Letters to and from the Editor

John B. Gibson, C. G. Nicholas Mascie-Taylor, James N. Thompson Jr., Arthur R. Jensen, Roland Fischer, Franklin C. Bing, Dwight J. Ingle, R. L. Hullinger, John F. Adams, F. A. Jenner, Claude A. Frazier

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LETTERS TO AND FROM THE EDITOR

DEAR SIR:

We feel it is necessary to correct a misleading impression given by Professor Jensen in his article, "The Phylogeny and Ontogeny of Intelligence" [1]. Jensen writes, "It is now generally accepted by geneticists, psychologists, and sociologists who have reviewed the evidence that social class differences in mental abilities have a substantial genetic component." This statement should be considered in relation to the following facts which at least many geneticists *would* accept: (i) The proportion of the IQ variance that arises from genetic variety in a population is less than 100 percent. (ii) IQ is a continuous variable, and it is not yet possible to determine gene frequencies. (iii) It follows "that we cannot at present answer the question whether there is any genetic component of social class or race differences in mean IQ" [2]. It is also impossible to say whether there is any environmental component, for this is, in fact, the same question. Thus, as it is impossible to quantify either the genetic or the environmental component of social class differences in mean IQ, we feel it is unwise to say that there is "a substantial genetic component" in these differences. We find it disturbing that Jensen chooses to ignore these facts.

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JOHN B. GIBSON
C. G. NICHOLAS MASCIE-TAYLOR
JAMES N. THOMPSON, JR.

Department of Genetics
University of Cambridge
Cambridge, England

DEAR SIR:

The first two statements listed by Gibson, Mascie-Taylor, and Thompson are certainly true. I disagree, however, with their third point. It is not a necessary condition either that there be 100 percent heritability of IQ or that we must be able to count single gene frequencies in order statistically to infer with a high degree of probability the existence of a genetic component in the mean IQ difference among socioeconomic classes. (I am here referring to social class differences *within* a given racial group. The question of intelligence differences between racial groups involves other methodological problems and types of evidence not discussed here.)

Several lines of evidence support with a high level of confidence the conclusions that social classes, on the average, differ to some degree in the genetic factors involved in intellectual development. Social classes may be viewed as Mendelian populations that have diverged genetically. When the population is stratified into five or six socioeconomic status (SES) categories, mainly according to occupational criteria, the mean IQs of the *adults* so classified, from the highest SES category (professional and managerial) to the lowest (unskilled labor), span a range of some 30–40 points. The standard deviation of IQs *within* SES groups averages about 9 or 10 points for the adult population, as compared with SD = 15 for the whole population. Children born into these SES groups, on the other hand, show a mean IQ difference, from the lowest to the highest class, of only 20–30 points; and the SD *within* classes for children is about 13 or 14 IQ points, which means there is almost as much IQ variation among children *within* social classes as we find in the total population.

The cause of the higher degree of correlation between SES and IQ among adults than among children is the high level of social mobility in each generation. In England and in the United States more than 30 percent of the adult generation are found to be of a different SES than that of their own parents [1–3]. In each generation some individuals move up in SES and some move down. Those who move up have higher IQs, on the average, than those who move down.

Since the heritability, h^2 (i.e., the proportion of genetic variance), of IQ in the total population is between .70 and .80, and since the correlation between phenotypes and genotypes is the square root of the heritability, it follows that IQ estimates genotypic intelligence with a reliability of between $\sqrt{.70}$ and $\sqrt{.80}$ (i.e., between about .84 and .89 [4, 5]). Conversely, the reliability with which IQ measures the *non*genetic component of intelligence variation is $\sqrt{1-h^2}$, or between about .45 and .55. If only nongenetic factors determined individuals' SES, then the maximum correlation that could exist between SES and IQ would be in the range of .45–.55. In fact, however, the correlations generally found are between .30 and .50 for children and between .50 and .70 for adults (depending largely upon how fine grained the SES measure is). Now, if the correlation between IQs and genotypes is between .84 and .89, and the correlation between IQ and SES is between .50 and .70, the correlation between SES and genotypes must be greater than zero. To maintain a strictly environmental hypothesis, at the very least one would have to assume that only the environmental component of intelligence played a part in persons' educational and occupational attainments (the chief determinants of SES). If we admit no genetic component in SES differences in IQ and still admit the high heritability of IQ, we are logically forced to argue that persons have been fitted to their SES (meaning largely educational and occupational attainments) almost *perfectly* according to their environmental advantages and disadvantages, which constitute only 20–30 percent of the variance in IQ; and it would have to be argued that persons' innate abilities, talents, and proclivities play no part in educational and occupational selection and placement. This is a most unlikely state of affairs.

Consider other, more direct, evidence.

1. Adopted children show only about half as much dispersion in mean IQ as

a function of SES of the adopting parents as that of children reared by their own parents [6].

2. Children reared from infancy in an orphange, with no knowledge of their parents, show nearly the same correlation between their IQs and their fathers' occupational status (graded into five categories) as children reared by their own parents [7].

3. Most of the IQ difference between siblings reared together is attributable to differences in genetic inheritance. (The genetic correlation between siblings is about .5–.6.) When siblings who are reared together move into different social strata as adults, it is the sib with the higher IQ who is more likely to move up and the sib with the lower IQ who is more likely to move down the SES scale [8].

4. Sons whose IQs differ most from their fathers' IQ are more likely to change SES, the higher IQs moving up, the lower moving down [9]. Waller [10] found a correlation of .368 $\pm$ .066 between the father-son disparity in IQ (both tested as schoolchildren) and father-son disparity in SES as adults, when only the middle three of five SES classes were considered (since in Classes I and V mobility is restricted to only one direction).

5. Genetically identical twins who were separated in infancy and reared apart in homes of different SES (over a range of six categories, from professional to unskilled), differ on the average by only 1 IQ point per each SES category difference, with a total range of about 6 IQ points difference between the highest and lowest SES categories [11]. Compare this difference, in which genetic factors play no part, with the difference of 20–30 IQ points generally found between children in the lowest and highest SES classes.

All this evidence is highly consistent with a model of social mobility in which the genetic factors involved in mental ability, through the processes of segregation and assortment, become selected into somewhat differing gene pools in various social and occupational classes. It is most improbable that genes are not carried along with the observed phenotypic social mobility of IQ. The complete agnosticism expressed by Gibson et al. therefore, seems to me to be quite unwarranted.

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Arthur R. Jensen

Institute of Human Learning
University of California, Berkeley

DEAR SIR:

A selective glance at the literature on the development of tolerance to morphine reveals two interrelated factors. Morphine displays a high affinity to (apparently) proteinous structures, since tolerance to (the analgesic effects of) the drug develops after a *single* dose; but two types of tolerance can be distinguished, one independent of and the other dependent on "drug-test interaction" [1]. This latter type can be referred to as "state-bound" [2] tolerance.

Probably, the high affinity of morphine or its metabolite for particular neuronal protein may result in permanent binding after repeated administration of the drug. This is consistent with the observation of Misra, Mitchell, and Woods [3], who found that twenty-four hours after a subcutaneous injection of 10 mg/kg ^{14}C-morphine-N-methyl all the radioactivity was due to a conjugated form of morphine.

"State-boundness," or the ability to reexperience an event associated with a particular state of (ergotropic or trophotropic) arousal [4],[1] is restricted to "variable" individuals, who can be characterized by variable performance on some perceptual and/or behavioral tasks [2].

Since we find that variability on a simple perceptual or behavioral measure, such as the standard deviation on handwriting area, follows a log-normal distribution, the population most liable to develop tolerance and, thus, favor conditions for the development of morphine addiction may be narrowed.

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ROLAND FISCHER

Drug Treatment and Research Center
Veterans Administration Hospital
50 Irving Street, N.W.
Washington, D.C. 20422

[1] "Ergotropic arousal . . . is characterized by increased activity of the sympathetic nervous system . . . [whereas] trophotropic arousal results from an integration of parasympathetic with somatomotor activities" [5].

DEAR SIR:

I do not think that anyone would quarrel with Ingle's view that "there is a risk of error in trying to describe the properties of a whole in terms of the properties of a part" [1]. The example he has selected to illustrate this fallacy, however, does not appear to be as well chosen or as adequately described as it might be. The impression conveyed could well be that a scientist believed that 46.5

percent of all children had rickets because that was the incidence which he, working in Baltimore, had found by examination of the bones of a limited number of children at necropsy. The source of these data must be the paper by Follis et al., written many years ago [2]. Dr. Edwards A. Park, who at the time was a leading authority on the histopathology of rickets, had examined the anterior costochondral junctions of the middle ribs of 230 consecutive children, who happened to be between the ages of two and fourteen, at necropsy over a period of several years. Signs of rickets were observed in the microscopic sections of the bones of 107 of the children. The authors believed that most of the children showing rachitic lesions had this condition before the onset of their fatal illnesses, which were most often of short duration. In only six of the cases could the diagnosis of rickets be made roentgenologically; even in these six children the condition had been unsuspected during life. I think that Park, and others at the time, regarded the data as important evidence, not of the incidence of rickets in an unexamined population, but as evidence of a need for vitamin D (and of course calcium and phosphorus) through all the years of linear growth of the bones.

Park's views influence our thinking today, largely because data on the nutritional status of the bones in children are still not as extensive as we would like. It seems important, therefore, to protect what little evidence we do have concerning the importance of providing physiological quantities of vitamin D to growing children. That is the reason I, whose thinking has been marred too often by the fallacies described by Ingle, bring this matter to your attention.

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Franklin C. Bing

2651 Hurd Avenue
Evanston, Illinois 60201

Dear Reader:

Franklin C. Bing has identified an error in my paper on fallacies and errors. I wrote about the study on rickets in a way that would lead the reader to infer that the authors concluded that 46.5 percent of all children had rickets. They did not. This inference was made by the sales education department of a pharmaceutical house which placed the conclusion in their advertising and in their sales pitch for vitamin products by company salesmen. I agree with all of the foregoing comments of Franklin Bing and thank him for the correction.

Dwight J. Ingle

Dear Sir:

And More on Sex

Students learn to engage in the educational environment much as they might learn to engage in physical sexuality.

"Go to school. It will help you," he is told. He indulges—not fully committed, not open, not free to give and receive. Love does not pervade the academy. "It" is left at home over the weekend.

And so many enter the classroom so defensive and so fearful, a most effective prophylactic. Some use the rhythm method, engaging in dialogue and openness only at safe periods—least-risk periods.

All works best when one doesn't want to create and produce. But *that* is the purpose of education—to create and produce.

But, alas, faculties are composed, in part, of witches with whom no one seeks to relate or untouchable celibates who induce a similar response.

Where are those who become involved and who deliberately take the risk?

R. L. HULLINGER

Department of Anatomy
Purdue University
Lafayette, Indiana 47907

DEAR SIR:

In the "brave new world" discussed by Glass in "Human Heredity and Ethical Problems" [1], it seems we are led to assume that it would require a technology removed to a distant future to keep the population from being overindependent and "hoodwink" people to react in predictable ways.

REFERENCE

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JOHN F. ADAMS

Great Lakes Naval Hospital
115 High Street
Highwood, Illinois 60040

DEAR SIR:

Dr. Siomopoulos's suggestion [1] that the free-will problem arises by the way brain processes become experiences raises at least as many problems as it solves. It would imply that the awareness was of no importance in determining the course of events. In evolution there was, therefore, no selection for enlarging the area of consciousness; that just happened somehow.

If, however, to be able, as it were, to think things out in the mind's eye is of advantage, as it does seem to be, then mental processes might be seen as influencing and changing the laws of physics and chemistry in certain situations. In which case why should we slavishly make enormous extrapolations to deny the apparent freedom we experience?

The problem of course is still more complex. The language we use makes us materialists and determinists, though it is naïve to believe we are able to describe reality accurately. This becomes particularly apparent in a physicist's study of the nature of ultimate particles.

The use of scientific language to achieve many ends is of proven value, its misuse to reduce man and the universe to a merely pointless predetermined charade is reprehensible and mistaken. Perhaps the pleasures of art, music, literature, mysticism, etc. dimly assure us of other things, and might we not reasonably ponder why the universe is such that awareness is possible anyway?

REFERENCE

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F. A. Jenner

Department of Psychiatry
Whiteley Wood Clinic
Woofindin Road, Sheffield 10, England

Dear Sir:

I am compiling case reports of allergic reactions to biting insects (i.e., mosquitoes, fleas, kissing bugs, bedbugs, gnats, and flies), including horsefly, sandfly, deerfly. I am also interested in reactions to fire ants.

I would appreciate it if physicians would supply me with case reports of any patients who have had reactions to such insects, including in those reports the history of the type of reaction and complications if any; the immediate treatment. If desensitization were attempted, what were the results? Please send to:

Claude A. Frazier, M.D.

4-C Doctors Park
Asheville, North Carolina 28801