

LETTERS

The Science Court Experiment

Earl Callen's letter (10 Sept., p. 951) expresses his fears about the potential for the science court to become an authoritarian instrument, stifling the ability of scientists to speak out on public policy matters. His views may be shared by many scientists. It is important that wide public debate be held on the science court concept so that this and other possible arguments against the court can be fully aired. The public session on the science court at the April meeting of the American Physical Society was a start in this direction. The Colloquium on the Science Court held from 19 to 21 September 1976 at Leesburg, Virginia, was another step.

As a member of the task force that has been developing the science court idea, I have, as Callen says, taken the position that the court should be regarded more as a set of procedures to be used as needed than as a continuing institution with a life of its own. It is my impression that this view is not uniquely mine among the members of the task force and, indeed, it is quite consistent with the discussion of the science court presented by the task force in the article "The Science Court experiment: An interim report" (20 Aug., p. 653).

It is incorrect to suggest, as Callen does, that my views are the basis of a different plan for a science court that is being considered by the Consumer Product Safety Commission. First of all, the only plan that I am aware of is the one being developed by the task force. Second, the Commission has not formally discussed the science court concept, nor has it considered any specific plan. I believe the science court has merit and that it could, if properly developed, be useful to the Consumer Product Safety Commission as well as other government regulatory bodies. However, I am only one of five Commissioners, and the question of use of a science court has yet to be addressed by the full Commission.

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Answers to the points made in Callen's letter are to be found in the Interim Report published in the 20 August issue of *Science*.

The public discussion that Callen calls for we hope has been stimulated by the Colloquium on the Science Court held at Leesburg, Virginia.

Callen talks about the science court issuing statements of "scientific Truth." The first page of the Interim Report says, "We have no illusions that this procedure will arrive at the truth, which is elusive and tends to change from year to year."

Callen asks "which facts" will be dealt with by the court. The procedure for selecting the facts to be dealt with is specifically discussed in the Interim Report, and a procedure has been suggested in which the case managers for either side propose the factual statements which will be considered by the science court. Thus, both sides will have full opportunity to bring forth those relevant facts they consider important.

Callen makes the broad statement that "In social policy questions it is impossible to separate facts from values." This is, of course, a question which has been debated by philosophers for centuries. We don't propose to enter into that debate, but simply to avoid issues where the distinction cannot be made.

Finally, Callen announces that "The science court will stifle public debate." The Interim Report points out that the process will be conducted entirely in public, and the only authority that will attach to its results will arise out of the credibility the public assigns to its procedures.

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Wald and the Cambridge City Council

Barbara Culliton's article in the 23 July issue of *Science* (News and Comment, p. 300) on the Cambridge City Council's involvement with recombinant DNA contains a small factual error in saying, "Wald went to see Mayor Vellucci, whom he persuaded that the potential threat of P3 recombinant experiments to the public health is a very real one." Mayor Vellucci needed no persuading. He had several days earlier put this matter on the docket of the next City Council meeting, on the strength of an article on genetic recombination in the Boston *Phoenix* of 7 June.

This is not an apology; but I do not want to be given unjustified credit for an event I did not bring about.

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Heritability of IQ

Feldman and Lewontin (19 Dec. 1975, p. 1163) make numerous references to my writings on the heritability of intelligence, often in ways that are so incomplete as to be inaccurate or misleading. Serious readers may find this out for themselves, since I have written in some detail on the various criticisms of the heritability concept as related to mental measurements mentioned by Feldman and Lewontin: the meaning of heritability in the behavioral sciences and the question of genotype-environment interaction (1), genotype-environment covariance (2), the heritability of differences between groups (1, 3), and the broader educational and social implications of the substantial heritability of IQ (4).

On one fundamental theoretical point on which I have not previously written in any detail, however, Feldman and Lewontin draw an unwarranted conclusion. They state that "... as selection progresses, the additive genetic variance is 'used up' so that the h^2_N [the narrow heritability or proportion of additive genetic variance] 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 [the broad heritability or the proportion of the total phenotypic variance due to all genetic factors, additive and nonadditive], which does not, how much selection has gone on." They then argue that the difference between the empirical estimates of 0.75 and 0.40 for the h^2_B and h^2_N , respectively, of IQ, forces the conclusions 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." These are weak inferences in the absence of knowledge about selection intensities, as Feldman and Lewontin rightly point out.

The one reasonable inference that can be drawn from the present evidence is that the intelligence measured by IQ is a fitness character—the genes involved in IQ variance have undergone selection in the course of human evolution.

But theoretically we are not justified in concluding, as do Feldman and Lewontin, that whatever it is that IQ measures

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has not been under intense selection for very long. As Feldman and Lewontin say, the presence of significant dominance variance generally indicates past selection. (Although dominance variance could conceivably come about in the absence of past selection as a result of dominant and recessive mutants, this seems a less likely explanation.) But since the proportion of dominance variance to total genetic variance depends upon the relative frequency of the dominant genes, and since we do not know the frequencies of additive, dominant, and recessive genes involved in IQ, we cannot draw any conclusions about the duration or intensity of selection. However, the presence of dominance variance, inferable from the difference between h^2_B and h^2_N , does indicate the effects of dominant genes and most probably of past selection. Further evidence of dominant genes for intelligence is the well-established finding of substantial inbreeding depression for IQ, indicating directional dominance, that is, the dominant genes enhance IQ (5, 6).

Even if all the additive genes had been "used up" by selection in the course of evolution, and even if there were complete dominance at all gene loci, the additive genetic variance could still be considerably greater than zero. More precisely, with *complete dominance* at all loci, the additive genetic variance will not be less than the dominance variance until the frequency of the dominant genes is more than twice the frequency of the recessive genes. As Falconer points out, "[t]he concept of additive variance does not carry with it the assumption of additive gene action; and the existence of additive variance is not an indication that any of the genes act additively (i.e., show neither dominance nor epistasis)" (7, p. 138).

One could even argue that a narrow heritability of 0.40 (which, according to the estimate cited by Feldman and Lewontin, means additive variance would constitute only 53 percent of the total genetic variance) indicates a comparatively low proportion of additive genetic variance. Consider the narrow heritabilities of characteristics that have been subjected to selection in domestic animals: milk yield of cows, 0.30; lardiness of pigs, 0.55; length of wool in sheep, 0.55; egg weight of chickens, 0.6 (7, pp. 167-188). In light of these figures for highly selected traits, the estimates of narrow heritability of IQ (most are in the range of 0.4 to 0.6) would seem to suggest considerable selection for IQ. Also, interestingly, inbreeding depression of IQ (that is, the decrease in mean

IQ per 10 percent increase of the coefficient of inbreeding as a percentage of the noninbred mean) is at least as large as comparable figures for highly selected characteristics of domestic animals (5, p. 295; 7, p. 249).

The additive genetic variance will approach zero as a result of natural selection only if the trait is perfectly correlated with fitness in the Darwinian sense and if there is zero mutation rate. The additive variance will attain some value greater than zero for traits which are imperfectly correlated with fitness. We would not expect extremely high correlations of human intelligence with fitness. It is even likely that selection for intelligence has probably been somewhat relaxed with the advance of civilization. In human cultures the fitness of persons of quite low intelligence may be enhanced by the contributions of relatively few individuals at the high end of the ability scale, for example, through inventions, advances in agriculture, hygiene, and so forth, which benefit everyone in the society, more or less, regardless of their level of intelligence.

Brain size is known to have more than doubled in size in the course of human evolution, from *Australopithecus* to present-day man, in whom there is a reliable correlation of about 0.30 between brain size and IQ (8).

Finally, the evidence for the substantial heritability of IQ does not depend upon complex analyses in quantitative genetics. The fact that genetic factors are strongly involved in individual differences in IQ is firmly established by numerous studies of adopted children, whose IQ's are much less correlated with the IQ's of their adoptive parents (and with assessments of their adoptive environments) than with assessments of their biological parents, with whom they have had no postnatal relationship (9), and by studies showing that identical twins reared apart are more similar in IQ than fraternal twins reared together (10).

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References and Notes

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The article by Feldman and Lewontin fairly teems with conspicuous inaccuracies. Here is a modest sampling.

1) They commence with a serious misrepresentation of Jensen's views. What Jensen actually concludes from the finding of a high heritability (h^2) of IQ is that *existing* differences in cognitive development are not generated to a significant degree by *existing* differences in the social environment, including those of income or cultural milieu. From this he sensibly infers that educational programs *of a certain kind*—namely, simple "enrichment" schedules aimed merely at delivering the normal "middle-class" cultural milieu to disadvantaged children—are unlikely to reduce cognitive differences by very much. This carefully *limited* inference is in no way fairly communicated by Feldman and Lewontin's summary of it: "... 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." This astounding paraphrasing conveniently obscures the consequential distinction between environmental interventions *of a certain specific kind*, as contrasted with those *of every conceivable sort*. With this latter meaning, their summarizing statement becomes, to be sure, an easy, even unworthy, beast to slay; but it becomes at the same time an imaginary creature of their own making.

As it happens, Jensen's conclusion was quite a fair prediction of the empirical results independently obtained from massive "enrichment" efforts of this very type. Therefore, the implication of high IQ heritability must now be taken into account in planning future attempts at improving this trait. Whereas this implication is commonly misinterpreted to mean that all such efforts might as well be abandoned as inevitably futile, the true implication is that eventual success in raising IQ will almost certainly come about only through environmental innovations that currently are rare or non-existent in the populations where a high h^2 has been observed. Future attempts should therefore focus on novel types of intervention. The work of Heber *et al.* (1) is a notable instance of such radical environmental changes, and they have in

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fact reported impressive success in raising IQ.

2) The specific models of genetic action discussed by Feldman and Lewontin in their figures 1 and 2 are bizarre, to say the least. First, by positing genotypes that differ greatly from each other in their phenotypic response to the same environment, they merely concede Jensen's basic contention that genetic factors are important in accounting for individual differences in mental abilities. Second, to posit that the expression of these genotypes is strongly influenced by changes in the environment ignores the empirical evidence we already have on this point, from reports on separated twins and other adopted children, showing that IQ is not strongly affected by the particular environmental intervals sampled in these studies. Obviously, these results do not exclude the possibility that IQ might be greatly changed by other kinds of environmental alteration; this is why future efforts at enhancing IQ should be concentrated here. But Feldman and Lewontin should at least make explicit that their proposition is purely a speculation about as yet untested environmental conditions, and that what data we do have concerning normal people moved around within common environmental situations show their norm of reaction for IQ to be in fact rather flat. Third, to posit that the same environmental change can evoke different changes in the respective phenotypes, even including changes in opposite directions, is simply another way of saying that the interaction of environment with genotype is nonadditive. It is reasonable to raise this possibility for discussion, but it is surely not reasonable to write, in the face of lengthy discussions of just this point by Jensen (2, pp. 48–54 and 173–179) and others (3), that “this situation is ignored by . . . Jensen”

3) The contention by Jensen 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” is correct. Consider the situation of two groups manifesting with respect to a given trait both great within-group variability and a large difference in the group means. If the trait were found to have negligible heritability within each of the groups (which would mean that only environmental factors caused this diversity), one would certainly be more hesitant to attribute a priori the group mean difference to genetic causes than in the contrasting case, where the within-group heritabilities were high. In the first case, environmental variation is the only source

that has as yet been established as an actual cause of some phenotypic diversity; that anything else has the capability to cause such diversity is pure speculation. In the second, complementary case, it is genetic variation that has been nailed down as an actual cause of some phenotypic diversity, while every other possible source remains entirely speculative. Thus, in order to entertain seriously a purely environmental interpretation for a difference in group means in the face of high within-group heritabilities, we are required to make two additional assumptions of a very specific kind: first, that there is a milieu factor that has the capability of producing phenotypic diversity; and second, that this influence acts in such a manner that it can wholly cause the group means to differ but cannot cause significant phenotypic variation within either group (or else, the within-group heritabilities would be high, as posited). If we apply the principle of parsimony, it is unarguable that this alternative is, a priori, the least probable one.

In the case of white-black IQ differences there is in fact a plausible environmental influence that could well be imagined, a priori, to act in just this fashion—namely, racial discrimination. It is when we move away from the a priori condition by proceeding with the systematic scrutiny of the operationally testable corollaries of this broad hypothesis that it encounters serious challenges. These have been very thoroughly explicated elsewhere (2, 4).

Let us suppose that we accept the thesis favored by Jensen, to wit, that individual differences in IQ, regardless of ethnic or racial group membership, are not strongly conditioned by existing environmental diversity. What then? There are two main, proper implications. The first one, previously mentioned, is that we must, to the extent that we continue to deem it of value to change IQ, look, in the manner of Heber, among new or rare environmental modifications for effective therapies. The second and probably most immediately fruitful one is that we must make the educational system mold itself around individual mental differences, rather than allow it to continue to ignore them as being brought about by superficial and easily reversible influences. This means that instructional methods must adapt as much as possible to each child's configuration of mental abilities; and since high-IQ children can already be taught comparatively well by known methods, the urgent need clearly is for preferential emphasis on devising techniques that can be effective for low-IQ children as well (5).

The potential thus exists for a considerable reduction in inequalities in IQ and realized school performance, and as a natural concomitant, in the social differences that correlate with the level of educational attainment. And by lowering such differences between individuals, one will also necessarily have lowered the differences between the groups they may compose (6). For this reason the genetic interpretation of mental differences offers us, not a counsel of despair as is—curiously—so often alleged, but instead a sound and realistic basis for solid educational and social advancement.

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5. W. R. Havender, *Alternative: Am. Spectator* **9**, 10 (April 1976).
6. The reader will find it instructive to compare these sentences with what Feldman and Lewontin, in their summary statement quoted earlier, purport to be the social implications of Jensen's contemplations.

Feldman and Lewontin argue two points, the first of which is that “no statistical methodology exists that will enable us to predict the range of phenotypic possibilities that are inherent in any genotype.” Seven equally bland variants of this statement can be generated by replacing “statistical” with “non-statistical,” “phenotypic” with “genotypic,” and “genotype” with “environment.” Their second point is that human biometrical genetics has been and will remain sterile, “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.” This resounding declamation is ambiguous: since not even a single gene with low penetrance can be demonstrated in man, their “low” should presumably read “high”; disproof of a simple mechanism is a convincing argument for a more complicated one; estimation of heritability is useful if and only if it leads to prediction or discrimination among genetic hypotheses.

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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 com-

plex 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

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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)].
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6. J. L. Lush, in *Thirty-Third Annual Proceedings of the American Society of Animal Production* (1940).
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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

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this is an area to which Jensen appears not to have given much previous thought. His remarks concern the issue of the change in genetic variance under natural selection. Our claim was that, since the additive part of the genetic variance for IQ would decrease toward zero under natural selection (in the absence of mutation) while other parts need not, it might be possible to infer how much selection has gone on. Jensen seems to be under the misapprehension that the mere presence of nonadditive genetic variance is a demonstration of the previous action of natural selection. R. A. Fisher has speculated (1) that for certain phenotypes the degree of dominance itself may be under the influence of natural selection, but this evolutionary modification of dominance should by no means be taken as a rule (2), nor does it mean that the existence of dominance must imply the previous action of natural selection. On the contrary most models of enzyme action lead directly to dominance as a consequence of the nonlinearity of enzyme-product relations. Thus Jensen is incorrect in claiming that the presence of dominance indicates past selection.

Jensen apparently does not understand that natural selection destroys all variance unless there is some sort of stable polymorphic equilibrium. In the latter case, some gene frequencies will be held at intermediate equilibria, with the consequence that there is no additive variance on the fitness scale (3), while additive variance may persist on the phenotype scale. If Jensen wants to maintain that the additive variance for IQ is present in the face of natural selection, he should also maintain that the relevant genes are maintained at intermediate equilibrium by some sort of balancing selection. In the face of this it would be most difficult for him to maintain his previous position on "dysgenic" trends.

Another elementary misconception is exhibited in Jensen's statement that the presence of dominance variance can be inferred from the difference between h^2_B and h^2_N . This is incorrect, since h^2_B includes contributions from the genotype by environment interaction variance, epistatic variance, and other terms, as well as the dominance variance (4). The other terms would have to be shown to be negligible before the difference in heritabilities could be attributed to dominance variance.

Jensen concludes his analysis of the selection problem with the statement, "We would not expect extremely high correlation of human intelligence with

fitness." It appears that, in his confusion over the fundamental theorem of natural selection, he has overlooked his earlier statement that we were wrong in claiming that IQ has not been under intense selection for very long.

At the conclusion of his letter, Jensen implies that he accepts the validity of the studies on identical twins reared apart. This is, of course, very much in line with his 1969 point of view (5). But it is quite inconsistent with his more recent writing (6), in which he has rejected a large part of the data he originally used.

Jensen devotes his comments to a segment occupying about 7 percent of our article. He ignores our discussion of between-group differences, a topic upon which he has written extensively in the social science literature, as well as our numerous other criticisms of his use of heritability.

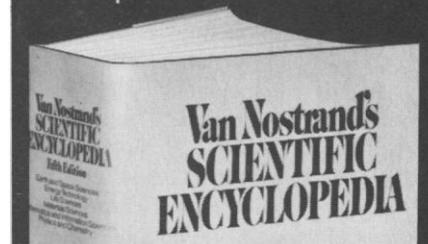
Havender, on the other hand, addresses a potpourri of Jensen's previous notions. He commences with his evaluation of what Jensen really means when he says "Compensatory education has been tried and it apparently has failed" (5) or when he entitles an article "The differences are real" (7). Havender's claim is that Jensen really meant *some forms of* "compensatory education" and *a few* "differences." If, in fact, this is what Jensen had in mind (and we find it difficult to extract this interpretation from the written words), then what has heritability to do with the problem? Havender would have us believe that Jensen's motive in promoting the importance of heritability of IQ has been to demonstrate the need for "novel types of intervention."

In fact Jensen has used the estimated heritability of IQ in white populations to justify his assumption of genetic differences for IQ between blacks and whites. As we have tried to point out in our article and elsewhere, both arguments are logically incorrect; h^2_B provides no information on the possible effect of intervention, nor on between-group differences.

As to whether our figures 1 and 2 are bizarre, it is sufficient to draw the reader's attention to the literature in population genetics on norms of reaction (8). Havender's claim that adoption studies show that IQ is not strongly affected by the environment is incorrect (9). The problem is how reliable such studies are, whatever their conclusion (10).

Havender, continuing the tradition of Jensen and his followers, fails to acknowledge that no information concerning group differences can be extracted from within-group heritability. If he

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finds it strange that “the genetic interpretation of mental differences” has been viewed as a “counsel of despair,” we recommend that he read the history of eugenics as applied to IQ in the early part of L. J. Kamin’s book (10). Perhaps then he will see how much “educational and social advancement” has been achieved as a result of such counsel.

The intemperate tone of Morton’s letter, in which he accuses us of cultivating “obscurity” and “clumsy harrying of biometrical genetics” is understandable, since he has spent so much of his own scientific energy in developing the methodologies that we question. Unfortunately his letter provides no substantive support for his polemic. Morton offers as his example of a case where genetic knowledge has improved risk prediction, of all things, hemophilia! But hemophilia is the result of a single recessive sex-linked mutation with complete penetrance. As we point out in our article, this is *precisely* the one situation in which genetic information is useful in predicting risks. The question is whether any genetic hypothesis *more complicated* than one or two Mendelian loci with high (Morton correctly points out our slip of the pen here) and constant penetrance, improves risk estimation. Rather than suggesting that those who are forced to use empirical risk calculations are “charlatans” and “quacks,” Morton might have helped us by giving us the evidence that the complex pedigree analyses in which he engages have, *in fact*, improved the practice of genetic counseling. The absence of such evidence and the question of what constitutes first- or second-rate service to patients must remain open (11).

Morton claims that “flogging” broad heritability is unnecessary. He need only read any issue of *Behavior Genetics*, not to mention numerous textbooks on genetics and behavior. As to whether any geneticist supposes that the heritability of group differences can be predicted from intragroup heritability, he might try comparing notes with Plomin and DeFries, who also have a letter to the editor in this issue of *Science*. We agree that there was nothing in our article that any competent geneticist does not know. But *knowing* and *saying* appear to be two quite different things.

Genetic counseling has an important function in serving to avoid human suffering. We must not reject any knowledge that will make such counseling more accurate; but we must not pretend to knowledge that we do not have nor assume that very complicated and impenetrable mathematical formalities are nec-

essarily closer to the truth by nature of their being farther from our understanding.

Plomin and DeFries make two points worth commenting on. First they offer the demonstration of heritability of schizophrenia as a counterexample to our claim that genetic analyses of “complexly determined behavior” are not useful. But they do not reveal what the use of this demonstration has been either in counseling or treatment. Perhaps it is their belief that the existence of such a heritability argues against psychotherapeutic treatment and in favor of some sort of physical intervention. The heart of our argument is that the existence of heritability is irrelevant to the possibility and form of therapy.

In their second point Plomin and DeFries persist in that incorrect claim that the formula connecting within-population and between-population heritabilities has some content. They seem to believe that any formula involving two variables (h^2_B, h^2_W) provides them with a meaningful connection. For example, let the variance in amount of manure produced by bulls in Iowa be σ^2_B and the variance in the number of words in letters to the editor of *Science* be σ^2_S . We then form the ratio $B_S = \sigma^2_B/\sigma^2_S$. By a simple rearrangement we have $\sigma^2_B = B_S \sigma^2_S$. Have we really shown that there is some meaningful relationship? This argument is logically identical to that which connects h^2_B and h^2_W . That is, their ratio is used to define the intraclass correlation, and then each by an algebraic rearrangement, h^2_B , is made to appear as a function of h^2_W .

Frankel raises the entirely spurious issue of scientific freedom and openness of inquiry. He tells us that “No person has a right to legislate . . . social attitudes for others, much less for a whole scientific community” and that “Scientific advocates of eugenics have the same right . . . to express their views as do Feldman and Lewontin.” But these are red herrings. Nowhere in our article do we “legislate” anything or speak about depriving anyone of the right to express any idea or view. What we have done is to point out that some “ideas” are incorrect, some even nonsense, and that scientific concepts have been *misused* and sometimes blatantly misrepresented for political ends. We reiterate that “in our opinion geneticists ought to dissociate themselves utterly from eugenics” for the reasons given in our article. Frankel implies that we wish to bury objective truth or prevent its discovery because we dislike or fear the social consequences. This is an often repeated er-

ror in discussions of genetics and race. We neither fear nor dislike any objective truth. What we fear and detest is the misuse of scientific concepts in order to justify misrepresentation of objective reality. The right to express views does not include the “right” to twist scientific concepts, the “right” to illogical reasoning, and the “right” to misrepresent data. On the contrary the community of scientific workers has the obligation to expose falsehood and to demonstrate the limitations that assumptions place on the applicability of conclusions.

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“Pregnancy Prevention”

Healey’s letter (9 July, p. 98) suggests that the incidence of gonorrhoea has declined more rapidly in Sweden than in Denmark because the Swedes refer to protective devices by a shorter word. Not to be outdone by the Swedes, the Danes also use the word *kondom*. The Danish term *svangerskabsforebyggende middel* is a general one that also refers to IUD’s, diaphragms, and pills. Furthermore, even though a purchaser would not ask for *kondoms* by the general term, it would be no more difficult for him to say than the equivalent, “pregnancy preventative,” is for English-speaking people.

I am sorry Healey’s theory does not hold water; it would be a great advance in medicine if diseases could be controlled by the introduction of new words into vocabularies.

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