prove average performance in that population. If h^2 is high but environments sampled in that population are largely unfavorable, then (again) simple environmental improvement will probably change the mean phenotypic level. If h^2 is high and the environments sampled are largely favorable, then novel environmental manipulations are probably required to change phenotypes, and eugenic programs may be advocated.

The most common misunderstanding of the concept "heritability" relates to the myth of fixed intelligence: if h^2 is high, this reasoning goes, then intelligence is genetically fixed and unchangeable at the phenotypic level. This misconception ignores the fact that h^2 is a population statistic, bound to a given set of environmental conditions at a given point in time. Neither intelligence nor h^2 estimates are fixed.

It is absurd to deny that the frequencies of genes for behavior may vary between populations. For individual differences within populations, and for social-class differences, a genetic hypothesis is almost a necessity to explain some of the variance in IQ, especially among adults in contemporary white populations living in average or better environments. But what Jensen, Shuey, and Eysenck (and others) propose is that genetic racial differences are necessary to account for the current phenotypic differences in mean IQ between populations. That may be so, but it would be extremely difficult, given current methodological limitations, to gather evidence that would dislodge an environmental hypothesis to account for the same data. And to assert, despite the absence of evidence, and in the present social climate, that a particular race is genetically disfavored in intelligence is to scream "FIRE! . . . I think" in a crowded theater. Given that so little is known, further scientific study seems far more justifiable than public speculations.

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References and Notes

1. For a review of studies, see L. Erlenmeyer-Kimling and L. F. Jarvik, *Science* **142**, 1477 (1963). Heritability is the ratio of genetic variance to total phenotypic variance. For human studies, heritability is used in its broad sense of total genetic variance/total phenotypic variance.
2. The *Harvard Educational Review* compilation includes Jensen's paper, "How much can we boost IQ and scholastic achievement?," comments on it by J. S. Kagan, J. McV. Hunt, J. F. Crow, C. Bereiter, D. Elkind, L. J. Cronbach, and W. F. Brazziel, and a rejoinder by Jensen. See also A. R. Jensen, in J. Hellmuth, *Disadvantaged Child*, vol. 3 (Special Child Publ., Seattle, Wash., 1970).
3. P. L. Nichols, thesis, University of Minnesota (1970). Nichols reports that in two large samples of black and white children, seven-year WISC IQ scores showed the same means and distributions for the two racial groups, once social-class variables were equated. These results are unlike those of several other studies, which found that matching socioeconomic status did not create equal means in the two racial groups [A. Shuey (*5*); A. B. Wilson, *Racial Isolation in the Public Schools*, vol. 2 (Government Printing Office, Washington, D.C., 1967)]. In Nichols's samples, prenatal and postnatal medical care was equally available to blacks and whites, which may have contributed to the relatively high IQ scores of the blacks in these samples.
4. By interaction, Eysenck means simply $P = G + E$, or "heredity and environment acting together to produce the observed phenotype" (p. 111). He does not mean what most geneticists and behavior geneticists mean by interaction; that is, the *differential* phenotypic effects produced by various combinations of genotypes and environments, as in the interaction term of analysis-of-variance statistics. Few thinking people are not interactionists in Eysenck's sense of the term, because that's the only way to get the organism and the environment into the same equation to account for variance in any phenotypic trait. How much of the phenotypic variance is accounted for by each of the terms in the equation is the real issue.
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7. *New York Times*, 8 Oct. 1971, p. 41.
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9. S. Scarr-Salapatek, *Science*, in press.
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12. J. Thoday, *J. Biosocial Science* **1**, suppl. 3, 4 (1969).
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14. T. E. Reed, *Science* **165**, 762 (1969); *Am. J. Hum. Genet.* **21**, 1 (1969); C. MacLean and P. L. Workman, paper at a meeting of the American Society of Human Genetics (1970, Indianapolis).
15. E. W. Reed and S. C. Reed, *Mental Retardation: A Family Study* (Saunders, Philadelphia, 1965); *Social Biol.* **18**, suppl., 42 (1971).
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17. J. C. DeFries, paper for the C.O.B.R.E. Research Workshop on Genetic Endowment and Environment in the Determination of Behavior (3–8 Oct. 1971, Rye, N.Y.).
18. R. Heber, *Rehabilitation of Families at Risk for Mental Retardation* (Regional Rehabilitation Center, Univ. of Wisconsin, 1969). S. P. Strickland, *Am. Ed.* **7**, 3 (1971).
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20. J. Rynders, personal communication, November 1971.
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22. I thank Philip Salapatek, Richard Weinberg, I. I. Gottesman, and Leonard I. Heston for their critical reading of this paper. They are not in any way responsible for its content, however.

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