Excerpts from . . .

The Nature of Human Variation

by Richard Lewontin

If IQ had been under natural selection of any intensity at all during the course of human history, all that additive variance should be gone.

The problem of assessing genetical variation in human populations is the same as for almost any other species. Our predictive theories—those which tell how genetical variation has accumulated, what is responsible for its present status, and what the future of genetical variation in a population will be—are framed entirely in terms of the frequencies of allelic substitutions at various loci [*alleles* are alternate forms of the same gene—for example, that which determines eye color]. To describe the genetical variation in populations in terms of those substitutions is a very difficult and, in fact, at the moment, impossible job . . .

In an attempt to measure allelic substitutions that have small effects on the phenotype [the appearance of an organism resulting from the interaction of the genotype and the environment] a number of people in the last few years have taken to trying to characterize the enzymes and proteins that are the direct products of gene action and to characterize them by their physical-chemical properties, which, although they may not have any marked effect on the phenotype of the organisms, are sufficiently marked in their effect under laboratory test conditions so that you can detect differences in different individuals. That is, you try to find a system which allows you to detect the differences in physical-chemical behavior of proteins despite the fact that the organism itself cannot detect them. That's what molecular biology does for us.

The result of this kind of study in a variety of organisms—mice, *Drosophila*, and so on—is to arrive at an estimate of what the typical genome [a single set of chromosomes] of a typical individual in a typical population looks like.

Excluding man for the moment, and thinking only of *Drosophila* or mice, which are the two best documented cases, we find that, as a minimum estimate, something like 40 percent of all the structural genes in the genome of a sexually reproducing species have some significant genetic segregation in any population; and that the average individual is himself, or herself, about 15 percent heterozygous [having dissimilar pairs of genes at some loci, only one of which can be transmitted to progeny]. So that each one of us, if we have, say, 10,000 genes, is a heterozygote at 1,200 to 1,500 of those loci . . .

How heterozygous is man, if we assume that human blood groups are a random sample of the human structural genome? . . .

What we can do is to estimate the proportion of all genes that are polymorphic (that have more than one allele in them) cumulatively . . .

The estimate of the average heterozygosity per individual appears to be leveling out at about 16 percent. And the average frequency of polymorphic genes per individual is leveling out at about 37 percent. That figure is remarkably like the figure from mice and from *Drosophila*. That is to say, between 30 and 40 percent of all genes are polymorphic, and something like 12 or 15 percent of every locus in every individual is heterozygous.

This, then, is the kind of information that gives you a solid picture of the amount of available variation on which natural selection and human evolution can operate . . .

I think we should stop talking about vast numbers of genes controlling traits, all genes being of equal effect. For behavioral characteristics the real facts of life may turn out to be that four, five, or six loci will turn out to contribute 80 or 90 percent of the variance for a behavior trait, and the rest of the genome contributes the 10 or 20 percent. I propose that if anybody is really interested in doing the genetics of behavior in any organism that is manipulable by genetic tricks, the first thing that must be done is to establish the dose-response curve for genome against variance . . .

R. A. Fisher, the British statistician, enunciated what he called the fundamental theorem of natural selection, in which he said, in effect, that during the course of selection, either natural or artificial, so-called additive variance is used up. Eventually, equilibrium gene frequencies are arrived at in which all of the additive variance is gone and the only variance left is the interaction variance and the environmental variance.

Now when we come across a character that has vast quantities of additive genetic variance, our first suspicion is that this character has never been under natural selection; or at least if it has, it has been under natural selection only very weakly or for a very short time. As a matter of fact, a character whose additive genetic variance is on the order of 50 or 60 or 70 percent of all the variance is a very unusual quantitative character. If IQ has indeed got 50 or 60 or 70 percent additive genetic variance, then I wish to call into question very severely our notions about the adaptive significance of this variation of intelligence. Because Fisher's fundamental theorem, which is only approximate but still qualitatively true, tells us that if IQ or the performances that we measure by IQ tests had been under natural selection of any intensity at all during the course of human history, all that additive variance should be gone. I leave it to you then to ponder on the meaning of the very large amount of additive variance for IQ . . .

The important genetic discovery of Thomas Hunt Morgan and Calvin Bridges, something which population genetics in general does not take into account, is that genes are *not* floating around as individual particles. If you make a theory of population genetics that includes the fact that genes are organized on chromosomes, then you get the curious result that no single locus can be shown to have any important natural selection, that the chromosome will evolve as a kind of block by the accumulation of very small effects, and that natural selection may be operating to stabilize the frequency of the genes within a population. But one will not find that out by examining the effect of a simple locus substitution at a single locus. One has to take into account the entire chromosomal array.