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The Heritability Hang-up

The role of variance analysis in human genetics is discussed.

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The nature-nurture issue has provided some of the most keenly contested debates in the fields of biology, psychology, sociology, and physics during the past 5 years. As is well known, the same questions have been a source of controversy for more than a hundred years in diverse political and social climates. From time to time, practical measures have been implemented whose nature has often depended on the relation between the political ideals of the regime of the time and the ideals of the contemporary "scientific" participants in the nature-nurture controversy. Some of the history of this interaction in the United States as it pertains to intelligence has been surveyed by Kamin (*1*) and Allen (*2*).

The most recent explosion of interest in the question is probably attributable to Jensen's (*3, 4*) contention that, since inequalities in cognitive performance are largely genetic in origin, environmental intervention through educational or social innovations will be of minimal value in reducing these inequalities.

The premise for this argument is based on Jensen's analyses of the data from a large number of empirical studies. From these analyses, Jensen also argues that there is probably a strong genetic component to the observed differences in mean IQ between black and white children in the United States.

The analyses and arguments that were made by Jensen for IQ, and by others for other quantitative traits in humans, are all based on a fundamental methodology that was invented by R. A. Fisher, the analysis of variance. The analysis of variance is meant to cope with the problem of dissecting the multiple causes of observed phenomena when the actual physical chain of causation of each individual event cannot be followed. What is observed is the variation in the phenomenon, measured qualitatively by the variance; the analysis partitions this variation into a proportion that is ascribed to the variation in each causal element and each combination of causal elements. Thus for IQ, the total variance in IQ scores in a population would be partitioned into an environmentally caused variance due to variation in the life experience of individuals, a genetic variance arising from variation in heredity among individuals, a genotype-environment interaction variance reflecting the lack of additivity of genetic and environmental deviations, and an error variance arising from uncontrolled variations in test procedures and, more important, developmental accidents that cannot be associated with specific, known environmental variables. As we show below, this partitioning of the causes of variation is really illusory, and the analysis of variance cannot really separate variation that is a result of environmental fluctuation from variation that is a result of genetic segregation. The genetic variance depends on the distribution of environments and the environmental variance depends on the distribution of genotypes.

The analysis of variance is, in fact, what is known in mathematics as a local perturbation analysis. It is assumed that the actual IQ of an individual is some unknown function of genotype (G) and environment (E)

$$\mathrm{IQ} = f(G,E)$$

In any given population, there is some joint distribution of genotypes and environments

$$\phi(G,E)$$

and this joint distribution is mapped onto a distribution of IQ scores $\theta(\mathrm{IQ})$ by a functional equation.

A complete analysis of the causes of variation would involve predicting the changes in the IQ distribution $\theta(\mathrm{IQ})$ from changes in the distribution of genotypes and environments $\phi(G,E)$. However, such an analysis would require that we know the first partial derivatives of the unknown function $f(G,E)$. What we substitute instead is an analysis of what would happen to the mean of $\theta(\mathrm{IQ})$ if very small perturbations were made in the mean of $\phi(G,E)$. The analysis of variance is a way of estimating the effects of these very small perturbations in the means, and the variance components estimated are directly related to the partial derivations of the unknown function $f(G,E)$. Thus the analysis of variance produces results that are applicable only to small perturbations around the current mean. It cannot make any predictions about any larger issues.

In the analysis of variance of genetic and environmental causation, a special term is used for the proportion of all variance that is partitioned into the genetic variance. This proportion is called heritability in the broad sense (h^2_B). The genetic variance itself can be further broken down into a contribution that is due to individual alleles (additive variance), a contribution that is due to pairs of homologous alleles at a locus (dominance variance), a contribution that is due to combinations of nonhomologous loci (epistatic variance), and so forth. The proportion of the phenotypic variance that is additive genetic variance is called heritability in the narrow sense (h^2_N).

We claim that this type of formulation is irrelevant to human population genetics on two counts. First, a model that is structured in this way cannot produce information about causes of phenotypic differences. Second, we do not, nor can we, use

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variance analysis in the resolution of those problems that are acknowledged to be central to the study of human population genetics.

Use and Estimation of Heritability

The broad-sense heritability is not used in practical applications such as plant and animal breeding. For the breeder, h^2_N, based on the additive genetic variance, is more important since Fisher's fundamental theorem of natural selection predicts that h^2_N determines the speed with which desired changes in a phenotypic measure can be produced by artificial selection. In fact, it determines, to a large extent, the selective breeding program to be undertaken. In situations of low h^2_N (especially with large families) family selection is usually preferred. In such a scheme, the family mean, rather than individual performances, is used as a selection criterion. When heritability is high, individuals are usually selected by the breeder according to their own phenotypic values. The estimate of h^2_N is obtained from phenotypic correlations that are observed among relatives, predicted correlations between individual relatives on the basis of Mendelian theory, and empirical correlations in phenotype between mates; environments are always randomized over genotypes.

The narrow heritability does not provide an index of the importance of an individual's genotype in determining the phenotype. It is merely an index of the amenability to selective breeding and, as such, is of practical use in the construction of breeding programs.

The analysis outlined above works because in animal and plant breeding experiments it is possible, by appropriate design, to eliminate those environmental correlations between relatives that would be confused with genetic correlations. Similarly, in agricultural applications correlation between genotype and environment is usually negligible due to randomization of the environmental effects—which is, of course, a major aim of the experimental design. To an extent, it is also possible to estimate genotype-environment interaction in controlled populations by carrying out selection programs and estimation procedures in many environments appropriate to the final conditions of agricultural production. Nevertheless, genotype-environment interaction remains a serious problem even in agricultural applications. If varieties are tested under a particular range of conditions, or a selection program is carried out over a limited range of environments, the selected material may be totally inappropriate for other conditions. This has been the case with the hybrid corn that is so successful in the 20-foot (6 meters) soils of the American corn belt but is poorly adapted to tropical conditions or the marginal rainfall and temperature conditions of the Soviet Union (*5*). So, too, the beef and dairy cattle breeds that are very productive in Europe and North America are completely unsuited to tropical Queensland where Zebu cattle, normally less productive, are the basis of cattle improvement (*6*).

In problems concerning the population genetics of human behavioral traits, the existence of a variance contribution from genotype-environment interaction and a genotype-environment correlation have long been recognized as major difficulties. On the one hand, the obvious problems these cause for estimation of h^2_N have led to the use of the h^2_B in discussion of such characters. On the other hand, as was implied by Falconer (*7*) and emphasized by Moran (*8*) and Layzer (*9*), the very existence of genotype-environment correlation precludes the valid statistical estimation of the genotypic, environmental, and interaction contributions to the phenotypic variance. That is because correlation makes it impossible to know how much of the phenotype similarity arises from similarity of genotype and how much from similarity of the environment. Thus in human population studies, where experimental controls are either impossible or unethical, statistical inference about the heritability of traits that are phenotypically plastic is invalid. We discuss these difficulties later from another point of view.

Human Diseases and Heritability

In principle, it may be possible to separate those phenotypes whose frequency of occurrence can be analyzed in terms of simple genetic probabilities from those requiring more complicated statistical analysis. The former class can be characterized by those traits whose deviation from their mean is due to rare deleterious alleles. The latter is characterized by those whose deviation from their mean is caused by contributions from many segregating gene loci, each of minor effect, and the interaction of the genotypes so produced with the environment. Segregation analysis attempts to distinguish between these two causes of phenotypic deviation. Whereas the first class is studied with the elementary tools of Mendelian ratios and the Hardy-Weinberg law, the second falls into the category of quantitative characters that were discussed above and would usually be studied by variance analysis and described in terms of their heritability.

In the study of human diseases that have some genetic basis, there may be room for discussion as to whether the distinction attempted by segregation analysis is possible. However, Cavalli-Sforza and Bodmer (*10*, figure 9.17) suggest that it is. The distinction is valuable for three medical and public health–related reasons: (i) we would like to cure the disease, (ii) some distress may be ameliorated by genetic counseling, and (iii) elimination of the disease is conceivable. In some ways, these are all interrelated, but we shall attempt to examine them in the order listed and ask how such a phenotypic variance analysis as we outlined above can help.

If the disease happens to be simply determined by a variant allele at a single locus, then we have a mathematically simple means of calculating the chance that an individual is genetically predisposed to the disease, given some family information. We are then led to questions about the biochemical environment of the individual and biochemical means of curing or treating the disease (*11*, pp. 259–265). More than a thousand clinical syndromes can plausibly be attributed (by segregation analysis) to the effects of single genes. Of those that can be treated at present (notably phenylketonuria, galactosemia, and fructosemia), the correction is made either by intervention in the diet of the infant patient or by replacement therapy. For those characters that show evidence of familial concentration but which are not inherited in a manner amenable to simple probabilistic prediction, the outlook for cure is not in the least helped by a knowledge of the proportion of variance that is genetic. Most such characters can be explained either in terms of single genes with reduced penetrance or in terms of multiple genes, each of which has a small effect. No matter what the heritability, the cure envisaged is always environmental manipulation; diabetes mellitus and celiac disease are examples of diseases with familial concentration, complicated inheritance patterns, and dietary treatments. The point is clear that for this second class of diseases, where it is usual to report heritability, this heritability is neither used in the search for a cure, nor, more importantly, in deciding whether treatment is possible.

For purposes of genetic counseling, if we know the precise mode of inheritance of the genetic disease, the procedure is relatively simple. We may suggest that individuals whose progeny are likely to be handicapped avoid reproduction. By knowledge obtained from amniocentesis, abortion of a

diseased fetus may be indicated. Of course, amniocentesis would be suggested in instances where pedigree information or other suggestions indicate that the fetus is at risk.

The techniques involved in calculating risks for diseases of unknown and possibly complex modes of inheritance are substantially more involved. Some of these are discussed by Smith (*12*) and in the references therein. These procedures often involve the use of an estimate of heritability. Here we encounter the major difficulty pointed out by Kidd and Cavalli-Sforza (*13*) in connection with their study of schizophrenia. There may be enormous differences between estimates of heritability that are obtained from assumptions of a single gene and a multigene model. These, in turn, generate very different estimates of the risk to progeny. For a number of genetic diseases with complex patterns of inheritance, there may be a genetic component, as indicated from familial concentrations. However, there is often not enough information to differentiate between the two models. For purposes of counseling about such traits, it may be dangerous to attach much significance to heritability, which may fluctuate widely depending on the genetic hypothesis that is used (*13, 14*).

Moreover, for characters of complex inheritance, we do not know whether a detailed genetic model provides a significant improvement over a purely empirical estimate in the assessment of risk. For common yet complex diseases like diabetes, excellent empirical data can be gathered and risks can be calculated separately for various ages, socioeconomic classes, cultural patterns, and the like. It is hard to see, in this case, how an inferred complex genetic model could be helpful unless it predicted and identified subgroups that were not taken into account in the empirical risk functions, and for which risk was substantially different from that of the population at large. We stress identification here because it is critical. Suppose, for example, that diabetes is a collection of etiologically different disorders, each with its own pattern of inheritance. If segregation analysis, pedigree analysis, or any other statistical analysis has led us to infer the existence of such groups, and has provided rules for deciding in any individual case to which etiological group the family belongs, then there is a double chance of error for the genetic counselor. First, he must decide to which etiological group the case belongs, and this cannot be done with certainty because there is a probability of misclassification. Second, given the classification, an error in the assigned risk is possible because the underlying genetic model that is inferred is only a guess at the real situation for this class. Thus the total error in assigning a risk may be no less, and in fact may be considerably greater, than if empirical risk functions only are used.

Finally, we come to the question of potential elimination of genetic disease, a topic that incorporates positive and negative eugenics and euphenics. In general, the effect of avoidance of reproduction in marriages with risk of disease to the progeny or abortion of the diseased fetus or both (both constitute negative eugenics) is to decrease the frequency of occurrence of the disease by an amount of the order of the mutation rate. This decrease, of course, occurs extremely slowly. For similar reasons, the dysgenic effect of relaxation of selection against formerly lethal recessive diseases is extremely slow. Changes in the frequencies of such diseases as diabetes mellitus, celiac disease, and schizophrenia, whose mode of inheritance is not known, are probably intermediate between the dominant deleterious and recessive deleterious diseases, and will therefore be largely determined by mutation rates. The influence of heritability in this case will be similar in magnitude to the influence of the selection coefficients of the heterozygotes in the simpler cases. The amount of time necessary for significant eugenic effects to be manifested must be so large that, with respect to the cultural changes occurring in the same period of time, the eugenic effects would most likely be negligible. We must remember that only 100 generations have passed since the Roman Republic.

The issue of eugenics is, at any rate, a political one and geneticists have by and large failed to understand it as such. If we are concerned with the public health question rather than the matter of individual suffering in individual families, which is the domain of the genetic counselor, then we are concerned with the effects of a given frequency of a genetic disorder on the collectivity. Geneticists have neither the right nor, more importantly, the power to determine the direction of public policy and legislation. Nor have the legislative and executive powers of the state made decisions induced by and based on genetic knowledge. What they have done (for example, in the Immigration Act of 1924) is to use genetic misinformation to rationalize a politically determined policy. When geneticists talk seriously of the implications of alternative eugenic schemes they are playing an academic game because there is no serious possibility that eugenic measures will be legislated as a result of scientific considerations. As usual, "eugenic" measures, such as the sterilization of welfare recipients, follow socioeconomic prejudices. In our opinion, geneticists ought to dissociate themselves utterly from eugenics because they can only give legitimacy (even if unwilling legitimacy) to pernicious social actions.

With respect to human genetic diseases, we conclude that variance analysis as summarized by heritability is irrelevant to attempts to cure and eliminate such diseases and is rarely applied in genetic counseling.

Normal Human Variation: Proteins

Harris (*11*) estimates that in man 30 percent of the loci which code for enzymes are polymorphic within populations. Lewontin (*15*) gives an estimate, based on blood groups in man, of 35 percent for the same quantity. The heterozygosity per locus per individual was 6 percent in the first study and 16 percent in the second study. One of the important conclusions to be drawn from the great amount of protein variation that was discovered is that we expect genetic variation among individuals within populations. In fact, Lewontin (*16*) has calculated the Shannon-Weaver diversity within populations, within races between populations, and between races, and has shown that 85 percent of human genetic diversity is attributable to variation between individuals within populations and only about 6 percent is due to variation between races. In the light of such quantitative results we must consider the statement by Jensen (*17*, p. 81) that "any genetically conditioned characteristic that varies among individuals within a subspecies (i.e. race) also varies genetically between different subspecies" with the implication that the variations within and between groups are somehow commensurate. The difficulty inherent in this statement is discussed below.

For the protein variations just mentioned, we are not interested in cure or counseling. Nor are we interested in changing allele frequencies by medical intervention. The primary reasons for studying this normal variation are those concerned with the elucidation of the evolutionary process. We are interested in the reconstruction of human biological history. We are interested in the correlations between cultural innovation and change and the spread of alleles. We are interested in determining whether a polymorphism is maintained by natural selection or is merely a transient phase in a stochastic process in which most selection acts to eliminate deleterious alleles. None of these issues has involved the use of phenotypic variance analysis, and it seems safe to say that, as an index, heritability is irrelevant here.

Normal Human Variation:

Quantitative Characters

Perhaps the best publicized, most controversial, and least understood area in which variance analysis has been applied is in the study of normal (nonpathological) quantitative phenotypic characters [that is, those that are not simply Mendelian but which have unknown or complex patterns of inheritance (or both)]. We have already introduced the concepts of broad and narrow heritability as they are used on characters of agricultural importance and animal breeding. In particular, the heritability of an economically valuable trait is useful in the design of breeding programs whose goal is to change the distribution of the phenotypic measure in a restricted population under a precise set of environmental conditions. We now present an analysis of why the application of this type of analysis to human behavioral traits cannot help to clarify the causes of a phenotypic measure. Our arguments are especially pertinent to the IQ controversy.

We start with the assertion that, for the quantitative characters in which we are now interested, differences in both genotypes and environment can be causes of phenotypic differences. What is not so well accepted, however, is that analysis of variance and its summary statistic, heritability, do not separate the two causes of variation in the phenotypic measure. This is because the analysis of variance is done (and heritability is calculated) with respect to a particular array of genotypes and environments in a specific population at a specific time. This array is usually a biased sample of the full array of genotypes and environments (*7*, p. 270). This problem has been extensively discussed and analyzed by Cavalli-Sforza and Feldman (*18*), Cavalli-Sforza (*19*), and Lewontin (*20*). Their verbal and mathematical models constructed to explicate the last point are similar in spirit. Let us discuss the one from Lewontin (*20*).

Figure 1 is a norm of reaction figure that gives the phenotype P as a function of the environment E for two different genotypes G_1 and G_2. Obviously, both genotype and environment influence the phenotype in this example. However, if the environments are symmetrically distributed around E_1 (Fig. 1), there will appear to be no average effect of genotype, while if the population is weighted toward an excess of G_1, the average phenotype across environments will be constant, as is shown by the dashed line. Thus the environmental variance depends on the genotypic distribution, and the genotypic variance depends on the environmental variance. This very important interdependence means that for

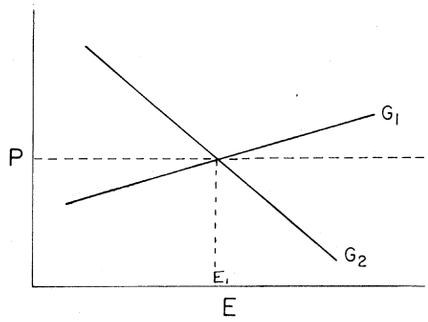

Fig. 1. Phenotype P plotted against an environmental variable E. If the environments are symmetrically distributed around E_1 there is no average effect of genotype. If there is an excess of G_1 in the population the average phenotype will be constant, as represented by the horizontal dashed line.

a character like IQ, where the norm of reaction, the present genotypic distribution, and the present environmental distribution are not known, we cannot predict whether an environmental change will change the total variation. Lewontin (*21*) gives an extreme example of the latter difficulty where all the variation between two populations is environmental, despite a heritability of 1.0 within each.

A further important point shown by Fig. 1 is that fixing either the environment or the genotype does not necessarily lead to a decrease in the total variance. For example, fixing genotype G_2 (and thus eliminating the genetic variance) increases the total variance because G_2 is more susceptible to environmental change. It is also easy to construct graphs like Fig. 1 in which environmental change improves the phenotype of both G_1 and G_2 but decreases the proportion of the variance that is genetic.

Figure 2 is a case such that, if the environments are weighted to the left, analysis of variance shows a strong genotypic effect. If the environments are weighted toward the right, thus producing improvement in both phenotypes, the proportion of

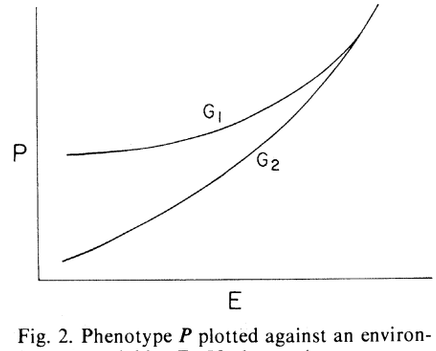

Fig. 2. Phenotype P plotted against an environmental variable E. If the environments are weighted to the left there is a strong genotypic effect. If the environments are weighted to the right the genetic variation is reduced.

variation that is genetic is reduced. This situation is ignored by both Jensen (*22*) and Herrnstein (*23*), whose discussion does not take account of this possible form of genotype-environment interaction. The specific model of Cavalli-Sforza and Feldman (*18*) incorporated genotype-environment interaction through a cultural contribution to the phenotype of an offspring which was determined by the phenotypes of the parents. In their model, the cultural component was itself transmitted from parents to offspring each generation. This cultural inheritance can have a pronounced effect on the phenotypic mean in a very short time. In fact, even in the absence of genetic variation, correlations between relatives may be expected from cultural effects alone. When both biological and cultural inheritance occur, separation of these effects is bound to be extremely difficult, especially in the absence of reliable data on adoptions (*18*). Our recent work indicates that cultural effects can strongly influence gene frequency changes as well as overcome the effects of strong natural selection in the sense that phenotypes acquired by learning or other modes of cultural transmission can spread through a population even though they lower the fitness of the individuals showing the phenotype. In the process, gene frequencies also change (*24*).

Heritability and Differences Between Groups

Jensen (*17*) states that "the fact of substantial heritability of IQ within populations does increase the a priori probability that the population difference is attributable to genetic factors." Many authors have pointed out the logical flaws in this statement and counterexamples have been presented. For purposes of argument, consider the case of skin color. If we estimate the heritability of skin color among white New Yorkers, including people of Italian, English, Irish, Puerto Rican, and Polish ancestry, we would find a high heritability. Suppose we now compare a group of New Yorkers who are left to winter in the city with a group of their well-to-do in-laws who spent the winter in Miami Beach. There would be a considerable skin color difference between groups, but no genetic causation.

Lush (*25*) introduced the concept of between-group heritability h^2_B in animal breeding. He was primarily interested in family groups and expressed the heritability of family means as

$$h^2_B = h^2_N \frac{1 + (n-1)r}{1 + (n-1)t} \quad (1)$$

where h^2_N is the narrow-sense heritability in the whole population, r is the intraclass genetic correlation among members of the same group, t is the analogous intraclass phenotypic correlation, and n is the group size. The heritability of within-family deviations (h^2_W) is

$$h^2_W = \frac{1 - r}{1 - t} h^2_N \quad (2)$$

so that when n is large

$$h^2_B \simeq h^2_W \frac{(1 - t)r}{(1 - r)t} \quad (3)$$

Again it should be stressed that the heritability here is always narrow sense and that the derivation of these equations (*7*, pp. 232–237) is made for the purpose of comparing family selection with individual selection in breeding work. There was no consideration of genotype-environment correlations which, if they exist, make it impossible to estimate r in Eq. 1, Eq. 2, and Eq. 3. From Eq. 3, it seems tempting, since t can be estimated in a standard way from analysis of variance, to plot h^2_B as a function of h^2_W and r for given t (*26*, p. 300). In this functional representation, h^2_B is a dependent variable while h^2_W, r, and t are independent variables. However, this is an inversion of the actual situation because the intraclass genetic correlation is defined by the relation of the variance within groups to the variance between groups. Thus Eq. 3 is a definitional tautology, not a causal relationship. In any sense of causation or of estimation, r is dependent on h^2_B and not vice versa. The suggestion that h^2_B is in some way predictable from h^2_W, or that the size of h^2_W somehow contains information about the size of h^2_B, is entirely spurious. However you look at Eq. 1, Eq. 2, and Eq. 3, under the restrictions necessary in the study of human behavioral traits we cannot obtain sufficient information to sustain the claim that from high heritability and large between-group differences it follows that the difference is largely genetic.

Differences in IQ performance between the races have long been accepted as an established fact. As we have pointed out, it is logically incorrect to argue, as Jensen does, that these differences are due to genetic factors. The recent study by Tizard (*27*) indicates in a practical way how flawed this argument may be. Tizard studied the IQ scores of white, black, and mixed-race children in English residential nurseries of high quality. In one study of 85 children, aged between 2 years 0 months and 4 years 11 months, all of whom had been in residence for at least 6 months, three tests were administered. These were a Reynell comprehension, a Reynell expression, and a Minnesota nonverbal. In all tests, the nonwhite children did better on the average, but only in the last test was the difference significant in favor of the nonwhite children.

In another study, Tizard obtained Wechsler preschool and primary scale of intelligence full-scale IQ's of 64 children, aged 4 to 5 years, all of whom had been institutionalized from the age of 4 months to at least 2 years. Three subgroups were examined: those still in the institution, those adopted by white families, and those restored to their natural mothers. In all the subgroups, nonwhites did better on the average than whites, but the differences were not significant. It has often been emphasized (*18*) that nonrandom adoptions may severely confound the results of such studies. Tizard considered whether the placements or adoptions had been made randomly and found no evidence for matching or attempted matching, although she gives no details of this comparison. If her claims of randomness of adoption are correct, then Tizard's work makes a substantial methodological advance.

In summary, studies of differences between the races at present leave us with two difficulties. First, as the studies by Tizard (*27*), Nichols and Anderson (*28*), and others indicate, the extent of differences between the races may vary widely with the environmental regimes of the groups. In fact, in their study of black children from southeastern United States, Kennedy *et al.* (*29*) point out that these children's low mean IQ should not be compared to the national norm. Second, we are unable to make any inferences from between-group differences and within-group statistics about the degree of genetic determination of the between-group differences. In other words, the concept of heritability is of no value for the study of differences in measures of human behavioral characters between groups.

Historical Reconstruction

To see a legitimate use of heritability analysis in human populations, we need to go back to Fisher's original ideas of genetic variance, and especially to his fundamental theorem of natural selection (*30*). This theorem states that the rate of change of fitness is equal to the additive genetic variance in fitness. It is a consequence of the definition of additive genetic variance as the regression of offspring phenotype on parental phenotype that a parallel theorem holds for any phenotypic character. The change in mean phenotype is equal to the ratio of the additive genetic variance to the total variance (that is, h^2_N) multiplied by the selection differential.

It can also be shown that as selection progresses, the additive genetic variance is "used up" so that the h^2_N is decreased finally to zero, or nearly so. A consequence of these theorems is that, if natural selection has long been in operation on a character, the additive genetic variance for the character should be small, and the only genetic variance left should be nonadditive (dominance and epistatic variance). Thus we may be able to judge, from the ratio of h^2_N, which goes to zero during evolution, to h^2_B, which does not, how much selection has gone on. For example, if we really believed the estimate of 0.75 for the h^2_B of IQ in European populations (we do not), and if we believed the single published estimate of h^2_N of 0.40 (we do not), we would be forced to conclude that whatever it is that IQ measures, it has not been under intense selection for very long. Conversely, if there is a great deal of nonadditive genetic variance, but very little additive, we may guess at a long and consistent history of selection.

Of course, these are only weak inferences since, in the absence of knowledge about selection intensities, we cannot specify what we mean by long and intense selection. In addition, because of genotype-environment interactions, especially in behavioral traits, a long history of selection in one set of environments may reduce the h^2_N to a very low value, but a recent change of environment may produce a new level of additive genetic variation.

Conclusions

The problem we have been examining is the degree to which statistical structures can reveal the underlying biological structure of causation in problems of human quantitative genetics. We must distinguish those problems which are by their nature numerical and statistical from those in which numerical manipulation is a mere methodology. Thus, the breeding structure of human populations, the intensities of natural selection, the correlations between mates, the correlations between genotypes and environments, are all by their nature statistical constructs and can be described and studied, in the end, only by statistical techniques. It is the numbers themselves that are the proper objects of study. It is the numbers themselves that we need for understanding and prediction.

Conversely, relations between genotype, environment, and phenotype are at base mechanical questions of enzyme activity, protein synthesis, developmental movements, and paths of nerve conduction. We wish, both for the sake of understanding and prediction, to draw up the blueprints

of this machinery and make tables of its operating characteristics with different inputs and in different milieus. For these problems, statistical descriptions, especially one-dimensional descriptions like heritability, can only be poor and, worse, misleading substitutes for pictures of the machinery. There is a vast loss of information in going from a complex machine to a few descriptive parameters. Therefore, there is immense indeterminacy in trying to infer the structure of the machine from those few descriptive variables, themselves subject to error. It is rather like trying to infer the structure of a clock by listening to it tick and watching the hands. At present, no statistical methodology exists that will enable us to predict the range of phenotypic possibilities that are inherent in any genotype, nor can any technique of statistical estimation provide a convincing argument for a genetic mechanism more complicated than one or two Mendelian loci with low and constant penetrance. Certainly the simple estimate of heritability, either in the broad or narrow sense, but most especially in the broad sense, is nearly equivalent to no information at all for any serious problem of human genetics.

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Humanizing Computerized Information Systems

Guidelines developed in a series of workshops are presented and their implications are discussed.

Theodore D. Sterling

The accumulation and control of information is a critical function for government and private, industrial and nonindustrial organizations. Yet the role of information as an organizational resource is not very well understood, especially as it is related to the organization's environment. What does appear is that computerized information systems have become a facilitating technology that interacts with organizational, historical, and environmental pressures and goals to shape not only the internal structure of an organization but also its interactions with society (*1, 2*). There is little doubt that the computerized or automated information system is revolutionizing the management of most, if not all, systems by which goods and services are produced or information is accumulated. This should be a source of great concern.

Weizenbaum (*3*) asked whether large computerized systems can be used by anybody except governments and really large corporations and whether such organizations will not use them mainly for antihuman purposes. The power of computerized information systems to control large enterprises answers the need to manage large systems and make them amenable to human control. By any criteria of management performance, computerization of a system permits its detailed control, and thus the computer is the ideal management tool. But the cost of the control is high. Start-up costs to redesign and computerize large-scale enterprises are immense. In concentrating on feasibility and workability and simultaneously minimizing costs, few systems designers seem to have been concerned about whether their products will be used for antihuman purposes.

In many ways, it is immaterial whether control over the management network is exercised by manual means or by automation. As long as official procedures are detrimental to human dignity, nothing is changed in converting to automation—except that individuals may shift the blame for their oppression from the human cog to the computer cog. It may be necessary, therefore, to clarify the dehumanizing components of a management system, which may be present whether or not the system has been automated, and to provide relief for any suffering they may have caused.

In a previous analysis (*4*) I pointed to two design strategies that account in large part for the presence of dehumanizing features in a management system. First, the efficiency of an enterprise is commonly increased by treating the recipients of the service and participants in the system as unpaid components whose time, effort, and intelligence do not appear in the cost accounting. Then, in order to maintain the efficiency of procedures once they have been established, the system is made exceedingly rigid, permitting freedom of action at only a few, usually hidden, focal points of real control. Dehumanizing features are thus already ingrained in most systems of management, and automation

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