The Intelligence of Heritability

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Abstract:

The influences of heredity and environment on behaviour are sometimes quantified as a heritability ratio, which assigns a percentage of variation in test scores to variation in the genotypes of individuals. There are compelling reasons, both biological and statistical, to doubt the validity of the common practice of partitioning variance in this manner. This paper outlines the conceptual foundations and explains the weaknesses of heritability analysis, reviews evidence of heredity-environment interaction during development, and argues for an alternative research strategy to detect and understand the functions of specific genes relevant for individual differences in behaviour.

Article:

The importance of heredity for human intelligence and other mental attributes is a perennial topic of debate in psychology with a history extending to Rousseau and earlier. Since 1970 these questions have been addressed by a specialization in psychology calling itself behaviour genetics and having its own professional society and journal. The dominant school of thought in this subdiscipline asserts that almost every human characteristic is determined by *both* the genetic inheritance and the life experience of the individual, and it seeks to estimate the relative strengths of these two factors. The index commonly used to summarize the results for a specific behaviour is the heritability coefficient, which is the most salient feature of the academic discipline of behaviour genetics. Studies of twins and adopted children have claimed substantial heritability of everything from time spent watching television to religious conservatism and what brand of beer you prefer (Plomin ct al., 1990; Rosen, 1987; Waller et al., 1990). At the 1991 Behavior Genetics Association meeting in St. Louis, among the 64 presentations involving studies of human beings, 45 involved heritability analysis. Recent feature articles in *Science* touted heritablity analysis as the state of the art in the discipline (Bouchard ct al., 1990; Plomin, 1990). Many articles have been devoted to the likely value of the heritability of intelligence in particular. The present article focusses on the concept of heritability itself and questions its intelligence.

Heritability in the broad sense (h_B^2) is said to estimate the proportion of variance in a measure of behaviour or other phenotype (V_Y) in a breeding population that is attributable to genetic variation ($h_B^2 = V_G/ V_Y$). The estimation of this parameter involves a model based on the inheritance of genes via the principles of Mendel, which are well established, plus assumptions about how genetic effects are related to environment and behaviour, which are still contentious.

Genetic Facts

Before presenting the main argument, a few genetic *facts* should be considered. A gene is a segment of a DNA molecule occurring at a specific locus or place along the DNA, and it codes for the structure of a polypeptide molecule that may function as a protein, enzyme or hormone. A person's genotype is the pair of genes he or she has at the locus, one coming from each parent. There are perhaps 50,000 (possibly as many as 100,000) different genes in the 23 human chromosomes, each coding for a specific polypeptide (McKusick, 1991). Of these, about 30,000 are expressed as distinct proteins in the brain and 20,000 are specific to the brain (Sutcliffe, 1988), which gives some idea of the complexity confronting those who hope to understand the relation of heredity to brain development and behaviour. There are also 37 genes in the mitochondria in the cell cytoplasm

deriving mainly from the mother (Wallace, 1991), although evidence from mice indicates a few mitochondria may he paternal (Gyllensten et al., 1991). As of October, 1990, there had been 1,909 chromosomal genes, perhaps 2% of the total number, identified and mapped to an approximate location on a chromosome (Stephens et at, 1990), and one year later the count was 2,144 (Chipperfield et al., 1991). The basic principles of genetic inheritance and gene action are well documented, but the era of discovering new genes has barely begun. In some instances people carry slightly different forms or alleles of a gene at a particular locus, such that each form codes for a slightly different structure of the protein molecule. When this occurs, the locus is said to cause a protein polymorphism in the population and can give rise to noteworthy differences between individuals in their blood types, immune system functions, brain structures or behaviours. Less than 5% of the human genetic loci are believed to cause substantial protein polymorphism. If 20,000 genes are unique to the brain, perhaps 19,000 will be effectively the same in almost all people, whereas only 1,000 will give rise to individual differences. From a genetic perspective, what people have in common vastly exceeds what makes us different; genetic differences between human beings and our close relative the chimpanzee are far greater than the modest variation among people of different ancestries (Vigil= et al., 1991). The methods of Mendelian genetics are sensitive only to the small minority of genes which arc polymorphic and tend to make us different; they allow no meaningful statements about the role of heredity in general. Sometimes a defect in a single gene may have obvious consequences, such as in colour blindness, phenylketonuria or cystic fibrosis. On the other hand, some genes have rather slight effects that require large samples and rigorously controlled experiments to detect. These unseen genes presumed to have minor effects are the domain of quantitative genetics and heritability analysis.

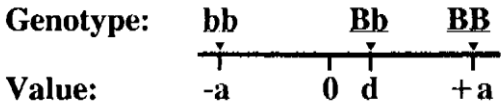

Fig. 1 The correspondence between each genotype at a hypothetical locus "B", where there are only two alleles in the population, and its contribution to the genetic component of phenotypic value of a behaviour on a continuous scale. By convention, the zero point is defined to be midway between genotypes *bb* and *BB*. Genotype *BB* is presumed to increase the individual's behaviour by an amount *a*, and the heterozygote *Bb* increases it by the degree of dominance *d*.

The Model and Assumptions

The validity of heritability analysis depends on the merits of several assumptions. First, Falconer (1981) and others propose a simple model of how each possible genotype at one locus specifies a quantitative genetic component of a phenotype, which may be a behaviour (see Fig. 1). Next, the effect of all polymorphic loci affecting a behaviour are combined by *adding* them to yield the total O, for an individual, which assumes genotype at one locus does not influence the action of genes at other loci. Then it is asserted that the measured score (or phenotype) of an individual on a psychological test (Y_i) is the *sum* of only two components, G_i determined by the genes and E_i specified by the person's environment; that is, G and E must not interact. Many sources introduce the model with a G×E interaction term attached to the end of the model, but they quickly drop this and proceed with the simple additive model as the basis for further analysis. Finally, if there is no correlation between the parental genetic values and the environments they provide for their children, or between the G and E of the children, it is possible to deduce the correspondence between the hypothetical heritability ratio and the observed correlations between test scores of relatives using algebra (see Falconer, 1981, for details). Given these derivations, the phenotypic correlations are computed from data and the heritability parameter is estimated from them. Advocates of this approach acknowledge that the parameter estimate from a single study applies only to the population sampled and the current environment.

Critics such as Goldberger (1978), Kempthorne (1978), Schönemann (1989) and Wahlsten (1979) have expressed grave doubts about the concepts, methods and algebra presented by leading authorities on behaviour genetics, while others have questioned the capacity of experimental designs to separate effects of heredity and environment with humans (Kamin, 1974; Roubertoux and Capron, 1990; Taylor, 1980). Nevertheless, it has

been asserted that most psychologists arc now convinced by heritability coefficients of the importance of genetics for individual differences in behaviour (Starr, 1987).

Emergence of Developmental Interactionism

Of course, many scientists thought that basic point had been made quite adequately 50 years ago by the selective breeding of Tryon's maze bright and maze dull rat strains and several human twin studies. Instead of becoming fixated on the question of *the size* of the genetic influence, they sought to learn *how genes* influence development of the individual. To achieve this objective, one must either have extraordinary control of breeding and rearing conditions, which can be done only with laboratory animals, or be able to identify specific genes relevant to a behaviour. Data from humans can in principle be assessed for effects of single genes, but this is not often done in behaviour genetics (only 6 papers at the 1991 St. Louis B. G. A. meeting) and pertinent methods are not included in leading texts in the field. Heritability analysis based on correlations among relatives is inadequate on both counts for a developmental analysis.

Two lines of investigation, heritability analysis and a more developmental approach, have emerged and evolved in parallel, aware of each other but separated by a conceptual barrier vis-à-vis heredity-environment interaction. The developmental perspective was evident in the pioneering behaviour genetic work of John Paul Scott and Benson Ginsburg who independently studied fighting behaviour of the same inbred mouse strains but under different laboratory conditions. Scott (1942) found that C57BL mice never attacked and C3H mice usually started a fight after several feints towards an intruder. This was just as the USA was entering World War it, and he annointed the strains "pacifist" and "aggressor." Ginsburg obtained the opposite results; C57BL mice always defeated C3H mice in a paired encounter. He also discovered that a series of defeats could render a formerly dominant animal submissive, and victories could inspire the meek to fearsome feats of furry fisticuffs (Ginsburg and Alice, 1942). When they compared their data, the authors realized the consequences of a difference in heredity must depend strongly on the environment. Ginsburg (1967) later demonstrated that small differences in the manner of handling the mice prior to weaning accounted for the differences in fighting after sexual maturity. Early in his career, perhaps during a critical period for the shaping of ideology, he became an interactionist, as did several others who learned the lessons of these early studies.

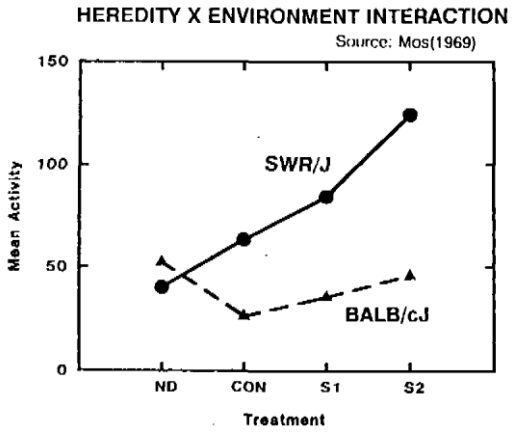

Fig. 2 Mean locomotor activity in an open field for two inbred mouse strains treated differently at 23 and 24 days of age, prior to testing at 50 days. Group ND was not disturbed prior to testing. Group CON was placed into an electric shock box but given no shock. Groups S1 and S2 received two levels of electric shock, S1 being mild and S2 moderate. Redrawn with permission from Mos (1969); additional details presented in Mos et al. (1973).

Scott went on to become director in 1946 of the Division of Behavioral Studies at the Jackson Laboratory, a world center of genetic research with mice and dogs, and Ginsburg joined his staff (Ginsburg, 1992). In 1948 Joseph Royce, a young doctoral student of Thurstone at the University of Chicago, went to the Jackson Lab as a research fellow to do a factor analysis of canine behaviour, and was exposed to interactionist ideas. Royce himself later became an ardent advocate of a multifactorial systems approach to understanding behaviour. His own studies of mice, summarized in his autobiography (Royce, 1978), convinced him that " ... heredity-environment *interactions*, which have not been adequately investigated for any behavioural phenotype to date, arc extremely subtle, sensitive, and important."(p. 229, emphasis in the original) Interactionism was recognized in his laboratory at the University of Alberta. For example, in the M.Sc. thesis of his student L. Mos (1969) it was argued that: ¯The phenotypic development of each genotype is determined by its ontogenctic environment."

(p. 4) Mos subjected two inbred strains of mice to four different treatments shortly after weaning and then tested numerous behaviours several weeks later. For open field activity, the early stimulation had dramatically different effects on the two strains (Figure 2). Among the 19 measures of behaviour he analysed, 18 showed strain by treatment interaction significant at $\alpha = .025$ or better.

A most influential example of heredity-environment interaction was provided by the McGill bright and dull rat lines selectively bred for learning of the Hebb-Williams mazes. Hughes and Zubek (1956) reported a large line difference at the 10th generation of selection with standard lab rearing. Cooper and Zubek (1958) later reared the two lines at the thirteenth generation of selection in either an enriched or restricted environment, and they found no significant line difference in either environment. This study is the most widely cited example of heredity-environment interaction (Platt and Sanislow, 1988), despite the fact that the global interaction term ($F = 3.07$, df = 2/59, P = .054) is not statistically significant (Wahlsten, 1990; see Note 2). The Cooper and Zubek article has been widely misinterpreted as evidence for a "reaction range" or a "reaction surface" in which the function relating mean value of a behaviour to each environment is essentially the same shape but with slightly different slope for each genotype and the rank order among genotypes is always maintained (Platt and Sanislow, 1988; see also Turkheimer and Gottesman, 1991). This study exemplifies a somewhat different concept, the norm of reaction (Lewontin, 1974), but does not prove rank orders are invariant. In fact, rank orders of strains are often changed when the environment is changed, as in the Mos(1969) study. When the same inbred mouse strains are tested for motor activity in different labs (Wahlsten, 1990; see Figure 3), certain strains are consistently high (c57BL/6) or low (BALB/c), but others are strongly affected by small details of the testing or rearing conditions. In a review of the literature, Erlenmeyer-Kimling (1972) concluded "that gene-environment interactions are numerous and that treatment effects are frequently reversed in direction for different genotypes."(p. 201)

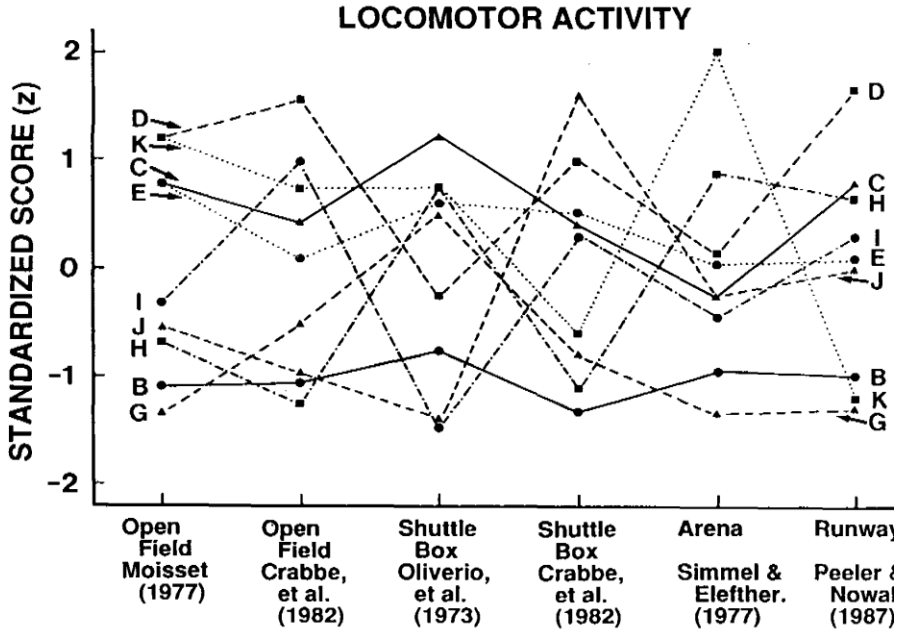

Fig. 3 Locomotor activity of the seven Bailey (By) recombinant inbred mouse strains (D, E, G, H, I, J, K) and their two progenitor strains BALB/cBy (B) and C57BL/6By (C). For each situation, the nine strain means were standardized to facilitate comparisons of different studies. Configurations of the test apparatus and rearing conditions varied slightly among laboratories. Reprinted with permission from Wahlsten (1990), *Behavioural and Brain Sciences*; copyright by Cambridge University Press.

Heritability Requires Separation of Causes Here we arrive at a conceptual barrier. Heritability analysis, as outlined above, requires an assumption that heredity and environment *do not* interact. The conceptual model used for this analysis posits two components, one determined by the individual's genes (G_i) and the other by the environment (E_i), which sum to determine a measured behaviour of an individual, such that $Y_i = G_i + E_i$. This corresponds to a causal model of development (Figure 4) in which the effects of the genes and environment are entirely separate and independent, and where the direction of causation is strictly from *G* and E to behaviour and never the reverse. Two strains of animals should differ by a preset amount when they are reared in the same

environment, no matter what environment it is. The consequences of a change in environment, be it dietary or psychological, should be the same for all people, regardless of genotype. The organism should not be able to actively choose or change its own environment.

The doctrine of the adders (G + E) is a theory of *two components* of behaviour which reduces to a doctrine of strict genetic determinism for one of the components, commonly identified with brain structure (Wilson, 1983). This is equivalent to a computer model of the brain, where there is a neat dichotomy between innate hardware and acquired memories or software. The theory of DNA-encoded brain structure was espoused recently by the editor of *Science* to justify spending billions of dollars on the Human Genome Project as a means for curing anti-social behaviour (Koshland, 1990).

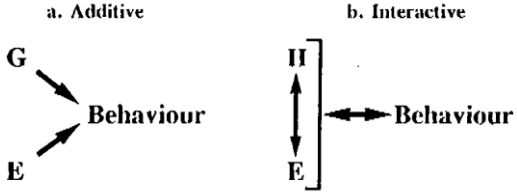

Fig. 4 Causal diagrams corresponding to two conceptual models. (a) The Y = G + E (additive) model. Effects of genotype (G) are said to be entirely separate from and unrelated to effects of environment (E) on the measure of a behaviour. (b) The interactive model. Two interdependent processes, heredity (H) and environment (E), jointly determine development of behaviour, and behavioural processes can influence environment-gene interrelations.

The additive model is not biologically realistic. There are so many instances where the response of an organism to a change in environment depends on its genotype or where the consequences of a genetic defect depend strongly upon the environment, that genuine additivity of the two factors is very likely the rare exception. The truth of this assertion is widely recognized (Bateson, 1987; Cairns et al., 1990; Fentress, 1981; Gollin, 1985; Gottlieb, 1991; Oyama, 1985), but not in human behaviour genetics. Abundant evidence at the molecular level now allows little room for doubt that circumstances can determine when and where a gene acts to influence the course of development and neural activity (Rusak et al., 1990; Schoups and Black, 1991; Sharp et al., 1991). The features of the environment most effective in turning a gene on and off, as well as the time of greatest sensitivity to modulation and the shape of the function depend on the specific gene and must be tested empirically. A well-documented example of this control is the production of the hormone melatonin (Vaughan, 1984). Light entering the eye stimulates the hypothalamus, which activates cells in the spinal cord, then a sympathetic ganglion and finally the pineal gland, where the production of several enzymes synthesizing melatonin is shut down, later to be resumed during darkness. The effect of light in the environment is mediated by the nervous system; environment rarely acts directly on a gene to affect its expression.

Perils of Partitioning Variance

In addition to being outdated biology, the assumption of additivity poses statistical perils in many situations. Only if an individual score is a *sum* of two components can the variance of the sum be divided meaningfully into separate parts. This is done routinely with statistical analysis to separate between and within group variance. Statistically significant superiority of one gender on a cognitive measure, for example, might conceal a trivially small effect If the mean scores of men and women differ by δ standard deviations, then the proportion of the total variation attributable to the gender difference is $\omega^2 = \delta^2 / (\delta^2 + 4)$ for large samples (Cohen, 1988). The meta-analysis by Feingold (1988) indicates that the magnitudes of gender difference on tests of numerical and spatial abilities, which were never much above $\delta = 0.5$, are gradually disappearing. When $\delta = 0.5$, about 6% of the variance arises from the group difference. Hence, the overwhelming bulk of the variance occurs among people having the same gender. This is a form of partitioning variance that generates little opposition because there is only one factor, gender. In this example, the size of the gender difference clearly involves cultural factors, and the small ω^2 value is not a precise separation of nature and nurture.

When we want to learn about the relative contributions of the *combined* effects of two or more distinct factors, partitioning of variance clearly becomes perilous. Let us use the term "heredity" or H rather than genotype because heredity in general entails more than the genes in the nucleus. Suppose a multiplicative relation is true for a behaviour. The simple formula $Y_i = H_iE_i$ gets at the essence of the difficulty, although many other kinds of interaction arc possible. Perhaps the ordinary family environment yields little benefit for a child with bad genes; the multiplicative model asserts the child low in H would benefit less from an improvement in E than would one high in H. Assigning children randomly to homes would be unethical, so let us rear two genetically uniform strains of animals with heredity values H_1 and H_2 in an environment worth E_l. For strain 1, the expected value of Y is H_1E_1, and for strain 2 it is H_2E_1. Thus, the difference in strain means, which should be proportional to the "heritability," will be $H_1E_1 — H_2E_1 = E_l (H_1 — H_2)$. Logically, a simple two group design like this should reveal the pure effect of a difference in heredity uncontaminated by the properties of the environment, but algebraically it is apparent that the size of the strain difference depends as much on the specific rearing environment as it does on the difference in H values (Wahlsten, 1990). For a multiplicative interaction between H and F, the main effects and interaction effect are not independent; although algebra can separate the sum of squares into three parcels, their magnitudes are joinly determined. When H and E interact, heritability is plastic and environmental plasticity is heritable. Only if H and E are truly additive can their effects be neatly separated with algebra. It has been shown that if certain kinds of interaction are present in the data, the estimated heritability coefficient which assumes no interaction will be inflated (Lathrope et al., 1984). A log transformation would render multiplicative variables additive, but this would not change reality; rather, it could obscure the truth (Wahlsten, 1990).

Some behaviour geneticists acknowledge the mathematics but argue that actual tests of interaction effects with analysis of variance (ANOVA) find they are usually not statistically significant and can therefore be ignored (Detterman, 1990). Unfortunately, for several realistic and psychologically interesting kinds of interaction the power of the test of the interaction term in an ANOVA is much lower than the power of the test of the main effects (Wahlsten, 1990, 1991). ANOVA treats interaction as what is left over after main effects have been taken into account. A nonsignificant interaction term is persuasive only if the test is based on a sufficiently large sample, which often is not the case. Increasing the range of each variable may help to reveal an interaction, but it generally cannot increase the sensitivity of the test of interaction until it equals the power of tests of main effects (Wahlsten, 1990); increasing the range also increases power of tests of main *effects,* and the power differential remains. For many kinds of interaction, the difference in power can be quite large.

MATERNAL ENVIRONMENT

Consider an experiment done in my laboratory. Using mice, we separated the effects of the prenatal and postnatal maternal environments by grafting the ovaries of an inbred strain into either the same strain or an F_1 hybrid, and then at birth fostering the pups to either an inbred or hybrid mother (see Table 1). Our working hypothesis was that the effects of differences in the maternal environment would be greater for inbred mice than hybrids; the hybrids should be better "buffered" against changes in the environment (Hyde, 1973). We tested numbers of animals that we could afford to test in a two year period, making this a very ambitious experiment with 337 mice or an average of 21 per group being run through several tests of complex behaviour and then processed for histology at 100 days after birth. Certain main effects were large but interactions usually fell short of significance (Bulman-Fleming, Wahlsten and Lassalle, 1991). Does this prove heredity and environment really were additive? One can readily estimate sample size needed to achieve an acceptable level of power when a one-degree-of-freedom interaction effect can be tested as a linear contrast (Ψ_c) among J group means (μ_1 to μ_1). If the contrast is $\Psi_c = c_1\mu_1 + c_2\mu_2 + \ldots + c_J\mu_J$ and the standard deviation within each group is σ, then sample size to yield power 1 - β of the test when Type I error probability a is given by Wahlsten (1991):

$$n \text{ (per group)} = \frac{(z_\alpha - z_{1-\beta})^2 \Sigma c_j^2}{(\frac{\Psi_c}{\sigma})^2} + 2$$

TABLE 1
Hypothesized Means of Brain Weight (mg) with Interaction Present and Contrasts to Test Various Effects

Geno	Pre	Post	Mean	BALB vs C57	Inbred vs Hybrid Pre	Inbred vs Hybrid Post	Geno (I vs H) × Post
B	B	B	500	−1	−1	−1	−1
B	B	H	510	−1	−1	1	1
B	H	B	505	−1	1	−1	−1
B	H	H	515	−1	1	1	1
C	C	C	490	1	−1	−1	−1
C	C	H	500	1	−1	1	1
C	H	C	495	1	1	−1	−1
C	H	H	505	1	1	1	1
BXC	B	B	530	0	−1	−1	1
BXC	B	H	535	0	−1	1	−1
BXC	H	B	532.5	0	1	−1	1
BXC	H	H	537.5	0	1	1	−1
CXB	C	C	520	0	−1	−1	1
CXB	C	H	525	0	−1	1	−1
CXB	H	C	522.5	0	1	−1	1
CXB	H	H	527.5	0	1	1	−1

Abbreviations: B, BALB/cWAH2; C, C57BL/6J; H, F_1 hybrid; I, Inbred (either BALB or C57); Geno, Genotype; Pre, Prenatal maternal environment; Post, Postnatal maternal environment. Model to obtain means: BALB − C57 = 10mg; I vs H prenatal effect = 5.0mg for inbred pups, 2.5mg for hybrid pups; I vs H postnatal effect = 10mg for inbred pups, 5mg for hybrid pups; hybrid pups − inbred pups = 30mg.

The group means shown in Table 1 were derived from the explicit hypothesis that the environmental effects on hybrids arc half the magnitude of those on inbreds. Under this hypothesis, the sample size required to detect the difference between the BALB and c57 inbred strains with a power of 90% when $\alpha = 0.05$, one-tailed, would be only 12, whereas for the interaction between genotype and postnatal environment it would be 80 mice *per group*, which is four times the number that exhausted the resources of my NSERC grant. Examples for many other kinds of interaction are described in Wahlsten (1991).

Turning to genetic studies of humans, suppose you could convince someone to finance a really large twin or adoption study. How could you test for the presence of gene-environment interaction? For behaviours of interest to psychologists, there is no generally acceptable test. If women were like nine-banded armadillos who always give birth to monozygotic quadruplets (Storrs and Williams, 1968), and if they were willing to turn their broods over to the state for controlled rearing, we might be able to devise a method. If we could identify a specific genetic locus relevant to a behaviour and specify who had which form of that gene, as occurs with phenylketonuria (Woo, 1991), a good test could be done. However, heritability analysis is employed when the investigator has no idea about how many genes may be involved, where they may be on the chromosomes or what they may do. Lacking an effective empirical test, the statistical model is not falsifiable.

Other Dubious Premises

Although the problem of interaction is lethal for heritability analysis, this is not the only dubious premise. There are at least four other major difficulties. (1) A gene codes for something at the molecular level and does not code for a definite part of a test score at the psychological level. Where a gene effect on IQ test score has been documented, as in PKU, the size of the effect is not fixed by the genotype; it depends on several other co-acting factors (Holtzman et al., 1986; Levy and Waibren, 1987). Enzymes, the products of genes, are arranged in complex metabolic pathways with numerous feedback loops and interactions galore (Lehninger, 1982). Studies with mice have documented large differences in the effects of a major gene when combined with different sets of genes at other loci (Coleman, 1981; Messer et al., 1991), which invalidates the simple summation of genetic effects across loci. (2) A behaviour is not the product of only two causal influences, H and E. A third source of

individual differences, sometimes called "randomness" of development, arises internally in the organism (Bookstein, 1988; Bulman-Fleming and Wahlsten, 1991; Gartner, 1990; Kurnit et al., 1987), and this factor interacts with H and E effects. (3) Genuine absence of gene-environment covariance is most unlikely in research with humans. Path analysis or LISREL can come to the rescue only if the aspects of the environment relevant to the behaviour in question are known and can be summarized in a simple number, which is unlikely (Wachs, 1983). Complex path models usually involve more unknown parameters than there are observed correlations and thus are underdetermined (Taylor, 1980). (4) Futhermore, there are good reasons to believe that behaviour can actively select and change the environment (Gottlieb, 1991; Odling-Smee, 1988). This bidirectionality of effects contradicts the model in Figure 4a, arid it yields a covariance of Ji and E that violates an assumption of ANOVA.

In view of all this, I would feel more secure riding a three legged moose over thin ice than relying on a heritability coefficient to help me understand the origins of individual differences or predict future levels of intelligence. These and other shortcomings recently prompted the quantitative geneticist Oscar Kempthorne (1990) to comment " that most of the literature on heritability in species that cannot be experimentally manipulated, for example, in mating, should be ignored."

Pursuit of Single Genes

If we eshew heritability analysis, what viable alternatives remain? Among those of us who work with laboratory animals, most devote their time to detecting new genes or studying their consequences. For example, in my laboratory, evidence has accumulated that absence of the mouse corpus callosum is attributable to three recessive genes (Livy and Wahlsten, 1991). Neumann and Collins (1991) have identified three genes making mice prone to sound induced seizures. It is particularly encouraging to note the excellent research being done by the younger generation of behaviour geneticists. Sokolowski and her students have detected and mapped a single gene *(for)* affecting foraging patterns in fruit fly larvae (de Belle et al., 1989), and they are now working to discover how it functions biochemically. Wilson and her students are studying the development of behaviour in mice with the obese gene *(ob),* which show a marked preference for warmer temperatures than their genetically normal siblings (Wilson et al., 1991). Rankin and her students are devising methods to study learning in single gene mutants of nematode worms (Rankin and Beck, 1992). If some psychologists see this work as esoteric and irrelevant to humans, they should be aware that dozens of genes identified in fruit flies are also found in vertebrates (Merriam et al., 1991), and that in mice there are large stretches of the chromosomes which are homologous with pieces of human chromosomes, containing the same genes in the same order (Lalley et al., 1989). The relevance of mouse research to human behaviour genetics is greater than ever, yet communiciation between the two groups is mainly in one direction, perhaps because the advocates of heritability analysis would prefer not to see what the "mousers" have found.

Generic versus Genetic Statistics

Should we conclude that scientists interested in heredity and behaviour ought to study mice, flies and worms? Perhaps many should, but there is a role for research involving human heredity in psychology. Knowledge of genetics can help to design elegant studies of nongenetic effects where exceptional control is possible and confounding by genetic effects is minimized. An obvious example is monozygotic twins (Mz) reared together but discordant for major disorders such as Alzheimer's disease (Creasey et al., 1989) or schizophrenia (Reveley et al., 1982). The pair has the same genotype and very similar early environments, so there must be some relatively subtle aspects of development which can yield a really large difference later in life. Another example is dizygotic twins (oz) compared with nontwin siblings having the same parents. Genetically, the two kinds of pairs are equally dissimilar. Twins are conceived and born on the same day, unlike sibs who make their appearance years apart This provides an excellent opportunity to study cohort effects due to societal changes (e.g. Flynn, 1987). Contrasting same-gender with male-female dizygotic pairs could help to evaluate generational changes in gender related environments.

The influence of heredity on behaviour can also be studied legitimately, provided the researcher is willing to use *generic* rather than *genetic* statistics. Studies involving twins and adoption can be analysed using the same

multiple regression methods we apply with environmental experiments. Suppose one compares MZ and DZ twins. Some would advocate use of the formula $h_B^2 = 2(r_{MZ} - r_{DZ})$ to assess the size of the genetic effect (Gray, 1991), but this requires faith in a host of disreputable assumptions, such as additivity of H and F,. Instead, evaluate the null hypothesis that genetic variation is irrelevant for the behaviour with two tests of significance: (a) r_{bZ} vs. 0, and (b) r_{MZ} vs. r_{DZ}. If both tests are significant, this supports the hypothesis that genetic variation is important but does not prove it because confounded environmental effects could produce or magnify the differences. To assess how important the effect might be, which depends strongly on the range of variables studied, we could examine the multiple R^2 in a regression equation which predicts the difference between scores of a pair, using effect coding for sib vs nonsib, twin vs nontwin sib, and MZ vs DZ twins. If the data indicate there is a substantial difference between kinds of relatives, this does not prove heredity is the cause, but it might show the apparent effect is large enough to justify proceeding with genetic segregation analysis (Lalouel et al., 1983) to see if a major gene is involved and, if it is, linkage analysis to find where it occurs on a chromosome (Ott, 1985). This line of investigation can lead to discoveries that may actually help those who suffer from a faulty gene (Desnick and Grabowski, 1981). Furthermore, a preliminary twin study using generic, general purpose statistics is much less likely to be misinterpreted and misused by the nonspecialist in genetics than one which cites a heritability coefficient. Although generic regression methods will not provide a failsafe prophylactic to prevent reification of a parameter estimate, readers will hopefully be more critical and cautious when matters are expressed in familiar terms rather than the more technical language of the geneticist. Simply using the term heritability implies acceptance of its inherent assumptions.

A search for single gene effects on human behaviour is likely to uncover only those with large effects which behave conventionally. As Lewontin (1974) has emphasized, gene-environment interactions can occur in myriad forms which make the more commonplace genetic polymorphisms devilishly difficult to uncover with humans beyond experimental control.

Conclusion

Heritability analysis of human behaviour has become the dominant paradigm in academic psychology and now appears prominently in introductory texts, where it is presented to naive students who have no understanding of the false assumptions inherent in the calculations. This preeminence of heritability analysis is the outcome of a power struggle, not the resolution of a debate among scientists. Apparently it has persuaded many academics that the importance of genes for behaviour can be understood without knowing anything about the cells or physiology which connect the two grossly disparate levels. Partitioning variance with ANOVA seems almost second nature to psychologists, which may help to explain why heritability analysis has found a receptive audience here rather than in developmental physiology where interaction is ubiquitous and calculus is fundamental.

It is somewhat ironic that this rise to prominence has taken place during a period when biological research on gene action has accumulated so much evidence of interaction. The theory of one gene → one character or the *mosaic* theory of heredity has been firmly rejected by biology in favour of one gene → one or more polypeptide molecules. The H + E model is a vestige of mosaic theory which claims that a gene codes for a fixed portion of the phenotype.

Interactionism, on the other hand, leads to a view of heredity and environment not as components but as dynamic and historically determined processes which give rise to structure and motion by virtue of the dialectical interplay of the internal and the external, the nucleus and the cytoplasm, the individual and society. Intricate structures emerge from relatively undifferentiated tissue through qualitative transformation. Attempts to dichotomize are continually defeated by new discoveries of how experience sculpts the pattern of synaptic connections in the cerebral cortex, how behaviour actively modifies and selects the environment, and how social interactions of the individual modulate the metabolic activities of genes.

Gene-environment interaction offers great possibilities for treating genetic disorders and deficiencies. It has exploded the old myth that the consequences of a genetic deficiency arc inevitable. If a child is inferior in

school because of an unknown genetic problem, the best kind of genetic research may help to design a program of effective treatment. Above all, it is ignorance of causes that makes poor development inevitable. Real genetic knowledge may enhance the modifiability of development ... *provided* we know precisely what gene is involved and how it works.

Notes:

1 Royce, like several other behaviour geneticists, was not entirely consistent in this respect. He stressed interaction in one context but often cited the concept of heritability and ignored interaction when theorizing about human behaviour (e.g., Royce, 1979).

2 The global interaction term may not be the best approach to assessing heredity-environment interaction in a 2 x 3 design. For the Cooper and Zubek (1958) data, it is reasonable to expect that the difference between the two strains would be greater in the normal lab environment prevailing during selective breeding than in either the restricted or enriched environment. Using raw data kindly provided by Dr. R. M. Cooper, a planned contrast testing that hypothesis reveals a significant interaction effect (t = 2.44, df = 59, p = .009).

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