Historically, the study of behavior and the study of heredity have shared contradictory relationships. Experimental behaviorists long denied the relevance of heredity, whereas for mental testers heredity was of overriding importance (Chase, 1980). With the advent of behaviorism, experimental psychology spent half the century trying to found a science on the denial of heredity, thus making it impossible to understand the diversity and individuality that characterize members of diploid cross-fertilizing species (Hirsch, 1963). Because all conspecifics were assumed to be born alike, they were expected to behave alike in similar circumstances (Hirsch, 1967). By assumption, behavior was independent of heredity. That was the context in which was launched the approach since called Behavior Genetics (Hirsch and Tryon, 1956). During the past 20 years many developments have converged to produce the work summarized by Ehrman and Parsons (1976). We are not concerned here with a review of the literature; instead, we shall consider basic concepts of behavior genetics and some misconceptions that have impeded progress.

Failure conceptually to appreciate and to integrate three fundamentals of biology—individuality, interaction and norm of reaction throughout ontogeny—underlies the long confusion. It is a fact that members of cross-fertilizing species are genotypically unique. Moreover, although it is a platitude to say that heredity and environment interact to produce the phenotype, it is just that interaction that has thwarted attempts to build for ideal organisms, models of behavior analogous to those built for ideal systems. Not only do genotypes differ in response to a common environment, but one genotype varies in response to different environments. Therefore, we need concepts to encompass behavior-genetic relations that are neither isomorphic nor independent: isomorphism might have justified the naive reductionism that led behavior genetics to racism, and independence might have justified behaviorism’s naive environmentalism.
Individuality and Diversity

The understanding of individuality and diversity is essential to the understanding of behavior genetics and its appropriate application. Ordinarily, members of a cross-fertilizing, sexually reproducing species possess a diploid, or paired, set of chromosomes. Most species whose behavior we study are sexually dimorphic. The genetic basis of this dimorphism resides in the distribution of the heterosomes, a homologous pair of sex chromosomes (XX) being present in the mammalian female and an unequal pair (XY), in the mammalian male. Sexual dimorphism guarantees that any population will be variable to the extent of at least two classes.

Chromosomes other than sex chromosomes are called autosomes. Every autosome is normally represented by a homologous pair whose members have identical genetic loci. Alternative forms of a gene any of which may occupy a given locus are termed alleles. If an individual receives identical alleles from both parents at homologous loci, he is said to be homozygous for that gene. If he receives two alleles that differ, however, he is said to be heterozygous for that gene. The process by which a gene changes from one allelic form to another is called mutation.

When a gene is represented in the population gene pool by two allelic forms, the population will be genotypically polymorphic to the extent of at least three classes. That is, individuals may be homozygous for either of two alleles or heterozygous for their combination.

Study of populations has revealed that often extensive series of alleles exist for a locus. Well-known examples are the three (actually more) alleles at the ABO-blood locus in man and a dozen or more alleles at the white-eye locus in Drosophila. In general, for each locus having \( n \) alleles in the gene pool, a population will contain \( n(n + 1)/2 \) genotypic classes. Mutation ensures variety in the gene itself.

Sexual reproduction involves meiosis—a complex cellular process resulting in a meristic division of the nucleus and formation of gametes (reproductive cells) having single genomes (a haploid chromosome set). One homolog in every chromosome pair in our diploid complement is of paternal origin and the other is of maternal origin. In meiosis, the homologs of a pair segregate and a gamete receives one from each pair. The assortment to gametes of the segregating homologs occurs independently for each pair. This process ensures diversity because it maximizes the likelihood that gametes will receive unique genomes. For example, gametogenesis in Drosophila willistoni produces eight alternative gametic genomes, which, if we represent the three chromosome pairs of this species by \( Aa, Bb \) and \( Cc \), we designate \( ABC, ABc, AbC, aBC, Abc, aBc, abc \). In general, \( n \) pairs of chromosomes produce \( 2^n \) genomes (if we ignore the recombination of gene linkages that actually occurs in crossover exchanges between chromosomes). Man, with 23 chromosome pairs, produces gametes with any of \( 2^{23} \) alternative
genomes. This makes vanishingly small the chances that even siblings (other than monozygotes) will be genetically identical. Since the gamete contributed by each parent is chosen from $2^{23}$ alternatives, the probability that the second offspring born to parents will have exactly the same genotype as their firstborn is $(1/2)^2$, or less than 1 chance in over 70 trillion! The probability that two unrelated individuals will have the same genotype, then, is effectively zero.

The argument for the genotypic uniqueness of members of populations is even more compelling, since other conditions such as physiological systems and ontogenic development contribute significantly to this already great individual diversity. However, natural selection works to eliminate diversity in prodigious quantities and to maintain a narrow fit to the available niche. For example, rodents (rats, mice, hamsters, etc.), of which there are over 300 genera and almost 3000 species, have litters varying in size from one or two up to 15 or 16 with an average below ten. Taking, for convenience, the number eight, which we obtain in some of our mouse and rat colonies, we can calculate how a population might grow:

<table>
<thead>
<tr>
<th>Generation</th>
<th>Parents</th>
<th>Offspring</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>$N_0$</td>
<td>2</td>
<td>8</td>
<td>$8 + 2 = 10 = 5 \times 2$ pairs</td>
</tr>
<tr>
<td>$N_1$</td>
<td>10</td>
<td>40</td>
<td>$40 + 10 = 50 = 25 \times 2$ pairs</td>
</tr>
<tr>
<td>$N_2$</td>
<td>50</td>
<td>200</td>
<td>$200 + 50 = 250 = 125 \times 2$ pairs</td>
</tr>
<tr>
<td>$N_3$</td>
<td>250</td>
<td>1000</td>
<td>$1000 + 250 = 1250 = 625 \times 2$ pairs</td>
</tr>
<tr>
<td>$N_4$</td>
<td>6250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_5$</td>
<td>31,250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_6$</td>
<td>156,250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_7$</td>
<td>781,250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_8$</td>
<td>3,906,250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_9$</td>
<td>19,531,250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N_{10}$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In general,

$$N_t = N_0 (1 + p)^t$$

$$N_{10} = 2(1 + 4)^{10} = 19,531,250 \approx 20\text{ million}$$

Given good conditions they reach sexual maturity in a few weeks, closer to 30 than to 60 days, and gestation lasts only about 20 days. So it is possible to have over three generations a year and individuals can live two or more years. It is no exaggeration whatsoever to say that the reproductive capacity of a single couple might reach 20 million within the time of their own possible lifespan, because not only can their grandchildren be actively reproducing within the first year, but they themselves can have three or four litters in a year. Of course, it was precisely this insight
transmitted by Malthus to both Darwin and Wallace which provided the key to evolution. Darwin even calculated that the slowest breeding mammal, the elephant, would have produced 15 million descendants from a single couple in five centuries. The message of the Malthusian calculation is simple: the reproductive capacity of any species is sufficient to flood the planet, and yet none of them does. Selection pressure is intense. Only a small proportion of the possible progeny survive in each generation.

However, man in civilization is doing ever more to limit reproduction and preserve the diversity, and through technology is spawning an ever increasing variety of cultural niches to accommodate his diversity. We are becoming maybe the most cosmopolitan species in the history of the animal kingdom. This is not a claim that human evolution has stopped—only that the pattern of selection pressures is no longer tailoring our species to a narrow niche of aggressive hunting and food gathering.

**Concepts in Quantitative Genetics**

**Polygenic Traits Versus Major Gene Effects**

In transmission genetics (the study of the inheritance of genes), a distinction is made between so-called major gene effects and polygenic effects. The study of the inheritance of major genes is the domain of classical Mendelian genetics, while the study of the inheritance of traits with continuous variation and polygenic correlates is the domain of quantitative genetics.

A major gene can be defined as an allele that alters in some distinctive way the "normal" or wild-type expression of a trait. The expression of a major gene is an either-or effect with either the wild-type or the "deviant" phenotype being manifested. The heterozygote may, however, show an intermediate phenotype. If appropriate matings are made, the major gene will show Mendelian segregation ratios. Examples of major genes in man are sickle-cell anemia, Huntington's chorea (a degenerative disease of the central nervous system) and phenylketonuria.

Polygenic traits are ones in which there is no obvious wild-type expression. Instead, these traits show continuous variation among all members of a population that is not due solely to environmental influences. Examples in man are height, pigmentation and intelligence. Polygenic correlates are often rated on a finer scale than are major gene effects. A trait like height, which is studied as a polygenic trait, may also be studied, in some cases, as a major gene effect. For example, "normal" height is the wild-type phenotype for a major gene like achondroplasia (human dwarfism).

By definition, polygenic traits are specified by a number of loci. These loci are usually independently assorting and each has two or more alleles. The loci act as if they form a related system so that each allele adds or subtracts a certain quantitative increment to the trait being measured. Polygenicity does not imply that there are any underlying interlinked biochemical systems between alleles,
perfect additivity of alleles, or the presence or absence of dominance. Polygenic systems may act in an additive manner, but this does not imply that any one allele adds one inch to height or five IQ points to intelligence, even though the mathematicogenetic models make it appear that such is the case.

The Case of One Locus with Two Alleles
To illustrate the thinking and mathematics behind quantitative genetics, we now consider the simplest case of one gene having two alleles at its locus. We also assume a random mating population with no migration, selection or mutation. That is, the population is in Hardy–Weinberg equilibrium, so that the genotypic frequencies are constant from one generation to the next. The two alleles are $A_1$ and $A_2$ with frequencies of $p$ and $q$, respectively.

Since the population is in Hardy–Weinberg equilibrium,

\begin{align}
  p + q &= 1 \\
  p^2 + 2pq + q^2 &= 1 
\end{align}

The frequencies of the three genotypes ($A_1A_1$, $A_1A_2$ and $A_2A_2$) are given by the square of the allelic array [Equation (1)]. This gives the genotypic array [Equation (2)].

The Average Effect of a Gene
Earlier we stated that polygenic systems are those genetic systems that act as if each allele adds or subtracts a certain increment to the phenotypic expression of a trait. The average effect of a gene is the estimated increment that a given allele from a parent contributes to the progeny phenotype. Parents do not transmit genotypes to their progeny. Genotypes are broken up by segregation and independent assortment. Therefore, new genotypes form in every generation. Predictions of progeny genotypic values can be made only if the parental genotypic values can be expressed in terms of genic values (i.e., the part of the genotypic value due to a single allele).

Each genotype has a corresponding observed phenotypic value designated here by $Y_{11}$, $Y_{12}$ and $Y_{22}$ (Table I). The genotypic value for each genotype is measured on a scale as deviations from a midpoint. The midpoint (MP) is defined as the scale value exactly intermediate between the phenotypic values of the two homozygotes $A_1A_1$ and $A_2A_2$. Thus, the genotypic values of the homozygotes are equal but opposite in sign. The genotypic value of the heterozygote is defined as $d$. (See Figure 1.)

Breeding Value
Since parents transmit only half their genes to their progeny, the mean genotypic value of their progeny is a function both of the
In terms of dominance relationships:

- if \( d = 0 \), there is no dominance.
- if \( d > 0 \) and \( d < |a| \), there is partial dominance.
- if \( d = |a| \), there is complete dominance.
- if \( d > |a| \), there is overdominance.

**Figure 1**

Arbitrarily assigned genotypic values [after Falconer (1960)].

Parents' alleles and of the average gene effects. The value assigned to an individual, as measured by the mean genotypic value of the progeny, is the breeding value or additive genetic value. The breeding value can be measured directly and, like the average effect of allele substitution, is a population parameter.

The additive genetic, or breeding, value can be expressed in terms of the average gene effects. It is simply the sum of the average effects of each allele at a locus. If more than one locus is involved, the breeding value is obtained by the summing of the average effects over alleles at all loci. This is the additive portion of the genotype and is the most important, since it determines the resemblance between relatives. The common elements relatives can share are independently acting genes, not unique combinations.

**Dominance Deviation**

For a trait in any population, it is possible to calculate the genotypic value of an individual as the deviation from the trait-scale midpoint and to calculate the breeding value from the mean of his (or her) offspring values. The observed difference between the genotypic value is the dominance deviation. For one locus with two alleles, the genotypic value is composed solely of the additive genetic value and the dominance deviation: \( G = A + D \). (Since both the additive genetic value and the dominance deviation are expressed as deviations from the mean, the genotypic values must be expressed the same way, Table I.)

A most important distinction is that between dominance as it applies to alleles at a locus and dominance deviation as described here. The presence of dominance deviation does depend on the presence of dominance at a locus (i.e., if \( d = 0 \), there is no
dominance deviation, e.g., Figure 1), but it also depends on the allelic frequencies in the population. The dominance deviations are given in terms of allele frequencies in Table I.

The dominance deviation is a population parameter and not a measure of the amount of dominance because it depends on both allelic frequencies and the magnitude of \( d \). Unlike dominance, which is manifested only in the heterozygote, dominance deviation is found in all genotypes.

The dominance deviation and the additive genetic values are uncorrelated. Therefore, knowing the additive value of any genotype does not help to predict the amount of its dominance deviation.

**Epistatic Interaction Deviation**

When only one locus is involved, the genotypic value can be expressed in terms of the additive value and the dominance deviation value: \( G = A + D \). With two or more loci, the genotypic value can be expressed as the additive and dominance deviation values summed across all loci: \( G = \sum A + \sum D \). If there is interaction between the loci, a third term, an interaction term, has to be introduced. Hence, \( G = \sum A + \sum D + \sum I \) (or just \( G = A + D + I \)).

These interactions are of several types, and the number of types of interactions increases with the number of loci. For two loci there are three types of interaction: interaction between additive values (\( AA \)), interaction between dominance deviation values (\( DD \)) and interaction between additive and dominance deviation values (\( AD \)). For more than two loci, the number of interactions rapidly increases. Thus, for three loci, there are also interactions among all three additive values (\( AAA \)), between two additive values and one dominance deviation value (\( ADA \)) and so forth. Since these separate interactions are difficult to measure, all interactions are usually clustered under the symbol \( I \).

**Variance**

The study of genetics is properly the study of variation and of variance. Where a trait shows no phenotypic variance, there is no evidence that it has genetic correlates. One important aspect of

**Table I**

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Frequency</th>
<th>Phenotypic value</th>
<th>Genotypic value</th>
<th>Genotypic value taken as deviation from the mean</th>
<th>Additive genetic value</th>
<th>Dominance deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A_1A_1 )</td>
<td>( p^2 )</td>
<td>( Y_{11} )</td>
<td>( a )</td>
<td>( 2q(a - pd) )</td>
<td>( 2\alpha_1 = 2q\alpha )</td>
<td>( -2q^2d )</td>
</tr>
<tr>
<td>( A_1A_2 )</td>
<td>( 2pq )</td>
<td>( Y_{12} )</td>
<td>( d )</td>
<td>( a(q-p) + d(1-2pq) )</td>
<td>( \alpha_1 + \alpha_2 = (q-p)\alpha )</td>
<td>( 2pqd )</td>
</tr>
<tr>
<td>( A_2A_2 )</td>
<td>( q^2 )</td>
<td>( Y_{22} )</td>
<td>( -a )</td>
<td>( -2p(a + qd) )</td>
<td>( 2\alpha_2 = -2pq\alpha )</td>
<td>( -2pq^2d )</td>
</tr>
</tbody>
</table>
quantitative genetics is the partitioning of the total phenotypic variance into smaller variance components.

The most common partitioning of variance is the division of total phenotypic variance into environmental and genotypic components. This partitioning is of little interest to geneticists but is used a great deal by psychologists.

The first partitioning of phenotypic variance is into genetic and environmental components: \( V_p = V_g + V_e \). The estimation of these two components is not easy. One technique involves the use of isogenic populations—either homozygous inbred lines or the \( F_1 \) cross between two such inbred lines. These isogenic populations must be reared in a variety of environments. Since all individuals in a single population have the same genotype, any variation between them must be due to environmental differences. Heterogenic populations must be reared in the same variety of environments. The heterogenic population variance is due to both genetic and environmental causes. The difference between the variances of the heterogenic \( (V_g + V_e) \) and isogenic \( V_e \) populations provides the estimate of the total genetic variance.

**Heritability**

No discussion of quantitative genetics would be complete without a consideration of heritability, especially since heritability is used in the human intelligence literature without regard for its conceptual basis. There are two types of heritability, broad \( (H^2) \) and narrow \( (h^2) \), each with a different conceptual basis and with a distinct and different usage. They are unfortunately confused and the symbol \( (h^2) \) is often misused to represent both of them.

Broad heritability \( (H^2) \) is the ratio of the total genetic variance (plus any genotype–environment correlation) to the total phenotypic variance: \( H^2 = V_g/V_p \) where \( V_g = V_a + V_d + V_i \). Since the genetic variance is a function of the allele frequencies of the gene correlates of a trait, any estimate of \( H^2 \) is valid only for a specified population (i.e., specified allele frequencies) and a particular environmental variance. Broad heritability is not a fixed quantity. On the contrary, it is a population descriptor that varies with allele frequencies and with changes in the environment.

Broad heritability is most often misused by psychologists in assuming it is a nature–nurture ratio or index to the causes of a trait. Heritability, however, describes the apportionment of variance: it does not describe how much of a trait is determined by heredity for any individual. Nor does it describe the average influence of heredity in determining the level of trait expression in a population. Broad heritability estimates only the contribution of genetic factors (additive, dominance deviation, interaction and genotype–environment covariance) to the total phenotypic variance for one trait in a specified population. Broad heritability is of little interest to geneticists and is only used (or misused) as described above.
Narrow heritability \((h^2)\) is the ratio of additive genetic variance to the total phenotypic variance. Its value, like that of \(H^2\) depends on the allelic frequencies in the population and applies only to a specified environmental variance. Narrow heritability is one of the most important parameters in quantitative genetics, since it can be used to predict the results of selective breeding for a given level of trait expression in a particular environment.

Only in one case, where \(d = 0\) and there is no interaction variance, do broad and narrow heritability have the same value. It is clearly wrong to use the terms interchangeably unless the above two conditions are explicitly stated. In most cases, \(H^2\) and \(h^2\) estimate different values, and \(H^2\) can be used at best only as an estimate of the upper limit of \(h^2\).

**Resemblances Among Relatives**

Genetically, the relationship of resemblance among members of a family is based on a probability of sharing identical alleles at each locus. Progeny receive one homologue of every chromosome pair from each parent. Therefore, an offspring shares with each parent 50% identical alleles with a probability approaching unity. On average, siblings also share half their alleles, but their relationship is more complex, because it is what we usually mean by probabilistic. Given in the gene pool four alleles at a locus and two parents, each heterozygous for a different combination of two of the four alleles, consider, for example, the mating \(A_1A_2 \times A_3A_4\), which can produce progeny of genotypes \(A_1A_3, A_1A_4, A_2A_3, A_2A_4\). A pair of siblings could have 16 possible genotypic combinations, such as,

1. \((A_1A_3, A_1A_3)\)
2. \((A_1A_3, A_2A_3)\)
3. \((A_1A_3, A_2A_3)\)
4. \((A_1A_3, A_2A_4)\)

Combination 1 has 100% identity; 2 and 3 have 50% identity; and 4 has 0% identity. Since the other 12 possible combinations have a similar distribution of percentages of identity, the average identity is 50%. Note, however, that unlike parent–offspring identity, which has no range and only one value, the sibling identity value is averaged over a range from 0 to 100%. The same relationship prevails at every independent autosomal locus. Relatives such as half-siblings, half-cousins and grandparent–grandchild are less closely related because they have a smaller range and lower average probabilities for sharing identical alleles. Attempts to estimate \(h^2\) for a trait (in some population) are best made by means of studies of the resemblances of relatives, because it is the additive gene effects that relatives share. At least, that is the usual recommendation. Therefore, we next consider how this is done and how the results thus obtained can be interpreted.

*Parent–offspring correlation.* Parent–offspring correlations can be used to estimate the narrow heritability. It is easier to use the regression of the offspring on the parents than intraclass correla-
tions since we are dealing with pairs of observations rather than with observations between groups:

\[ b_{op} = \frac{\text{cov}(op)}{V_p} \]

where \( b_{op} \) is the regression of offspring on parent, \( \text{cov}(op) \) is the covariance of parent and offspring, \( V_p \) is the parental variance.

The covariance between the parent and the offspring is the sum of the cross-products of the genotypic values. If we ignore epistatic interaction, the genotypic value of the parent is \( G = A + D \). The genotypic value that an offspring shares in common with a parent is \( \frac{1}{2} A \), since the child inherits half the parental alleles and none of the parental dominance deviation.

\[ \text{cov}(op) = \Sigma \left( \frac{1}{2} A \right) (A + D) \]

\[ \text{cov}(op) = \frac{1}{2} \Sigma (AA) + \frac{1}{2} \Sigma (AD) \]

since additive and dominance deviation values are uncorrelated:

\[ \text{cov}(op) = \frac{1}{2} (AA) \]

The covariance of \( A \) with \( A \) is simply the additive variance. Thus, the regression of offspring on parent gives an estimate of one-half the narrow heritability:

\[ \text{cov}(op) = \frac{1}{2} (AA) = \frac{1}{4} V_a \]

\[ b_{op} = \frac{\text{cov}(op)}{V_p} \]

\[ b_{op} = \frac{\frac{1}{4} V_a}{V_p} \]

\[ b_{op} = \frac{1}{2} h^2 \]

The measurement of the regression of the offspring on the parent gives an unbiased estimation of the narrow heritability. This is one of the easiest sets of measurements to make, but to be valid the measurements have to be made on both parent and offspring at the same age under the same environmental conditions and, of course, with replication of genotype observations. This may be practical for fast-maturing species but is certainly not so for man.

**Midparent–offspring correlations.** A second way to look at the resemblances between parent and offspring is to look at the resemblances between the offspring and the mean value of the parental trait expression. This mean value is usually designated the midparent. The covariance between the midparent and offspring also estimates \( \frac{1}{2} V_a \). The regression of the offspring on the midparent estimates \( h^2 \), since the variance of the midparent is only one-half the variance of the single parent:

\[ b_{op} = \frac{\frac{1}{2} V_a}{\frac{1}{2} V_p} \]

\[ b_{op} = h^2 \]

Midparent–offspring correlations have the same disadvantages for human studies as single-parent–offspring correlations. In addi-
tion, they require that additional measurements be made so that the scores of both parents are known.

**Half-sib correlations.** When one is dealing with the correlations among siblings, its often easiest to compare the variances between and within the families. Thus, one uses the intraclass correlation \(t\) rather than regression.

The between-group or between-family variance is that variance common to all members of a group or family. The within-family variance is that variance among family members and is a measure of the uniqueness of individuals.

The intraclass correlation can be used to estimate the heritability as illustrated in the path diagram in Figure 2. The correlation between the phenotypic values of the half-sibs Progeny 1 and Progeny 2 is a function of the alleles that they share in common and the relationship between those common additive genetic values and their phenotypes. Half-sibs have an average genetic relationship of \(\frac{1}{4} (r = \frac{1}{4})\). The regression of phenotype on the additive genetic value is the square root of the heritability \((\sqrt{\frac{1}{4}} = h)\). The intraclass correlation between half-sibs estimates \(\frac{1}{4} h^2\).

The correlation between half-sibs is another unbiased estimator of the narrow heritability. It is easier to measure half-siblings at the same age, but they are fairly rare. In addition, it is difficult to equalize the environments of half-sibs to make the measurements of the traits meaningful, and there remains the problem of replication.

**Full-sib correlations.** The problem of estimating \(h^2\) becomes much more complicated in the case of resemblance between full-sibs.
Full-sibs have an average additive genetic correlation of \( r = \frac{1}{2} \), so it seems logical that they would estimate \( \frac{1}{2} h^2 \), since half-sibs estimate \( \frac{1}{4} h^2 \). As explained above, however, full-sibs inherit alleles from both parents, so that they have the possibility of having the identical genotype at any given locus. Full-sibs have not only one-half of their additive genetic variance in common but also share one-quarter of their dominance deviation variance. Since full-sibs are more highly related than one-half, the intraclass correlation gives a biased estimate of \( h^2 \).

Full-sib correlation is one of the easiest to measure. It is easy to locate full-sibs, the environments can be fairly well equalized, and often sibs can be measured at the same age, but there remains the problem of replication. Since the full-sib correlation is biased by dominance deviation variance, it can be used only as the upper limit to heritability.

**Difficulty of measurement.** In experimental studies, one attempts to set up a series of replicated controlled matings to get several different estimates of the heritability. A common mating scheme is to mate a number of sires to a greater number of dams. This will give a series of full-sib families for each dam, both full- and half-sib families for each sire, and unrelated families among sires. By a nested analysis of variance, estimates of heritability based on full- and half-sib correlations can be obtained. The half-sib estimate is unbiased but imprecise; the full-sib estimate is more precise but biased. In general, an average of the two estimates of \( h^2 \) is taken as a rough estimate of the heritability.

For human studies, such nested analysis is nearly impossible. Controlled human matings with replication are not feasible. Such a nested system might be possible in polygamous cultures or with second marriages where there are children by each marriage, but this offers little hope for human heritability estimates. The other available techniques also have severe limitations. Full-sibs are the easiest to measure but provide a biased estimate. Half-sib pairs are difficult to locate and have the problem of the controlling of environments. Parent–offspring correlations are useful but only if measurements are obtained at the same age.

Even with well-designed studies, the estimation of \( h^2 \) is not very accurate and the standard error is usually quite large. Klein, DeFries and Finkbeiner (1973) have presented tables of standard errors for different methods of estimating \( h^2 \). The numbers of subjects that would have to be measured are quite large. For example, to get an estimate of \( h^2 = 0.6 \) within 95% confidence intervals, for any method appropriate to human populations, would require measurements on a minimum of 800 individuals from 400 different families. To our knowledge, no studies of this magnitude have been attempted in human populations.

Heritability is an important parameter in quantitative genetics applied to agriculture. If the \( h^2 \) of a trait in a population is known
then some prediction can be made about the response of that population to selective breeding for some expression of that trait in the environment in which the heritability was calculated. Heritability values \( h^2 \) and \( H^2 \) are not nature–nurture ratios. There are no such ratios, since each genotype is unique and has developed a phenotype through continued idiosyncratic interaction with the environment.

Theoretical Misconceptions

Our misconceptions are due to the piling up of errors in theoretical and methodological foundation literature. In what follows we give a few examples and correct several misconceptions.

A literature has built up that, though incorrect, is widely believed to provide answers to an unanswerable question: What are the proportional contributions of heredity and environment to trait expression in human populations and to differences among races in the expression of those traits? The readers have been informed that the unfortunate situation, in which Jensen revived the discredited argument for "Negro" racial inferiority by claiming that both "intelligence" itself and average IQ differences between races were 80% genetic, is "the most important paper in [psychology] since Pavlov and Freud . . . a masterful summary of evidence that has been gathering for several decades" (McConnell, 1970). This occurred in an intellectual climate permeated by the myth of two cultures: "A distinction often made between science and the humanities is that in science, a cumulative discipline, one need study only the latest paper on any subject to obtain all the background necessary for further investigation" (Bonner, 1961).

We have since learned to our chagrin, however, that believers of this fallacy also assumed that scientists act with an integrity and a humility, which Jensen (Hirsch, 1976) and others, (Stoker 1976, see Burt discussion below) have not shown.

Formula Error in Determining Heritability from Twin Data

Jensen (1967) presented the formula \( h^2 = (r_{MZ} - r_{DZ})/(1 - p_{oo}) \) without any theoretical justification that it measures heritability, broad or narrow. In this formula, \( r_{MZ} \) and \( r_{DZ} \) are phenotypic correlations between monozygotic and dizygotic twins respectively and can be observed directly. However, \( p_{oo} \), the genetic correlation between sibs, cannot be observed. It can be obtained from their phenotypic correlation if heritability is known, which of course it is not.

To overcome this difficulty Jensen (1967) suggested that the formula \( p_{oo} = (1 + p_{pp})/(2 + p_{pp}) \), where \( p_{pp} \) is the genetic correlation between mates, should be used to find \( p_{oo} \). He gives Li (1955, Chapter 13) as reference for this formula. It, however, does not appear there and when consulted, Professor Li replied (private communication to AV) that he had never seen it before it was
brought to his notice. This formula lacks theoretical justification. Moreover, it does not resolve the problem of finding genetic correlation. In order to use it, we require the genetic correlation between mates which can be obtained from their phenotypic correlation only if heritability is known. Thus, to use Jensen's formula for finding heritability of a trait, a previous estimate of that heritability is required.

How did Jensen (1967) resolve the dilemma? He assumed that the genetic correlation between mates is .25 without revealing how this figure was obtained from their phenotypic correlation of .6. The value of $p_{MZ}$, actually $p_{w0}$, which Adams et al. (1976) recently used, is based on this assumed genetic correlation. The value of .8 for the broad heritability of IQ, so often quoted by Jensen and others, is also based on this value. It is difficult to find a scientific justification for the method or to have any faith in estimates of heritability based on it.

**Genotype–Environment Interaction**

Jinks and Fulker (1970) described a method for estimating genotype–environment interaction from twin data. Using this method, they concluded that no such interaction exists for IQ. Jensen (1970) applied their method to studies of monozygotic twins reared apart (see below) and also concluded that no such interaction exists. Jensen's replication and the nonexistence of genotype–environment interaction for IQ are often cited. Two points concerning this method need to be stated clearly, and Professor Jinks has generously accepted both.

To begin, there was a slip in the algebra on p. 314 of Jinks and Fulker's paper. When their error is corrected and incorrect covariance values are replaced by correct ones, there is no unambiguous interpretation of the zero covariance of sums and differences from MZ twin scores (on which their estimate of zero heredity–environment interaction depended). Their method should always yield zero correlation between genotype and environment if the distribution of twin scores is bivariate normal, i.e., the result is an artifact. Thus investigators, who find no correlation between the sums of twin IQ scores and their absolute differences, only confirm something that is expected on statistical reasoning with no genetical meaning. It is therefore not surprising that all replications of the method show heredity–environment interaction for IQ to be approximately zero. Needless to say, this proves nothing except that the twin distribution for IQ might be approximately bivariate normal.

Secondly, Jensen's (1970) replication combined data from four well-known studies. Not only does his attempt fail because of the faulty method, but also because pooling those data was unjustified.
Norm of Reaction

Interaction and norm of reaction describe aspects of the complex genotype–phenotype relationship. The latter focuses on the fact that a genotype may develop different phenotypes in different environments. The former includes the latter and considers at one time many genotypes and many environments. For an array of genotypes and various sets of environmental conditions, it calls attention to how, out of the variety of possible distributions of phenotypic outcomes, the particular one obtained will depend on which genotype develops under which conditions. Haldane analyzed the interaction concept and formulated its quantitative interpretation. He showed that $m$ genotypes in $n$ environments generate $(mn)!/(m!n!)$ kinds of interaction. McGuire and Hirsch (1977) show that the proportion of these permitting generalization is small and diminishes as $m$ and $n$ grow. The latest failure to justify a generalized superiority–inferiority hierarchy (Plomin et al., 1976) does so by baldly asserting the following nonsense: “This truism for the individual (heredity–environment interaction) is simply false for individual differences in a population.”

Within and Between Population Heritabilities

The independence of mean and variance of a normal distribution is a well-known statistical fact. On the relationship between the heritability of a population and the mean difference among populations, the position is succinctly stated by Bodmer (1975), who says, “Heritability measurements within a population bear no relationship to the question of genetic differences between populations.” Yet DeFries (1972) claims that such a relationship exists. His solution is illusory, however, and the results of his proposed analysis would be uninterpretable, because the formulas used make the following assumptions: (1) that the conditions under which human populations breed are comparable to the controlled conditions of animal and plant breeding experiments; (2) that the variances of two populations compared are equal; (3) that populations are mating randomly; and (4) that populations share a common environment. None of the foregoing assumptions can be justified. Human breeding conditions are not controlled. In the case of IQ, where DeFries (1972) used Falconer’s formulas, the assumption about variances is not valid. Even Jensen (1972) reported the phenotypic variance of IQ in the American black population to be about two-thirds of that in the white population. Furthermore, with respect to IQ, both populations mate assortatively, not randomly, which will result in correlation between genotypes. Also, the two populations have different environments and might have different coefficients of assortative mating. There is no gainsaying that, so long as heredity and environment interact or
are correlated, both of which occur with human intelligence, heritability cannot be defined.

**Misapplication of Heritability**

Heritability, broad as well as narrow, is a technical term in quantitative genetics. Moran (1973) in a brief but excellent note pointed out that this precise concept cannot be used when genotype–environment covariance is present. Moreover, if the environmental component is correlated with families, Fisher’s (1918) model cannot be used. Since Morton (1976) has now alleged that “comments by Moran on genotype–environment covariance . . . were subsequently corrected,” by Holroyd, it is important for it to be known that Moran was correct and Holroyd was confused. Moran’s (1973) simple analysis shows that, “for characteristics such as human intelligence in which genetic and environmental components are correlated, ‘heritability’ cannot be defined. . . .” Holroyd (1975) has objected, contending that “there is no logic for . . . [Moran’s] statement since if heritability \( h^2 \) is defined as the ratio of the total genetic variance to the total phenotypic variance. . . . This simply puts the covariance term in with the non-genetic variance which, in fact, is the usual procedure in Psychology” (Jensen, 1972). Holroyd’s “correction” was based on what he alleged psychologists usually do—include the genotype–environment covariance with the nongenetic variance—and he cited Jensen as his authority. Jensen (1972), however, had stated exactly the opposite of what Holroyd attributed to him, for he had said, “Since most estimates of heritability of intelligence are intended to reflect the existing state of affairs, they usually include the covariance in the proportion of variance due to heredity” (italics added).

So we see that use of heritability in human behavior genetics has been indiscriminate and counterproductive. In quantitative genetics, heritability in the narrow sense \( h^2 \) is defined as the ratio of additive genetic variance to phenotypic variance in the absence of either correlation or interaction between genotype and environment. However, when correlation exists, either (1) between genetic and environmental contributions to trait expression or (2) between environmental contributions to trait expression in both members of a parent–child or sib pair, heritability is not defined. Furthermore, when heritability can be defined, for example in well-controlled plant and animal breeding experiments, it has no relevance to measured differences in average values of trait expression between different populations: heritability estimates throw no light upon intergroup comparisons!

Obviously, heritability as genetically defined is not suited to the requirements of behavior genetics, for which other concepts will have to be developed. It also needs to be clearly understood that “High or low heritability tells us absolutely nothing about how a
given individual might have developed under conditions different from those in which he actually did develop. Heritability provides no information about norm of reaction” (Hirsch, 1970).

The Misunderstanding of Regression
Since regression of offspring score from parental value toward the population mean has so often been misconstrued as providing evidence for genetic determination of trait expression [see Vetta (1975) for examples], we explain why that is a misinterpretation. Consider the simplest conceivable case of complete genotype–phenotype isomorphism, no dominance and panmixia, and let a group of fathers be selected to reproduce whose average value on a trait is \( x \), measured as a deviation from the population mean. They will have offspring whose average value on the same trait scale is \( \frac{1}{2} x \). The observed regression arises because both sexes contribute in reproduction, but we selected fathers only. Since mothers were unselected, their average deviation value on the trait is zero. The average progeny value is the average of the two parental values, or \( \frac{(x + 0)}{2} = x/2 \). However, if we select the sexes so that \( x \) is the average for both mothers and fathers, then the progeny average will be \( \frac{(x + x)}{2} = x \), and there will be no regression to the mean. That is, when the genetic contribution from both parents is taken into account, regression to the mean is not an index of genetic determination and genetic determination does not imply that there will be regression to the mean.

Jensen (1973), comparing the IQs of sibs of chosen groups of children from two ethnic groups, showed some confusion concerning sibling regression to the mean. Thoday (1973) clarified this confusion.

A Classic Revised
Since 1918, Fisher’s paper, “The correlation between relatives on the supposition of Mendelian inheritance,” has been recognized as the source of the variance analysis technique—used for quantitative genetic and behavior–genetic analyses of phenotypic variance. Besides being received as a technical tour de force, it was considered to be a conceptual landmark, because it was supposed to have reconciled what was previously believed to be irreconcilable—the biometric and Mendelian points of view. Also, it has been widely believed that Fisher analyzed the components of the total variance in a Mendelian population by showing that Mendelian inheritance will lead to the observed correlations. This notion, however, is misleading because it has achieved, among geneticists and others, a status not unlike that of Vedas among Hindus. It is revered but not read. Few geneticists would claim to have read it and even fewer to understand it.

Fisher assumed a large number of independent factors having similar effects with partial dominance. He investigated correlations among relatives assuming that the population was mating at
random and developed a model of assortative mating for a population in equilibrium. He showed that assortative mating will increase the genetic variance and obtained formulas for correlations between relatives. His model involves three parameters: \( \mu \) the phenotypic correlation between mates, \( c_1 \) the ratio of total genetic variance to phenotypic variance and \( c_2 \) the ratio of additive genetic variance to total genetic variance. For a population mating assortatively \( \mu \) is usually known; \( c_1 \) and \( c_2 \) can be obtained from the parent–child and sib correlations. Of course, what Fisher actually did was to use observed phenotypic parent–child and sib correlations to estimate values of \( c_1 \) and \( c_2 \) for the population. He did not show that the Mendelian inheritance will lead to the observed correlations.

Burt and Howard (1956) appear to have been the first to use Fisher's model to analyze IQ data. Actually it is not correct to say that they used his model. As already stated, the purpose of the model is to estimate the parameters \( c_1 \) and \( c_2 \). Burt and Howard, however, assumed the values of those parameters. Unfortunately many behavior geneticists have used their papers as a guide for the application of Fisher's model. [It is now revealed that in addition to unsubstantiated data, Margaret Howard as well as another phantom collaborator may have been invented by Cyril Burt. See Gillie (1979).]

The 1918 paper was Fisher's first major paper in genetics and apparently he did not appreciate fully the implications of his model. His formulas for parent–child and sib correlation are not correct for his model. Moreover, the correct formulas for these correlations for his model suggest another interpretation when the value of \( c_1 \) exceeds one, as was the case in the example Fisher drew from the Pearson and Lee data on human height and span. Fisher assumed additive deviations to be the only causes of resemblance between parent and child and thus failed to take account of the correlation between the additive and dominance deviations of parent and progeny. However, Wright (1952) has pointed out that, "Assortative mating introduces a correlation between dominance deviations of parents and offspring and between dominance deviations of either and additive deviations of the other."

It has now been shown that in Fisher’s model of assortative mating there is a (small) correlation between additive and dominance deviations of parent and child. Thus, the assumptions on which Fisher obtained his formulas for parent–child correlation \([ = \frac{1}{2}c_1c_2(1 + \mu)]\) and sib correlation \([ = \frac{1}{4}c_1(1 + c_2 + 2c_2A)]\) are not correct for his model. Recently it has been possible to obtain formulas that are better for Fisher's model (Vetta, 1976). In Fisher’s notation these are

\[
\text{parent–child correlation} = \frac{1}{2}c_1c_2 \left[ 1 + A(1 - A)^2 \right] + \frac{1}{4}c_1(1 - c_2)A(1 - A)
\]

\[
\text{sib correlation} = \frac{1}{2}c_1c_2 \left[ 1 + A(1 - A)^2 \right] + \frac{1}{4}c_1(1 - c_2)
\]
In both formulas the first term represents the contribution of additive deviations to correlation and the second term the contribution of dominance deviations. The contribution made by dominance deviations is smaller for parent–child than for sib correlation, because $A(1-A) \leq \frac{1}{4}$. This is another reason why the concept of heritability (defined as additive variance/phenotypic variance) cannot be applied to human populations mating assortatively.

If the coefficient of assortative mating $\mu$ is known, values of $c_1$ and $c_2$ can be obtained from parent–child and sib correlation formulas. Effectively, the difference between the two correlations gives a fraction of the dominance variance. The formulas given here and those obtained by Fisher, however, are for genetic correlations that are not known and phenotypic correlations have been used instead. If the environments of sibs are more alike than those of parent and child, the difference between phenotypic correlations will also contain a fraction of the environmental variance. When the phenotypic correlational difference is too large to be explained by the amount of dominance variance in the population, the value of $c_1$ will exceed one. Thus, such a value, far from being evidence for no environmental effects, as Fisher believed, actually indicates their presence. However, a value less than one for $c_2$ does not, of itself, exclude environmental effects. It could mean that the contributions of dominance deviations, as well as of environmental effects, are small.

Johnson’s (1974) dissertation provides a paradigm for serious human behavior–genetic analyses. The unexpected problems and analytic subtleties, to cope with which the literature and prior experience had left us totally unprepared, provided a most sophisticated but duly sobering experience. A consideration of some of the problems and results from this unique investigation can be of immense heuristic value. Only the most salient and general features are considered here. Thompson (1968) has distinguished three features of behavior: it is complex, continuously variable and developmentally fluid. It is the third property of ontogenetic flexibility, reflecting norm of reaction, that contributes so much confusion to the long agonized debate over the heredity–environment pseudoquestion. Accordingly, as a heuristic exercise from the behaviorist’s perspective, a human trait, embodying the first two properties but not the third, was studied, thus circumventing the intractable problem of estimating the proportion of trait variation attributable to postnatal environment—a phenotype stable from birth to death whose expression is uninfluenced by where an individual grows up. The human fingerprint (dermatoglyph) is one such trait. It has a fascinating complexity and is interindividually variable but intra-individually postnatally stable in expression. It provides an intergenerational transmission–genetic perspective for studying segregation and assortment of similarities and differences in trait expression.
Table II
Distributions of Loop (L), Whorl (W) and Arch (A) Pattern Types
Cumulated over Ten Fingers in Johnson’s Total Illinois Sample and in That Sample Subdivided by Generations

<table>
<thead>
<tr>
<th>Generation</th>
<th>Cumulated over ten fingers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>L</td>
</tr>
<tr>
<td>Parent</td>
<td>.66</td>
</tr>
<tr>
<td>Offspring</td>
<td>.67</td>
</tr>
<tr>
<td>Total of both</td>
<td>.66</td>
</tr>
</tbody>
</table>

Observations were made on 10,580 individual fingerprints of 1,058 subjects in 212 nuclear families (both parents and two or more offspring). For ease of exposition our discussion considers only analysis of the dermatoglyphic pattern-type trait. It illustrates well the problems besetting this complicated field.

Analysis of three widely recognized alternative forms of trait expression—loop, whorl and arch—(shown in Table II) yields their distributions both throughout the entire sample and across the two generations separately. Also Table III shows a comparison with

Table III

<table>
<thead>
<tr>
<th>Pattern Type</th>
<th>Study</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
<td>Johnson</td>
<td>.63</td>
<td>.55</td>
<td>.72</td>
<td>.63</td>
<td>.87</td>
</tr>
<tr>
<td></td>
<td>Ford–Walker</td>
<td>.64</td>
<td>.56</td>
<td>.74</td>
<td>.61</td>
<td>.85</td>
</tr>
<tr>
<td></td>
<td>Holt</td>
<td>.68</td>
<td>.61</td>
<td>.76</td>
<td>.64</td>
<td>.86</td>
</tr>
<tr>
<td></td>
<td>Johnson</td>
<td>.32</td>
<td>.31</td>
<td>.16</td>
<td>.34</td>
<td>.11</td>
</tr>
<tr>
<td>W</td>
<td>Ford–Walker</td>
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<td>.33</td>
<td>.17</td>
<td>.37</td>
<td>.12</td>
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<tr>
<td></td>
<td>Holt</td>
<td>.27</td>
<td>.30</td>
<td>.16</td>
<td>.33</td>
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<tr>
<td></td>
<td>Johnson</td>
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<td>.14</td>
<td>.12</td>
<td>.03</td>
<td>.02</td>
</tr>
<tr>
<td>A</td>
<td>Ford–Walker</td>
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<td>.11</td>
<td>.09</td>
<td>.03</td>
<td>.03</td>
</tr>
<tr>
<td></td>
<td>Holt</td>
<td>.04</td>
<td>.10</td>
<td>.09</td>
<td>.03</td>
<td>.02</td>
</tr>
</tbody>
</table>

Figure 3
Distributions for ten fingers of proportions of parental mating type combinations (pie chart areas coded by shading for six pattern-type combinations). Distributions for ten fingers of proportions of offspring pattern types (heights of bars) and proportions of parental mating combinations from which offspring descended (shade-coded areas of bars).
two other studies at the population level. The data clearly reveal a polymorphism stable across generations and populations.

When the focus shifts from the population to the family pedigree level of analysis, other problems arise. Three alternative forms of trait expression on each of ten fingers generate a sample space of $3^{10} = 59,049$ possible pattern combinations of fingerprint phenotypes for individuals and $(3^{10})^2 = 3^{20} = 3,486,784,401$ parental mating possibilities. Because the ten fingers do not show perfect correlation of trait expression, for each finger in every family the parents have been classified separately as a mating combination. The three forms generate a minimum per finger of six such parental combinations.

Some of the complexity is revealed by the phenotypic analysis in Figure 3, showing results for ten fingers separately. For each finger it presents (1) the distribution of parental mating combinations by pattern type, (2) the offspring distribution of the three pattern types, and (3) the proportions of each pattern type among offspring of the six parental combinations. The relative frequencies both of parental combinations and of their offspring phenotypes vary over the several fingers. Though the data do not show dermatoglyphics to be independent of heredity, neither do they suggest any obvious rule of transmission of pattern type from parent to offspring.

A *sine qua non* for any study of heredity is proof positive of the presumed biological relationship, i.e., ascertainment of the biological validity of the designated kinships, such as parent–offspring, sibling, etc. Available time, funds and subject cooperation permitted testing on four blood factors the members of 38 of the 212 families—about one-sixth of the sample. No less than 13%, that is, five out of the 38 families, have children who cannot be the biological offspring of at least one of the putative parents. Subsequently we learned of a British report (Philipp, 1973) disqualifying, as fathers of the family children, 30% of husbands (private communication from Professor Tizard of London to J.H., 30 September 1975).

Unfortunately, in addition to its preoccupation with heritability, the extant human behavior–genetics literature is vitiated by being almost totally devoid of fundamental control observations. Every difficulty encountered in the dermatoglyphic study must be faced in any serious human behavior–genetic analysis. Moreover, such work cannot ignore the ontogeny of behavior, which, in contrast to dermatoglyphics, is most sensitive indeed to postnatal environmental conditions.

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309–322.


Nonvocal Social Signals
and Clinical Processes

Robert M. Adams

Nonverbal behavior that communicates information to other persons is currently receiving much attention from a variety of research disciplines, including communication, social and clinical psychology, psychiatry and human ethology. The 1977–1978 Subject Guide to Books in Print lists 56 titles under nonverbal communication. Recent reviews have included general overviews (Weitz, 1974; Leathers, 1976; LaFrance and Mayo, 1978; Harper et al., 1978) and reviews of specific topics such as gaze (Argyle and Cook, 1976), interpersonal spacing (Altman, 1975), body movement and posture (Spiegel and Mochotka, 1974) and facial expression (Ekman and Friesen, 1975; Izard, 1971). The level of the reviews has ranged from critical analyses of the empirical literature to what Harrison (1975) has termed “psychopornography.”

In recent years several authors [e.g., Ekman and Friesen (1968)] have seen the relevance of nonvocal (i.e., other than speech or speech-related) behavior to the causes, assessment and treatment of the problems that bring individuals to clinical practitioners. Here I intend to provide a general overview of some of the existing literature in this area as well as a more detailed look at a specific problem area—that which has been referred to as assertiveness. Assertiveness was selected as an area of research that appeared to have clear parallels in the dominance systems discussed extensively in the ethological literature on humans and nonhuman primates.

An assumption implicit in the discussion of deviant nonvocal social signals in relation to interpersonal problems is that there are a set of behavioral signals that are relatively uniform within the culture and perhaps to some extent across cultures. Eibl-Eibesfeldt (1975) has been one of the strongest advocates of the universality of nonvocal signals, citing the crosscultural similarity of a large number of behaviors. While other authors [e.g., Birdwhistell (1970)] argue against universality, the argument is typically in support of culturally determined forms of the behavior and at least predicts relative uniformity of signals within the culture to which indi-