To understand their argument we must consider it in detail.

Feldman and Lewontin begin by terming the analysis of variance a local perturbation analysis, as indeed it is under certain assumptions (and geometry and the natural sciences likewise). Then they introduce broad heritability, which can be determined only from the study of identical twins reared apart in random environments, provided that gene-environment covariance and differential effects of prenatal environment are negligible. Since in practice broad heritability is not estimable, flogging it seems unnecessary. They next conclude that "statistical inference about the heritability of traits that are phenotypically plastic is invalid." What does this mean when heritability is the complement of plasticity? They cite approvingly two comments by Moran on genotype-environment covariance, both of which were subsequently corrected (*1*). A valid treatment of gene-environment covariance was introduced more than half a century ago by Wright and later refined (*2*).

I take greatest exception to the section of the article in which the authors advocate a purely empirical method of calculating the risk of genetic disease, thereby attacking a promising development in genetic counseling—the use of genetic models. Most genetic disease is of complex etiology. Until recently, recurrence of such conditions could be estimated only by empirical calculation of risks. This method depends on no detailed genetic analysis, considers only the child immediately following the proband, and pools families of different composition, ignoring normal siblings, more remote relatives, sex, age, quantitative information, and etiological heterogeneity. The dictionary definition of "empiric" is "one who deviates from the rules of science or accepted practice; one who relies upon practical experience alone, disregarding all theoretical and philosophic considerations; hence a quack, a charlatan"—the very apotheosis of local perturbation.

Hemophilia illustrates the way in which the empirical calculation of risks can be first a step forward, then backward. Almost 2000 years ago the Talmud used empirical risk calculation: later-born sons of a woman who had lost two boys due to bleeding were not to be circumcized, nor were the sons of her sisters; but paternal half-sibs were treated as normal individuals. While remarkably accurate for its day, this is less predictive than the determination of genetic risks based on detection of carrier women, which does not require the signal of two prior deaths. Faults of empirical risk calculation are rectified in complex segregation analysis, which gives specific and precise estimates of genetic risks (*3*). One of the required parameters is heritability. Feldman and Lewontin's statement that "confusing risks can be calculated separately for various ages, socioeconomic classes, cultural patterns, and the like," does not convey to the reader that affection of family members is the central factor in genetic counseling. The counselor who follows the advice of Feldman and Lewontin and prefers the empirical calculation of risks to the more complete specification provided by genetic analysis is giving his patient second-rate service.

After this fallacy, so damaging to medical genetics, discussion of gene-environment interaction and intergroup differences is anticlimactic. Interaction diminishes family resemblance and need not concern those whose task is to explain resemblance, not dissimilarity. The heritability of group differences cannot be predicted from intragroup heritability, but no geneticist supposes that it could.

Feldman and Lewontin have generalized their attack on a particular psychologist to include a significant part of science. They are concerned about possible abuse of genetics by nongeneticists, forgetting how often dire prophecies are dispelled by investigation (*4*). The evil they fear thrives in the obscurity they cultivate. Their clumsy harrying of biometrical genetics is entirely unbecoming and does only senseless harm to the cause of science and humanity (*5*).

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In contrast to Feldman and Lewontin, we welcome the recent swing of psychology from the environmental excesses of the past to a more balanced view of the biological bases of behavior (*1*). Behavioral genetics is but one part of a zeitgeist that is bridging the gap between the study of behavior and the study of biology, a movement that includes both sociobiology and psychobiology (*2*).

Contrary to the impression that Feldman and Lewontin create, it is not difficult to find examples of the usefulness of

genetic analyses in the study of complexly determined behavior. Psychopathology is an obvious example. Before the mid-1960's, psychologists continued to look for environmental causes of schizophrenia and other psychoses. In 1966, a single behavioral genetic study turned the field around. Heston (*3*) studied the adopted offspring of 47 schizophrenic women and compared them to a matched control group of adopted children whose biological parents had no known psychopathology. Of the adopted children with a schizophrenic heritage, five were diagnosed as schizophrenic; none of the control children was schizophrenic. Regardless of whether one likes the concept of heritability, this behavior is clearly influenced by genetic factors. That is a fundamental piece of knowledge. Behavioral genetic studies have also led to important discoveries concerning the manic-depressive psychoses (*4*).

In addition to asserting that heritability does not advance either cures or counseling, Feldman and Lewontin reiterate the common knowledge that heritability estimates are limited to the population sampled and that genotype-environment interaction and correlation may be important. These points are misinterpreted by Feldman and Lewontin to mean that quantitative genetic analyses are, therefore, of no use. The conclusion does not follow (*5*). The very purpose of quantitative genetic studies is to *describe* genetic variability *in a specific population* and to ascribe that variability to environmental differences and genetic differences in that population (*6*). The question of generalizing to other samples and other times can only be answered empirically (the evidence with respect to cognitive abilities suggests considerable generalizability). Feldman and Lewontin seem to be more concerned with the question of *what could be* rather than *what is*. That is a legitimate concern, of course, but it should not be the basis for a critique of quantitative genetic analysis.

One aspect of their article that was most disturbing to us was its polemical nature. Feldman and Lewontin imply that the motivation of geneticists is eugenic and that they are the dupes of politicians who "use genetic misinformation to rationalize a politically determined policy." Rather than attempting to discredit research in behavioral genetics, the authors could better serve science by encouraging the search for specific genotype-environment interactions or genotype-environment correlations that they assume to be so important.

In addition to these general issues, it is necessary to address one technical point

concerning Feldman and Lewontin's discussion of the relationship between within-group heritability (h^2_W) and between-group heritability (h^2_B), which they also use to symbolize heritability in the broad sense). Although not cited by Feldman and Lewontin, h^2_B was first expressed as a function of h^2_W (their equation 3) by DeFries (*7*). DeFries made two points: (i) There *is* a mathematical relationship between h^2_B and h^2_W, contrary to what Lewontin (*8*) had previously asserted; and (ii) nevertheless, high h^2_W by no means implies high h^2_B. Feldman and Lewontin agree with the second point, but they state that the first point is "entirely spurious" because equation 3 does not describe a "causal relationship." Surely they cannot mean that all noncausal mathematical relationships are entirely spurious (*9*).

Although we disagree with many of the assertions contained in their article, we share Feldman and Lewontin's interest in reliable data on adoptions. We believe that well-designed adoption studies can provide the best information about the relative importance of heredity as a cause of individual differences in human behavior, as well as the first solid information concerning the importance of genotype-environment correlations and interactions (*5*).

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References and Notes

1. See, for example, the 1975 presidential address of the American Psychological Association [D. T. Campbell, *Am. Psychol.* **30**, 12 (Dec. 1975)].
2. E. O. Wilson's highly touted tome, *Sociobiology: The New Synthesis* (Harvard Univ. Press, Cambridge, Mass., 1975), is one example of this trend.
3. L. L. Heston, *Br. J. Psychiat.* **112**, 819 (1966).
4. J. Angst and C. Perris, *Int. J. Ment. Health* **1**, 145 (1972); J. Mendlewicz and J. L. Fleiss, *Biol. Psychiatr.* **9**, 3 (1975).
5. For a discussion of the possible effects of genotype-environment interaction and correlation on quantitative genetic analyses and the use of adoption data to assess these effects, see R. Plomin, J. C. DeFries, J. C. Loehlin, *Psychol. Bull.*, in press.
6. J. L. Lush, in *Thirty-Third Annual Proceedings of the American Society of Animal Production* (1940).
7. J. C. DeFries, in *Genetics, Environment, and Behavior*, L. Ehrman, G. S. Omenn, F. Caspari, Eds. (Academic Press, New York, 1972), pp. 5–16.
8. R. C. Lewontin, *Bull. At. Sci.* **26**, 2 (March 1970).
9. Causality of the intraclass genetic correlation (r) and h^2_B is irrelevant to the existence of a relationship between h^2_B and h^2_W. Nonetheless, the assertion that r "is dependent on h^2_B and not vice versa" is wrong. J. C. Loehlin, G. Lindzey, and J. N. Spuhler [in *Race Differences in Intelligence* (Freeman, San Francisco, 1975), pp. 290–291] have rephrased DeFries' argument in terms of four quantities to be estimated: (A) genetic variance between groups, (B) genetic variance within groups, (C) environmental variance between groups, and (D) environmental variance within groups. It can be stated that r = A/(A+B) and h^2_B = A/(A+C). Thus, the two parameters are essentially coordinate in status.

Feldman and Lewontin conclude their consideration of eugenics with the statement, "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." This statement contains the implication that scientists might properly withhold scientific views that are judged to have undesirable social consequences. In the past, this opinion would have had interesting consequences. Recall, for example, the social impact of Darwin's theory of natural selection. The "social Darwinists," led by the most influential sociologist of the time, Herbert Spencer, reinterpreted the concept of fitness to imply that the poor were unfit, the rich fit. The theory of natural selection, thus popularized and (mis)interpreted, provided a rationale for exploitative, laissez-faire capitalism. Darwin thus gave legitimacy, presumably unwilling, to a social theory that we, at least, would consider "pernicious." Ought he to have desisted for that reason?

Eugenics, of course, is not in itself a purely scientific issue; however, its scientific component is not negligible, as attested by the paragraph in Feldman and Lewontin's article that precedes the above quotation. But other geneticists, notably H. J. Muller and his followers, might assess the scientific issues somewhat differently. Scientific advocates of eugenics have the same right, and even obligation, to express their views as do Feldman and Lewontin.

Maintenance of open discussion of scientific issues impinging on sensitive social issues is doubly important because, whereas scientific conclusions are at least in principle demonstrable, the judgment of which social consequences are desirable and which "pernicious" is intrinsically subjective. No person has a right to legislate such social attitudes for others, much less for a whole scientific community.

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Jensen first claims that our references to his work are inaccurate or misleading. But he does not offer a single example. It is therefore difficult to take this blanket condemnation seriously. The references he offers, far from answering our objections, more usually repeat the errors we discuss. He devotes most of his letter to a theoretical point on which he has not previously written in any detail. Population geneticists will quickly see that