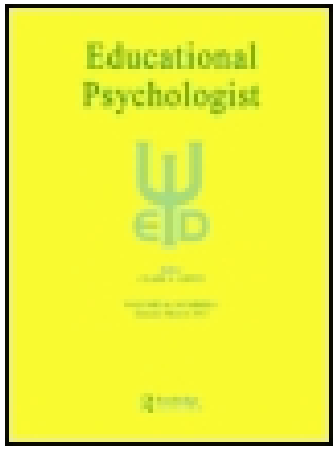

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The meaning of heritability in the behavioral sciences

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THE MEANING OF HERITABILITY IN THE BEHAVIORAL SCIENCES[1]

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ABSTRACT

The concept of heritability in quantitative genetics is defined and discussed in terms of its implications for individual and group differences in behavioral traits, with particular reference to studies of the heritability of IQ. Common misconceptions concerning the relevance of heritability analysis for individual scores and the roles of genotype x environment covariance and interaction are clarified. Some of the popular criticisms of heritability analysis as applied to mental ability are shown to be misconceived.

Background

The application of the models and methods of quantitative genetics to the investigation of individual differences in mental measurements, particularly the IQ, has stimulated new critical scrutiny of the concept of heritability. In its technical sense, heritability is merely one aspect of a model in biometrical genetics which attempts to account for the observed differences in covariance between various kinships on metrical or continuous characteristics.

The essential elements of the biometrical model were set forth by R. A. Fisher (1918), who showed that the correlation between relatives in continuous traits could be accounted for in terms of Mendelian principles by extending the simple 2-allele model to two or more loci and multiple alleles, thereby building up a polygenic model which is theoretically continuous with single-gene Mendelian inheritance. Each of the polygenes is assumed to have a small effect on the expression of the trait; the model also includes the possibility of dominance (or partial dominance) and epistasis. The sum total of the polygenic effects are in principle analyzable into additive and interaction components: interactions between alleles at the same locus (dominance) and between alleles at different loci (epistasis). When a large number of genes are involved, making for continuous rather than discrete expression of the trait, the effects of the multiple genes in a population are most conveniently described in terms of the variance of the trait, and the degree of resemblance between relatives is expressed as covariance, or as a coefficient of correlation, which is the ratio of covariance to the total variance.

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1 This article is based on a talk given at a plenary symposium on "Heritability and Human Affairs" at the Annual Convention of the American Society of Human Genetics, October 16, 1974, in Portland, Oregon. The author's address is Institute of Human Learning, University of California, Berkeley, CA 94720.

Definition of Heritability

The most familiar biometrical model, sometimes called the additive or linear model, analyzes the individual's phenotype (i.e., measurement) on a given trait into a number of additive effects (or components), both genetic and environmental. More precisely, what is analyzed is never the absolute measurement of the phenotype, but the *deviation* of the measurement from the grand mean of all such measurements in the population. Consequently, all of the genetic and environmental components of the phenotype are represented quantitatively as deviations from the population mean. With the elements of the model expressed as deviations, an individual's phenotypic deviation P from the population mean is:

$$P = G + E + I + e \tag{1}$$

where G is the individual's genotypic deviation,
E is the environmental deviation,
I is deviation due to interaction (i.e., non-additive effects of G and E),
e is measurement error.

The genotypic deviation G may be analyzed into additive and dominance deviations (thus, $G = A + D$). Further, the additive deviation may be broken into two components, one that would exist if mating were random plus an addition due to assortative mating. Assortative mating is the mating of similar phenotypes, which, if the trait is heritable, implies some genetic correlation between mates. Of course, only the genetic part of the correlation between parental phenotypes contributes to the additive genetic variance. For notational simplicity, at the moment, I will assume there is no dominance or assortative mating, though the model explicitly makes provision for these effects.

The population variance in phenotypes, therefore, is also analyzeable into the corresponding components of variance:

$$\sigma p^2 = \sigma_G{}^2 + \sigma_E{}^2 + 2r_{GE}\sigma_G\sigma_E + \sigma_I{}^2 + \sigma_e{}^2 \tag{2}$$

Heritability h^2 is defined as the proportion of the total phenotypic variance attributable to genetic variance, thus $h^2 = \sigma_G{}^2 / \sigma_P{}^2$.

Geneticists distinguish between narrow and broad heritability. Narrow heritability is the proportion of only the additive genetic variance under random mating. Broad heritability includes the effects of dominance (and epistasis) as well as the additive variance, including the component due to assortative mating. Since broad heritability includes *all* the genetic components determining the phenotype, it is also termed the coefficient of genetic determination.

Heritability of IQ

The methods of biometrical genetics have been applied in numerous studies to intelligence test scores obtained on various kinships such as monozygotic twins (reared together and reared apart), dizygotic twins, siblings, unrelated children reared together, parent-child, and other relationships. Most such studies find values of h^2 (broad heritability) in the range from about .60 to .90.

The same methods of estimating h^2 when applied to height measurements show only slightly higher h^2 for height than for IQ. The pattern of kinship correlations for height

is very similar to that for IQ. Weight is even more similar to IQ in this respect. The main difference is that IQ has a higher degree of assortative mating. The presence of substantial dominance variance in IQ suggests that whatever IQ tests measure involves a fitness character which has undergone some degree of selection in the course of human evolution. Directional selection for a given trait "uses up" its additive genetic variance, since selection acts directly on phenotypes, which are much more highly correlated with the additive than with the nonadditive genetic determinants. Dominance for higher IQ accounts for the phenomenon of "inbreeding depression" of IQ in the offspring of cousin matings and incestuous matings.

Misconceptions About Heritability

Much of the controversy arising from the finding of a substantial heritability for IQ stems from a few fundamental misconceptions about the interpretation of this index.

The most troublesome misconception is that a high heritability necessarily means immutability of the trait in question, implying a hopeless fatalism, against which some people tend to react emotionally. A heritability index simply tells us that in the particular population sampled, for the particular measurement obtained, under the conditions it was obtained, a certain proportion h^2 of the total variance is attributable to genetic factors.

Genetic determination refers to those aspects of the individual's internal environment which are traceable, at least in principle, through a complex chain of causality going back in the course of the individual's development to the DNA transmitted in the parental gametes. The proportion of non-genetic variance, $1\text{-}h^2$, is attributable to all other factors outside the DNA, acting since the moment of conception. The estimation of h^2 *per se* tells us nothing at all about those non-genetic factors. Nor does it preclude the possibility that under some environmental circumstances which differ from those in the population for which h^2 has been determined, h^2 could assume a quite different value. The methods of biometrical genetics, of course, have no power to predict h^2 under as yet untried interventions in the internal or external environments. It does give an indication of the relative influence of *existing* environmental sources of variance, and, if h^2 is very high, it tells us that merely reallocating individuals in existing environments will not have much effect in the rank ordering of individual differences. It should be kept in mind that h^2 is a *ratio* of two variables: genetic variance σ_G^2 to genetic plus environmental variance σ_E^2. And either σ_G^2 or σ_E^2 or both of these variances can change.

The obverse of the immutability misconception is the idea that the non-genetic variance, $1\text{-}h^2$, (but excluding measurement error) has the same meaning as "environment" in the popular sense of this term. In biometrical genetics, the environmental variance σ_E^2 is simply the residual non-genetic variance, nothing more. There is nothing in the heritability analysis which tells us specifically the source of σ_E^2. We cannot assume *a priori* that it represents those aspects of the environment which are the most salient or the most easily manipulable. σ_E^2 is not necessarily due to environmental influences associated with socio-economic status or other aspects of the environment most often mentioned in discussions of the disadvantaged. Discovery of the precise nature of environmental influences on IQ (or on any other trait) requires quite another type of investigation, involving the experimental manipulation of specific hypothesized sources of environmental

variance. It is possible that some substantial part of the nongenetic variance in IQ is so fortuitous or "microenvironmental" as to practically defy control, and will thus remain as residual, nongenetic (and non-error) variance.

It should also be apparent that $1\text{-}h^2$ says nothing about the actual magnitude of environmental variation, but tells us only the effect of that variation (whatever it is) on the trait in question, relative to the variance due to genetic factors. Thus, high h^2 could mean that the trait is possibly sensitive to environmental variation but little such variation exists in the tested population, or it could mean that the trait is in fact insensitive to a wide range of environmental variation. Experimental, rather than purely statistical, procedures are needed if we are to answer such questions.

Another popular misconception is that h^2 can apply only to populations and not to individuals. Equation 1, representing the components of an individual score, clearly contradicts this notion. An individual's genetic value (i.e., genotypic deviation from the population mean) can be calculated, given his phenotypic value and h^2 as well as the assumption that the individual is a member of the population for which the value of h^2 has been estimated. The estimate is, of course, probabilistic, and we can state the probability that the individual's estimated genetic value differs by X amount from the true value. Just as in psychometrics we use the standard error of measurement in estimating the probability that an obtained score is within X units of the true score, so in genetics we can use the square root of the estimated nongenetic variance, $\sqrt{1\text{-}h^2}$, in a way analogous to the standard error of measurement to estimate the individual's genetic value. For example, we can say that with $h^2 = .75$, the chance that a person with an IQ of 80 is genotypically equal to or higher than the average genotypes of all persons with an IQ of 100 is less than one in 200.

Heritability Within Groups and Between Groups

Another point of confusion has been the implications of heritability of individual differences *within* groups for the heritability of differences *between* groups, when the groups are various subpopulations such as races and social classes.

It is generally agreed that heritability within groups, h^2_W, has no logically necessary implication for heritability between groups, h^2_B. This does not imply, however, that there may not be probabilistic implications of h^2_W for h^2_B or that there is no theoretical connection whatsoever between h^2_W and h^2_B, given knowledge of certain other parameters.

Generally, for highly heritable characteristics within groups, phenotypic mean differences between groups also show a heritable component, even when there are obvious environmental differences between the groups. Often there is a positive correlation between genotypes and the environmental factors most relevant to the characteristic, e.g. skin pigmentation and amount of exposure to ultraviolet radiation.

Instances are rare where the direction of genotypic means is the opposite to that of the phenotypic means; more often phenotypic and genotypic means are positively correlated. If within-group heritability is high (i.e., greater than 0.5), a purely environmental theory of the between-groups difference must posit a much larger environmental difference between the groups, than the genetic difference posited by a genetic theory of the groups difference, in order to explain the phenotypic difference between the group means, unless the environmental theory also posits an additional hypothesis that the mean difference

between the groups is due to environmental factors which are not the same as those responsible for environmental variance *within* the groups.

A reasonable presumption (though certainly not proof) of genetic group differences seems to be related to the magnitude of the group difference and the heritability of the trait in question, as seen in the fact that few persons believe that the average difference in stature between Pygmies and Watusis is not largely genetic, despite their very different habitats, diets, and customs. The fact that the group mean difference is large (relative to the standard deviation within groups) and involves a trait of very high heritability, makes it seem reasonable to believe that the group difference is largely genetic. (I don't know of any other evidence that it is genetic.) The same kind of "reasonable hypothesis" must also apply to other characteristics, including behavioral traits, in which there are substantial phenotypic differences and substantial heritability within groups, although, of course, the degree of plausibility will depend upon the magnitudes of the group difference and of the within-groups heritability of the trait in question, as well as upon other factors such as the nature and extent of environmental differences, if these are known and their causal relationship to the trait in question is established.

Formulation of Between-Groups Heritability as a Function of Within-Group Heritability

The standard formula (e.g. Lush, 1968) for the relationship of between-groups heritability, h_B^2 (i.e., the genetic fraction of the variance among the phenotypic group means) and the heritability in the whole population (i.e., the combined groups) is:

$$h_B^2 = h^2 \left[\frac{1 + (n\text{-}1)r}{1 + (n\text{-}1)t}\right] \tag{3}$$

where h^2 is the narrow heritability in the whole population
n is the sample size
r is the intraclass correlation among the genic values (for the particular character in question) of members of the same group.
t is the intraclass correlation among the phenotypic values of the same group.

When nr and nt become large,

$$h_B^2 \cong h^2 (r/t). \tag{4}$$

The heritability within groups, h_W^2, can be expressed as:

$$h_W^2 = h^2 \frac{(1\text{-}r)}{(1\text{-}t)} \tag{5}$$

From Equations 2 and 3, the geneticist De Fries (1972) derived the following formula for the heritability between groups:

$$h_B^2 \cong h_W^2 \left[\frac{(1\text{-}t)r}{(1\text{-}r)t}\right] \tag{6}$$

If there is a positive correlation between heredity and environment, this expression underestimates the heritability of the group difference.

If the correlation between heredity and environment is negative, h_B^2 is overestimated by the formula. The relationship of between-group to within-group heritability for two groups with equal variance, normal distributions of the trait, and a mean difference of one standard deviation, can be shown graphically as in Figure 1.

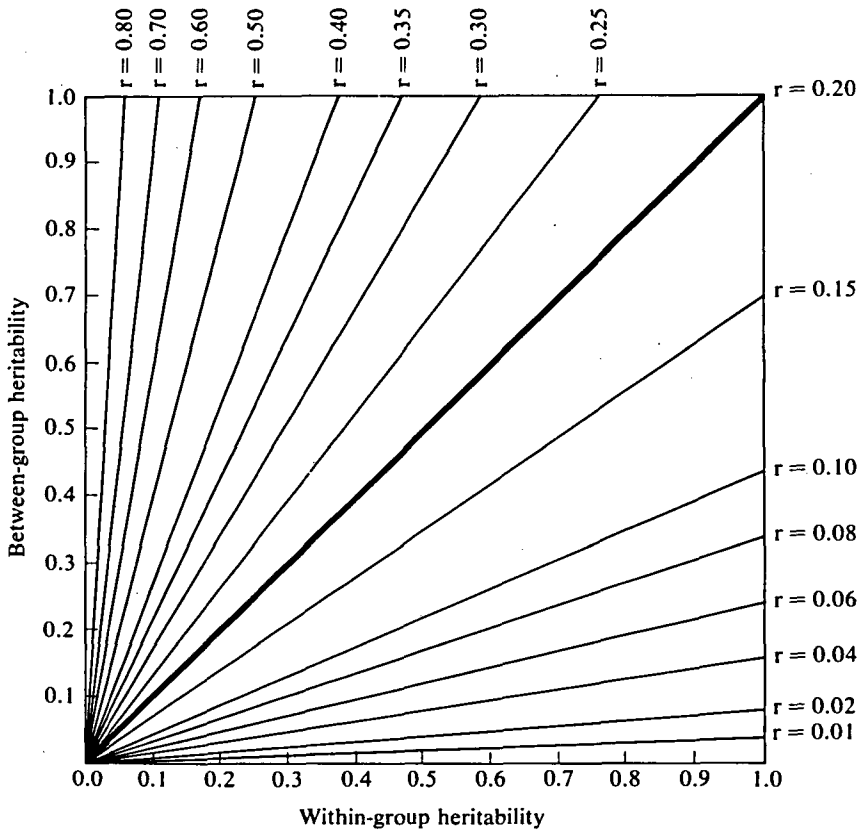

Fig. 1. Between-group heritability expressed as a function of within-group heritability and the genetic correlation of members of the same group (r). See text for explanation. (From *Introduction to behavioral genetics* by G. E. McClearn and J. C. DeFries. W. H. Freeman and Company. Copyright ©1973. Reproduced with permission of publisher and authors.)

The formula is obviously only of theoretical interest, since we lack information on one of the parameters, r, the intraclass genic correlation for the trait in question. Thus the formula gets us nowhere, unless, of course, one wishes to speculate concerning the probable value of r. But this is the very point in question. If the groups do not differ at all genetically, r will be zero and h_B^2 will be zero. For groups whose means differ by one standard deviation, the *phenotypic* intraclass correlation, t, is 0.20. (The intraclass correlation t = .20 is most easily obtained from a one-way analysis of variance which partitions the total variance (say, of IQ) between-groups and within-groups. If the group means differ by 15 IQ points and the σ within each group is 15 IQ points, then the between-groups variance σ_B^2 will be $(15/2)^2 = 56.25$, and the within-groups variance σ_W^2 will be

$15^2 = 225$. The intraclass correlation is $t = \sigma_B^2/(\sigma_B^2 + \sigma_W^2) = 56.25/(225 + 56.25) = 0.20$.

The genic intraclass correlation r, however, is unknown. Unless one assumes that all the genetic difference between groups in the trait of interest is purely a result of random genetic drift (which affects all gene loci equally, on the average), or that the causes of selection are the same within and between groups, there is no way to estimate r for any particular polygenic trait. We cannot assume the same selection pressures have existed in all races, and in fact there is reason to suspect the contrary. The traits in which we are most interested psychologically probably do not involve exclusively neutral genes, subject only to random drift. If they did involve only neutral genes and the trait were highly polygenic, then there would be no reason to expect any appreciable systematic genetic difference between large population groups. The size of r will of course differ for various traits which have been subjected to different selection pressures over many generations. Thus it is pointless to try to estimate r for one characteristic and expect it to be generalizable to others. The De Fries formula therefore is useless empirically. Those who believe there are no genetic differences can say $r = 0$. If one makes the unwarranted assumption that genetic group differences are not confounded with environmental differences, then it might be said that $r = t/h^2$ (where h^2 is the heritability in the whole population). And if one makes the assumption that the between-groups environmental effects are of the same nature as within-groups environmental effects, one could say that $r = h^2 t$. But we don't know h^2 in the whole population, either. It is a function of h_W^2 and h_B^2, and it is h_B^2 that we can't determine for lack of knowing r. Because of this lack, we must conclude that, at present, attempts to infer the magnitude of heritability between groups from the determinable heritability within groups is a blind alley.

Criticisms of h^2 as Applied to IQ

Heritability analysis of the IQ and other mental test scores has been subjected to a variety of criticisms bearing mostly on methodological matters. Four main criticisms of h^2 that have been put forth are described under the following headings.

IQ Not an Absolute Scale. This is perhaps the most misconceived criticism of all. It has been claimed that h^2 cannot be computed from measures which are not on an absolute or ratio scale (Layzer, 1972, 1974). It is true that the IQ is not measured on an absolute scale with a true zero point and that at best it is only an interval scale. Most kinds of test scores are only ordinal scales denoting rank order. But there is nothing in the logic of heritability analysis which requires that the measures be absolute. The basic method of genetical analysis is the analysis of variance, in which the analyzed variables are all simply deviations from the grand mean, so that any linear transformations of the scale will have no effect on the outcome. IQs are perfectly suitable data for analysis by the methods of biometrical genetics, and no valid argument has been made to the contrary.

Different Environmental Correlations for MZ and DZ Twins. The fact that *MZ* twins reared apart have much more similar IQs that *DZ* twins reared together leaves really no doubt of the heritability of IQ. But twins reared apart are rare and so most of the evidence for the heritability of IQ is based on the difference in the covariance (or correlations) between *MZ* and *DZ* twins, where the twins are reared together. A common formula is $h^2 = 2(r_{MZ} - r_{DZ})$, which is a value lying within the limits between broad and narrow heritability. (It underestimates broad heritability because it does not take account of

assortative mating and it overestimates narrow heritability because it does not take account of dominance and epistasis.) It has been argued in this case that the environments of *MZ* twins are more alike than of *DZ* twins and that this greater environmental correlation for *MZ*s is confounded with the genetic variance in any estimate of h^2 based on the difference between the *MZ* and *DZ* correlations. The same claim could be made of any other kinships as well, and in principle it is correct. The question, therefore, is really an empirical one, *viz.* is the environment, in fact, more similar for *MZ* than for *DZ* twins in ways that would influence IQ? For example, *MZ* twins are more often dressed alike, often are mistaken for one another, and spend more time with each other than do *DZ* twins. On the other hand, there is some evidence that *MZ* twins have less similar prenatal environments (Price, 1950). Also, since the true zygosity of twins is frequently unknown or incorrectly perceived by their parents, it would seem unlikely that the overall difference in the social treatment of *MZ*s and *DZ*s would be sufficient to account for the large difference between their IQ correlations (on the average about .90 and .55, respectively), which is of about the same order as for height and is considerably greater than the correlational differences for more family influenced variables such as scholastic achievement.

To get some idea of the effect that differences in environmental correlation might have on the estimated heritability, one can hypothesize varying degrees of environmental correlation for *MZ* and *DZ* twins, enter these various values into sets of simultaneous equations and solve the equations for the values of the genetic and environmental variance. This procedure can be best described in the next section in connection with the treatment of the covariance of genotype and environment, since the environmental correlations interact with the genotype-environment covariance in determining the heritability.

G X E Covariance Must Be Zero. It has been argued that a necessary condition for heritability analysis, i.e., obtaining an estimate of the genetic variance or h^2, is the absence of a genotype-environment correlation (Layzer, 1974).

The part of the total phenotypic variance which is attributable to a correlation between genotypes and environments has two components:

(1) One, in which the environmental variation, though correlated with the genotype, is not a result of the genotype, and

(2) the other, in which the environmental variations are selected or created by particular genotypes. The first aspect would be illustrated by the genotypically bright child who also has the additional environmental advantage of being reared by highly intelligent parents. The second aspect would be illustrated by the case of a genotypically bright child who, because of his brightness, tends to become unusually engrossed in intellectually challenging pursuits and invests a lot of his time and energy on them, which may also happen to be of some advantage in performing on an IQ test. Some geneticists hold that the second type of genotype-environment covariance should be included as part of the genetic variance. In any environments allowing a range of choices and opportunities, it would be virtually impossible either to eliminate or to enforce the kind of genotype-environment covariance that results from genotypic differences in self selection and utilization of different aspects of a highly varied environment. It should be realized, too, that in a natural environment any part of the $G \times E$ covariance, since it is not included in the variance due to the direct effect of the environment, which is by definition independent of the genotype, must be regarded as evidence that some part of the environment is in-

fluenced by the genotype—either that of the individual in question, or of his parents, or of some correlated genotype. Since we know that the psychological or social environment does not affect the genotype, the $G \times E$ covariance must in some way be accountable to genetic factors.

We can see the relationship between genetic variance and $G \times E$ covariance, and their mutual relationship to the environmental correlations for *MZ* and *DZ* twins, by solving simultaneous equations representing the genetic model for the *MZ* and *DZ* twin covariances, as follows:

$$Cov_{MZ} = r_{MZ}\sigma_p^2 = \rho_{GG'}\sigma_G^2 + \rho_{EE'}\sigma_E^2 + 2\,\rho_{GE}\sigma_G\sigma_E \tag{7}$$

$$Cov_{DZ} = r_{DZ}\sigma_p^2 = \rho'_{GG'}\sigma_G^2 + \rho'_{EE'}\sigma_E^2 + 2\,\rho_{GE}\sigma_G\,\sigma_E \tag{8}$$

where r_{MZ} is the observed correlation between MZ twins,

r_{DZ} is the observed correlation between DZ twins,

σ_p^2 is the total phenotypic (observed) variance,

σ_G^2 is the genetic variance (unobserved),

σ_E^2 is the environmental variance (unobserved),

$\rho_{GG'}$ is the genetic correlation (unobserved but derived from Mendelian theory), with a value of 1.00 for *MZ* twins and of 0.5 for *DZ* twins in a randomly mating population (higher values than 0.5 under assortative mating, but lower with dominance and epistasis),

$\rho_{EE'}$ is the environmental correlation (unobserved) between twins,

ρ_{GE} is the correlation between genotype and environment.

As an example, we can use the median of all *MZ* and *DZ* twin correlations reported in the literature to solve this set of equations for σ_G^2 and σ_E^2, varying $\rho_{EE'}$ and ρ_{GE} over a range of likely values, and assuming different degrees of assortative mating. The review by Erlenmeyer-Kimling and Jarvik (1963) gives .87 as the median correlation for IQ of all studies of *MZ* twins reared together and .56 as the median for *DZ* twins. Multiplying these correlations by the total variance for IQ (i.e. 225) gives Cov_{MZ} = 195.75 and Cov_{DZ} = 126.00. The results of solving the set of simultaneous equations for σ_G^2 and σ_E^2, assuming a range of values of $\rho_{EE'}$ and ρ_{GE}, are shown in Figures 2, 3, and 4, for assortative mating coefficients of 0, .20, and .40, respectively. (These assortative mating coefficients are the *genetic* correlation between mates, which is estimated by the product of the narrow heritability and the phenotypic correlation between mates. The products of empirical estimates of the narrow heritability and the phenotypic assortative mating coefficients for IQ fall within the range from .20 to .40.) We see that the estimate of genetic variance (and hence the heritability, h^2) increases as a function of degree of assortative mating. But the effect of $G \times E$ covariance depends upon both the degree of assortative mating (having less effect for higher assortative mating) and upon the discrepancy

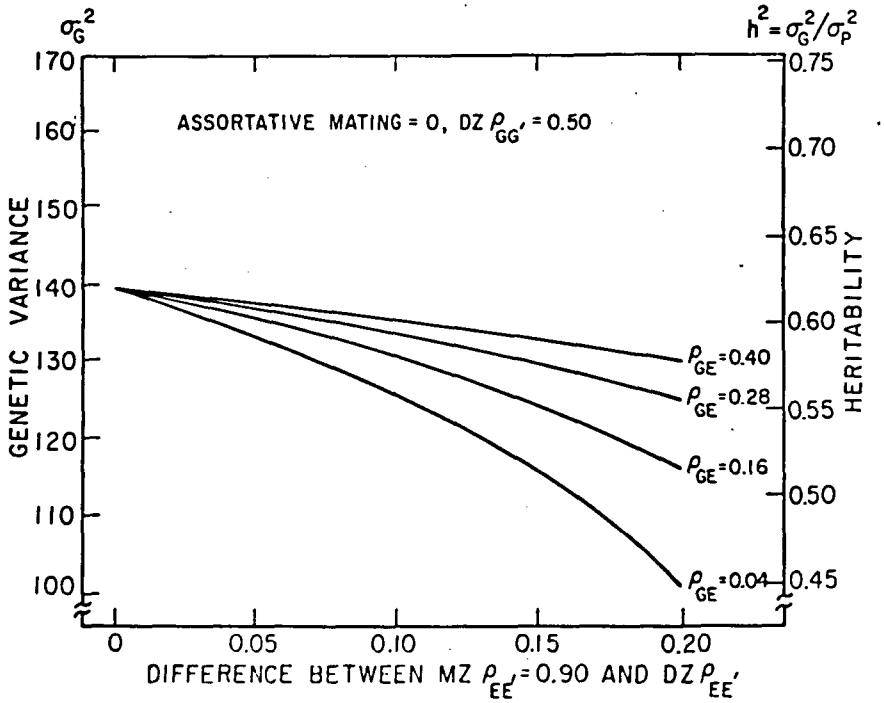

Fig. 2

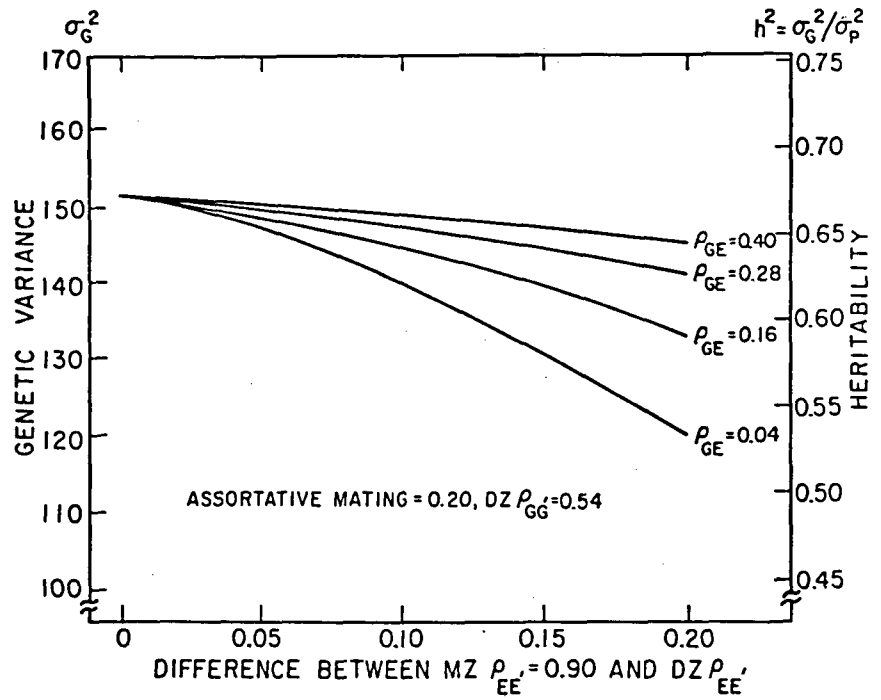

Fig. 3

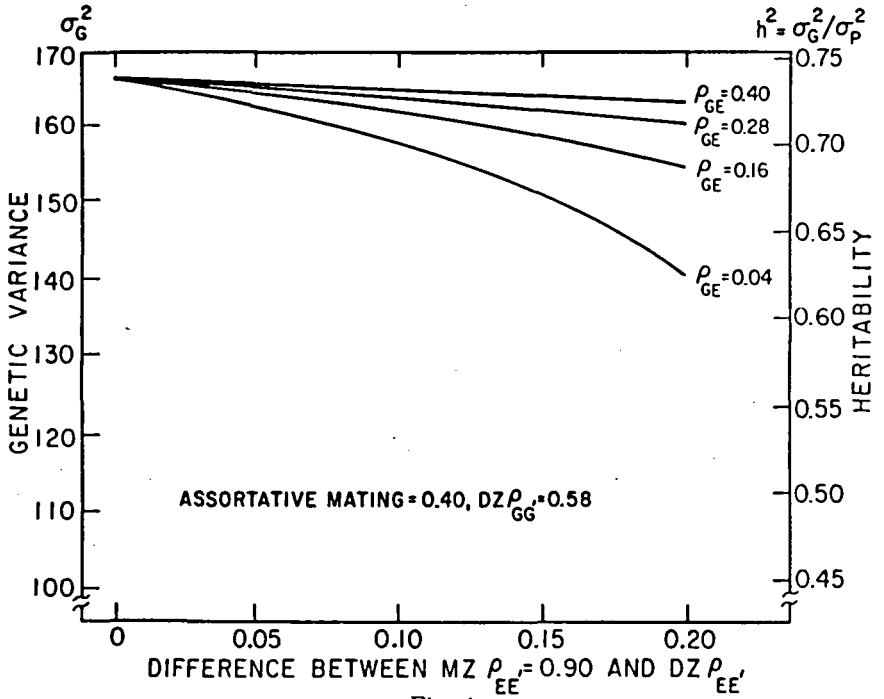

Fig. 4

Figs. 2-4. Genetic variance and broad heritability based on *MZ - DZ* twin comparisons under different assumed values of the parameters $\rho_{EE'}$ (environmental correlation between twins), ρ_{GE} (correlation between genotypes and environments), and assortative mating.

between the *MZ* and *DZ* environmental correlation. The greater this discrepancy, the greater is the effect of the *G* X *E* covariance. But it may seem surprising and counter-intuitive that the greater the *G* X *E* covariance, the greater is the genetic variance and consequently the higher is the heritability. Figures 2 to 4 show the most probable range of values for heritability as estimated by the twin method, assuming a range of reasonable values of *MZ-DZ* differences in $\rho_{EE'}$ and for ρ_{GE}. The values of h^2 thus range from about 0.50 to 0.75. With assortative mating of .40, an environmental correlation of .90 for *MZ* and .70 for *DZ* twins, and a genotype-environment correlation of .40, h^2 is .74. Corrected for attenuation, assuming a test reliability of .93 (the average reliability of the Stanford-Binet IQ), h^2 is .80. However, I am using this merely as an illustration of the inter-relationship of Cov_{GE} with other parameters involved in heritability estimation, rather than as an attempt to arrive at a best estimate of h^2. For this we would want to take much more of the existing kinship data into account. The important point demonstrated here is that larger *G* X *E* covariance does not always diminish h^2. (I have discussed the genotype x environment correlation, with empirical examples, in much greater detail elsewhere [Jensen, in press].)

G X E Interaction. Statistical interaction of genotype x environment consists of the non-additive effects of genetic and environmental factors. These non-additive effects can, in theory, take many forms, as clearly explicated by Lewontin (1974). A substantial amount of such *G X E* interaction would, of course, result in a poor fit of the linear or additive model of genetic and environmental effects for the trait in question. The main types of these non-additive effects, or *G X E* interactions, if they exist, should be statistically detectable by means of a trend analysis of the relationship between the means and differences of the measurements for pairs of identical twins, ideally twins reared apart. The magnitude of the difference between *MZ* twins reflects purely environmental effects. The mean of the twins' scores reflects mainly genetic effects, but also some environmental component. The regression of twin differences on twin means can be subjected to a trend analysis for linear, quadratic, cubic, quartic, etc. components. A statistically significant level of any of these components would indicate the presence of *G X E* interaction, which would then be assessed in terms of the proportion of variance it accounts for. I did such a trend analysis, up through the quintic component, on all the published data on *MZ* twins reared apart and failed to find any significant trends. The same kind of analysis applied to a large sample of sibling IQs also revealed no significant *G X E* interactions. Of course specific interactions, unique for each individual, would not be revealed by this kind of analysis, which can detect only systematic interaction effects. Specific interactions in any domain of scientific measurement always remain indistinguishable from errors of measurement or other "noise."

If systematic interactions did show up, one would investigate the possibility that they are merely scale artifacts and would determine whether the interaction could be eliminated by rescaling the data. This is standard procedure in quantitative genetics. It has not proved necessary so far in the case of IQ data, since no one has yet succeeded in showing any evidence of systematic *G X E* interaction for IQ.

If *G X E* interaction is held up as a criticism or limitation of the applicability of heritability analysis to mental test data, the burden of demonstrating the presence of substantial *G X E* interactions in such data must be assigned to the critics. In science, many valid forms of measurement and analysis could be made unworkable by some hypothetical set of conditions. For example, radar measurements of planetary orbits *could* be distorted by hypothetical electromagnetic fields in outer space. If anyone wishes to advance this hypothesis, let him adduce the evidence for it. The evidence for the linear model of heritability analysis is that it fits the existing data very well indeed and has not yet been superceded by any improved or tested model. As geneticists Rao, Morton, and Yee (1974, p. 357) have remarked about *G X E* interaction, "Since armchair examples of significant interaction in the absence of an additive effect are pathological and have never been demonstrated in real populations, we need not be unduly concerned about interaction effects. The investigator with a different view should publish any worthwhile results he may obtain."

The Larger Significance of Heritability in the Behavioral Sciences

The concept of heritability has taken on a greater importance than can be warranted in a strictly technical sense, because it has focused attention on the importance of biological and genetic factors in human behavior, particularly human abilities. This attention

comes at a time when a large segment of the behavioral sciences has ignored or shunned genetical thinking. Yet genetic analysis in the broad sense, combined with the tools of psychometics and experimental psychology, is highly relevant and necessary to the study of behavior.

Many features of the recent controversy concerning the heritability of IQ are merely symptomatic of the estrangement, since about 1930, of the behavioral and social sciences from their proper continuity with biology, evolutionary theory, and genetical analysis. The heritability concept is only one of the many possible links between the biological and behavioral sciences. The basic issues raised by the controversy concerning the inheritance of mental abilities are much broader than the technical concept of heritability *per se*. The future development of the behavioral sciences will depend upon dealing with these matters within a scientific rather than within an ideological context.

REFERENCES

DE FRIES, J. C. Quantitative aspects of genetics and environment in the determination of behavior. In L. Ehrman, G. S. Omenn, & E. W. Caspari (Eds.), *Genetics, environment and behavior: Implications for educational policy*. New York: Academic Press, 1972.

ERLENMEYER-KIMLING, L., & JARVIK, L. F. Genetics and intelligence: a review. *Science*, 1963, *142*, 1477-1479.

FISHER, R. A. The correlation between relatives on the supposition of Mendelian inheritance. *Transactions of the Royal Society of Edinburgh*, 1918, *52*, 399-433.

JENSEN, A. R. How much can we boost IQ and scholastic achievement? *Harvard Educational Review*, 1969, *39*, 1-123.

JENSEN, A. R. The problem of genotype-environment correlation in the estimation of heritability from monozygotic and dizygotic twins. *Acta Geneticae Medicae et Gemellologiae*, in press.

LAYZER, D. Science or superstition? A physical scientist looks at the IQ controversy. *Cognition*, 1972, *1*, 265-300.

LAYZER, D. Heritability analyses of IQ scores: Science or numerology? *Science*, 1974, *183*, 1259-1266.

LEWONTIN, R. C. Annotation: The analysis of variance and the analysis of causes. *American Journal of Human Genetics*, 1974, *26*, 400-411.

LUSH, J. L. Genetic unknowns and animal breeding a century after Mendel. *Transactions of the Kansas Academy of Science*, 1968, *71*, 309-314.

MCCLEARN, G. E., & DE FRIES, J. C. *Introduction to behavioral genetics*. San Francisco: W. H. Freeman, 1973.

PRICE, B. Primary biases in twin studies: A review of prenatal and natal difference-producing factors in monozygotic pairs. *American Journal of Human Genetics*, 1950, *2*, 293-352.

RAO, D. C., MORTON, N. E., & YEE, S. Analysis of family resemblance. II. A linear model for familial correlation. *American Journal of Human Genetics*, 1974, *26*, 331-359.