ELSEVIER

Cognition 56 (1995) 99–128

COGNITION

How heritability misleads about race

Ned Block*

Department of Linguistics and Philosophy, Massachusetts Institute of Technology, 77 Massachusetts Avenue, Cambridge, MA 02139-4307, USA

Received 2 February 1995, final version accepted 12 May 1995

Abstract

The Bell Curve revives and elaborates an argument given by Jensen to the effect that facts about heritability of IQ in whites dictate that blacks are genetically inferior in IQ. But clarification of the concept of heritability shows that this reasoning is fallacious. Heritability is an uninteresting measure that only misleads us about race.

1. Introduction

The Bell Curve's main argument for black genetic inferiority in IQ (Herrnstein & Murray, 1994) depends for its persuasive force on conceptual confusions that have been tacitly accepted to some degree even by many of the book's sharpest critics. The book contains two lines of thought. One, which I will accept for present purposes, is: that IQ tests substantially measure "general intelligence", that IQ is socially important and that IQ is 60% heritable within whites. (I'll explain heritability below) The second main line of thought – which I will be contesting – is the argument for genetic inferiority of American blacks. Before I get to their argument for this conclusion, I want to be clear about the conclusion itself. Murray has recently complained about misinterpretation in an article entitled "The real bell curve" (Murray, 1994). He grumbles about critics, such as Stephen Jay Gould, who read the book as saying that racial differences in IQ are mostly genetic. He quotes from the book:

> If the reader is now convinced that either the genetic or environmental explanations have won out to the exclusion of the other, we have not done a sufficiently good job of presenting

* E-mail: block@psyche.mit.edu.

SSDI 0010-0277(95)00678-8

one side or the other. It seems highly likely to us that both genes and environment have something to do with racial differences. What might the mix be? We are resolutely agnostic on that issue: as far as we can determine, the evidence does not yet justify an estimate." (*Bell*, p. 311)

But the *resolute agnosticism* we see here is on the issue of which is *more* responsible for the low IQs of blacks, bad environment or genetic inferiority. What they are very much *not* agnostic about is that *part* of the IQ difference between blacks and whites is genetic, and (given their way of thinking about the matter) to the extent that *any* of this difference is genetic, blacks are genetically inferior.[1]

What is their argument? It proceeds from two facts: the first is the 60% heritability of IQ within whites and the second is the average 15-point IQ difference between whites and blacks.[2] A crude version of their argument goes like this. If IQ were completely genetic in whites and completely genetic in blacks, the IQ difference between whites and blacks would have to be completely genetic; but given that IQ is *largely* genetic (at least in whites), surely it is very probable that the IQ difference is partly genetic in origin. There is more to their argument than that; they raise issues about the pattern and the magnitude of the differences that I will get to later.

Herrnstein's and Murray's argument depends on thinking of the 15-point IQ difference as divisible into a genetic chunk and an environmental chunk. This way of thinking dictates the following three alternatives:

(1) Extreme environmentalism: Blacks are genetically on a par with whites, so all of the IQ gap is environmental.
(2) Extreme geneticism: Blacks are environmentally on a par with whites, so all of the IQ gap is genetic.
(3) The reasonable view: Blacks are worse off *both* genetically and environmentally: some of the gap is genetic, some environmental.

Option 1, extreme environmentalism, is thought to be excluded by the 60% heritability of IQ. Option 2, an equally extreme geneticism, is excluded by well-known environmental effects on IQ together with differences between black and white environments that are acknowledged by Herrnstein and Murray. So we are left with option 3, which on this way of putting the matter is the reasonable view – and it postulates *some black genetic inferiority*. But their way of putting the alternatives blots out a *crucial possibility*,

[1] I am going along with a dangerous way of thinking here. As I will point out in the last section of this paper, this talk of part of an IQ difference is deeply problematic.

[2] This is a widely accepted figure, but Nisbett (1995) gives some plausible arguments that the gap has narrowed. As he says, no study in the last two decades indicates a narrowing of the gap by less than 4.2 points. I am using Herrnstein's and Murray's figure, putting aside disagreements about the data so as to concentrate on the conceptual issues.

namely that blacks are *much* worse off than whites environmentally and actually *better* off genetically. Allowing this option, we get a different set of alternatives: *genetically, blacks are worse off – or better off – or equal to whites.*

The idea of the missing alternative arises from the possibility that genetic IQ differences and actual measured IQ differences go in *opposite directions*. Even if you think that races are likely to differ genetically in IQ, the question remains of the *direction* of the genetic difference. In this scenario, black environments for the development of IQ are on the average sufficiently worse than white environments as to lower average black IQ *more* than 15 points. I don't say that this is *likely*, but I do say it is *possible* and its possibility is important. What you consider as *possible* affects what you think is an *extremist* position. The critics of Herrnstein and Murray have tended to trip over this possibility.

For example, in a *New York Times* op. ed. critique that describes *The Bell Curve* as "bogus" and "nothing but a racial epithet" (Herbert, 1994), Bob Herbert insists that "the overwhelming consensus of experts in the fleld is that environmental conditions account for most of the disparity when the test results of large groups are compared". In effect, he uses known environmental effects on IQ to argue for a *low degree of genetic inferiority* in blacks. Even Stephen Jay Gould (in an otherwise excellent article in *The New Yorker* – Gould, 1994) missteps here. Apparently accepting *The Bell Curve's* way of conceiving of the issue, he complains that Herrnstein and Murray wrongly minimize the large environmental malleability of IQ. He says that they turn "every straw on their side into an oak, while mentioning but downplaying the strong circumstantial case for substantial malleability and little average genetic difference". Gould does little to guard against the natural interpretation of "little average genetic difference" in the context of discussion of *The Bell Curve* as *little average genetic inferiority of blacks*. Jim Holt's critique in the *New York Times* (Holt, 1994) asks "How then do we account for the sizable gap in measured IQ?" and then goes on to emphasize prenatal explanations of most of "the racial gap". (Low birth weight is almost three times as common in black babies as in white babies – see Lieberman, 1995.) But if one accepts the Herrnstein and Murray framework, it is a *stretch* to appeal to prenatal differences to reduce the size of the genetic inferiority all the way to zero. A somewhat different version of the same fallacy is a misplaced agnosticism. *US News and World Report* (Leo, 1994), attempting to be neutral and unbiased, says of the IQ gap that "we don't know . . . how much is genetic, or how much environmental factors are responsible". That is, we don't know *how genetically inferior* blacks are. The *expectation*, that is, is that blacks are genetically inferior to *some* extent, but lacking information, we don't know *how much*. A number of the critics in *The New Republic* (31 October 1994) in turn, wonder about the size of the "genetic component of the black–white difference" (p. 10), thereby buying into the same way of thinking. Again, we can see that what you take

as possible affects what you take as actual. As with the passage from Herrnstein and Murray that I quoted earlier, agnosticism ends up as agnosticism about just how genetically inferior blacks are.

If you accept *The Bell Curve's* way of putting the options, evidence for environmental effects at best shows that the amount of genetic inferiority is little or nothing, and so the critics find themselves up against the wall of zero genetic difference. Given the set of alternatives provided by Herrnstein and Murray, the idea that the environmental difference between blacks and whites is big enough to account for 15 IQ points looks like an *extremist* hypothesis. But given the *actual* alternatives, that blacks are genetically on a par with whites, or worse off, or better off, zero genetic difference doesn't seem extremist at all.

But isn't the idea of black genetic superiority in IQ desperate and pathetic, merely a logical possibility? Roses are red, violets are blue. Color is genetic, so the color difference is genetic. Is there any real possibility that the genetic color difference is the opposite of the observed one? Toe number is genetic in sloths and in humans, and humans are observed to have five toes whereas sloths (that is, diurnal sloths) are observed to have three toes. Is there any real possibility that the genetic toe difference between humans and sloths goes in the *opposite direction* from the observed toe number gap? Well, there is a very remote possibility that the three-toed sloth evolved six toes, but the three toes we observe are a result of a thalidomide-like chemical which has polluted their food during the years in which we have observed them.[3] But this possibility is only worth mentioning as an example of something *extremely unlikely*. And that leads to a principle that underlies all of Herrnstein's and Murray's thinking on race even though it is never articulated. Recall that the crude form of their argument is that if IQ were completely genetic in blacks and whites, then the black-white difference would have to be completely genetic as well; so given that IQ is 60% heritable within whites, very likely the black–white difference is partly genetic. The underlying principle is: *if a characteristic is largely genetic und there is an observed difference between two groups, then there is "highly likely"* (I allude to their term from the passage quoted in the first paragraph of this article) *to be a genetic difference between the two groups that goes in the same direction as the observed difference.* So given the substantial heritability of IQ, if East Asians are superior in measured IQ, then they are highly likely to be *genetically* superior; and if blacks are inferior in measured IQ, then they are highly likely to be *genetically* inferior in IQ.

Here is a roadmap to the rest of this article. The principle just mentioned

[3] Roses are red, violets are blue, color is genetic, so the color difference is genetic. This sounds like a tautology, but it isn't. Perhaps the easiest way to see that is to think of what it is for color to be genetic to implicitly involve appeal to the idea of a normal environment. If the environment is not normal (as in the thalidomide example) the conclusion may be false even when the premises are true.

is right on one sense of "genetic" and wrong on another. However, the sense in which it is *wrong* is the one that employs the figure of 60% heritability. In the sense in which the principle is right, IQ is not genetic. In the next section, I will explain the ambiguity in "genetic". Then I will explain why the principle just mentioned is wrong in the sense of "genetic" that really counts for their argument: *heritability*.

2. Heritability versus genetic determination

The key to part of the fallacy of *The Bell Curve* is the distinction between two concepts: *genetic determination* and *heritability*. The concept of inheritance which allows us to speak of number of toes as genetic is the ordinary commonsensical idea of *genetic determination*. But the *scientific concept*, on which all Herrnstein's and Murray's data rely, is *heritability* and not *genetic determination*. Heritability has to do with what causes *differences* in the value of a characteristic, whereas genetic determination is a matter of what causes a particular value of the characteristic *itself*. Heritability is *defined* as a fraction: the ratio of *genetically caused* variation to *total* variation (environmental and genetic). *Genetic determination*, by contrast, is an informal and intuitive notion that has no quantitative definition (for reasons that I will explain later). It depends on the idea of a normal environment. A characteristic could be said to be genetically determined if it is coded in and caused by the genes and bound to develop in a normal environment. Genetic determination in a single person makes sense: my brown hair color is genetically determined. By contrast, heritability makes sense only relative to a population in which individuals differ from one another. You can't ask "What's the heritability of *my* IQ?"

We can get a handle on the difference by noticing that the number of fingers on a human hand or toes on a human foot is genetically determined: the genes code for five fingers and toes in almost everyone, and five fingers and toes develop in any normal environment. However, interestingly, the *heritability* of number of fingers and toes in humans is almost certainly very *low*. What's going on? If you look at cases of unusual numbers of fingers and toes, you find that most of the variation is environmentally caused, often by problems in fetal development. For example, when pregnant women took thalidomide some years ago, many babies had fewer than five fingers and toes. And if we look at numbers of fingers and toes in adults, we find many missing digits as a result of accidents. Genetic coding for six toes is rare in humans, though apparently not in cats. Heritability, you will recall, is a fraction: the ratio of genetically produced variation to total variation (total variation = variation due to all causes, genetic and environmental). If the genetically caused variation is small compared to the environmentally caused variation, then the heritability is low, even when the characteristic in

question is genetically determined. So toe number is genetically determined but nonetheless low in heritability.[4]

Conversely, a characteristic can be *highly heritable* even if it is *not genetically determined*. Some years ago when only women wore earrings, the heritability of having an earring was high because differences in whether a person had an earring were "due" to a chromosomal difference, XX versus XY. Now that earrings are less gender-specific, the heritability of having an earring has no doubt decreased. But neither then nor now was having earrings genetically determined in anything like the manner of having five fingers. The heritability literature is full of high measured heritabilities for characteristics whose genetic determination is doubtful. For example, the same methodology that yields 60% heritability for IQ also yields 50% heritability of academic performance and 40% heritability of occupational status (Plomin, DeFries & McClearn, 1990 p. 393). More significantly, a child's *environment* is often a heritable characteristic *of the child*. If degree of musical talent is highly heritable and if variation in the number of music lessons a child gets depends on variation in musical talent, then the number of music lessons that a child gets may be heritable too, despite not being genetically determined. This is not an idle speculation. Recent studies of heritabilities of various features of children's environments show substantial heritabilities for many environmental features, for example, the "warmth" of the parents' behavior toward the child. Even number of hours of TV watched and number and variety of a child's toys show some heritability (Plomin & Bergeman, 1991; see also Scarr & McCartney 1983). If this seems unintelligible, think of it this way: variation in these environmental properties is in part due to variation in heritable characteristics of the child, and so the environmental characteristics themselves are heritable. People who read *The Bell Curve* often suppose that a heritable characteristic is one that is passed down in the genes, but this identification is importantly flawed. The number and variety of a child's toys is not passed down in the genes. Heritability is a matter of the causation of differences, not what is "passed down".

By contrast, a characteristic is genetically determined if the characteristic itself – not differences in it – is caused by the genes. A characteristic can be genetically determined even if there is no genetically caused variation. Number of heads in humans is genetically determined even if there is no genetic variation in number of heads at all. I've mentioned physical characteristics, but there are many mental features that are genetically determined too. For example, if you put a pacifier of one of a number of shapes (e.g., spherical or cubical or knobbly) in a newborn baby's mouth, the baby will look preferentially at a picture of the same shape. So

[4] I am using a very liberal notion of genetic determination. On a finer grained notion, one might want to distinguish between "the genes code for X" and "the genes code for Y, but the result, given normal development, is X".

something about the coordination between vision and feel is very likely genetically determined. But there may be little or no genetic variation in such mental characteristics, so they are genetically determined without, perhaps, being heritable.[5]

What "genetic determination" comes to depends heavily on what we count as a "normal" environment, as Kitcher (1996) notes. For example, we could think of a normal environment as one that is usual, or, rather differently, as one that allows people to thrive, even if it is uncommon. On the former notion, phenylketonuria (PKU) was once a genetically determined form of mental retardation. But on the latter, given that retardation can be avoided by a diet that is low in phenylalanine, it is not genetically determined. This example illustrates something that we will return to in the last section of the paper, namely that (as Kitcher notes), increasing knowledge often leads us to move from thinking of the genes determining X to the genes determining the tendency to develop X in certain environments. We now tend to think of what is genetically determined in the case of PKU not as retardation but rather as an inability to metabolize phenylalanine that can cause retardation if there is phenylalanine in the diet.

So what is genetically determined depends on what we count as a normal environment. But not so for heritability. It depends *not at all* on what environments are normal, but only on the balance of genetic and environmental differences in the causation of differences in the characteristic. We will return to this point later.

I have given examples of traits that are genetically determined but not heritable and, conversely, of traits that are heritable but not genetically determined. But the reader may be suspicious: what relevance do these weird examples have to the case of IQ? Maybe there is a range of normal cases, of which IQ is an example, for which the oddities that I've pointed to are just irrelevant?

Not so! In fact IQ is a great example of a trait that is highly heritable but not genetically determined. Recall that what makes toe number genetically determined is that having five toes is coded in and caused by the genes so as to develop in any normal environment. By contrast, IQ is enormously affected by *normal environmental variation* (and in ways that are not well understood). As Herrnstein and Murray concede, children from very low socio-economic status (SES) backgrounds who are adopted into high-SES backgrounds have IQs that are dramatically higher than their parents. The point is underscored by what Herrnstein and Murray call the Flynn effect: IQ has been rising about 3 points every 10 years worldwide. Since World

[5] What psychologists usually have in mind when they say that aspects of syntax are genetically determined (Chomsky , 1975; Fodor, Bever, & Garrett, 1974) or that aspects of the concept of an object are genetically determined is that the source of the *information* that people end up with is the genes. This model does not apply to IQ.

War II, IQ in many countries has gone up 15 points, about the same as the gap separating blacks and whites in the USA. As Herrnstein and Murray note, no one knows why IQ has been rising. In some countries, the rise has been especially dramatic. For example, average IQ in Holland rose 21 points between 1952 and 1982 (Flynn, 1987a). In a species in which toe number reacted in this way with environment (e.g. a centipede-like creature which grows legs depending on how much it eats) I doubt that we would think of number of toes as genetically determined. The dramatic rises in IQ in Holland (and other countries) is a very significant fact which I will return to.

I said that these dramatic increases in IQ show that IQ is enormously affected by normal environmental variation. But this claim may appear to be in conflicit with the following fact: that the correlations among adopted children in the same family are very small, near zero, by late adolescence (see Plomin & Daniels, 1987; Plomin & Bergeman, 1991). Doesn't that show that normal environmental variation has no effect? This is the opposite of the correct conclusion from these data. The low correlations among adopted children show that normal variation of the sort that exists even *within* families has a large effect. It should also be noted that the bottom of the socio-economic distribution is scanted in these data because this segment of the population is not favored by adoption agencies. Unfortunately, this segment of the population is all too normal. So what does not appear to have much of an effect on IQ (after adolescence) is variation in SES within the middle range and, in addition, number of books in the home, cultural activities of the parents and the like – in short, the sort of thing that psychologists measure about shared family environment.

One very important conclusion from both the Flynn data and the low correlation data just mentioned is that no one understands very much about how environmental variation differentially affects IQ. The cause of the large increases in Holland is simply unknown. Even Herrnstein and Murray concede that of the environmental variation in IQ "relatively little can be traced to the shared environments created by families. It is, rather, a set of environmental influences *mostly unknown at present*, that are experienced by individuals as individuals" (*Bell*, p. 108; emphasis added).

The crucial factor that has enabled the research that Herrnstein and Murray report to exist at all is the fact that one can measure the heritability of a characteristic without even having much of an idea of what the characteristic is. We needn't know what IQ tests measure in order to calculate the heritability of IQ – we need only be able to measure IQ, whatever it is, in various circumstances. Suppose you could place two pairs of genetically identical (one-egg) twin human fetuses in randomly chosen wombs, and then give them IQ tests after they had grown up in different environments (including different prenatal environments). Suppose twins Sally and Sarah were reared in very different environments – one impoverished, the other rich in intellectual stimulation – yet both ended up

with the same high IQ; and twins Fred and Ted, also reared in very different environments, both had the same low IQ. That would indicate that the genetic differences in the population were contributing more to IQ variation than the environmental differences. So heritability of IQ would be high. The opposite procedure would be rearing genetically unrelated individuals in the same environment (note: same environment, not similar family, since that appears not to provide the same environment). To the extent that heritability is high, unrelated children reared in the same environment should not resemble one another any more than people chosen at random from the population. To the extent that heritability is low, their IQs should be highly correlated.

A common method for measuring heritability relies on comparisons of the correlations of IQ among one-egg twins raised by their biological parents compared with two-egg twins raised by their biological parents. Suppose you give IQ tests to two children and they get the same score. One has a one-egg (identical) twin, the other has a two-egg (fraternal) twin. Suppose that you can predict the score of the one-egg twin reliably, but that your prediction of the score of the two-egg twin is much less reliable. This would be an indication of high heritability of IQ because one-egg twins share all their genes whereas two-egg twins normally share half their genes.

Heritability studies of IQ *within white* populations in the USA and northern Europe have tended to yield moderately high heritabilities. Herrnstein and Murray's 60% is a reasonable figure. But it is important to note that no one would do one of these heritability studies in a mixed black/white population. Why not? If you place a pair of black one-egg twins in different environments "at random", you automatically fail to randomize environments, because the black twins will bring part of their environment with them; they are both black and will be treated as black.

I mentioned that heritability, unlike genetic determination can be very different in different populations. For example, the heritability of IQ could be decreased if half the population were chosen at random to receive IQ lowering brain damage. The quantity a/b gets smaller if b *gets larger.* By damaging the brains of some people, you make the environmentally caused variation larger. Another example that illustrates the same point: suppose we could make a million clones of Newt Gingrich, raising them in very different environments so there would be some variation in IQ, all environmentally caused. The quantity a/b is zero if a is zero. So heritability in that population would be zero because the ratio of genetic variation to total variation is zero if the genetic variation is zero. To take a real example, the heritability of IQ increases throughout childhood into adulthood. Plomin (1990a) gives heritability figures of under 20% in infancy, about 30% in childhood, 50% in adolescence and a bit higher in adult life. (Plomin notes that the results are not a consequence of increasing reliability of IQ tests.) Studies of older twins in Sweden (Pedersen et al., 1992) report an 80% heritability figure for adults by age 50 as compared to a 50% heritability for

children. One possible reason for the rise in heritability is that although the genetic variation remains the same, environmental variation decreases with age. Children have very different environments; some parents don't speak to their children, others are ever verbally probing and jousting. Adults in industrialized countries, by contrast, are to a greater degree immersed in the same culture (e.g., the same TV programs). With more uniform environments, the heritability goes up.[6] Heritability can even be different in men and women. Suppose, for example, that variation in hair length in women depends on heritable variation in the hair itself, whereas hair length in men depends on non-heritable variation in conventions in peer groups. Then hair length would have higher heritability in women than in men. (I'll give an example later on which there are actual data.) I hope these points remove the temptation (exhibited in *The Bell Curve*) to think of the heritability of IQ as a constant (like the speed of light). Heritability is a population statistic just like birth rate or number of TVs and can be expected to change with changing circumstances. There is no reason to expect the heritability of IQ in India to be close to the heritability of IQ in Korea.

These issues are pathetically misunderstood by Murray, as shown by a recent CNN interview reported in *The New Republic* (Wright, 1995). Murray declared "When I – when we – say 60 percent heritability, it's not 60 percent of the variation. It is 60 percent of the IQ in any given person." Later, he repeated that for the average person "60 percent of the intelligence comes from heredity" and added that this was true of the "human species", missing the points made above that heritability makes no sense for an individual and that heritability statistics are population-relative. In a letter to the editor that complains about being quoted out of context (Murray, 1995), Murray quotes more of what he had said, namely: ". . . your IQ may have been determined overwhelmingly by genes or it may have been – yours personally – or overwhelmingly by environment. That can vary a lot from individual to individual. In the human species as a whole, you have a large genetic component." Though Murray embarrasses himself, *The Bell Curve* itself does not make these crude mistakes. Herrnstein, the late co-author, was a professional on these topics. But part of the upshot of this essay is that the book's main argument depends for some of its persuasive force on a more subtle conflation of heritability and genetic determination. And Murray's confusion serves to underscore just how difficult these concepts can be even for someone so numerate as Murray.

Again, the critics of the book are often confused on this topic. A recent op. ed. critique of Herrnstein and Murray in the *New York Times* (Hofer, 1994) describes the basic thesis of the book as the claim that "genes, rather than experience, primarily determine the development of the complex sets

[6] Another possibility mentioned by Scarr and McCartney (1983) is that older children and adults make their own environments to a larger extent than younger children do.

of behaviors that underlie general intelligence". The article goes on to describe the book as linking "inherited intelligence" with race, and as drawing unwarranted conclusions about "genetic influences on general intelligence". Throughout the article, the language of genetic determination is substituted for the language of heritability. No wonder the article ends up conceding "the small differences in IQ between races and other genetically distinct groups that Mr. Murray and Professor Herrnstein set forth in endless detail".

What's the upshot of the distinction between genetic determination and heritability[7] for the argument of *The Bell Curve*? Let's go back to the sloth example: toe number is genetic in sloths and in humans; there is a difference in toe number (three versus five); so the toe number difference is genetic. This is a good argument: it strains the imagination to suppose that the genetic toe difference between sloths and humans goes in the opposite direction from the actual observed toe difference. The idea that our genes code for two toes despite the five toes we see at the beach is ludicrous. So in this sense the Herrnstein and Murray argument works for the concept of *genetic determination*. But the data on genes and IQ *do not* concern genetic determination, but rather *heritability*.

Is IQ genetically determined as well as heritable? No! As I already pointed out, IQ is very reactive to changes in environments in the *normal* range. Recall the example of the large rise in Holland. Surely both 1952 and 1982 count as *normal*! (Of course it is one thing for IQ to be plastic to environmental changes and quite another thing for the changes to be ones that we know how to *manipulate*; in fact, as Herrnstein and Murray note, no known environmental intervention short of adoption changes IQ very much.) Further, the claim that IQ is genetically determined, is not the kind of quantitative claim on which Herrnstein and Murray would want to base their claims about genes and race.

Where are we? Here is an outline of the argument so far:

(1) The basic Herrnstein and Murray argument says: IQ is largely genetic in whites; there is a 15 point black/white difference; so part of the difference is probably genetic.
(2) "Genetic" is ambiguous.

[7] The distinction between genetic determination and heritability might be challenged as follows: whenever a characteristic is genetically determined but not highly heritable, we can always increase its heritability by expanding the relevant population. Thus, number of digits has low heritability in people, but high heritability in the population that includes both people and animals that have different numbers of digits. (This line of thought appears in Sober, 1993, p. 190.) Briefly, there are two problems with this reasoning. First, there is no guarantee that there will be other species that differ in the required way. To appeal to merely *possible* species would be a mistake, since *everything* is heritable relative to *some* range of possible species. Second, this reply does nothing to counter the cases of characteristics that are heritable but not genetically determined.

(3) In the sense of *genetic determination*, IQ is not genetic in whites or anyone else, and in any case the issue is not quantitative.
(4) In the sense of *heritable*, IQ is largely genetic (among whites in the USA at least). But, I will argue below, in this sense of "genetic", the argument does not work.

I said at the outset that the confusion between genetic determination and heritability is partially responsible for the persuasive force of Herrnstein and Murray's argument. But even when this confusion is cleared up, their argument can appear very persuasive. In what follows, I will be leaving behind the issues just discussed about the distinction between genetic determination and heritability. I will be focusing on the logic of the argument from heritability to racial genetic differences. The upshot will be that the logic of the argument is flawed and that heritability is an uninteresting and misleading statistic.

3. Heritability and race differences

Arthur Jensen's 1969 article in the *Harvard Educational Review* started off the current controversy by arguing from heritability within whites to genetic differences between whites and blacks. In 1970, Richard Lewontin gave a graphic example that illustrates why this is a mistake (Fig. 1). Suppose you buy a bag of ordinary seed corn from a hardware store. This is ordinary genetically variegated (not cloned) corn. You grow one handful of it in a carefully controlled environment in which the seeds get uniform illumination and uniform nutrient solution. The corn plants will vary in height and since the environment is uniform, the heritability of height will be 100%. Now take another handful of corn from the same bag which you grow in a similarly uniform environment but with a uniformly poor nutrient

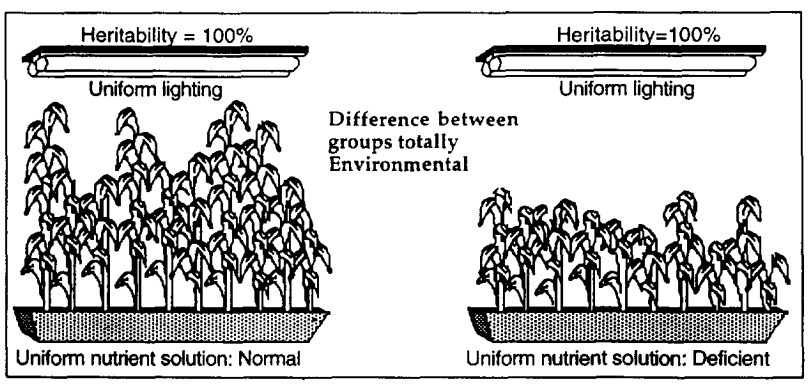

Fig. 1. Heritability can be high within each of two groups even though the difference between the groups is entirely environmental.

solution. The plants will vary in height again but all will be stunted. The heritability of height in both groups is 100%, but the difference in height between the groups is entirely environmentally caused. So heritability can be *total* within groups even if there is no genetic difference between groups.

The application to race is obvious: heritability is high within whites. But high heritability within groups licenses no conclusion about differences between groups.[8]

So we see that high heritability within groups does not dictate any genetic difference between groups. Nor does high heritability within groups dictate the *direction* of any genetic difference between groups. The stunted corn could have been genetically taller, with the genetic advantage outweighed by the environmental deprivation.

In reply to Lewontin's critique, Jensen focused on a principle very much like the one that underlies Herrnstein and Murray's reasoning. He said:

> The real question is not whether a heritability estimate, by its mathematical logic, can prove the existence of a genetic difference between the two groups, but whether there is any probabilistic connection between the magnitude of the heritability and the magnitude of group differences. Given two populations (A and B) whose means on a particular characteristic differ by x amount, and given the heritability (h^2_A and h^2_B) of the characteristic in each of the two populations, the probability that the two populations differ from one another genotypically as well as phenotypically is some monotonically increasing function of the magnitudes of h^2_A and h^2_B. (Jensen, 1970, p. 104)

This argument has been repeated many times, by Jensen and others.[9]

Now if we knew nothing at all about two groups, *except* that they differed by 15 points in IQ and that IQ has some heritability in both, and we had to *guess* the causes, for all I've said so far, it would make sense to guess that the lower scoring group was disadvantaged *both* genetically and environ-

[8] Herrnstein and Murray mention Lewontin's point, but they do so in a way that distorts its crucial features. They leave out the 100% heritability within groups. In their version of the example, two handfuls of "genetically identical seed corn" are planted, one in Iowa and the other in the Mojave Desert (*Bell*, p. 298). So the heritabilities within the groups are zero (or undefined if all the corn in the Mojave dies). Also, their statement of the point conflates genetic determination with heritability. They say "That a trait is *genetically transmitted in individuals* [emphasis added] does not mean that group differences in that trait are also genetic in origin" (p. 298). However, the effect of Herrnstein and Murray having mentioned Lewontin's point, even in a distorted way, is that the critics have ignored it. And the effect of that is that the audience doesn't know about it.

[9] The latest incarnation is an article in *The Wall Street Journal* (Arvey et. al., 1994) by 52 behavior geneticists and psychometricians which is intended to correct misinformation in the debate about *The Bell Curve*. They mention the high heritability of IQ within whites and they add: "The reasons that blacks differ among themselves in intelligence appear to be basically the same as those for why whites (or Asians or Hispanics) differ among themselves. Both environment and genetic heredity are involved." And they hint at a conclusion much like that of Jensen and of Herrnstein and Murray about a probable black genetic deficiency. Herrnstein and Murray, however, are more cautious in not making claims about heritability within blacks.

mentally. (In the last section of the paper I'll show that even this weak principle is wrong.) However, the principle has no application to the *actual* racial question because we know *more* than nothing: we know that the environment can have huge effects on IQ (e.g., the Flynn effect of 3 points per decade and the 21-point increase in Holland) and that blacks are environmentally disadvantaged in a way that has been shown to count. But without being able to *measure* the effect of being treated as subnormal and the effect of a legacy of slavery and discrimination from the past, how do we know whether its average effect is sufficient to lower black IQ 15 points, or less than that – or *more* than that? Given the social importance of this issue, *guessing* is not appropriate.

I said that if we knew nothing except that there is a difference between two groups in a characteristic and that the characteristic has some heritability in both groups, we could guess that the lower group was disadvantaged both genetically and environmentally. Of course the reasoning is equally good for what Plomin, DeFries and McClearn call "environmentality", the "converse" of heritability (i.e., the proportion of variation due to environmental differences in the population). The danger of such guess-work in the absence of an evaluation of the relative environments is illustrated by gender and height. Women are shorter than men and height has some heritability and some "environmentality" in both groups. But the guess that women are deprived in environmental variables that affect height would be wrong. And an evaluation of these environmental variables would save us from a wrong guess here.

It is worth emphasizing the solidity of the data about the large increases in IQ in Holland. The 21-point increase reported by Flynn is based on comprehensive testing of all Dutch 18-year-olds who pass a medical exam (and there has been no change in the pass rate). The test used is Raven's Progressive Matrices, a widely respected "nonverbal test that is an especially good measure of *g*" (*Bell*, p. 273). Even Richard Lynn, the arch-Jensenist who is the source of much of *The Bell Curve*'s data on race, concedes this point. He says "The magnitude of the increase has generally been found to be about three IQ points per decade, making fifteen points over a fifty year period. There have, however, been some larger gains among 18 year-old conscripts in The Netherlands and Belgium amounting to seven IQ points per decade." Lynn also mentions that similar results have been found in France (Lynn, 1992, p. 382.) Herrnstein and Murray concede that "In some countries, the upward drift since World War II has been as much as a point a year for some spans of years" (p. 308), In an area where the facts are often contested, it is notable that this set of facts seems to be accepted by both sides.

Another fact that is widely accepted by all sides is that when it comes to specific identified genes and their products (e.g., genes for blood types) only about 7% of all human genetic variation lies between the major races of Africa, Asia, Europe and Oceania (Lewontin, 1982). About 85% of this

genetic variation is within ethnic groups, and about 8% is between ethnic groups within a single race (e.g., between Spaniards and Italians).

Herrnstein and Murray have heard appeals to the legacy of slavery and discrimination. They have a response, and their response forms part of their argument that I have not yet mentioned. They appeal to the pattern and the magnitude of racial differences. First, the pattern. They remind us that the black/white IQ difference is smallest at the lowest socio-economic levels (see Fig. 2), and this leads them to ask:

> Why, if the B/W difference is entirely environmental, should the advantage of the "white" environment compared to the "black" be greater among the better-off and better-educated blacks and whites? We have not been able to think of a plausible reason. An appeal to the effects of racism to explain ethnic differences also requires explaining why environments poisoned by discrimination and racism for some other groups – against the Chinese or the Jews in some regions of America, for example – have left them with higher scores than the national average (*Bell*, p. 299).

But these facts are actually not hard to understand. Blacks and whites are to some extent separate cultural groups, and there is no reason to think that a measure like SES means the same thing for every culture. Herrnstein and Murray mention the work of John Ogbu, an anthropologist who has distinguished a number of types of oppressed minorities (Ogbu, 1986). A key category is that of "caste-like" minorities who are regarded by themselves and others as inferior, and who, if they are immigrants, are not voluntary immigrants. This category includes the Harijans in India, the Buraku and Koreans in Japan and the Maori in New Zealand. He

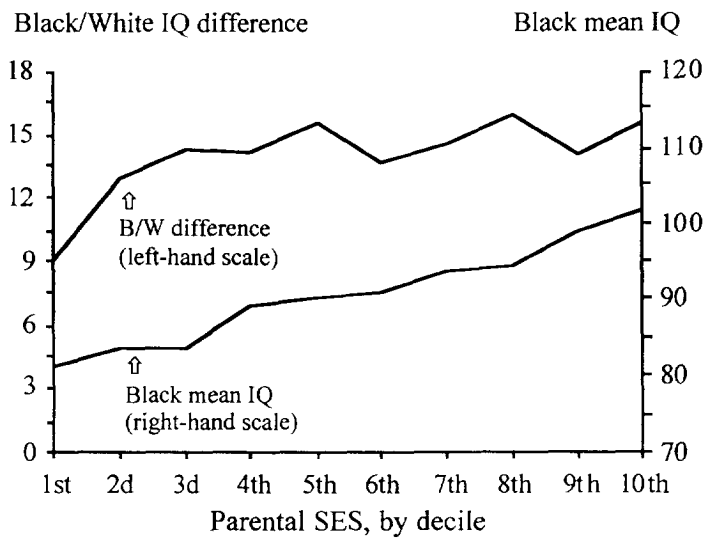

Fig. 2. The difference between average black and white IQ is smallest for the lowest 30% of SES. (Adapted from *Bell*, p. 288.)

distinguishes them from groups like Chinese and Jews who are voluntary immigrants and have a culture of self respect. If higher-SES blacks still are to some extent part of a caste-like minority then they will be at an environmental disadvantage relative to higher-SES whites. But low-status blacks and whites are more likely to share a caste background. Note in Fig. 2 that for the bottom 30% in SES, blacks and whites are more similar in IQ than for the top 70%. As Henry Louis Gates points out (Gates, 1994), affirmative action has had the effect of quadrupling the size of the black middle class since 1967. Most middle class blacks have arrived in the middle classes relatively recently, many of them under less than ideal conditions for the development of self respect. It would be surprising if children of these newly middle-class blacks were to have fully escaped their caste background in so short a time.

Ogbu notes that where IQ tests have been given "the children of these castelike minorities score about 10–15 points . . . lower than dominant group children". He notes further that differences remain "when minority and dominant group members are of similar socioeconomic background". But when "members of a castelike minority group emigrate to another society, the twin problem of low IQ test scores and low academic achievement appears to disappear" (Ogbu, 1986, pp. 32–33). Data suggest that the Buraku who have emigrated to this country do "at least as well at school and the work place" as other Japanese.[10]

That was the pattern: now let's move to the magnitude issue. Herrnstein and Murray calculate that "the *average* environment of blacks would have to be at the 6th percentile of the distribution of environments among whites. . . for the racial differences to be entirely environmental . . . differences of this magnitude and pattern are implausible", (p. 299). That is, 94% of whites would have to have an environment that is better for the development of IQ than the environment of the average black – if the 15-point difference is to be explained environmentally. Herrnstein and Murray think this is implausible because when you look at measures of the environment such as income of parents, quality of schools and the like, you do not find that 94% of whites have a better environment than the average black. But this calculation ignores the effect of being in Ogbu's category of a caste-like minority. Compare the Dutch 18-year-olds of 1982 with their fathers' cohort, the 18-year-olds of 1952. The difference is entirely environmental despite the probable substantial heritability within each group. Using the same procedures as Herrnstein and Murray, Flynn calculates that 99% of the 1982 group had to have a better environment for the development of IQ than the average member of the 1952 group (Flynn, 1987b).

[10] In a personal communication, Ogbu tells me that it is difficult to get data on Buraku immigrants to the USA partly because there are not very many of them and partly because no one wants to be identifed as a Buraku. One of the studies he cites in Ogbu (1986) was actually published under a pseudonym.

Given differences of this magnitude among people of a uniform culture who are separated by only a single generation, is it really so implausible that 94% of whites have an environment better than a black at the 50th percentile?[11]

Let me sum up the argument of this section:

(1) If we knew nothing at all about two groups *except* that they differed by 15 points in IQ and that IQ is moderately heritable in both, then, for all I've said so far, we could guess that the lower scoring group was disadvantaged both genetically and environmentally.
(2) We know that IQ has risen 3 points per decade in very many industrialized countries since World War II (the Flynn effect) and that in some countries IQ has risen 7 points per decade for three decades or more. These large changes in IQ (equal to or larger than the black/white IQ gap) cannot be due to any genetic change.
(3) We know that only 7% of identified human genetic variation is within races.
(4) It appears that when members of cast-like minorities emigrate, the IQ gap narrows or disappears.

Conclusion: environmental differences, including the sort that affect blacks in the USA, are known to have large effects on IQ. We have no way at present of quantifying this effect. So we should draw no conclusion about the probability of any black genetic IQ advantage or disadvantage.

4. Indirect heritabiliy

Earlier I said that genetic determination is not quantitative. Now I will explain why. Genetic determination is a species of causation. And causation is context-relative, as John Stuart Mill taught us.

To see this, imagine that a man is shot in the chest and dies. What caused his death? Of course he was shot, but, in addition, many other parts of the causal net – both conditions and events – were part of the mechanism by which the death was produced, e.g. bullet-permeable clothes, the intentions of the assassin, or (going further back in time) the invention of gunpowder. We do not usually think of such factors as causes, but in some contexts we do. You can cause someone's death by replacing some of the kevlar in his bullet-proof vest with cotton when you know he will be targeted by a sharpshooter who aims for the heart.

[11] Herrnstein and Murray have a number of other independent arguments for black genetic inferiority, some of which are directly relevant to the point about caste-like minorities. In particular, they claim that the IQ of blacks in African countries in which they are not an oppressed caste is lower than that of American blacks. I am not impressed with the data that they provide. See Kamin (1995) on this topic.

The *entire* causal net – the totality of factors that influence an event – is an objective matter, but which *parts* of the net count as "the cause" or even "a cause" are dependent on context. Notice that this context-relativity of what counts as a cause is no mere verbal matter. It really counts in ethics, law and politics. If you kill someone by replacing the kevlar in his vest with cotton, you can be convicted of murder. The bullet permeability of his vest caused his death. To quantify causation, to ascribe $X\%$ of the causation to the bullet and $Y\%$ to the bullet permeability of his vest, would require some way of standardizing contexts and interests, and then the notion would not be very useful for other contexts and interests.

But wait! Heritability is a causal notion too! It is the ratio of variation *caused by* genetic differences to total variation. So doesn't the same point apply to heritability? Why is heritability quantifiable? The answer is that the actual methods for measuring heritability involve the *tacit acceptance* of a uniform policy as to what will count as a cause and what will not that has nothing to do with our ordinary socially important ideas of causation and is often violently in conflict with them.

An example (Jencks et al., 1972) will help to make this point. Consider a culture in which red-haired children are beaten over the head regularly, but all other children are treated well. This effect will increase the heritability of IQ because red-haired identical twins will tend to resemble one another in IQ (because they will both have low IQs) no matter what the social class of the family in which they are raised. The effect of a red-hair gene on red hair is a "direct" genetic effect because the gene affects the color via an internal biochemical process. By contrast, a gene affects a characteristic *indirectly* by producing a direct effect which interacts with the environment so as to affect the characteristic (see Fig. 3). In the hypothetical example, the red-hair

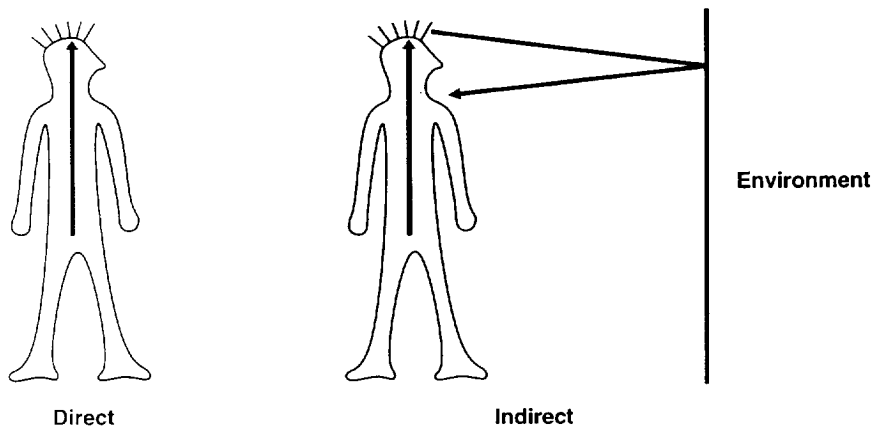

Fig. 3. A gene affects IQ directly via an internal biochemical process. A gene affects IQ indirectly by having a direct effect on something else (hair) which interacts with the environment so as to affect IQ.

genes affect IQ indirectly. In the case of IQ, no one has any idea how to separate out direct from indirect genetic effects because no one has much of an idea how genes and environment affect IQ. For that reason, we don't know whether or to what extent the roughly 60% heritability of IQ found in white populations is *indirect heritability* as opposed to direct heritability. (I coined the term "indirect heritability" many years ago (Block & Dworkin, 1974), but it is now sometimes called reactive heritability (Tooby & Cosmides, 1990).)

As I mentioned, we typically think of some but not other events or conditions in a causal chain as causes. The methodology used to measure heritability counts differences in characteristics as caused by genetic differences if there is a genetic difference, even if there is *also an environmental difference*, thus distorting the ways in which we normally think about causation. For instance, the heritability methodology focuses on the difference between the red-hair genes and genes for other hair colors, not on the fact that red-haired children – unlike blond children – are beaten.

Recall that earlier I said that wearing earrings used to be highly heritable because differences were "due" to the XY/XX difference. I put "scare quotes" around "due" because it is a by-product of the methodology for measuring heritability to adopt a tacit convention that *genes are taken to dominate environment*. Variation in ear-rings was as much a social matter as a genetic matter, but it still counted as highly heritable. If there is a genetic difference in the causal chains that lead to different characteristics, the difference counts as genetically caused even if the environmental differences are just as important. If we adopted the opposite convention, the convention that any environmental difference in two causal chains shows that the difference counts as environmentally caused, then we could not use current methodology for measuring heritability, because we have no general method of detecting indirect genetic effects using current techniques. Heritabilities using the two different conventions would be radically different if there are substantial indirect genetic effects.

Recall the examples mentioned earlier about the *measured heritabilities* of such quantities as number of hours of watching TV. No one should suppose that there is variation in genes for watching TV; this is a matter of indirect effects. Here is a good example of the never-never land of indirect heritability. A recent questionnaire study showed large heritabilities for many surprising "environmental" variables. (The method used was comparisons of one-egg and two-egg twins in non-adoptive environments. The more similar one egg twins are to one another compared to how similar two egg twins are to one another, the higher the heritability.) The twins were asked to indicate "things you have done during the past year". For "baby sat" the heritabilities were 35% for males and 47% for females. For "had your back rubbed", heritabilities were 92% for males and 21% for females (Schönemann & Schönemann, 1991, 1994). How could heritability be different for males and females? Suppose (just for illustration) that male

babysitters are chosen on the basis of weight and female babysitters are chosen on the basis of height. Height is more highly heritable than weight, so the indirect pathway would explain the difference. The result is *intelligible*, but it does show that heritability is a *strange* statistic.

Consider the fact that no one would do a heritability study on a mixed black/white population. I mentioned earlier that if you place a pair of black one-egg twins in different homes, you automatically fail to randomize environments, because the black twins will bring part of their environment with them; they are both black and will be treated as black. This is an indirect genetic effect *par excellence*. Implicitly, everyone in this field recognizes that, yet more subtle possibilities of indirect effects are typically ignored.

Recall that heritability is defined as a fraction: variation due to genetic differences divided by total variation. The measure of variation that is always used (though alternatives are available) is a statistical quantity known as variance. One factor that raises variance is a positive correlation between genetic and environmental variables. Consider, for example, a phenomenon of double advantage and double disadvantage (Jencks, et al., 1972). Suppose that children whose genes give them an advantage in musical talent tend to have parents who provide them with an environment conducive to developing that talent, music lessons, concerts, a great CD collection, musical discussion over dinner, etc. Suppose further that other children who have a genetic disadvantage also have an environrnent that stultifies their musical talents. Hence there will be a correlation between genes and environment that will move children towards the extremes of the distribution, increasing the variance in musical skills. Variance due to gene/environment correlation (gene/environment "covariance") should not be counted in the genetic component of the variance, and there are a variety of methods of separating out such variance. It is common in behavior genetics to distinguish among a number of different types of covariance (Loehlin, & DeFries, 1987; Plomin, DeFries, & Loehlin, 1977; Plomin, 1990a). The kind just mentioned in which parents provide genes for musical talent and an environment that develops it is called "passive", covariance because it doesn't depend on what the child does. Reactive covariance is a matter of the environment reacting to the child's qualities, as when a school gives extra music classes to musically talented children. Active covariance is a matter of the child creating a gene-environment correlation, as when a musically talented child practices musical themes in the imagination or pays attention to the musical environment. Passive covariance can be controlled in heritability calculations by attention to adoption studies in which the double-advantage/double disadvantage does not exist. But reactive and active covariance cannot be measured without specific hypotheses about how the environment affects IQ. And it is just *a fact about IQ that little is known* about how the environment affects it. So reactive and active covariance is on the whole beyond the reach of the empirical methods of our

era's "behavior genetics", for those methods do not include an understanding of what IQ *is* (e.g., whether it is information-processing capacity) or how the environment affects it.

Perhaps you think that my claim that no one understands very much about how the environment affects IQ would be hotly contested by behavior geneticists who agree with Herrnstein and Murray. *I doubt it!* Recall that earlier I mentioned that most of the environmental variation that affects IQ variation (in whites) is *within* families rather than *between* families. I mentioned the low correlations of adopted children raised in the same families. And I quoted Herrnstein and Murray agreeing that, of the environmental variation in IQ, "relatively little can be traced to the shared environments created by families. It is, rather, a set of environmental influences, *mostly unknown at present*, that are experienced by individuals as individuals." (*Bell*, p. 108; emphasis added.) Recall also that Herrnstein and Murray concede that no one has explained the Flynn effect.

The points about covariance just made assume that there are genes *for IQ* and that these genes may affect the environment so as to produce effects on IQ that are correlated with the ones that the genes themselves produce. But to think of the issue this way is to very seriously underestimate its significance. For as the red-hair example illustrates, indirect genetic effects needn't work through anything that should be thought of as "IQ genes".

Since we don't know much about how variation in environment differentially affects IQ, we can only guess about how variation in genes differentially affects IQ indirectly, via the environment. Suppose, for example, that a child's perceived attractiveness and self confidence strongly affect how adults interact with children in a way that largely accounts for the variation in IQ. Scarr and McCartney (1983), for example, say "It is quite likely that smiley, active babies receive more social stimulation than sober, passive infants. In the intellectual area, cooperative, attentive preschoolers receive more pleasant instructional interactions from the adults around them than uncooperative distractible children [p. 427]. . . . The social psychology literature on attractiveness . . . would seem to support our view that some personal characteristics evoke differential responses" (p. 433). Of course, adults could give some children more attention than others without making a difference to the children's IQs. That is, differential response on the part of adults does not show differential effect on IQ. But it could be so.

Suppose further that personal attractiveness and self confidence are highly heritable. Then we would have an indirect effect *par excellence*, and such an effect could, for all we know, largely account for the heritability of IQ. Without an understanding of how the environment affects IQ, we simply have no way of determining how much of the variance in IQ is indirect genetic variance of this sort. Of course, if we knew that some specific adult behavior that is triggered by some specific heritable property of children was responsible for a large component of IQ variation, then we could measure that behavior. But there is no theory of intelligence or IQ that would allow

us to have any synoptic grip on such factors. The point is underscored by the possibility that the differences in environment might be partly *internal* to the child's interaction with stimuli rather than a matter of differences in the stimuli themselves. As Scarr and McCartney (1983) note, "The toddler who has "caught on" to the idea that things have names and who demands the names for everything is experiencing a fundamentally different verbal environment from what she experienced before, even though her parents talked to her extensively in infancy" (p. 425).

This point is often (at least partially) acknowledged by behavior geneticists. For example, Plomin et al. (1977, p. 321) say: "Because it is not possible to measure all aspects of the environment (including everybody and everything) that might correlate with childrens' genotypes, it will probably never be possible to assess completely the effects of active and reactive genotype-environment correlations."

The upshot is that there may be a large component of heritability due to indirect genetic effects, including (but not limited to) gene–environment correlation, that is outside the boundaries of what can be measured given the mainly atheoretical approach that is available today. Where does the "gene–environment covariance" show up in heritability calculations? Answer: active and reactive effects that we don't know how to measure inevitably are included in the genetic component. This is often regarded by behavior geneticists as perfectly OK. In an often quoted passage, Roberts (1967) says:

> The genotype may influence the phenotype either by means of biochemical or other processes, labelled for convenience as "development", or by means of influencing the animal's choice of environment. But this second pathway, just as much as the first, is a genetic one; formally it matters not one whit whether the effects of the genes are mediated through the external environment or directly though, say, the ribosomes." (p. 218)

Jensen (1973) notes "Generally, CovGE [i.e., gene–environment covariance] is included in h^2 [heritability] . . . either on the assumption that the covariance is due to the genotype and/or because the particular method of estimating h^2 does not permit separation of V_G [genetic variance] and CovGE" (p. 369). Indeed, Jensen argues that "much if not most, of this effect should be included in the genetic variance, because, in part, r_{GE} [the correlation of genes and environment] is a result of the genotype's selective utilization of the environment" (p. 54). In practice, if researchers were actually to identify an "un-meritocratic" effect such as the red-hair indirect effect mentioned earlier, they would no doubt ignore Roberts' advice, counting the variance produced by the effect as covariance rather than genetic variance. Of course, we have *no idea* how much of the 60% of the variance in IQ that is said to be genetic is of this sort. So in actual practice, covariance due to indirect effects that people actually know how to measure – at least if it is flagrantly non-meritocratic – is not counted in the heritability. But other indirect effects are counted as genetic. So what counts

as genetic variance (inflating heritability) is a matter of *value judgements* and a matter of what effects we actually know about. Surely this makes heritability a lousy scientific concept.

In effect, the field has adopted as an axiom that *heritability of IQ can be measured by current methods*. Without this assumption, the right conclusion would be that since we cannot separate indirect genetic effects (including certain kinds of gene/environment covariance) from pure genetic variance, no heritability estimate can be made. Why does the field adopt this axiom? I cannot help thinking that part of the explanation is that behavior genetics is a young field (see Plomin, 1990a) struggling for acceptance and funding, and heritability is a flag that attracts attention to it.

Let us return to the speculation mentioned above that the 60% heritability of IQ (within whites) is entirely indirect and due to differential treatment of children on the basis of heritable characteristics. Then the direct heritability of IQ would be zero and we would have no reason to think that anything that could be called genes *for* IQ (e.g., genes for information-processing capacity) vary in the white population, and no reason to look for such differences to explain the 15-point difference between blacks and whites.

Indeed, we would have reason to look for differences in the ways adults interact with children to explain the black–white IQ difference. So indirect heritability suggests an environmental hypothesis about the measured black–white IQ difference, maybe one that could be the object of social policy. Are there reasons to expect indirect genetic effects in the black-white difference? I mentioned the obvious example of genes for skin color above. But there may be less obvious indirect effects as well. I mentioned earlier that there are many more low birth weight black babies than white babies. Nothing known appears to rule out a genetic explanation (Lieberman, 1995). If blacks are more likely to have genes for low birth weight babies, perhaps the effect could be neutralized by diet or by drug intervention in pregnancy. Certainly, no one should think of genes for low birth weight as "IQ genes".[12]

Let us return to the topic of the last section, the issue of whether heritability within groups yields any probability judgement about the genetic differences between groups. I commented that (for all I'd said so far) if we knew nothing at all about two groups *except* that they differed by 15 points in IQ and that IQ is heritable in both, and we had to *guess* the causes, it would make sense to guess that the lower scoring group was disadvantaged

[12] This is an oddball case but I still count it as an an indirect genetic effect. The mother's genes produce a direct effect on her reproductive system that has an additional effect on the environment of the baby. It should be noted that the direct/indirect distinction, as with any distinction involving causation, is itself context-relative. All direct genetic effects involve the environment in some way (if the environment had been different in the appropriate way the effect would have been different) so in some contexts such environmental dependencies can make the effect indirect.

both genetically and environmentally. But the points I've just made about indirect heritability show that any such guess would be misguided. The reasoning behind the guess assumed that the heritability of IQ within whites reflected differences in IQ caused by differences in *IQ genes*. But what the points about indirect heritability show is that we don't know whether *any* of the variation within whites is due to variation in IQ genes. If we have no real grip on what kinds of causal mechanisms result in the 60% heritability within whites, we can have no confidence in any extrapolation to blacks. Here is a very closely related point: the Jensen–Herrnstein–Murray reasoning assumed that there was a well/defined space of alternative genotypes for IQ that vary within whites and that can be used to apportion probabilities. Any reasoning about probability in the absence of data requires some way of dividing the possibilities into equi-probable alternatives. But what the direct/indirect distinction reveals is that the well-behaved space of IQ genotypes that would be required for such reasoning cannot be assumed to exist.

Let's call a person's genome (his total set of genes) genetically inferior with respect to IQ if that genome yields low IQ in any normal environment. But what is to count as a normal environment? In the example discussed earlier, genes for red hair yield low IQ within environments that are normal in the environment of the hypothetical society, but in environments that we would consider normal, the red-hair genes are irrelevant to IQ. What if the heritabilities observed for IQ are a result of indirect effects that can be changed by changing social practice? Then phrases like "genetically inferior in IQ" and "genetic disadavantage in IQ" will only apply to genomes such as that of Down's syndrome that yield low IQ no matter what the social practices.

Another consequence of the point about indirect heritability is to cast doubt on Herrnstein and Murray's ideas about genetic social stratification within whites based on IQ. If the 60% heritability does not reflect IQ genes, then there is no reason to suppose that social classes differ at all in IQ genes. Herrnstein and Murray worry about pollution of the gene pool by immigrants and by large numbers of children of low-IQ parents. But if the heritability of IQ is mainly indirect, their emphasis on genes is misdirected. If we lived in a culture that damages the brains of red-haired children, should we complain about genetic pollution when large numbers of red-haired immigrants arrived? No, we should try to change the social practices that deprive those with certain genes of an equal chance.[13]

[13] The ideas in this section were developed from my contribution to Block and Dworkin (1974, section 1) in lectures that I gave in the 1970s, and in part stimulated by Jencks et al. (1972). While this paper was in press I read Jencks (1980), which makes a number of overlapping points.

5. Genetic bucket theory

Though IQ is not genetically determined, something close to it might be said to be genetically determined: namely *potential IQ*. The genes, it might be said, determine the size of the mental bucket and then the environment fills the bucket to one level or another. (Herrnstein and Murray are far from alone in talking this way. Vernon, 1969, for example, introduced the term "intelligence A" for the size of the bucket.)

To see what is wrong with this idea, and also to see why heritability is much less relevant to social policy than many have supposed, we need the concept of a *norm of reaction*. Suppose that of the many plants growing on a mountain, some do well at high altitudes but poorly at low altitudes, and others are exactly the opposite. We could graph the situation as in Fig. 4. One type of plant, genotype G_1 thrives at low altitudes (as measured by the weight of the plant) and the other thrives at high altitudes. The norm of reaction is simply the function from environment to phenotype for a specific genotype. Two norms of reaction, one for each type of plant, are illustrated in the diagram.

What is most truly genetically determined is the norm of reaction for a trait rather than any specific trait, as every population geneticist knows. The concept of the norm of reaction allows us to see that heritability data can tell us nothing about the genetic bucket theory. Consider some *hypothetical* norms of reaction for IQ as in Fig. 5. (Note: $G_1$and G_2 are not intended as a guess about any actual races.)

One point illustrated is that current environments may have nothing to do with anything that could be thought of as *maximum* capacity, the potential size of the genetic bucket. That might be a ceiling we bump up against only in environments very different from ones available to us now (or perhaps

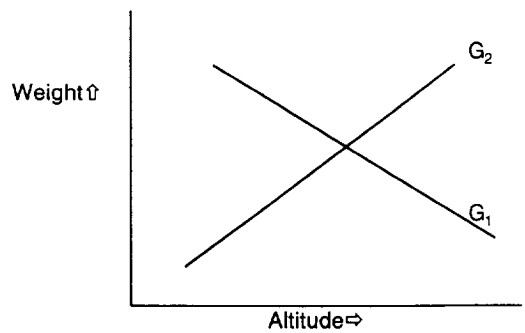

Fig. 4. One genetic type of plant does better at high altitudes, another does better at low altitudes

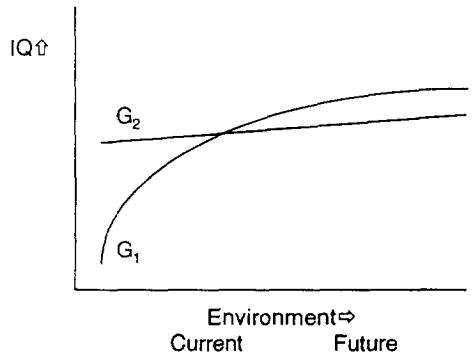

Fig. 5. Two hypothetical genotypes show a large difference in IQ in current environments, but the difference decreases and the order changes in another environment.

there is no ceiling at all). If this seems unlikely to you, remember the Flynn effect. The IQ gap between us now and ourselves and our relatives of 1950 is about the same as between blacks and whites now. Another point that doesn't depend on IQ: when Gödel's theorem and general relativity were young, it was said that there were only a handful of people smart enough to understand them. Now any reasonably industrious and with-it college undergraduate has a good grasp of both within his reach. The change is improved understanding on the part of the intellectual community which allows difficult ideas to be made digestible. A similar point could be made with respect to athletic skills. The techniques (diet, shoes, training regime) required to run a 4-minute mile put an achievement within the reach of many runners that was not available to the very top runners of a hundred year ago.

A second point illustrated by the graph is a much more significant one. Consider a population clustered on the left-hand side in the current environments section. If the population is equally split between G_1 and G_2, we can expect substantial heritability. But as the environment improves and moves into future environments, we get the following results: heritability decreases, IQ increases and G_1 and G_2 reverse. The point of course is not that anyone knows that this is what norms of reaction for IQ look like. The point is just the opposite – that no one does know how any human genotypes may react to environments that involve new intellectual machinery, new software for the brain. As Lewontin (1974; see also Feldman & Lewontin, 1975) has emphasized, heritability gives a local analysis that depends on the environmental conditions and the distribution of genotypes that happens to exist at a given time. It tells us nothing about the effect of introducing environments that are either new or not now common. If we want to know the effects of a new or uncommon environment, we should *ignore heritability* and just try changing the environments. This is yet another reason why heritability is a bad statistic.

A third point (closely connected to the previous two) is that the talk of genetic advantage and disadvantage that I allowed at the outset of this paper is problematic. Even if some genotypes determine lower IQs than others in some environments, that tells us nothing about what will happen in other environments. Perhaps in ideally favorable environments most genotypes yield more or less the same phenotype. Talk of part of the black/white difference being genetic is even more problematic. We could try to make sense of the notion of part of an IQ difference being genetic as follows. Suppose there is a 10-point IQ difference between you and me. If we were both raised in an average environment, the difference would have been only 2 points. Then we might be tempted to say that the environmental part is 80% of the difference and the genetic part is 20%. But as we can see from the curves of the last figure, if our actual environment is far from the average, what happens at the average environment may have little or nothing to do with the actual causes of our IQ difference. Indeed, you may have a "genetic advantage" in our actual environments, but I may have a "genetic advantage" in the average environment. Further, any statistic that depends on the average environment has the problem of the heritability statistic – that it provides a local analysis that may have nothing to do with environments outside the current range.

Genetic determinists often react to data like those that I mentioned earlier on the large IQ changes in Holland by saying that though IQ scores are very environmentally malleable, the relative positions of individuals and groups are not. In effect, what they are supposing is that the norms of reaction are always parallel. There is some evidence for this within a very narrow range of environments of the current correlational studies. (See Plomin, 1990b for the standard behavior genetics point of view on this, but see also Wahlsten, 1990 for a discussion of problems with this point of view.) But there is no evidence at all outside that range. I think the right thing to say is that we don't know what the shape of the norms of reaction are, and if we are curious we should ignore heritability statistics and simply *try out improved environments*.

Further, it is not difficult to think of possiblities in which norms of reaction for IQ might not be parallel. Suppose, for example, that the brain is in a certain respect like a muscle: the activity of abstract thinking makes dendrites sprout whereas not thinking makes them shrivel. Then the more one *does* think, the better one *can* think. (Perhaps this would help to explain why college graduates appear to be at lower risk for Alzheimer's disease.) Then over the long run, differences in confidence and motivation will make for differences in ability. If there are environments that encourage some genotypes but discourage others, then we could expect to see norms of reaction for cognitive abilities that are far from parallel (and to the extent that IQ tests tap cognitive abilities, we could expect to see the same for IQ norms).

I mentioned earlier Kitcher's observation that though it was once said that

PKU was a form of genetic retardation, we now feel that what is genetic in the case of PKU is the inability to metabolize phenylalanine properly. More generally, the more we know about the mechanisms of causation, the more we tend to see that what is genetic is not any specific phenotypic characteristic, but rather a tendency to develop it in certain environments. The notion of a norm of reaction allows us to see why that is the right way to think. Generally, what is genetic is simply a mapping from environment to actual (phenotypic) characteristics.

I would like to end with a brief comment on affirmative action. Herrnstein and Murray suppose that affirmative action policies depend on an assumption of genetic equality. But the main justifications for affirmative action do not so depend. Affirmative action is justified as a remedy for current discrimination, to make up for past discrimination and for the provision of role models. Issues about a genetic involvement in race differences have no relevance to these justifications.

Acknowledgements

I am grateful to the following people for their comments on earlier drafts: Alex Byrne, Susan Carey, Noam Chomsky, Josh Cohen, Delia Graff, Ned Hall, Michael Hardimon, Paul Horwich, Emily Jenkins, Johanna Jenkins, Philip Kitcher, Leonard Katz, Peter Klein, Amy Lang, Richard Lewontin, Karen Neander, Charles Nesson, Wayne O'Neil, Steven Pinker, Georges Rey, Sandra Scarr, Elliott Sober, Judith Thomson and an anonymous referee for this journal.

References

Arvey, R., et al. (1994, December 13). Mainstream science on intelligence. *Wall Street Journal.*

Block, N., & Dworkin, G. (1974). IQ, heritability and inequality. Part II. *Philosophy and Public Affairs, 4*(1), 40–99. Reprinted in Block & Dworkin (1976).

Block, N., & Dworkin, G. (1976). *The IQ Controversy.* New York: Pantheon.

Chomsky, N. (1975). On cognitive capacity. In *Reflections on Language* (ch. 1). New York: Pantheon. Reprinted in N. Block (1981) *Readings in Philosophy of Psychology* (Vol. 2, pp. 305–323). Harvard University Press: Cambridge.

Feldman, M., & Lewontin, R. (1975). The heritability hangup. *Science, 190,* 1163–1168.

Flynn, J.R. (1987a). Massive IQ gains in 14 nations: What IQ tests really measure. *Psychological Bulletin, 101,* 171–191.

Flynn, J.R. (1987b). Race and IQ: Jensen's case refuted. In S. Modgil & C. Modgil (Eds.), *Arthur Jensen: consensus and controversy* (pp. 221–232) Falmer Press: London.

Fodor, J., Bever, T. & Garrett, M. (1974). The specificity of language skills. In J. Fodor, T. Bever, & M. Garrett (Eds.), *The Psychology of Language* (pp. 436–441, 449–462). McGraw-Hill: New York. Reprinted in N. Block, *Readings in Philosophy of Psychology* (Vol. 2, pp. 324–338). Harvard University Press: Cambridge.

Gates, H.L., Jr. (1994, October 31). Why now? *The New Republic, 211,* no. 18, issue 4163, p. 10.

Gould, S.J. (1994). Curveball. *The New Yorker, LXX*, 139–149. Reprinted in S. Fraser (Ed.), *The Bell Curve Wars* (1995, pp. 11–22), Basic Books: New York; and in R. Jacoby and N. Glauberman (Eds.), *The Bell Curve Debate* (1995, pp. 3–13), Random Books: New York.

Herbert, R. (1994, October 26). Throwing a curve. *New York Times*. op.-ed. page.

Herrnstein, R.J., & Murray, C. (1994) *The Bell Curve*. New York: Free Press.

Hofer, M. (1994, December 26). Behind the curve. *New York Times*, op.-ed. page.

Holt, J. (1994, October 19). Anti-social science. *New York Times*, op.-ed. page.

Jencks, C., Smith, M., Acland, H., Bane, M., Cohen, D., Gintis, H., Heyns, B., & Michelson, S. (1972). *Inequality: a Reassessment of the Effect of Family and Schooling in America*. New York: Basic Books.

Jencks, C. (1980). Heredity, environment and public policy reconsidered. *American Sociological Review*, 45, 723–736.

Jensen, A.R. (1969). How much can we boost IQ and scholastic achievement? *Harvard Educational Review*, *39*, 1–123.

Jensen, A.R. (1970). Race and the genetics of intelligence: a reply to Lewontin. *Bulletin of the Atomic Scientists*. Reprinted in Block & Dworkin (1976), 93–107.

Jensen, A.R. (1973). *Educability and Group Differences*. New York: Harper & Row.

Kamin, L. (1995). Lie, damned lies, and statistics. In R. Jacoby and N. Glauberman (Eds.), *The Bell Curve Debate* (pp. 81–105). Random House: New York.

Kitcher, P. (1996). Fascinating genetalk. In *An Unequal Inheritance* (Ch. 11). New York: Simon & Schuster.

Leo, J. (1994, October 24). Why IQ isn't destiny/The return of the IQ wars. *US News and World Report*, 73–80/24.

Lewontin, R. (1970). Race and intelligence. *Bulletin of the Atomic Scientists*, *26*(3), 2–8. Reprinted in Block & Dworkin (1976).

Lewontin, R. (1974). The analysis of variance and the analysis of causes. *American Journal of Human Genetics*, *26*, 400–411. Reprinted in Block & Dworkin (1976).

Lewontin, R. (1982). *Human Diversity*. Scientific American Library: New York.

Lieberman, E. (1995). Low birth weight: not a black-and-white issue. *New England Journal of Medicine 332*, 117–118.

Loehlin, J. & DeFries, J. (1987). Genotype–environment correlation and IQ. *Behavior Genetics 17* (3), 263–277.

Lynn, R. (1992). Lynn replies to Flynn. In J. Lynch, C. Modgil & S. Modgil (Eds.), *Cultural Diversity and the Schools*. Falmer Press: London.

Murray, C. (1994, December 2). The real Bell curve. *Wall Street Journal*.

Murray, C. (1995, January 30). Letters to the editors. *The New Republic*, 5.

Nisbett, R. (1995). Race, IQ and scientism, S. Fraser (Ed.), *The Bell Curve Wars* (pp. 36–57). Basic Books: New York.

Ogbu, J. (1986). The consequences of the American caste system. In U. Neisser (Ed.), *The School Achievement of Minority Children: New Perspectives* (pp. 19–56). Hillsdale, NJ: Lawrence Erlbaum.

Pedersen, N.L., Plomin, R., Nesselroade, J., & McClearn, G. (1992). A quantitative genetic analysis of the cognitive abilities during the second half of the life span. *Psychological Science*, *3*, 346–353.

Plomin, R. (1989). Environment and genes. *American Psychologist*, *44*, 105–111.

Plomin, R. (1990a). *Nature and Nurture*. Brooks-Cole: Pacific Grove, CA.

Plomin, R. (1990b). Trying to shoot the messenger for his message. *Behavioral and Brain Sciences*. *13*, 144.

Plomin, R., & Bergeman, C. (1991). The nature of nurture: genetic influence on "environmental" measures. *Behavioral and Brain Sciences*, *14*, 373–386.

Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences 10*, 1–60.

Plomin, R., DeFries, J., & Loehlin, J. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*, 309–322.

Plomin, R., DeFries, J., & McClearn, G. (1990). *Behavioral Genetics: a Primer*. Freeman: New York.

Plomin, R., Loehlin, J., & DeFries, J. (1985). Genetic and environmental components of "Environmental Influences". *Developmental Psychology 21*, 391–402.

Roberts, R.C. (1967). Some concepts and methods in quantitative genetics. In J. Hirsch (Ed.), *Behavior–Genetic Analysis* (pp. 214–257). McGraw-Hill: New York.

Scarr, S. (1995). Human differences and political equality: the dilemma of group differences. *Issues in Science and Technology*, in press.

Scarr, S., & McCartney, K. (1983). How people make their own environments: a theory of genotype→ environment effects. *Child Development 54*, 424–435.

Schönemann, P., & Schönemann, S. (1991). We wondered where the errors went. *Behavioral and Brain Sciences 14*, 404–405.

Schönemann, P., & Schönemann, S. (1994). Environmental versus genetic models for Osborne's personality data on identical and fraternal twins. *Cahiers de Psychologie Cognitive/European Bulletin of Cognitive Psychology*, *13*, 141–167.

Sober, E. (1988). Apportioning causal responsibility. *Journal of Philosophy*, *85*, 303–318.

Sober, E. (1993). *Philosophy of Biology*. Westview: Boulder, CO.

Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: the role of genetics and adaptation. *Journal of Personality 58*, 17–67.

Vernon, P. (1969). *Intelligence and Cultural Environment*. London: Methuen.

Wahlsten, D. (1990). Insensitivity of the analysis of variance to heredity–environment interaction. *Behavioral and Brain Sciences*, *13* 109–120.

Wright, R. (1995, January 2). Has Charles Murray read his own book? *The New Republic*, 6.