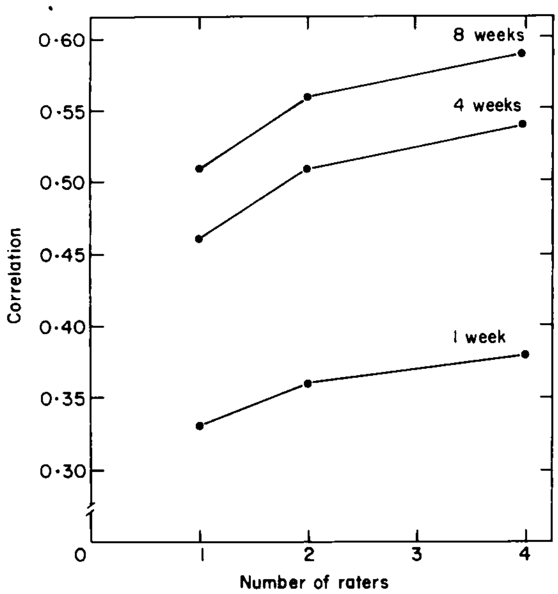


Fig. 1. Validity of ratings of interpersonal dominance as a function of number of raters and weeks of observation (after Moskowitz & Schwarz, 1982).

different assessment techniques exhibit convergent validity (as in the Hartshorne & May studies cited previously).

Moskowitz and Schwarz (1982) have reported that judges' ratings of individual differences in social dominance have high predictive validity when the ratings are averaged over several respondents and when the number of groups of observations is high (Figure 1). Eron (1980) found that average peer ratings of aggressiveness at age 8 correlated 0.45 with the average of a different set of peer ratings of aggressiveness at age 19. Moreover, those who had been rated as aggressive at age 8 were three times as likely to have police records by age 19 than the others.

Questionnaire measures also have predictive validity when criterion measures are reliably assessed. Fishbein and Ajzen (1974) found that although various attitude scales did not correlate highly with single behaviors, their correlations with aggregated behavioral measures ranged from 0.70 to 0.90. Comparable findings are reported by Jaccard (1974) who demonstrated that the dominance scales of the California Psychological Inventory and the Personality Research Form predicted self-reported dominance behaviors better in the aggregate than they did at the single item level. While both personality scales had a mean correlate of 0.20 with individual acts, the aggregated correlations were 0.58 and 0.64.

The heritability of personality

Given that behavioral traits exist and are consistent over time and across situations, we may ask where such consistencies originate. One approach to this question lies in evolutionary biology. While it has long been recognized that individual differences in intelligence are partly inherited, it is not always realized that there is a growing literature showing a heritable component in other personality traits.

Several procedures are available for estimating the proportion of variance in a set of measurements that is attributable to the genes rather than the environment (Eaves *et al.*, 1978; Falconer, 1981; Fulker, 1981; Plomin *et al.*, 1980). The basic assumption is that phenotypic variance in measurements can be partitioned into environmental (E) and genetic (G) components, which combine in an additive manner. The model usually also allows for a non-additive, or interaction, term ($G \times E$) to deal with possible non-additive combinations of genetic and environmental effects. Symbolically:

$$\text{phenotypic variance} = G + E + [G \times E].$$

The estimate of the genetic contribution to phenotypic variance is often referred to as a heritability coefficient and represented as h^2. The heritability of individual differences in behavior may be assessed by several methods. For example, selective breeding studies of animals may be undertaken, using crossfostering to control for upbringing. In humans, correlations may be calculated between scores on the trait in question and the degree of relatedness within the family, the best known example being twin studies. Adoption studies also permit the investigator to separate the effects of environment and heredity. Finally, the trait in question may be studied in infancy to ascertain whether individual differences emerge early and remain stable over time. When studies such as these have been carried out, a significant degree of genetic influence has been detected (Goldsmith, 1983; Plomin, 1983; Plomin *et al.*, 1980; Rushton, 1984*a*). In short, the evidence from converging methods implicates the role of heredity in human personality.

Adoption studies and the comparison of twins are the most widely used procedures for estimating h^2. In twin studies, monozygotic (MZ) twins are assumed to share 100% of their genes and dizygotic (DZ) twins are assumed to share, on average, 50% of their genes (we argue later that this assumption is likely to be an underestimate). By comparing such twins on a set of measures, estimates of h^2 can be made. If the correlation between scores on a trait is higher for the MZ than for the DZ twins, the difference can be attributed to genetic effects if it is assumed that the differential environment of each type of twin is roughly equal. Doubling the difference between the MZ and DZ twin correlations is one widely used estimate of h^2 (Falconer, 1981). Some have argued that the equal environment assumption is not valid. A review by Scarr and Carter-Saltzman (1979) suggests that this criticism is of limited importance: when parents and twins incorrectly classify zygosity, the degree of twin similarity is better predicted by true zygosity (defined by blood and fingerprint analyses) than by social definition. Moreover Loehlin and Nichols (1976) showed that when measures of the differences that do exist in the treatment of twins are correlated with personality and other scores, there is no evidence that differences in treatment have any effect.

Mittler (1971) reviewed available twin data using the concordance method. This involves finding a twin with a clearly established disorder (e.g. in mental hospital) and then determining whether the co-twin displays the same disorder. The weighted averages of the concordance rates from these studies are presented in Table 1. There appears to be a significant heritable component to most of these behavioral categories. Subsequent reviews of concordance data by Plomin *et al.* (1980) and Willerman (1979) provide further support for this conclusion.

The typical strategy for calculating heritabilities is to use questionnaire data on which to compare MZ and DZ twins reared together. For example, Loehlin and Nichols (1976) compared 514 pairs of MZ twins with 336 pairs of DZ twins who, as high school students, had taken the National Merit Scholarship test. Each participant took a wide variety of personality, attitude and interest questionnaires. The results showed the MZ

Table 1. The percentage of monozygotic (MZ) and same sex dizygotic (DZ) twins falling into the same category as their co-twins (adapted from Mittler, 1971)

Behavioral category	Number of studies	Number of pairs			% Concordant	
		Total	MZ	DZ	MZ	DZ
Adult crime	6	225	107	118	71	34
Alcoholism	1	82	26	56	65	30
Childhood behavior disorder	2	107	47	60	87	43
Juvenile delinquency	2	67	42	25	85	75
Male homosexuality	1	63	37	26	100	12
Manic depressive psychosis	5	518	168	350	73	12
Mental subnormality	2	586	197	389	96	56
Neurosis	10	1267	560	707	22	11
Schizophrenia	13	1251	503	748	53	11

twins to be roughly twice as much alike as the DZ twins over a wide range of personality measures—exactly as would be predicted by genetic theory.

Dramatic examples of identical twin similarity have been found by Bouchard and his colleagues (e.g., Bouchard *et al.*, 1981). The focus of their study is on identical twins, separated at birth and raised apart. Bouchard (1983) reports that the 34 pairs of identical twins raised apart studied to date demonstrate almost as much similarity on such objective personality scales as the Differential Personality Questionnaire, the Minnesota Multiphasic Personality Inventory, and the California Psychological Inventory as do identical twins raised together. Although individual cases must be interpreted with great caution, many remarkable similarities of lifestyle, personal preferences and idiosyncracies between members of these twin pairs have also been documented (Holden, 1980).

In the remainder of this section, a brief review is offered on the heritability of individual differences on several dimensions: activity level, aggression, altruism, chronogenetics, criminality, dominance, emotionality, intelligence, locus of control, political attitudes, sexuality, sociability, values, and vocational interests.

Activity level

Several investigations have found evidence that individual differences in activity level are in part inherited. These include studies by Buss *et al.* (1973), Owen and Sines (1970), Scarr (1966) and Willerman (1973). In one of these, Scarr (1966) assessed activity using a cluster of related measures including ratings, experimental tasks and interviews. The subjects were 61 pairs of MZ and DZ girls between six and ten years of age. Although the particular heritabilities differed from measure to measure, the average heritability was found to be 0.31. In another study, Willerman (1973) tested 93 sets of same sex twins and found the heritability of activity level to be close to 0.70. Additional studies, reviewed by Buss and Plomin (1984), also suggest there is substantial heritability for this measure.

Aggression

Several studies have been conducted on the heritability of individual differences in aggressiveness (Eysenck & Eysenck, 1976; Loehlin & Nichols, 1976; Owen & Sines, 1970; Rushton *et al.*, in press; Scarr, 1966). In Scarr's study, parents complete the Adjective Check List to describe their children. On this measure, aggresiveness had a heritability of 0.40. In Loehlin and Nichols' investigation with 850 twin pairs, cluster analyses were performed on self-ratings of various traits. Two clusters that Loehlin and Nichols labelled 'argumentative' and 'family quarrel' showed the MZ twins to be about twice as alike as the DZ twins. Rushton *et al.* (in press) gave a 47-item questionnaire measuring both aggressiveness and assertiveness to 573 adult twin pairs and found about 50% of the variance on each scale to be associated with genetic effects. Finally, psychoticism, a dimension correlated with hostility, had a reported heritability of 0.50 (Eysenck & Eysenck, 1976; Fulker, 1981).

Altruism

At least three studies have investigated the possible existence of genetically based individual differences in human altruism (Loehlin & Nichols, 1976; Matthews *et al.*, 1981; Rushton *et al.*, in press). Using cluster analysis of self-ratings, Loehlin and Nichols found a heritability of 0.44 on a dimension labelled 'kind' among their 850 twin pairs. Matthews *et al.* (1981) analyzed twin responses to a self-report measure of empathy and estimated a heritability of 0.72. In Rushton *et al.*'s study, three separate questionnaires measuring self-reported altruistic behavior, empathy, and nurturance were completed by 573 twin pairs. Approximately 50% of the variance on each scale was found to be associated with additive genetic influences.

Chronogenetics

Genetic mechanisms turn on and off over the course of a lifetime. Common phenomena that reflect such genetic clockworks are the age of onset of puberty and menopause. Identical twins are highly concordant for both events, whether reared apart or together (Bouchard, 1982). A more dramatic example is Huntington's chorea, a degenerative disorder of the central nervous system caused by a dominant gene. Age of onset varies from 5 to over 75, but family studies show that it is under genetic control. Age of first sexual intercourse is similarly influenced by the genes (Martin *et al.*, 1977). Chronogenetics is also important for cognitive development. Wilson (1983) examined genetic influence on the developmental spurts and lags so characteristic of growth. He compared nearly 500 pairs of MZ and DZ twins and their siblings from 3 months to 15 years of age, with measures made of height and mental development. The synchronies in development averaged about 0.90 for MZ twins but only about 0.50 for DZ twins or for other siblings, demonstrating the high heritability of these developmental trajectories.

Criminality

The possibility that genetic factors are among the causes of criminal behavior has long been conjectured (Eysenck, 1977). Studies of the concordance rates of MZ and DZ twins support this hypothesis (see Table 1). Additional support derives from adoption studies. For example, Mednick *et al.* (1984) found a statistically significant correlation for criminal convictions of property crimes between adoptees ($N = 14{,}427$) and their biological parents, while not finding one between adoptees and adoptive parents. Moreover, siblings adopted separately into different homes tended to be concordant for convictions, especially if the shared biological father also had a record of criminal behavior. Other reviews and evidence provide support for the heritability of crime (Ellis, 1982; Plomin *et al.*, 1980). For example Ellis (1982) reviewed the evidence from four classes of research design bearing on the genetics of criminality: general pedigree (or family) studies, twin studies, karyotype studies land adoption studies. He concluded that 'most of the evidence is extremely supportive of the proposition that human variation in tendencies to commit criminal behavior is significantly affected by some genetic factors' (p. 43).

Conversely, support for the inheritance of law-abiding behavior comes from studies assessing the heritability of such scales on the California Psychological Inventory as responsibility, socialization, and self-control. A review of several studies using these dimensions demonstrates heritabilities ranging from 0.30 to 0.40 (Carey *et al.*, 1978).

Dominance

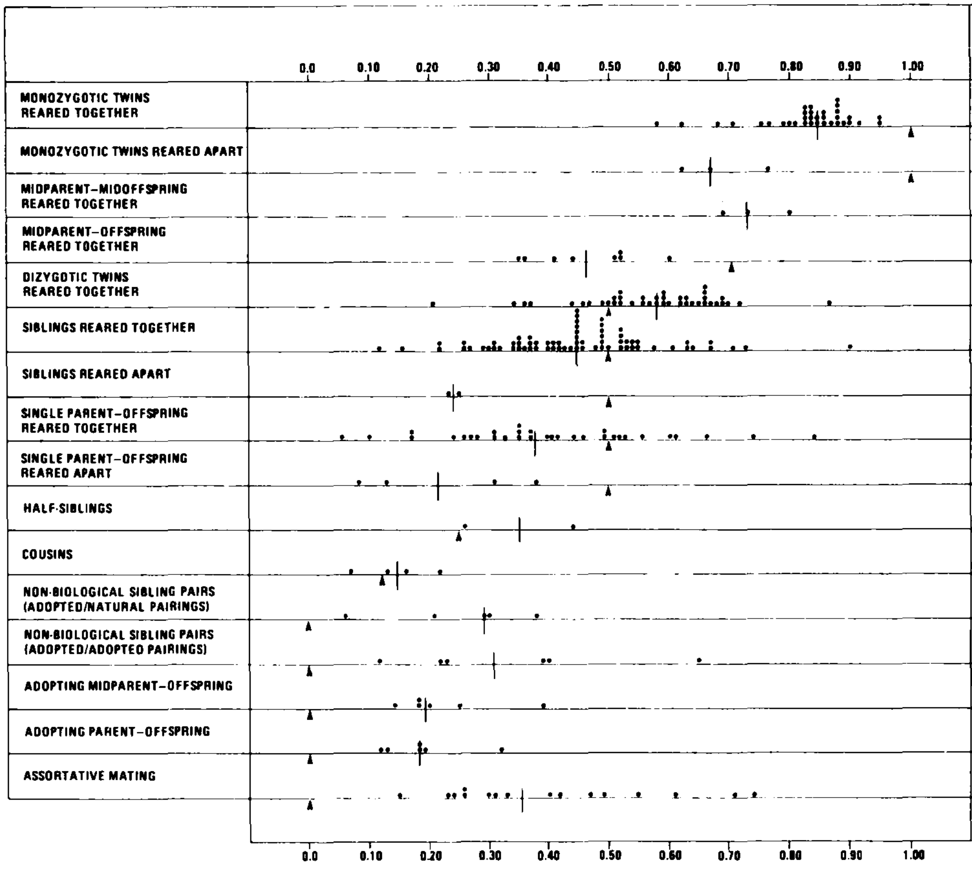
Using a variety of assessment techniques, several studies have found individual differences in interpersonal dominance to be largely inherited (e.g. Gottesman, 1963; 1966; Loehlin & Nichols, 1976). In a longitudinal study of 42 twin pairs, Dworkin *et al.* (1976) found that individual differences in dominance, as assessed on the California Psychological Inventory, remained stable over a 12-year time period, as did the heritability estimate. Carey *et al.* (1978), in a review of the literature, reported that, of all traits, dominance is one of those most reliably found to be heritable, with a weighted mean heritability coefficient, over several samples, of 0.56.

Emotionality

Individual differences in emotional reactivity have long been thought to be partly inherited and several studies have reported substantial heritability coefficients (e.g., Buss *et al.*, 1973; Dworkin *et al.*, 1976; Fulker, 1981; Scarr, 1966; Vandenberg, 1962). The largest heritability study of this trait was carried out by Floderus-Myrhed *et al.* (1980). They administered the Eysenck Personality Inventory to 12,898 unselected twin pairs of the Swedish Twin Registry. The heritability index for neuroticism was 0.50 for men and 0.58 for women. The opposite side of the coin, emotional stability (measured by the California Psychological Inventory's Sense of Well-Being Scale), has also been found to have significant heritabilities, both in adolescence and 12 years later, as in the previously mentioned study reported by Dworkin *et al.* (1976).

Intelligence

Ever since Galton (1869), more heritability estimates of intelligence have been computed than of any other trait. The data published prior to 1963 were reviewed by Erlenmeyer-Kimling and Jarvik (1963) and were compatible with an estimated heritability as high as 0.80. Many of these studies were subsequently criticized by Kamin (1974), who argued that flaws in them required an estimation of the heritability of intelligence to be closer to zero. Newer data and reviews (e.g., Loehlin & Nichols, 1976; Plomin & DeFries, 1980; Teasdale & Owen, 1984), however, have confirmed the high heritability of intelligence. The most extensive review is that by Bouchard and McGue (1981) based on 111 studies identified in a survey of the world literature. Altogether there were 652 familial correlations, including 113,942 pairings. The results were in accord with a polygenic model of the inheritance of IQ. Figure 2 displays the correlations between relatives, biological and adoptive, in the 111 studies.



	NO. OF CORREL-ATIONS	NO. OF PAIRINGS	MEDIAN CORREL-ATION	WEIGHT-ED AVER-AGE
MONOZYGOTIC TWINS REARED TOGETHER	34	4672	.85	.86
MONOZYGOTIC TWINS REARED APART	3	65	.67	.72
MIDPARENT–MIDOFFSPRING REARED TOGETHER	3	410	.73	.72
MIDPARENT–OFFSPRING REARED TOGETHER	8	992	.475	.50
DIZYGOTIC TWINS REARED TOGETHER	41	5546	.58	.60
SIBLINGS REARED TOGETHER	69	26,473	.45	.47
SIBLINGS REARED APART	2	203	.24	.24
SINGLE PARENT–OFFSPRING REARED TOGETHER	32	8433	.385	.42
SINGLE PARENT–OFFSPRING REARED APART	4	814	.22	.22
HALF-SIBLINGS	2	200	.35	.31
COUSINS	4	1,176	.145	.15
NON-BIOLOGICAL SIBLING PAIRS (ADOPTED/NATURAL PAIRINGS)	5	345	.29	.29
NON-BIOLOGICAL SIBLING PAIRS (ADOPTED/ADOPTED PAIRINGS)	6	369	.31	.34
ADOPTING MIDPARENT–OFFSPRING	6	758	.19	.24
ADOPTING PARENT–OFFSPRING	6	1397	.18	.19
ASSORTATIVE MATING	16	3817	.365	.33

Fig. 2. Familial correlations for IQ. The vertical bar in each distribution indicates the median correlation; the arrow, the correlation predicted by a simple genetic model. [Source: From T. J. Bouchard, Jr., and M. McGue (1981), Familial studies of intelligence: A review, Science **212**, *1055–1059. Copyright 1981 by the American Association for the Advancement of Science. Reprinted by permission.]*

Locus of control

The Internal–External Locus of Control Scale (I–E Scale) was developed as a continuous measure of the attitude with which individuals relate their own behavior to its contigent reward or punishment (Rotter, 1966). That one's own actions are largely affected by luck or chance or some more powerful force was labelled a belief in external control. The converse attitude, that outcomes are contingent on one's own behavior, was termed internal control. A recent study by Miller and Rose (1982) reported a twin family study in variation of locus of control. In this study, the heritability estimates based on the comparison of MZ and DZ twins were corroborated by also estimating the heritability through the regression of offspring on parent and the correlation between non-twin siblings. The results revealed heritability estimates greater than 0.50.

Political attitudes

It has generally been assumed that political attitudes are for the most part environmentally determined. However, in a twin study of social and political attitudes, Eaves and Eysenck (1974) found that a dimension of radicalism–conservatism had a heritability of 0.65; tough-mindedness, a factor identifiable with ideological commitment, had a heritability of 0.54; and the tendency to voice extreme views, irrespective of right or left wing bias, had a heritability of 0.37. In an Australian twin study, (N = 3800 pairs), Martin and Jardine (1984) found heritabilities of 0.38 for men and 0.49 for women for the endorsement of a wide variety of specific conservative social and political attitudes.

Sexuality

A study of twins, using questionnaire measures of strength of sex drive found evidence that inheritance plays a substantial role in accounting for individual differences in strength of sex drive (Eysenck, 1976). Differences in sex drive were found to be predictive of many phenomena, including age of first sexual intercourse [itself shown to be under genetic influence (Martin *et al.*, 1977)], intercourse frequency, and total number of partners.

Sociability

This is another well-researched trait and again the evidence favors the hypothesis of a large genetic component. Using different paper and pencil indices of the trait, some studies have found greater than 50% of the variance in individual differences in sociability to be inherited (Carey *et al.*, 1978; Dworkin *et al.*, 1976; Eaves & Eysenck, 1975; Floderus-Myrhed *et al.*, 1980; Fulker, 1981; Gottesman, 1963; 1966; Owen & Sines, 1970; Scarr, 1969). In the largest of these studies, Floderus-Myrhed *et al.* (1980) gave the Eysenck Personality Inventory to 12,898 unselected twin pairs of the Swedish Twin Registry. The heritability index for extraversion, highly related to measures of sociability, was 0.54 (men) and 0.66 (women).

Values and vocational interests

Loehlin and Nichol's (1976) study of 850 twin pairs provides evidence for the heritability of both values and vocational interests. Values such as the desire to be well-adjusted, popular and kind were found to have a significant genetic component. Having science, artistic, and leadership goals were similarly found to be genetically influenced as were a range of career preferences, including those for sales, bluecollar management, teaching, banking, literary, military, social service, and sports. Bouchard (1983) reported that, on measures of vocational interest, his 34 MZ twins raised apart were just as alike as MZ twins raised together. Moreover, both types of MZ twins were twice as similar as related individuals who share half their genes and live together (e.g. parents compared with offspring, or siblings, including DZ twins, compared). Adoption studies also confirm the heritability of vocational interests. Grotevant *et al.*, (1977) contrasted 194 adopted with 237 biological siblings, all of whom had spent an average of 18 years in their families. While biological siblings shared modestly similar interests, adoptive siblings did not.

Supergenes and the origins of personality traits

So far, we have considered evidence which leads to the conclusion that, although environmental factors have a part to play, the role of heritability in the formation of personality traits cannot be ignored. Following Russell *et al.* (1984), we now suggest ways in which personality traits could arise in accordance with established genetic principles: where characteristics are dependent upon the effect of a single gene, there is likely to be little variability within a population except in the case of balanced polymorphisms. Where they are dependent upon the effects of a group of linked genes, the processes of selection may operate on this group as well as on the individual gene and a range of stable individual differences may be maintained within the population. This notion may account for the formation of complex human characteristics such as personality traits, which, insofar as they are subject to genetic factors, are likely to be polygenic in nature.

Suppose that there are two mutations at different chromosomal locations. If either of these new genes increases the chances of its possessor successfully reproducing, then it is likely to increase in frequency until every member of the species possesses it. If either is disadvantageous, it will disappear. Whichever of these outcomes occurs, after an interval of time, there will be no individual differences due to differential possession of these genes. If, however, the possessor of *one* of these new genes is at a disadvantage compared with the person who posseses neither of them or both of them, different consequences may ensue. If the genes are close on the same chromosome, their inheritance may become linked: in time, some people may inherit both and some neither, but few people are likely to possess only one. If they are far apart on the same chromosome, an inversion may put them close together. If they are on separate chromosomes, translocation may occasionally put them on the same chromosome. As before, the possessors of neither or both will be at an advantage compared with the people who have only one.

The argument can be extended to more than two genes. Thus a collection of linked genes, a 'supergene' (Ford, 1976), may be formed. Individuals in the population will tend to differ from each other in terms of their possession of supergenes, each of which will produce a tendency to exhibit more than one behavioral quality, the cluster constituting a useful collection of attributes. Examples of this process are given by Dobzhansky (1970) and by Dawkins (1976). Dawkins cites the case of mimicry in butterflies, where members of an innocuous species have evolved to resemble distasteful species as a defense against predators. Within certain species, a curious strategy has evolved: some individuals resemble those of one distasteful species, and some another. Here, separate genes have come together in such a way that one cluster contains the genes concerned with mimicking one of the distasteful species; the other cluster contains the genes concerned with mimicking the second. As Dawkins suggests, all the different characteristics required for mimicry, such as color, wing shape, patterning, and various aspects of behavior, must have a high probability of being inherited as a single unit in order for the mimic to succeed and reproduce. In practice, intermediate forms very rarely occur.

Furthermore, they would clearly be at a disadvantage, in terms of avoiding predators, compared with either extreme.

We do not wish to imply that this is the only evolutionary process by which traits can be formed. For example, when a large number of genes all have an additive effect on some useful quality, the increase in frequency of each gene will be very slow. Until all of them have replaced their less useful alleles, the population will have a variable amount of the quality influenced by those genes. An example of a variable human quality probably resulting from this process is cranial capacity, which has been increasing for a long time (Passingham, 1982).

To recapitulate, where a mutation has an additive effect on reproductive success, it will eventually prosper or die out, and a state will be reached where individuals do not differ. When two or more mutations on the same chromosome have an interactive effect on reproductive success, stable individual differences may result, with each extreme consisting of an adaptive pattern of correlated behavioral tendencies. Thus general psychological traits or types may be expected to arise. It is of interest to psychology to discover not only whether such traits exist, but also the ways in which they are functional.

Genetic similarity theory

We have suggested that personality traits arise from supergenes which can be more variable than genes. So far we have discussed this in the context of individual fitness through reproductive success. Another way for supergenes to propagate themselves, of course, is through inclusive fitness, i.e. the reproductive success of kin. The idea of kin selection is not new (Haldane, 1932; Hamilton, 1964), but it has only recently become more widely known (Dawkins, 1976; Wilson, 1975), and forms a key element in sociobiological theory.

Kin selection essentially means that genes may ensure their own survival, not only by causing the organism of which they form a part to reproduce, but also by causing it to act in such a way that its relatives reproduce more than they would have done without its action. It is strange that attention has been focused on this particular example (kin selection) of a more general principle which may be stated as follows: a gene or supergene may ensure its own survival by acting so as to bring about the reproduction of *any* organism in which copies of itself are to be found. What we refer to as genetic similarity theory (GST) states that, rather than merely protecting known kin at the expense of known strangers, an organism could have a tendency to detect other genetically similar organisms and to exhibit protective behavior toward these strangers as well as toward its own relatives. In order to pursue this general strategy, it must, in effect, be able to detect copies of its genes in other organisms.

Many aspects of genetic similarity theory have been suggested previously (e.g., Dawkins, 1976; 1982; Hamilton, 1964; Lopreato, 1981; Thiessen & Gregg, 1980; Samuelson, 1983; Rushton, 1984*a*, *b*; Rushton *et al.*, 1984; Russell *et al.*, 1984). Dawkins (1976; 1982), for example, suggested a thought experiment in which a gene had two effects: it causes individuals possessing it to have a green beard, and to behave altruistically toward green-bearded individuals. The green beard serves as a recognition cue for the altruistic gene. Altruism therefore can occur with no necessity for the individuals concerned to be related. Thus with humans, components of similarity (personality, attitudes, physical appearance) may constitute a 'green beard' effect. Similarly, Thiessen and Gregg (1980) theorized that: 'The flow of altruistic behaviors, the ease of information transfer, and the genetic benefits of positive assortative mating are linked to the degree to which interacting individuals share homologous genes' (p. 111).

In the following sections we use GST to order several areas of investigation. These include: (a) kin recognition studies of animals raised apart; (b) assortative mating; (c) intrafamilial relations; (d) human friendship and altruism; and (e) ethnic nepotism.

Kin recognition studies in animals

A number of studies have recently appeared concerned with kin recognition in animals raised apart. In one, Greenberg (1979) provided a striking demonstration of the ability of the sweat bee, *Lasioglossum zephyrum*, to discriminate between conspecifics of varying degrees of relatedness even though they have not previously met. Guard bees of this species can effectively block the nest to prevent an intruder entering. In this study bees were first bred for 14 different degrees of genealogical relationship with each other. Then they were introduced near nests that contained either sisters, aunts, nieces, first cousins, or more distantly related bees. The results demonstrated a strong linear relationship ($r = 0.93$) between being able to pass the guard bee and the degree of genetic relatedness. In other words, the greater the degree of genetic similarity the greater the proportion of bees that were allowed to enter the hive. Thus the guard bees must, in effect, be able to detect the degree of genetic similarity between themselves and the intruder. In subsequent kin recognition studies of the honey bee, *Apis mellifera* (Breed, 1983; Getz & Smith, 1983), a genetic base to the behavior is suggested by the discrimination shown between full and half sisters raised in neighbouring cells.

There is also evidence that the ability to detect genetic similarity exists in species such as tadpoles, birds, deermice, ground squirrels and macaques. Consider the studies of Blaustein and O'Hara carried out on tadpoles of the frog *Rana cascadae* (Blaustein & O'Hara, 1981; 1982; O'Hara & Blaustein, 1981). Typically, the tadpoles are separated before hatching and reared in isolation. Subsequently the individual tadpoles are placed in a rectangular tank with two end compartments created by plastic mesh. Siblings are placed in one end compartment and nonsiblings are placed in the other. The tadpoles spend more time at the sibling end of the tank than at the other. Because the tadpoles were separated as embryos, a genetic similarity detection ability is implicated. Similar findings have been reported for tadpoles of the toad *Bufo americanus* (Waldman, 1982; Waldman & Adler, 1979). Moreover, kin preference behavior continues to be present after metamorphosis (Blaustein *et al.*, 1984).

Kin recognition has been reported for avian species by Bateson (1982), using Japanese quail, and by Radesater (1976), using Canada geese. Bateson (1982) found that quail reared with siblings and tested with individuals of the opposite sex preferred first to third cousins and both of these to unrelated conspecifics. In mammals, sibling recognition has been reported for three species of ground squirrel (Davis, 1982; Holmes & Sherman, 1982), for spiny mice (Porter & Wyrick, 1979), and for deermice (Grau, 1982). A study on pigtail macaques, *Macaca nemestrina*, by Wu *et al.* (1980), found that more interest was shown in paternal half-siblings than in non-relatives by individuals who had been separated from their mothers five minutes after birth, raised in incubators, and then reared with totally unrelated peers. Interest was measured by the amount of time the animals spent interacting with each other in apparatus containing several compartments. Because the half-siblings were paternally related, common prenatal experience cannot explain the preferences.

Assortative mating

A well known phenomenon explained by GST is assortative mating. It could be argued that assortative mating has nothing to do with genetic similarity, but occurs as a result of common environmental influences. This argument has difficulty accounting for the incidence of assortative mating in species ranging from insects through birds to primates in laboratory as well as natural settings (Thiessen & Gregg, 1980). Moreover, that assortative mating in animals is based on genetic similarity is illustrated by a group of experiments showing that individuals can detect known chromosomal differences in conspecifics. Nevo and Heth (1976) report that estrous females of the mole rat *Spalax ehrenbergi*, given a choice between males of two chromosomal forms, significantly preferred a male of their own chromosomal form. Majers *et al.* (1982) found that preferential mating was involved in the maintenance of the color polymorphism of the two-spot ladybird, *Adalia bipunctata*, with a strong female preference for the melanic rather than the non-melanic form. The proportion of females showing this preference could also be increased by selective breeding. Chromosome studies on wild populations of *Drosophila melanogaster* (Stalker, 1976) have found evidence for strong assortative mating for certain chromosomal inversions and their associated phenotypes.

As far as humans are concerned, the evidence regarding the detection of genetic similarity is incomplete. However, it is perfectly reasonable to suppose that a genetic similarity detection mechanism operates in humans too. Most instances of genetic similarity detection in humans are likely to be based on cues which are both complex and multifarious. As we have previously indicated, for example, judges are able to give reliable estimates of a subject's position on a partly inherited personality dimension. Likewise, people can reliably rate themselves. Thus people can compare themselves to others. Unless one adopts the implausible idea that humans detecting similarity are responding purely to the environmental components of the trait, one can conclude that the ability of humans to detect genetically similar others has been demonstrated.

It is widely accepted that human assortative mating occurs on the basis of such characteristics as race, socioeconomic status, physical attractiveness, ethnic background, religion, social and political attitudes, level of eduction, and IQ (Jensen, 1978; Thiessen & Gregg, 1980; Vandenberg, 1972). The median assortative mating coefficient for IQ, for example, averaged over 16 studies involving 3817 pairings is 0.37 (see Figure 2). Although the coefficients appear to be lower than for IQ, there is also assortative mating for personality traits, values, and vocational interests. Vandenberg (1972), Jensen (1978), and Thiessen and Gregg (1980) have reviewed the literature. While most coefficients are extremely small in magnitude, it is consistently found that significantly more are positive than negative. We provide, in Table 2, a summary of additional studies reported by Cattell (1982). While each dimension may add only a fractional amount to the total genetic variance shared by spouses, the cumulative effects, of course, could be considerable.

One recent study examined cross-racial marriages in Hawaii, and found there was more similarity in personality test scores among males and females who married across ethnic groups than those marrying within (Ahern *et al.*, 1981). The authors posit that, given the general tendency toward homogamy, cross-racial/ethnic couples marrying heterogamously on this dimension tend to 'make up' for this dissimilarity by choosing spouses more similar to themselves in other domains than do persons marrying within their own racial/ethnic group.

Assortative mating is also found for a variety of abnormal traits, including criminality (Guze *et al.*, 1970), alcoholism (Hall *et al.*, 1983; Rimmer & Winokur, 1972), the affective

Table 2. Weighted average of 5 studies of assortative mating on 795 U.S. and British married couples collapsed across stable and unstable marriages (adapted from Cattell, 1982)

Sixteen Personality Factor Questionnaire		r	Sixteen Personality Factor Questionnaire		r
A.	Affectia	0.18	M.	Autia	0.23
C.	Ego strength	0.17	N.	Shrewdness	0.13
E.	Dominance	0.22	O.	Guilt proneness	0.13
F.	Surgency	0.23	Q1.	Radicalism	0.21
G.	Superego	0.21	Q2.	Self-sufficiency	0.16
H.	Parmia	0.12	Q3.	Self-sentiment	0.17
I.	Premsia	0.19	Q4.	Ergic Tension	0.13
L.	Protension	0.15			

disorders (Baron *et al.*, 1981), and schizophrenia (Kallman & Mickey, 1946; Kreitman, 1968). It is, perhaps, also surprising that assortative mating coefficients are present for many anthropometric features. Table 3 presents coefficients for a number of straightforward physical variables which have been calculated from data given by Spuhler (1968). Given our discussion of individual differences in chronogenetics (see p. 69), it is of particular interest that Table 3 demonstrates higher assortative mating for *ages* of maximum and minimum weight than for either maximum or minimum absolute weight or weight at present marriage. Nonintuitive assortment is also found for family size and longevity (Thiessen & Gregg, 1980). In contrast to the studies cited above, it is interesting to note that humans do not appear to choose spouses on the basis of similarity of ordinal position within the family (Kemper, 1966; Spuhler, 1968), presumably a nongenetic variable.

Table 3. Assortative mating coefficients for physical features (adapted from Spuhler, 1968)

Physical measure	Number of studies	Number of pairs	r
Hair color	4	1544	0.28
Eye color	5	1774	0.21
Skin color	1	218	0.37
Weight	7	2507	0.25
Maximum weight	1	205	0.08
Age at maximum weight	1	205	0.52
Minimum weight	1	205	0.22
Age at minimum weight	1	205	0.39
Weight at present marriage	1	205	0.23
Body area	1	107	0.41
Lung volume	1	107	0.22
Stature	24	6168	0.21
Cervical height	1	205	0.29
Sternal height	1	649	0.09
Span	4	1924	0.18
Total arm length	1	78	−0.04
Upper arm length	2	726	0.17
Forearm length	4	1920	0.21
Hand length	3	917	0.09
Hand breadth	1	205	0.18
Middle finger length	1	205	0.61

Biacromial breadth	3	927	0.17
Bi-iliac breadth	3	906	0.18
Bicondylar breadth (elbow)	1	205	0.28
Bicondylar breadth (knee)	1	205	0.20
Minimum neck circumference	2	850	0.20
Chest circumference	7	1357	0.14
Pubic-epigastric height	1	628	0.31
Xiphoid-epigastric height	1	653	0.46
Minimum waist circumference	2	852	0.26
Maximum hip circumference	2	312	0.18
Maximum arm circumference	1	205	0.29
Maximum forearm circumference	1	205	0.22
Minimum wrist circumference	1	205	0.55
Maximum calf circumference	1	205	0.07
Minimum ankle circumference	1	205	−0.02
Chest breadth	2	721	0.20
Chest breadth (xiphoid-epigastric)	1	347	0.19
Chest breadth (epigastric point)	1	652	0.19
Chest depth	2	717	0.12
Chest depth (xiphoid-epigastric)	1	343	0.20
Chest depth (epigastric point)	1	647	0.16
Foot length	2	843	0.08
Sitting height	7	1378	0.23
Head circumference	8	1460	0.12
Head length	11	2961	0.07
Head breadth	11	2978	0.10
Head height	2	282	−0.21
Minimum frontal breadth	2	282	0.08
Bizygomatic breadth	9	2449	0.15
Bigonial breadth	2	282	0.12
Physiological face height	1	655	0.09
Total face height	3	910	0.05
Upper face height	3	904	0.15
Nose height	3	930	0.09
Nose breadth	3	931	0.11
Interpalpebral breadth	1	205	0.51
Bipalpebral breadth	1	205	0.25
Interpupillary breadth	1	205	0.20
Ear length	1	205	0.40
Ear lobe length	1	205	0.40
Trunk length	1	107	0.40

Family relationships

It would appear from the previous section that humans assortatively mate on a wide variety of characteristics including both personality and physical qualities. Given that many of the dimensions are partly inherited, it means that spouses are genetically more similar to each other than they are to an average person. According to GST this means that there will be altruism between spouses. This has important implications for family life. Several studies have shown that not only the occurrence but also the stability of relationships can be predicted by the degree of matching on personality characteristics (Bentler & Newcomb, 1978; Cattell & Nesselroade, 1967; Hill *et al.*, 1976; Meyer & Pepper, 1977; Terman & Buttenwieser, 1935*a*, *b*). Moreover, several studies have suggested that degree of assortative mating is correlated with fecundity (Thiessen & Gregg, 1980). Positive relations between number of children and similarity of spouses have been found, for example, for anthropometric variables (Clark & Spuhler, 1959), intelligence test scores (Spuhler, 1967), educational attainment (Kiser, 1968), and even family size (Garrison *et al.*, 1968).

It would appear that it is adaptive for parents to be genetically similar to each other, a conclusion supported by the fact that assortative mating has evolved independently several times over in a variety of different species (Thiessen & Gregg, 1980). The upper limit on the fitness-enhancing character of assortative mating, of course, 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 (Bateson, 1983; van den Berghe, 1983). Optimal fitness, then, consists in selecting a mate who is genetically similar but not actually a relative. It would be of interest to quantify the ideal percentage; van den Berghe (1983) speculates that it is about 12.5%, or the same as that between first cousins.

One expectation from the GST view of assortative mating involves parental care of their offspring. A general prediction is that the more genetically similar the parents are to each other the more genetically similar they and their children will be, and the more within family altruism will occur. Conversely, the less genetically similar the parents are to each other the less genetically similar they and their children will be, and the less within-family altruism will occur. This proposition could be tested in at least two ways: parents who are first cousins may be more protective toward their children than less related parents; and in multiethnic countries, the greater the disparity in ethnicity between parents (and, presumably, on average, the lower the genetic similarity), the less protectiveness and care for the children there will be.

A different test of GST could be made by examining preferences within families. Although each parent will have a minimum of 50% of his or her genes in common with each offspring, upward variations on this percentage will be expected. Some children will be genetically more similar to one parent than the other. This can readily be demonstrated. Suppose that the father gives the child 50% of his genes, 2% of which are shared with the mother, and the mother gives the child 50% of her genes, 8% of which are shared with the father. If this occurred, the child would share 52% of his genes with the mother and 58% of his genes with the father. It is also expected that while siblings will be at least 50% genetically similar to each other, fluctuations will occur. Parents and siblings can be expected to favor the child who is most similar to them. Favoritism within families is an unexplored topic. GST may render it an important one.

Evidence in favor of the GST predictions for differential within-family altruism comes from studies of rhesus monkeys growing up in large, captive, outdoor-living, multimale, multifemale, social troops in which adults of both sexes are promiscuous. Suomi (1982) cited studies in which rhesus mothers reacted differently to their infant being touched, depending on the interloper's degree of relatedness to their offspring: parental half-siblings were chased away less often than were unrelated juveniles. There is also evidence that males 'recognise' their own offspring, for Suomi (1982) reported that, despite male and female promiscuity, males proferred preferential treatment to their own offspring compared with nonoffspring. In these studies, the degree of genetic relatedness was established by blood tests. Suomi (1982) also reported ongoing research in which it was predicted that full siblings would interact more frequently with each other than they would with half-siblings, who, in turn, would interact more frequently with each other than they would with nonrelated peers. The results should provide useful tests of GST in these nonhuman primates.

In the case of humans, children dissimilar to a parent are at risk. A disproportionate number of battered babies are stepchildren (Lightcap *et al.*, 1982). Adoptions are more likely to be successful where the parents perceive the child as similar to them (Jaffee & Fanshel, 1970). Finally, anthropological data has shown that when paternity is uncertain (that is, when there is a considerable risk of low genetic similarly between a man and his wife's children), extreme measures may be taken: more resources may be invested in the children of a sister than of a wife; in many societies studied adultery constitutes grounds for infanticide (Daly & Wilson, 1983).

A genetic basis to friendship

GST may also have predictive power where relationships outside the family are concerned, especially since friendships appear to be formed on the basis of similarity. This holds for similarity as perceived by the friends (La Gaipa, 1977). It also holds for similarity on a variety of measured characteristics. For example, Berkowitz (1969) found that friends tend to be of similar height. It has been more usual to assess similarity by questionnaire. Using such methods, friendship or liking has been linked to similarity of activities (Karylowski, 1976), needs (Seyfried & Hendrick, 1973), personal constructs (Neimeyer & Neimeyer, 1981) and attitudes (Newcomb, 1961). Having reviewed available data, Richardson (1939) concluded that friends were of generally similar personality. Recent data tends to support this view (e.g. Gibson, 1971).

Experimental studies in which perceived similarity has been manipulated have shown it to be a powerful predictor of liking (Byrne, 1971). Apparent similarity of personality, or of any of a wide range of beliefs, has been found to be positively related to liking in subjects of varying ages and from many different cultures (Berscheid & Walster, 1978; Byrne, 1971). Given the above, and on the assumption that friends benefit each other, it seems reasonable to hypothesize that friendship is based on genetic similarity and leads people to help others. Certainly in young children it has been demonstrated that friendship sociograms correspond closely to sociograms based on altruism patterns (Strayer *et al.*, 1979).

Much altruism and friendship is reciprocal in nature. On the basis of comparative anthropological data, Mauss (1954) concluded that three types of obligations are widely distributed in human societies in both space and time: (a) the obligation to give, (b) the obligation to receive, and (c) the obligation to repay. Reciprocal exchanges breed cooperation and good feelings. A failure (or inability) to reciprocate, on the other hand, breeds bitterness and dislike (Fisher *et al.*, 1981). Numerous studies have demonstrated the tendency of individuals to reciprocate favors (Rushton, 1980). The tendency appears to be there even among preschoolers (Strayer *et al.*, 1979). Trivers (1971) has provided an analysis of how reciprocal altruism could evolve through natural selection even in the absence of genetic relatedness. All that is posited is that the performance of an altruistic act will result in a return of altruistic behavior. Axelrod and Hamilton (1981) have proposed a model of cooperative reciprocity that can even be extended to bacteria. Genetic similarity theory interacts with the theory of the natural selection of reciprocal altruism and predicts that the more genes shared by organisms, the easier reciprocal altruism and cooperation will develop. There would be no necessity for strict reciprocity.

We have implied that the function of friendship is to promote altruism. The most direct test of the validity of GST in this context is to see if genetic similarity produces altruism. We know of no appropriate direct test. However, it is possible to ask whether or not altruism is generally increased by actual or perceived similarity. Stotland (1969) reported studies in which subjects observed another person apparently receiving electric shocks. By manipulating the subjects' beliefs about similarity to the confederate, Stotland demonstrated appropriate covariations between this and physiological reactions and reported empathy. Subsequently, Krebs (1975) found that apparent similarity increased not only physiological measures indicating empathy but also willingness to reward the victim.

Ethnic nepotism

We only wish to touch briefly on the notion of ethnicity. Obviously, however, the very notion of ethnicity is based on the idea of extended kinship. Two individuals within the same ethnic group will, on average, be more genetically similar than two from different ethnic groups. The implications of this for relations between ethnic groups may be far reaching. There will be, for example, a biological basis for what van den Berghe (1981) has characterized as 'ethnic nepotism'. Ethnic nepotism is manifest in many ways. It explains why group members prefer to congregate in the same geographical area. Ethnic nepotism also predicts clear patterns of altruism—charitable donations, for example, are predicted to be made in greater quantities within ethnic groups than between them. Many studies have found that people are more likely to help members of their own race or country than members of other races, or foreigners (Brigham & Richardson, 1979; Feldman, 1968).

GST: the strong and weak versions

So far, we have been concerned to demonstrate the possibility that humans and other species may possess the ability to detect genetic similarity in others and to respond differentially to them on this basis. It is now appropriate to make explicit the ways in which this ability could arise (see also Blaustein, 1983; Dawkins, 1982). A strong version of the theory implies that individuals possess recognition alleles which confer the ability to recognize genetic similarity in the absence of previous familiarity or other proximal mechanisms (Dawkins's 1976 'greenbeard effect'). Thus, some phenotypes are inherently more attractive to the organism than are others. A weak version of GST implies that phenotypic preferences are learned.

If a strong version of GST is correct, it follows that similarity based on genetic traits would predict altruism more than similarity based on nongenetic causes. This deduction could be tested in the context of friendship for, as we have suggested, friendship is a means of promoting altruism. Freedman (1979) cites studies in which respondents report that their intention would be to help close kin over distant kin and distant kin over strangers. GST predicts that friends may be responded to with at least as much altruism as distant kin and that the greater the genetic similarity between friends, the more altruism would be expected. To test this prediction estimates of genetic similarity are needed. Biological techniques such as chromosome analysis would be ideal, while blood antigen analysis may provide a reasonable approximation. Cruder estimates are also possible. For example, similarity on major polygenic traits known to have high heritabilities should be more predictive of friends' altruism than similarity on equivalent traits of lower heritability. Unfortunately, the differential heritability of personality traits is by no means established, nor can one estimate the relative number of genes contributing to each trait. However, it may be possible to construct two alternative forms of questionnaire measures of the same personality trait, one composed of items of high heritability, and another of items of low heritability.

The weak version of GST implies that the ability to detect genetic similarity is acquired through a process of exposure to appropriate stimuli. Again, there is evidence that this mechanism may also operate. For example, Zajonc (1980) has experimentally demonstrated that the more familiar a variety of stimuli are (across a range of species, including humans), the greater the preference shown. Bateson (1983) has suggested that sexual preferences in humans may be established early in life by means of an imprinting-like process. Thus, as Blaustein (1983) and Dawkins (1982) discuss, the kin-recognition studies we cited earlier need not be interpreted as 'innate' or genetic. With unfamiliar siblings it may be achieved by familiarity with the individual's own odor or that of a close relative (Dawkins's 1982 'armpit effect'); individuals that smell as self could be distinguished from those that smell differently. Porter and Moore (1981) have demonstrated that bodily odors may also be salient stimuli for kin-recognition among humans. They found that T-shirts worn by individual children were correctly identified by the siblings and mothers of those children through olfactory cues alone. Furthermore, parents correctly distinguished between the odors of otherwise identical shirts worn by two of their children. To the degree to which individual odors and odor preferences are genetically determined, however, a preference for those similar to one's own may support the 'strong version' of GST.

It is possible to envisage situations in which the genetic similarity detection mechanism could be deceived, especially when it is customarily established by imprinting and learning processes. Redican and Kaplan (1978) applied synthetic odors to female squirrel monkeys and found that the social preferences of their infants, as measured by a series of visual discrimination tests, were affected over the first few months of life. Furthermore, preferences for the familiar synthetic rearing odor over an unfamiliar synthetic odor were demonstrated in month 5 post-partum. Finally, Kareem and Barnard (1982), in a study on mice, and O'Hara and Blaustein (1982) in a study on tadpoles, found that previously established kinship interaction preferences disappeared when other animals were allowed to become familiar.

We can at present only speculate on the extent to which these differing processes may operate in humans. Both the strong and the weak versions of GST necessarily involve phenotypic cues; how these mediate behavior, however, requires investigation. It should, in any case, be emphasized that we do not necessarily regard them as mutually exclusive. If there are evolutionary advantages to be derived from the ability to detect genetic similarity, it may well be the case that both distal and proximal mechanisms are employed.

Conclusions

We have reviewed evidence to the effect that personality traits exist, are stable over time, can be reliably measured, and are to some extent inherited. On the basis of these findings, we have developed a theory which suggests, firstly, how personality traits might arise through changes taking place at the gene level, and, secondly, how individuals might employ information about these characteristics in order to detect genetic similarity in others and respond accordingly. Genetic similarity theory is, in our view, capable of providing an extension to certain aspects of sociobiological theory and its applications to human behavior. Indeed, given recent theorizing about gene-culture coevolution (Lumsden & Wilson, 1981), profound interactions between inherited differences in personality and environment are to be expected. The central argument coevolutionary theory is that genes causally affect culture and that culture, in turn, causally affects relative gene frequencies. Thus we expect different personalities to create different environments for themselves and, in turn, differentially thrive therein (Rushton & Russell, 1984). As we have discussed, assortative mating provides a good example of this process (see also, Buss, 1984).

Genetic similarity theory need not, of course, be confined to those areas of psychology which we have chosen to discuss; the theory also has considerable implications for the study of social behavior in small groups and even within and between nations. Much of the evidence we have cited could be explained in a variety of different ways. The idea of GST does not necessarily conflict with these alternative explanations, but appears to be one of a few theories which is currently capable of providing a parsimonious explanation of a whole range of disparate findings.

References

Ahern, F. M., Cole, R. E., Johnson, R. C. & Wong, B. (1981). *Behav. Genet.* **11**, 181–194.

Axelrod, R. & Hamilton, W. D. (1981). *Science* **211**, 1390–1396.

Baron, M., Mendlewicz, J., Gruen, R., Asnis, L. & Fieve, R. R. (1981). *J. affective Disorders* **3**, 167–171.

Bateson, P. P. G. (1982). *Nature* **295**, 236–237.

Bateson, P. P. G. (Ed.) (1983). *Mate choice*. Cambridge: Cambridge University Press.

Bentler, P. M. & Newcomb, M. D. (1978). *J. consult. clin. Psychol.* **40**, 1053–1070.

Berkowitz, W. R. (1969). *Psychological Reports* **24**, 373–374.

Berscheid, E. & Walster, E. (1978). *Interpersonal Attraction* (2nd edition). Reading, MA: Addison-Wesley.

Blaustein, A. R. (1983). *Am. Nat.* **121**, 749–754.

Blaustein, A. R. & O'Hara, R. K. (1981). *Nature* **290**, 246–248.

Blaustein, A. R. & O'Hara, R. K. (1982). *Anim. Behav.* **30**, 1151–1157.

Blaustein, A. R., O'Hara, R. K. & Olson, D. H. (1984). *Anim. Behav.* **32**, 445–450.

Block, J. (1961). *The Q-sort Method in Personality Assessment and Psychiatric Research*. Springfield, IL: Charles C. Thomas.

Block, J. (1971). *Lives through Time*. Berkeley, CA: Bancroft Books.

Block, J. (1981). In (Rabin, A. I., Aronoff, J., Barclay, A. M. & Zucker, R. A. Eds), *Further Explorations in Personality*. New York: John Wiley.

Bouchard, T. J. Jr. (1982). In *1983 Yearbook of Science and the Future*. New York: Encyclopedia Britanica.

Bouchard, T. J. Jr. (1983). Paper presented to the Fourth International Congress on Twin Studies, London, England, June 28 to July 1.

Bouchard, T. J. Jr., Heston, L., Eckert, E., Keyes, M. & Resnick, S. (1981). In (Gedda, L., Parisi, P. & Nance, W. E. Eds) *Twin Research 3: Part B. Intelligence, Personality, and Development*. New York: Alan R. Liss.

Bouchard, T. J. Jr. & McGue, M. (1981). *Science* **212**, 1055–1059.

Breed, M. D. (1983). *Anim. Behav.* **31**, 86–91.

Brigham, J. C. & Richardson, C. B. (1979). *J. appl. Psychol.* **9**, 314–322.

Buss, A. H. & Plomin, R. (1984). *Temperament: Early Developing Personality Traits*. Hillsdale, NJ: Erlbaum.

Buss, A. H., Plomin, R. & Willerman, L. (1973). *J. Personality* **41**, 513–524.

Buss, D. M. (1984). *J. Personality social Psychol.* **47**, 361–377.

Byrne, D. (1971). *The Attraction Paradigm*. New York: Academic Press.

Carey, G., Goldsmith, H. H., Tellegan, A. & Gottesman, I. I. (1978). *Behav. Genet.* **8**, 299–313.

Cattell, R. B. (1982). *The Inheritance of Personality and Ability*. New York: Academic Press.

Cattell, R. B. & Nesselroade, J. R. (1967). *J. Personality soc. Psychol.* 7, 351–361.

Clark, P. J. & Spuhler, J. N. (1959). *Hum. Biol.* **31**, 121–137.

Daly, M. & Wilson, M. (1983). *Sex, Evolution, and Behavior*. Boston: Willard Grant Press.

Davis, L. S. (1982). *Behav. Ecol. Sociobiol.* **11**, 65–70.

Dawkins, R. (1976). *The Selfish Gene*. Oxford: Oxford University Press.

Dawkins, R. (1982). *The Extended Phenotype*. San Francisco, CA: Freeman.

Dobzhansky, T. (1970). *Genetics of the Evolutionary Process.* New York: Columbia University Press.

Dworkin, R. H., Burke, B. W., Maher, B. A. & Gottesman, I. I. (1976). *J. Personality soc. Psychol.* **34**, 510–518.

Eaves, L. J. & Eysenck, H. J. (1974). *Nature* **249**, 288-289.

Eaves, L. J. & Eysenck, H. J. (1975). *J. Personality soc. Psychol.* **32**, 102–112.

Eaves, L. J., Last, K. A., Young, P. A. & Martin, N. G. (1978). *Heredity* **41**, 249–320.

Ellis, L. (1982). *Criminology* **20**, 43–66.

Epstein, S. (1979). *J. Personality soc. Psychol.* **37**, 1097–1126.

Epstein, S. (1980). *Am. Psychol.* **35**, 790–806.

Erlenmeyer-Kimling, L. & Jarvik, L. R. (1963). *Science* **142**, 1477–1479.

Eron, L. D. (1980). *Am. Psychol.* **35**, 244–252.

Eysenck, H. J. (1970). *The Structure of Human Personality.* New York: Macmillan.

Eysenck, H. J. (1976). *Sex and Personality.* London: Open Books.

Eysenck, H. J. (1977). *Crime and Personality* (3rd edition). London: Routledge & Kegan Paul.

Eysenck, H. J. (Ed.) (1981). *A Model for Personality.* New York: Springer-Verlag.

Eysenck, H. J. & Eysenck, S. B. G. (1976). *Psychoticism as a Dimension of Personality.* London: Hodder & Stoughton.

Falconer, D. S. (1981). *Introduction to Quantitative Genetics* (2nd edition). London: Longman.

Feldman, R. E. (1968). *J. Personality soc. Psychol.* **10**, 202–214.

Fishbein, M. & Ajzen, I. (1974). *Psychological Review* **81**, 59–74.

Fisher, J. D., DePaulo, B. M. & Nadler, A. (1981). In (Rushton, J. P. & Sorrentino, R. M., Eds.), *Altruism and heping behavior: Social, personality, and developmental perspectives.* Hillsdale, N. J.: Lawrence Erlbaum Associates.

Floderus-Myrhed, B., Pedersen, N. & Rasmuson, I. (1980). *Behavior Genetics* **10**, 153–162.

Ford, E. B. (1976). *Genetics and Adaptation.* London: Edward Arnold.

Freedman, D. G. (1979). *Human Sociobiology: A Holistic Approach.* New York: The Free Press.

Fulker, D. W. (1981). In (Eysenck, H. J., Ed.), *A Model for Personality.* New York: Springer-Verlag.

Galton, F. (1869). *Hereditary Genius: An Enquiry into its Laws and Consequences.* London: Macmillan.

Garrison, F. J., Anderson, V. E. & Reed, S. C. (1968). *Eugen. Q.* **15**, 113–127.

Getz, W. M. & Smith, K. B. (1983). *Nature* **302**, 147–148.

Gibson, H. B. (1971). *Br. J. soc. clin. Psychol.* **10**, 213–220.

Goldsmith, H. H. (1983). *Child Dev.* **54**, 331–355.

Gottesman, I. I. (1963). *Psychol. Monogr.* **77** (9), whole no. 572.

Gottesman, I. I. (1966). *J. Child Psychol. Psychiat.* **7**, 199–208.

Grau, H. J. (1982). *Anim. Behav.* **30**, 497–505.

Greenberg, L. (1979). *Science* **206**, 1095–1097.

Grotevant, H. D., Scarr, S. & Weinberg, R. A. (1977). *J. Personality soc. Psychol.* **35**, 667–676.

Guze, S. B., Goodwin, D. W. & Crane, J. B. (1970). *Am. J. Psychiat.* **126**, 115–118.

Haldane, J. B. S. (1932). *The Causes of Evolution.* London: Longmans, Green.

Hall, R. L., Hesselbrock, V. M. & Stabenau, J. R. (1983). *Behav. Genet.* **13**, 373–382.

Hamilton, W. D. (1964). *J. theor. Biol.* **7**, 1–52.

Hartshorne, H. & May, M. A. (1928). *Studies in the Nature of Character: Vol. 1. Studies in Deceit.* New York: Macmillan.

Hartshorne, H., May, M. A. & Maller, J. B. (1929). *Studies in the Nature of Character: Vol. 2. Studies in Self-Control.* New York: Macmillan.

Hartshorne, H., May, M. A. & Shuttleworth, F. K. (1930). *Studies in the Nature of Character: Vol. 3. Studies in the Organization of Character.* New York: Macmillan.

Hill, C. T., Rubin, Z. & Peplau, L. A. (1976). *J. soc. Issues* **32**, 147–168.

Holden, C. (1980). *Science* **207**, 1323–1328.

Holmes, W. G. & Sherman, P. W. (1982). *Am. zool.* **22**, 491–517.

Hrdy, S. B. (1977). *The Langurs of Abu: Female and Male Strategies of Reproduction.* Cambridge: Harvard University Press.

Jaccard, J. J. (1974). *J. Res. Personality* **7**, 358-367.

Jaffee, B. & Fanshel, D. (1970). *How They Fared in Adoption: A Follow-up Study.* New York: Columbia.

Jensen, A. R. (1978). In (Osborne, R. T., Noble, C. E. & Weyl, N., Eds.) *Human Variation: The*

Biopsychology of Age, Race and Sex. New York: Academic Press, pp. 51–105.

Kallman, F. J. & Mickey, J. S. (1946). *J. nerv. ment. dis.* **104**, 303–315.

Kamin, L. J. (1974). *The Science and Politics of IQ*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Kareem, A. M. & Barnard, C. J. (1982). *Anim. Behav.* **30**, 594–601.

Karylowski, J. (1976). *Personality soc. Psychol. Bull.* **2**, 71–74.

Kemper, T. D. (1966). *J. Marriage Family* **28**, 346–349.

Kiser, C. B. (1968). *Eugen. Q.* **15**, 98–112.

Krebs, D. L. (1975). *J. Personality soc. Psychol.* **32**, 1134–1146.

Kreitman, N. (1968). *Br. J. Psychiat.* **114**, 699–718.

La Gaipa, J. J. (1977). In (Duck, S. Ed.), *Theory and Practice in Interpersonal Attraction*. London: Academic Press.

Lightcap, J. L., Kurland, J. A. & Burgess, R. L. (1982). *Ethol. Sociobiol.* **3**, 61–67.

Loehlin, J. C. & Nichols, R. C. (1976). *Heredity, Environment, and Personality*. Austin: University of Texas Press.

Lopreato, J. (1981). *Ethol. Sociobiol.* **2**, 113–126.

Lumsden, C. J. & Wilson, E. O. (1981). *Genes, Mind and Culture: The Coevolutionary Process*. Cambridge, MA: Harvard University Press.

Majerus, M. E. N., O'Donald, P. & Weir, J. (1982). *Nature* **300**, 521–523.

Martin, N. G., Eaves, L. J. & Eysenck, H. J. (1977). *J. biosoc. Sci.* **9**, 9–97.

Martin, N. G. & Jardine, R. (1984). *Behav. Genet.* **14** 608–609.

Matthews, K. A., Batson, C. D., Horn, J. & Rosenman, R. H. (1981). *J. Personality* **49**, 237–247.

Mauss, M. (1954). *The Gift: Forms and Function of Exchange in Archaic Societies*. Glencoe, IL: Free Press.

Mednick, S. A., Gabrielli, W. F. & Hutchings, B. (1984). *Science* **224**, 891–894.

Meyer, J. P. & Pepper, S. (1977). *J. Personality soc. Psychol.* **35**, 331–342.

Miller, J. Z. & Rose, R. J. (1982). *J. Personality Soc. Psychol.* **42**, 535–540.

Mischel, W. (1968). *Personality and Assessment*. New York: John Wiley.

Mittler, P. (1971). *The Study of Twins*. Harmondsworth, Middlesex: Penguin.

Moskowitz, D. S. & Schwarz, J. C. (1982). *J. Personality soc. Psychol.* **42**, 518–528.

Neimeyer, G. J. & Neimeyer, R. A. (1981). *J. Res. Personality* **15**, 427–435.

Nevo, E. & Heth, G. (1976). *Experientia* **32**, 1509–1511.

Newcomb, T. M. (1961). *The Acquaintance Process*. New York: Holt, Rinehart and Winston.

O'Hara, R. K. & Blaustein, A. R. (1981). *Anim. Behav.* **29**, 1121–1126.

O'Hara, R. K. & Blaustein, A. R. (1982). *Behav. Ecol. Sociobiol.* **11**, 43–49.

Owen, D. R. & Sines, J. O. (1970). *Behav. Genet.* **1**, 235–248.

Passingham, R. E. (1982). *The Human Primate*. Oxford: W. H. Freeman.

Plomin, R. (Ed.) (1983). *Child Dev.* **54**, 253–435.

Plomin, R. & DeFries, J. C. (1980). *Intelligence* **4**, 15–24.

Plomin, R., DeFries, J. C. & McClearn, G. E. (1980). *Behavioral Genetics: A Primer*. San Francisco, CA: Freeman.

Porter, R. H. & Moore, J. D. (1981). *Physiol. Behav.* **27**, 493–495.

Porter, R. H. & Wyrick, M. (1979). *Anim. Behav.* **27**, 761–766.

Radesater, T. (1976). *Can. J. Zool.* **54**, 1069–1072.

Redican, W. K. & Kaplan, J. N. (1978). *Physiol. Behav.* **20**, 79–85.

Richardson, H. M. (1939). *Psychol. Bull.* **36**, 104–142.

Rimmer, J. & Winokur, G. (1972). *Dis. nerv. Syst.* **33**, 509–511.

Rotter, J. B. (1966). *Psychol. Monogr.* **80**(1), whole no. 609.

Rushton, J. P. (1980). *Altruism, Socialization, and Society*. Englewood Cliffs, NJ: Prentice-Hall.

Rushton, J. P. (1984*a*). In (Royce, J. R. & Mos, L. P, Eds.) *Ann. theor. Psychol.*, Vol. 2. New York: Plenum Press.

Rushton, J. P. (1984*b*). In (Royce, J. R. & Mos, L. P., Eds.) *Ann. theor. Psychol.*, Vol. 2. New York: Plenum Press.

Rushton, J. P., Brainerd, C. J. & Pressley, M. (1983). *Psychol. Bull.* **94**, 18–38.

Rushton, J. P., Fulker, D. W., Neale, M. C., Nias, D. K. B. & Eysenck, H. J. (in press). *J. Personality soc. Psychol.*

Rushton, J. P. & Russell, R. J. H. (1984). *Behav. Brain Sci.* **7**, 740–741.

Rushton, J. P., Russell, R. J. H. & Wells, P. A. (1984). *Behav. Genet.* **14**, 179–193.

Russell, R. J. H., Rushton, J. P. & Wells, P. A. (1984). In (Royce, J. R. & Mos, L. P., Eds.) *Ann. theor. Psychol.*, (Vol. 2). New York: Plenum Press.

Samuelson, P. A. (1983). *J. soc. biol. Struct.* **6**, 3–15.
Scarr, S. (1966). *Child Dev.* **37**, 663–673.
Scarr, S. (1969). *Child Dev.* **40**, 823–832.
Scarr, S. & Carter-Saltzman, L. (1979). *Behav. Genet.* **9**, 527–542.
Seyfried, B. A. & Hendrick, C. (1973). *Sociometry* **36**, 207–220.
Spuhler, J. N. (1967). In (Spuhler, J. H., Ed.) *Genetic Diversity and Human Behavior*. Chicago: Aldine.
Spuhler, J. N. (1968). *Eugen. Q.* **15**, 128–140.
Stalker, H. D. (1976). *Genetics* **82**, 323–347.
Stotland, E. (1969). In (Berkowitz, L., Ed.) *Advances in Experimental Social Psychology*, Vol. 4. New York: Academic Press.
Strayer, F. F., Wareing, S. & Rushton, J. P. (1979). *Ethol. Sociobiol.* **1**, 3–11.
Suomi, S. J. (1982). In (Lamb, M. E. & Sutton-Smith, B., Eds) *Sibling Relationships*. Hillsdale, NJ: Erlbaum.
Teasdale, T. W. & Owen, D. R. (1984). *Nature* **309**, 620–622.
Terman, L. M. & Buttenwieser, P. (1935*a*). *J. soc. Psychol.* **6**, 143–171.
Terman, L. M. & Buttenwieser, P. (1935*b*). *J. soc. Psychol.* **6**, 267–289.
Thiessen, D. D. & Gregg, B. (1980). *Ethol. Sociobiol.* **1**, 111–140.
Trivers, R. L. (1971). *Q. Rev. Biol.* **46**, 35–57.
Vandenberg, S. G. (1962). *Am. J. hum. Genet.* **14**, 220–237.
Vandenberg, S. G. (1972). *Behav. Genet.* **2**, 127–157.
van den Berghe, P. L. (1981). *The Ethnic Phenomenon*. New York: Elsevier.
van den Berghe, P. L. (1983). *Behav. Brain Sci.* **6**, 91–123.
Waldman, B. (1982). *Anim. Behav.* **30**, 700–713.
Waldman, B. & Adler, K. (1979). *Nature* **282**, 611–613.
Willerman, L. (1973). *Child Dev.* **44**, 288–293.
Willerman, L. (1979). *The Psychology of Individual and Group Differences*. San Francisco, CA: W. H. Freeman.
Wilson, E. O. (1975). *Sociobiology: The New Synthesis*. Cambridge: Harvard University Press.
Wilson, R. S. (1983). *Child Dev.* **54**, 298–316.
Wu, H. M. H., Holmes, W. G., Medina, S. R. & Sackett, G. P. (1980). *Nature* **285**, 225–227.
Zajonc, R. B. (1980). *Am. Psychol.* **35**, 151–175.