Genetic Theories and Influences: Comments on the Value of Diversity

JAMES F. CROW, *University of Wisconsin*

Professor Crow agrees "for the most part with Jensen's analysis." He does suggest qualifications when drawing inferences from existing studies in biometrical genetics. First, he notes his reservations about the reality of the mathematical assumptions implicit in analysis of variance models. Second, he draws attention to the limited sample size available in studies of twins and siblings reared apart and asks how representative such groups are. Third, he notes that predictive models have inherent limits when new, qualitatively different, treatments are introduced into the environment.

Biometrical genetics has become quite a sophisticated subject with a substantial body of mathematical theory. One reason for this development is that the simple, mechanistic nature of Mendelian inheritance is very inviting to probabilistic model-builders. A second reason is that, because complex traits depend on more genes than can be individually identified, some sort of statistical treatment is necessary.

The foundations for the theory of biometrical genetics were laid by R. A. Fisher and Sewall Wright, using methods depending mainly on correlation and variance analysis. The procedures are widely used in animal breeding, thanks especially to J. L. Lush, who has been conspicuously successful in adapting these procedures to use in practical breeding problems. The general theory and methodology in this field are described with insight and lucidity by Falconer (1960).[1] Jensen's article, together with many others that he has written recently on this subject (see his bibliography), constitutes a thorough review and synthesis of

[1] This and subsequent references are to articles and books cited in Jensen's article.

the various attempts to apply these methods to human intelligence and scholastic achievement. Jensen has become a leader in this field, and I as a population geneticist, admire his understanding of the methods and his diligence and objectivity in bringing together evidence from diverse sources. He presents the evidence fairly, relying on empirical data in preference to introspection or traditional wisdom, and is very careful to distinguish between observation and speculation.

I shall confine my comments mainly to the genetic aspects of the article. I agree for the most part with Jensen's analysis. Any differences could probably best be described by saying that, in general, I have somewhat less confidence than he in the quantitative validity of the methods—more reservations about the reality of the necessary assumptions. I don't mean by this that I would reach opposite conclusions; I am simply more agnostic. This is especially true as regards intergroup comparisons and, in particular, the importance of genetic factors in racial differences.

The Concept of Heritability

Much of Jensen's article is concerned with the heritability of intelligence (I am trying to use the term intelligence in the same technical sense as he does). The word heritability has been used for some time by psychologists studying twins, but the measures—such as Holzinger's H-index and various modifications thereof—have not usually corresponded to the geneticist's definition. Jensen has done a great deal to clarify this point.

Heritability, in the geneticist's terms, can be described in three equivalent ways, depending on whether the viewpoint is that of analysis of variance, regression, or correlation analysis: (1) the ratio of the genetic variance to the total variance, (2) the regression of genotype on phenotype, and (3) the square of the correlation of genotype with phenotype. Jensen uses mainly the first.

As he says, the total or phenotypic variance (V_P) can be analyzed into genotypic (V_H) and environmental (V_E) fractions:

$$V_P = V_H + V_E + \text{interactions and error.}$$

The genotypic variance (V_H) can be subdivided further into the additive or genic variance (V_G), dominance (V_D), and inter-locus interaction (epistasis) (V_I). (See Jensen, p. 37.) Thus,

$$V_H = V_G + V_D + V_I.$$

V_G is defined as the best linear representation of the phenotypic values (best in the least squares sense), and V_D and V_I are treated as deviations from it. This procedure for subdividing V_H has two important advantages: The first is that V_G, V_D, and V_I defined this way are independent and we do not have to worry about covariances among them.

The second advantage is that V_G provides a means for predicting future generations when there is selection. The reason for this lies in the nature of the Mendelian mechanism. What is transmitted by a parent to his progeny is not an intact genotype, but a random sample of genes. Therefore the best prediction is a linear estimate of the average value of the contribution of the individual genes, the variance of which is measured by V_G. I might add, parenthetically, that the situation is not really as tidy as the above sentences may imply. It is difficult to identify the contribution of epistasis, particularly when one considers the complications of linkage between genes on the same chromosome. In many cases the breeder gets satisfactory predictions by simply ignoring epistasis, a fact which may be caused by one of several conditions. It may be that the gene loci act approximately additively on the chosen measurement; it may be that various gene interactions are in opposite directions and therefore cancel each other; or, as is often the case, it may be that the numbers are small, so that even a large discrepancy is regarded as a satisfactory fit, simply because there is not enough statistical power to make a finer discrimination.

The other interactions, between genotype and environment and between environmental components, are not automatically taken care of and have to be considered specifically. It is important conceptually to distinguish, as Jensen does, between *interaction* of heredity and environment (as when a good genotype gets more of a boost from a good environment than a poor genotype does) and *covariation* of heredity and environment (when a good genotype tends to be located where the environment is good). The components due to errors of measurement can usually be ignored if the correlations are corrected for attenuation.

It is important to emphasize that heritability can be defined in two ways:

$$\text{heritability in the narrow sense: } h^2 = \frac{V_G}{V_P}$$

$$\text{and heritability in the broad sense: } \quad H^2 = \frac{V_H}{V_P} .$$

For mnemonic convenience, I shall use h^2 to indicate the narrow definition, which is always quantitatively smaller, and H^2 for the broader (and larger) definition.

Finally, I designate the environmental fraction of the variance as

$$E^2 = \frac{V_E}{V_P}$$

The plant or animal breeder is interested in h^2 because it helps him to predict the expected gains from selection and to estimate the effectiveness of a breeding program. The psychologist is likely to be more interested in H^2 (and E^2) because it partitions the variance into genotypic and environmental components and may thereby afford some insight into each cause. E^2 gives some guidance as to the amount of influence that environmental differences are having and, among these, specific factors may be identified. (See Jensen's discussion of heritability, pp. 33-43.)

Animal and plant experiments have shown that heritability estimates have reasonably good predictive accuracy when the numbers and statistical design are such as to provide a powerful test. However, the prediction is valid for only this particular situation, because heritability is a function of gene frequencies, the mating system, and existing environmental influences. As such, it will change when these change. This means, among other things, that the initial heritability will not be a good guide for long-time selection programs. The program, if successful, will change gene frequencies, and therefore the heritability may change. Furthermore, the environment may change, and this can also change the heritability.

How Valid Are Heritability Measurements of Intelligence?

The animal or plant geneticist gets rid of some of the most troublesome covariances by experimentally designed randomization. This is clearly out of the question in dealing with man. Correlations between relatives are caused by both genetic and environmental similarities. Jensen's general formula for H^2—a great improvement over those of earlier authors, in my opinion—is

$$H^2 = \frac{r_1 - r_2}{\rho_1 - \rho_2},$$

where r_1 and ρ_1 are the observed and theoretical correlations for one degree of relationship, and r_2 and ρ_2 are the corresponding quantities for another degree. If we ignore interaction, the correlation will be

$$r_i = \rho_i H^2 + \rho_i' E^2 ,$$

where ρ_i' is the environmental correlation for the ith degree of relationship. As a simple illustration of what happens when the ρ's are not independent, suppose that $\rho_i' = k\rho_i$, where k is a constant. Then

$$\frac{r_1 - r_2}{\rho_1 - \rho_2} = H^2 + kE^2 ,$$

which, instead of measuring H^2, includes an unknown fraction of the environmental variance, thus limiting the usefulness of such a formula. It is likely, for example, that cousins have environmental similarities that are less than those for siblings but more than for children in unrelated families.

If the formula is used to compare monozygous and dizygous twins, there are the often-discussed uncertainties as to whether intra-family environmental differences are the same for the two kinds of twins, as the formula assumes. Identical twins may more often share experiences than dizygotic twins. But, as many authors have pointed out, environmental similarity for monozygotic twins is not necessarily greater than for dizygotic, especially when intra-uterine environment is considered. For example, the likelihood of an unequal blood supply is greater in monozygous twins. Finally, the value of ρ is uncertain. For monozygotic twins it is clearly 1. But for dizygotic twins it is not known exactly. It is roughly 1/2—but decreased by dominance and epistasis and increased by assortative mating, both by unknown amounts.

Most of these difficulties could be removed if individuals of close genetic relationship could be randomized with respect to environments and if unrelated children could be reared in identical environments. These conditions are partially met by studies of twins and siblings reared in different households and by unrelated children reared in the same. As Jensen mentions, the Burt study appears closest to the ideal of placing the separated identical twins into random environments at an early age. There is some reassurance to the skeptic (such as I have been) in that H^2, as estimated by the correlation of one-egg twins reared apart, and E^2, estimated by the correlation between unrelated children reared together, add up to approximately 1—as they should if everything is simple ($.75 + .24 = .99$ in Jensen's Table 2; $.86 + .25 = 1.11$ in Burt's individual measurements). Other crosschecks are also in reasonable agreement, but the numbers are small.

If we take the results from many investigations at face value, there is a great deal of consistency, as Jensen points out, and H^2 averages about .8. Furthermore the dominance and epistatic components appear to be small. That the heritability is large is a justifiable conclusion at this stage, although the precise value

must remain in doubt for the various reasons given. We shall have to be content with measures that are only approximate, pending more evidence on the reality of the assumptions. I agree with Jensen in deploring an uncritical assumption that only environmental factors are important and that genetic differences are negligible.

I admire the diligence of Newman, Shields, and Burt in finding twins and siblings reared apart. Particularly useful, as Jensen has emphasized, would be data on half-siblings reared in different homes. Any excess of similarity of those with a common mother over those with a common father would provide a measure of prenatal and neonatal maternal influences. Though difficult to obtain, it would also be necessary to have data on the correlations between the non-common parents. There will always be some doubt, however, as to whether children from broken homes—separated twins and siblings—and from foster home environments can be regarded as representative of the normal population.

How Important Is It to Measure Heritabilities?

I share Dr. Jensen's interest in trying to determine H^2 and h^2, especially if this information can be extended to other populations. Particularly interesting is his suggestion that heritability be used as one criterion of the culture-fairness of a test. At the same time there are many social decisions that do not depend on a precise knowledge of the heritability of intelligence.

If society decides to improve IQ by eugenic means, h^2 will be useful in providing estimates of the expected gain. I believe that we already know enough to predict that a selection program to increase IQ or *g* would work. There would be an increase, but the amount would be uncertain, because of uncertainties both in the true value of h^2 and in the asumptions underlying its use as a predictor. However, society is clearly not ready to embark on a eugenic program of sufficient scope to make very much difference, even if heritability were equal to 1.

What guidance does H^2 (or E^2) offer for predicting the effect of improvement in the environment? E^2 tells us how much the variance would be reduced if the environment were held constant. It does not directly tell us how much improvement in IQ to expect from a given change in the environment. In particular, it offers no guidance as to the consequences of a new kind of environmental influence. For example, conventional heritability measures for height show a value of nearly 1. Yet, because of unidentified environmental influences, the mean

height in the United States and in Japan has risen by a spectacular amount. Another kind of illustration is provided by the discovery of a cure for a hereditary disease. In such cases, any information on prior heritability may become irrelevant. Furthermore, heritability predictions are less dependable at the tails of the distribution.

A high heritability of intelligence does not necessarily mean that a program of compensatory education is destined to fail, although it may necessitate a larger or more innovative environmental change than if E^2 were larger. Measuring heritability may be less important than getting empirical data on the effects of specific environmental factors. If environment acts as a threshold, as Dr. Jensen suggests, then it would be especially important to identify environmental influences that may be of great influence at the end of the scale, but less so within the normal range.

I am not acquainted with the compensatory education studies Jensen reviews nor am I professionally competent in that area, but my view from the outside is that we should not give up too easily. Perhaps the programs are too little and too late. There are surely a variety of ways of intensifying and improving the effectiveness of education. Also a small change in IQ, especially if accompanied by increased motivation and achievement, may be of great social benefit. Jensen expresses much the same idea:

> Thus it seems likely that if compensatory education programs are to have a beneficial effect on achievement, it will be through this influence on motivation, values, and other environmentally conditioned habits that play an important part in scholastic achievement, rather than through any marked direct influence on intelligence *per se*. The proper evaluation of such programs should therefore be sought in their effects on actual scholastic performance rather than in how much they raise the child's IQ. (p. 59)

Group Differences, Especially Racial Differences

Heritability studies have been confined almost exclusively to white populations and largely to normal environments. How relevant are they to other populations and environments? We are currently especially concerned about culturally disadvantaged groups and racial minorities. Strictly, as Jensen mentions, there is no carryover from within-population studies to between-population conclusions.

I agree that it is foolish to deny the possibility of significant genetic differences between races. Since races are characterized by different gene frequencies, there

is no reason to think that genes for behavioral traits are different in this regard. But this is not to say that the magnitude and direction of genetic racial differences are predictable.

It is clear, I think, that a high heritability of intelligence in the white population would not, even if there were similar evidence in the black population, tell us that the differences between the groups are genetic. No matter how high the heritability (unless it is 1), there is no assurance that a sufficiently great environmental difference does not account for the difference in the two means, especially when one considers that the environmental factors may differ qualitatively in the two groups. So, I think, evidence regarding the importance of heredity in determining group mean differences must come from other kinds of studies.

The failure, thus far, to find identifiable variables that, when matched, will equalize the IQ scores does not prove that the mean difference is hereditary. It can be argued that being white or being black in our society changes one or more aspects of the environment so importantly as to account for the difference. For example, the argument that American Indians score higher than Negroes in IQ tests—despite being lower on certain socio-economic scales—can and will be dismissed on the same grounds: some environmental variable associated with being black is not included in the environmental rating. Behavioral scientists can be expected to disagree, and they do, as to when enough identifiable environmental factors have been shown to be insufficient that the remaining differences should be regarded as mainly genetic. To me, the evidence on this question is not at all conclusive.

Final Comments

One of the goals of a democratic society, I believe, ought to be to provide each individual with the maximum opportunity to satisfy his needs and desires and to contribute to society's betterment through his special abilities. A population with a variety of phenotypes (and genotypes) ought to be more rewarding, and certainly more interesting, than one that is homogeneous. I do not go to the extreme of saying that all variation should be encouraged—I shall be quite happy if the gene for muscular dystrophy becomes extinct—but in general I believe that diversity is good, not bad. In any case, we have it.

Society should recognize that there is a great deal of genetic variability for all kinds of traits, including intelligence and special talents. I think that J. B. S.

Haldane once said that liberty is the practical recognition of human variability. We should also realize that to whatever extent society is successful in its goals of providing equality of opportunity, to that extent the heritability will increase. In view of this fact, I fully agree with Jensen that, rather than uniformity, the goal should be diversity of educational opportunity with maximum individual opportunity for finding the right niche, and that the reality of individual differences need not and should not mean rewards for some and frustration for others.