Heritability and Indirect Causation

Neven Sesardic†‡

Genetic differences can lead to phenotypic differences either directly or indirectly (via causing differences in external environments, which then affect phenotype). This possibility of genetic effects being mediated by environmental influences is often used by scientists and philosophers to argue that heritability is not a very helpful causal or explanatory notion. In this paper it is shown that these criticisms are based on serious misconceptions about methods of behavior genetics.

Does the fact that a given phenotypic trait is heritable entail that it is genetic (i.e., that the differences in that trait are due to genetic differences)? Many influential social scientists and philosophers (Christopher Jencks, Ned Block, Elliott Sober, Allan Gibbard and others) give a negative answer to that question. They all support this answer by using an essentially same example, which originates from Jencks et al. (1972).

Imagine that red-haired children are for some reason singled out for abuse and are frequently hit on the head by parents and teachers. As a result they get a lower IQ on average than other children. Now, although it is in a certain sense true that in this situation having a gene for red hair leads to a lower IQ, it just doesn't sound right to say that the IQ deficit of red-haired children is genetic. For, the manifestly critical influence here is the environmental one (the abuse), which explains the deficit, and furthermore explains it *completely*. Jencks and his followers claim that according to the way "heritability" is used in behavior genetics, the IQ difference between red-haired and other children in this hypothetical example would be heritable and counted as genetic. Since this strongly conflicts with common sense (which regards that difference as 100 percent environmental) they conclude that heritability is a somewhat Pickwickian causal notion,

†To contact the author, please write to: Department of Philosophy, Lingnan University, Hong Kong; e-mail: sesardic@ln.edu.hk.

‡The work on this paper was supported by a grant from the Research Grants Council of the Hong Kong Special Administrative Region, China (Project No. LU3005/01H).

Philosophy of Science, 70 (December 2003) pp. 1002–1014. 0031-8248/2003/7005-0012$10.00
Copyright 2003 by the Philosophy of Science Association. All rights reserved.

https://doi.org/10.1086/377384 Published online by Cambridge University Press

and that for that reason, the heritability of a trait by no means implies that the trait is also genetic in the important sense of that word which we ordinarily use to divide causal responsibility between genes and environments. Here are several characteristic passages:

> If, for example, a nation refuses to send children with red hair to school, the genes that cause red hair can be said to lower reading scores. . . . Attributing redheads' illiteracy to their genes would probably strike most readers as absurd under these circumstances. Yet that is precisely what traditional methods of estimating heritability do. If an individual's genotype affects his environment, for whatever rational or irrational reason, and if this in turn affects his cognitive development, conventional methods of estimating heritability attribute the entire effect to genes and none to environment. (Jencks et al. 1972, 66–67)

> [I]t is a by-product of the methodology for measuring heritability to adopt a tacit convention that *genes are taken to dominate environment*. . . . 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. (Block 1995, 117)

> But the methods used to assess the heritability of IQ automatically count variance produced by genetic variation as genetically caused variance even if it is also environmentally caused (Block and Dworkin 1976, 480)

> [I]magine [that] blue-eyed children are fed to lions, but some of them survive, maimed. If eye color is inherited and this grim ritual is the predominant cause of anyone lacking a leg in that population, then non-two-leggedness, in that population has substantial heritability. (Gibbard 2001, 169)

> If blacks are badly treated because of their skin color, and their skin color is genetic, then the lower IQ will be assigned to genes, not to environment. (Sober 2001, 74; cf. Sober 2000, 366)

For further examples, see also Levins (in Callebaut 1993, 249–250); Jencks 1980, 730; Garfinkel 1981, 119–120; Jencks 1992, 106; Taylor 2001, 179; Wachbroit 2001, 39; Moore 2001, 46.

Summarizing his views on the heredity-environment controversy, Jencks (1980, 731) gave a two-by-two table containing a taxonomy of various kinds of phenotypic effects. For our present purposes it is worth noting that differences resulting from sexual discrimination are classified there as being—*genetic!* These differences are of course usually regarded as being purely environmental in origin. The surprising outcome of labelling them as

https://doi.org/10.1086/377384 Published online by Cambridge University Press

genetic directly follows from Jencks's interpretation of heritability, according to which any variation ultimately caused by genetic variation is heritable (and hence genetic).

Clearly, sexual discrimination is initially triggered by a genetic difference. Roughly, there are four factors in the causal sequence: (1) two X chromosomes → (2) being female → (3) being discriminated against → (4) being less paid. In this scenario, the causal link between (2) and (3) stands out for us so saliently as the *nervus explanandi* of the observed income difference that any account mentioning only factor (1) would strike us as a joke, stupidity, or perhaps just a nonstandard causal attribution. And this is exactly the point that Jencks, Block, Sober and others want to make: If the logic of heritability is so permissive that it counts even consequences of sexual or racial discrimination as "genetic" effects, this means that heritable differences (in the technical and counterintuitive sense of behavior genetics) are not necessarily genetic effects *in our usual sense of the word*. Conclusion: Heritability estimates, whatever their magnitude, are never by themselves evidence that genes play an important explanatory role simply because even variation that is 100 percent heritable (i.e., ultimately due to genetic differences) may well be 100 percent proximately explained by much more relevant environmental differences.

I will try to show that far from being so semantically perverse, the term "heritability," when properly understood, actually accords quite well with our common-sense etiological ascriptions. Before embarking on this task, however, I have to do some preliminary definitional work, and explain three different kinds of genotype-environment correlation (or covariance):[1] passive, reactive, and active. These terms were introduced in Plomin et al. 1977, although a very similar distinction was informally made earlier and often discussed through examples.

Genotype-environment correlation is present when organisms with a given genotype tend to find themselves more often in one type of environment than do organisms with another genotype. I will follow the usual practice and illustrate the three kinds of G-E covariance with hypothetical and nontendentious examples involving IQ. First, if parents with higher IQ give to their children *both* genes for higher IQ *and* intellectually more stimulating environment at home, this is passive G-E correlation. It is called "passive" because neither the children's behavior nor their genotype is a causal factor that could account for the correlation. Second, if other people react to children with genotypically higher IQ by, say, imposing on them more intellectually demanding conversations and otherwise challenging their ability even further, this is reactive (or evocative) G-E correlation. Finally, active G-E correlation occurs when brighter children

1. The correlation is standardized covariance.

https://doi.org/10.1086/377384 Published online by Cambridge University Press

themselves seek and eventually select those experiences and environments that they find specially stimulating.

Passive G-E correlation is not relevant for the Jencks-Block-Sober argument, as they all acknowledge (Jencks 1980, 725; Block 1995, 118; Sober 2001, 73). In this case, the common cause of the children's double (dis)advantage (genetic and environmental) is the parental genotype, which produces the environmental effect on children through the parental phenotype, and the genetic effect through meiosis and conception. So, the two (dis)advantages accrue to children in a way that is not mediated by their own genotype at all. One of the reasons why the critics of heritability exclude passive covariance from their consideration is that the presence of this kind of G-E covariance is relatively easy to test with traditional methods of behavior genetics (the adoption design), and there is consequently little temptation to treat this recognizably separate source of variance analytically as a component of heritability.

What about active and reactive G-E covariance? Well, behavior geneticists are indeed inclined to subsume some instances of the former under genetic variance. However, they are reluctant to do the same with the latter, particularly when it comes to those types of reactive covariance that would make the notion "heritability" misbehave conceptually in a way described by Jencks, Block, and Sober. (In fact, the red-haired example is not the case of G-E correlation in the strict ANOVA sense, because there are no separate contributions to phenotypic variance from genes and environment, but I will ignore this subtlety here.)

In the case of active G-E correlation, the environments that lead to a phenotypic difference are *selected* by subjects themselves, whereas in reactive correlation they are *imposed* by others. Under some circumstances this distinction may affect the way we decide to apportion causal responsibilities. Namely, the influences of those environments that are *chosen* on the basis of genotype are typically difficult to keep apart from the influence of genotype itself. In many instances the selection of these environmental influences can be plausibly regarded as just a way a genotype is expressed, and hence as "a more or less inevitable result of genotype" (Jinks and Fulker 1970, 323). Therefore, phenotypic effects of such environments are indeed sometimes classified as heritable, on the grounds that they are practically inseparable from direct genetic effects and that they merely represent the self-realization of genotype (Jinks and Fulker 1970, 323; Jensen 1969, 39; Jensen 1973, 54, 368; Jensen 1976, 92–93; Rowe 1994, 90–92). In contrast, the key illustrations used by Jencks and others (red-haired children, sexual and racial discrimination) all involve reactive covariance in which environments are *arbitrarily imposed* from the outside. Now, the assertion that behavior genetics incorporates environmental influences into genetic variance *in this sort of situation* (imposed environments) surely

https://doi.org/10.1086/377384 Published online by Cambridge University Press

cannot be justified by merely giving examples where behavior genetics does that in a *different* type of situation (in cases of active G-E correlation, i.e., selected environments).

I have to forestall a possible misinterpretation here. I do *not* want to suggest that active and reactive covariance differ intrinsically from one another with respect to their causal status, whereby, as it were, active G-E covariance should as a matter of principle be always treated as a part of genetic variance, whereas reactive G-E covariance ought never to be subsumed under it. Rather, my claim is that G-E covariance is in the first place a source of variance that is *sui generis* and, as such, distinct from either the genetic or the environmental component of phenotypic variance (because G-E covariance involves *both* genetic and environmental influences). However, in certain specific situations researchers may wish to include the G-E covariance into genetic variance, but when they do that, they are typically guided by common-sense notions about causal attributions, rather than going against them. Namely, in some cases of active G-E covariance, G leads to E, which in turn leads to P, and all this unfolds in such a way that the genotype-environment correlation strikes us as just a self-actualization (or natural manifestation) of the genotype. Consequently, some behavior geneticists do tend to interpret phenotypic differences arising in this manner as resulting from genetic differences (that is, as being heritable) simply because they think that in that type of situation the role of the environment degenerates into its being a mere reflection of the genotype, or the way the genotype expresses itself: "To what extent could we ever get a dull person to select for himself an intellectually stimulating environment to the same extent as a bright person might?" (Jinks and Fulker 1970, 323)

The red-haired children example is totally different. In the cases of such blatant discrimination and abuse it would be obviously impossible to use the "self-realization" argument to incorporate the ensuing phenotypic variation into genetic variance. Actually, I am unaware that any serious scholar ever defended the idea that the indirect effects of Jencks-type scenarios should be treated as heritable.[2] On the contrary, a prominent behavior geneticist, a coauthor of the first behavior genetics

2. True, Loehlin et al. (1975, 87–88), discussing a fictional example of discrimination on the basis of eye color, say that since there the phenotype can be predicted from the knowledge of genotype, "in that sense it is not entirely incorrect to speak of a high heritability of this trait." However, the phrase "not entirely incorrect" clearly indicates that they do regard this way of speaking as misleading (it is incorrect, but not entirely!), and that they are uncomfortable with describing a phenotypic difference arising in this way as "heritable" or "genetic." Indeed, they consistently put scare quotes around these expressions in that context.

https://doi.org/10.1086/377384 Published online by Cambridge University Press

textbook, resolutely rejected the proposal, addressing this very issue head-on:

> In our human societies discriminatory practices are often based upon superficial physical characteristics or upon cultural stereotypes. In these instances a G-E correlation will result if, and only if, the criterion for discrimination is heritable in a genetic sense. If the criterion is a superficial physical trait (skin pigmentation, for example) it is of trivial behavioral interest. *Any correlation between it and behavior is logically attributable to environmental influences*. (Fuller 1979, 472; italics added)

In the face of this explicit repudiation of the Jencksian construal of heritability by the leading authority in the field, the reader will surely wonder whether the critics actually offered any textual support for their "paradoxical" reading of that crucial concept of behavior genetics. As a matter of fact, there has been surprisingly little effort to document the charge with quotations from relevant sources. To make things worse, even in those few rare cases where the attempt was made to provide evidence, a closer look into these "probative" texts always reveals that they were taken out of context and seriously misinterpreted. Here are some of these exegetical miscarriages.

Both Jencks and Block make much of the following quotation from R. C. Roberts: "it matters not one whit whether the effects of the genes are mediated through the external environment or directly, through, say, the ribosomes" (Roberts 1967, 218). On the face of it, the quoted sentence does seem to express the view that it makes absolutely no difference for behavior-genetic analysis how genes cause phenotypic differences, the only important thing being that genes are the ultimate causes. While Jencks quotes just that statement in isolation, Block gives a fuller version, and ironically the context he thereby supplies clearly reveals that even Roberts does not think that anything ultimately caused by genes should be automatically treated as a genetic effect. Here is the fuller version:

> 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, through, say, the ribosomes. (Block 1995; italics added)

Roberts considers here just two kinds of genetic influence on phenotype, the one internal to the organism and the other via the animal's *choice*

https://doi.org/10.1086/377384 Published online by Cambridge University Press

of environment. Apparently, the latter is *not* meant to include *any* indirect causation that starts with genotype and also involves environment, but only processes that involve genotype-*selected* environments (i.e., active G-E correlation). In other words, a more careful reading of Roberts's statement shows that it is restricted in content and that it does not apply to the cases of *imposed* environments at all. Therefore, it cannot be used (as it is by both Jencks and Block) to impute to behavior geneticists the counter-intuitive interpretation of heritability, according to which even effects of blatant discrimination would be heritable.[3] In Roberts's article there is another passage (not quoted by Jencks or Block) that might appear to corroborate their diagnosis:

> [T]he environment is *defined* as that which affects the phenotype independently of the genotype. If an effect stems from the genes, it is genetic; any other effect is an environmental one. (Roberts 1967, 218)

Again, a closer look changes the picture. First, in this context Roberts again seems to have in mind *active* G-E correlation because he speaks about "habitat *selection*" and "the animal's *choice* of environment."[4] Second, the whole paper is manifestly focused on research on animals where, for obvious reasons, the complex interactions characteristic for the Jencks-Block scenarios (like the red-haired children example) do not come into play. Third, Roberts's position is subtler than could be judged from that single quotation. A few pages later he issues an explicit warning that the possibility of unrecognized G-E covariance could lead to overestimation of heritability: "The overriding concern at this stage is to avoid environmental sources of covariance that would lead to the wrong answer by inflating the estimate of the heritability. . . *There is no substitute for common sense in avoiding the pitfalls in this respect*" (Roberts 1967, 234–235; italics added). And fourth, Roberts is an ill-chosen example for representing methods of behavior genetics simply because, strictly speaking, he is not a behavior geneticist at all, but a quantitative geneticist who, as he himself says, was just asked on that occasion to "look over the wall into the field of behavior genetics."

Jencks's criticism of behavior geneticists is basically that they have changed the ordinary meaning of "environment" (1980, 726). Allegedly,

3. Block's attempt to fortify that imputation with a quotation from Jensen (1973, 54) fails for the same reason. Again, it escapes Block's attention that in the very passage he quotes, Jensen only says that what should be included in the genetic variance is that part of the G-E correlation that is "a result of the genotype's *selective utilization of the environment*" (italics added). Clearly, Jensen had in mind the active, not reactive correlation.

4. Later he explicitly argues that passive G-E correlation should be excluded from genetic variance (Roberts 1967, 234–235). He never discusses anything that we would now call "reactive covariance," which actually represents the key issue for Jencks and Block.

https://doi.org/10.1086/377384 Published online by Cambridge University Press

they narrowed the connotation of the term so that it came to include only the environments that are *not* correlated with genotype. Jencks also claims that, as the arising terminological ambiguity is not readily recognizable, heritability information will frequently appear more interesting than it actually is:

> Narrowing the definition (while retaining the term itself) is certain to mislead all but the most attentive and sophisticated readers. Indeed, it is only a slight exaggeration to say that narrowing the definition of a term that has traditionally had a very broad meaning is *meant* to mislead—that is, meant to make one's results sound more significant than they really are. (Jencks 1980, 726)

One would expect such a sweeping and damning criticism to be documented with a long list of references illustrating this massive semantic shift in the nature-nurture debate. However, Jencks gives *only one* example where the redefinition of "environment" is openly defended. Rather surprisingly, the source in question is actually his own article from 1977 (coauthored with Marsha Brown). Needless to say, the fact that in that article Jencks indeed introduced the idiosyncratically narrow definition of "environment" does not begin to show that the same conception has been widely shared by others or that it is endemic to the whole field. Moreover, the suggestion from that article that "environment" should be treated as a remainder term after genetic effects are directly estimated was later explicitly rejected in a seminal paper on genotype-environment correlation:

> If some appreciable fraction of the variation of a trait is due to the covariation of genes and environments, *that portion of the variance can be assigned neither to heredity nor to environment*—it is attributable jointly to both. If one's analytic method proceeds by estimating the genetic effect directly and the environmental effect by subtraction, the presence of GE correlation could lead to a substantial underestimation of the latter. (Loehlin and DeFries 1987, 264; italics added)

Although Block (1995) cites the paper by Loehlin and DeFries, he seems to be unaware of their unequivocal refusal to subsume G-E correlation under genetic variance, which directly contradicts his allegation that in behavior genetics, "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."[5]

5. That the methods of behavior genetics are not so crude as painted by Block is further confirmed in an article from a book that also happens to be in his list of references: "The issues of genotype-environment correlation and interaction are complex—*not nearly as simple as saying that their effects are incorporated wholly in estimates of genetic variance*" (Plomin 1987, 43; italics added).

https://doi.org/10.1086/377384 Published online by Cambridge University Press

Elliott Sober also thinks that the semantics of heritability conflicts with common sense: "Quantitative geneticists differ from the rest of us in the way they tend to use the term 'environment,' and this difference in usage will probably persist. This means that when quantitative geneticists say that the variation in some phenotype has a genetic component, the rest of us must be very careful" (2001, 75; cf. Sober 2000, 366). In discussing the example with the abuse of redheads, he warns that geneticists are forced to describe that kind of situation in a bizarre way, and that they have to interpret the lowering of IQ, counter-intuitively, as a genetic effect: "Quantitative geneticists do not regard abuse as an environmental factor [. . .] The lower IQ of redheads [. . .] is said to be genetic, rather than environmental, on the grounds that individuals experience abuse because of their genes (Falconer 1981)" (Sober 2001, 73). Although the reference to Falconer's *Introduction to Quantitative Genetics,* placed so directly in the context of the redheads example, might be read as suggesting that Falconer both discusses that very example and claims that in such cases the result of the abuse should be regarded as a genetic effect, in fact Falconer does not address that kind of issue at all. His treatment of G-E correlation is very general and superficial (and is condensed to just one paragraph).

The only two examples of G-E correlation that he mentions deal with cows' milk yield (where cows are fed according to their yield, the better phenotypes being given more food) and with human intelligence (where genotypically mediated phenotypes of the parents affect the environment in which the children grow up). In the latter case, Falconer explicitly refuses to count an individual's environment as a part of its genotype, "because the environmental effects of the children are not a consequence of their own genotype, but of their parents' genotype" (that is, because it is *passive* G-E correlation). In the former case (with cows), which in our taxonomy would correspond to reactive G-E correlation, he does include the environmental effect into genetic variance, but *only* under the assumption that the G-E correlation is "in practice unknown." So, there is no suggestion, even here, that the effects of those environments *known* to be just ultimately but superficially associated with a given genotype should be imperatively attributed to that genotype (as Jencks, Block, and Sober would have it).

Most importantly, although in the context of selective breeding of cattle it might perhaps be all right to regard all consequences that ultimately originate from genetic differences as genetic effects (because the purpose here is to enhance the genotypic superiority as much as possible), it by no means follows that the same causal analysis would be acceptable in incomparably more complex situations involving people's behavior and intricate social interactions. Certainly, nothing Falconer says warrants the belief that he would endorse such an extrapolation. Besides, Falconer

https://doi.org/10.1086/377384 Published online by Cambridge University Press

actually seems to be the wrong source for this discussion. Namely, to know how researchers would describe the redheads example and similar cases, it doesn't seem appropriate to refer to a standard introduction to quantitative genetics, which will necessarily be too general and too coarse-grained to be a useful guide for situations of that level of social complexity. Rather, one is much better advised to look into those (numerous) publications in behavior genetics that try to deal precisely with such tangled mixtures of genetic and social influences. My search of this literature has uncovered no evidence that would corroborate the "paradoxical" reading of heritability advocated by many critics of behavior genetics.

What then about this disciplinary revision of concepts "environment" and "heritable" hypothesized by Jencks, Block, Sober, and others?[6] Have these words really undergone a momentous change of meaning in behavior genetics, whereby "environment" so shrank in content that it started to exclude many bona fide environmental effects, while "heritability" correspondingly expanded to the point of getting, as Block would put it, violently in conflict with our ordinary socially important ideas of causation (Block 1995, 116)? I think that this whole story is a myth. It is, for example, clearly belied by the terminological practice in that large and notorious segment of the heredity-environment controversy, the race and IQ issue. In this debate, literally no one would say that explaining the black-white IQ gap by appeal to discrimination would show that the difference is heritable, although this is exactly how the situation should be described by using the "narrow definition" of environment. On that definition, if discrimination is correlated with a genetically mediated difference of skin color, its effects should not be regarded as an environmental influence (analogously to the red-haired children example). But no one is tempted to use the narrow definition here, and for a very good reason. Labelling the IQ gap resulting from discrimination as heritable would defeat the purpose of the term. It would make the IQ difference between the races heritable by definition, and then one would have to invent a new concept ("*really* heritable"?) to discuss the central question whether genes are explanatorily involved in a stronger and more interesting way than just giving rise to a superficial characteristic that, in turn, becomes a target of discriminatory practices.

Besides, contrary to what Jencks and others assert, nothing pushes the notion "heritability" to be extended to cases like this. True, there is a tendency to take some part of the variance due to G-E correlation and divide it between genes and environments. Thus active G-E covariance is occasionally subsumed under genetic variance, and passive covariance is assigned to the environmental side of the equation. However, it is important

6. Jencks speaks about "redefinition," and Block and Sober about "tacit convention" and "convention."

https://doi.org/10.1086/377384 Published online by Cambridge University Press

to stress that this redistribution is not a necessary consequence of some esoteric methodology for calculating heritability. Rather, it is a practical decision primarily guided by an attempt to follow the common-sense way of apportioning causal responsibility. If there happens to be any doubt about how to classify G-E correlation, the regular fallback position is just to treat it as a distinct component of variance, separate from heritability and environmentality. But even in those cases where a researcher opts for subsuming it under one of the two main effects, this is always just a pragmatic decision. There is no "correct" answer that is dictated, as it were, by the logic of heritability (cf. Jensen 1969, 39).

To see that the way of causal attribution in behavior genetics is not really anomalous or aberrant, look at one of the many examples where common sense would treat indirect causation in basically the same manner. Suppose that in a certain region a positive correlation is observed between rainy weather and the number of injured drivers. Consider the following two possible explanations of that correlation: (1) rain makes roads slippery, and this leads to injuries; and (2) there is a crazy gang of psychopaths who go out during rain to drop bricks on cars from highway bridges, and this leads to injuries.

In (1), which corresponds to the effects of self-selected environments, it seems perfectly natural to say that rain causes injuries, essentially because rain is regarded as almost inseparable from roads becoming slippery, which in turn is an immediate cause of injuries (rain → slippery roads → injuries). However, in (2), which corresponds to the effects of imposed environments, it sounds wrong to say that rain causes injuries, although here, again, rain *is* an indirect cause of the greater number of injuries (rain → psychopaths with bricks → injuries). Why? I submit that one reason is that now rain has only a tenuous link with the mediating cause, and consequently the mediating cause gains on independence, salience, and explanatory importance. As a result, rain can no longer account for the effect on its own, i.e., without mentioning the middle link. Put differently, we would not regard the bricks falling from the bridges as a "natural" manifestation of rain.

Another contrast to (1), and an additional similarity to the "imposed environments" scenario, is that in (2), the issue of blame is involved, and in such cases we usually want to focus on the human action responsible for the final outcome, and not just on the antecedent conditions of that action. This is all admittedly pretty vague, and I am not sure how intuitions underlying our different approach to these two kinds of cases should be refined further and made more precise. Fortunately this doesn't really matter, for I only want to claim that in dealing with G-E correlations, behavior geneticists are by and large guided by the common-sense con-

https://doi.org/10.1086/377384 Published online by Cambridge University Press

siderations about causality, with all their characteristic vagueness and ambiguities.

It is somewhat ironic that the phenomenon of G-E correlation has been used to argue that the notion of heritability is, as Block says, in the violent conflict with common sense. For, in fact, behavior geneticists rely on nothing but common sense whenever they subsume (active) G-E correlation under heritability, i.e., whenever they decide to abandon the default option of treating G-E correlation as a separate, sui generis source of phenotypic variance, which is neither genetic nor environmental.[7]

REFERENCES

Block, Ned (1995), "How Heritability Misleads about Race", *Cognition* 56: 99–128.
Block, Ned, and Gerald Dworkin (1976), "IQ, Heritability and Inequality", in N. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House.
Callebaut, Werner (ed.) (1993), *Taking the Naturalistic Turn*. Chicago: University of Chicago Press.
Falconer, Douglas S. (1981), *Introduction to Quantitative Genetics.* New York: Longman.
Fuller, John L. (1979),"Comment on Wahlsten", in J. R. Royce, and L. P. Mos (eds.), *Theoretical Advances in Behavior Genetics*. Alphen aan den Rijn: Sijthoff and Noordhof.
Garfinkel, Alan (1981), *Forms of Explanation*. New Haven: Yale University Press.
Gibbard, Allan (2001), "Genetic Plans, Genetic Differences, and Violence: Some Chief Possibilities", in: Wasserman and Wachbroit (2001).
Jencks, Christopher, et al. (1972) *Inequality*. New York: Basic Books.
Jencks, Christopher (1980), "Heredity, Environment, and Human Policy Reconsidered", *American Sociological Review* 45: 723–736.
——— (1992), *Rethinking Social Policy*. Cambridge: Harvard University Press.
Jensen, Arthur R. (1969), "How Much Can We Boost I.Q. and Scholastic Achievement?", *Harvard Educational Review* 39: 1–123.
——— (1973), *Educability and Group Differences*. New York: Harper and Row.
——— (1976), "The Problem of Genotype-Environment Correlation in the Estimation of Heritability from Monozygotic and Dizygotic Twins", *Acta Geneticae Medicae et Gemellologiae* 25: 86–99.
Jinks, John L., and David W. Fulker (1970), "Comparison of the Biometrical Genetical, MAVA, and Classical Approaches to the Analysis of Human Behavior", *Psychological Bulletin* 73: 311–349.
Loehlin, John C., and John D. DeFries (1987), "Genotype-Environment Correlation and IQ", *Behavior Genetics* 17: 263–277.
Loehlin, John C., Gardner Lindzey, and J. N. Spuhler (1975), *Race Differences in Intelligence*. San Francisco: W. H. Freeman.
Moore, David S. (2001), *The Dependent Gene*. New York: W. H. Freeman.
Plomin, Robert (1987), "Genetics of Intelligence", in S. Modgil and C. Modgil (eds.), *Arthur Jensen: Consensus and Controversy*. New York: The Falmer Press.

7. There is another way of seeing indirect genetic effects as raising a difficulty for heritability estimates. Rather than relying on *semantics* (i.e., suggesting that indirect genetic effects are part of the meaning of heritability) some critics make a *methodological* claim that the problem with heritability is that as a matter of fact direct and indirect genetic effects cannot be empirically separated. This argument deserves a separate discussion.

https://doi.org/10.1086/377384 Published online by Cambridge University Press

Plomin, Robert, et al. (1977), "Genotype-Environment Interaction and Correlation in the Analysis of Human Behavior", *Psychological Bulletin* 84: 309–322.
Roberts, R. C. (1967), "Some Concepts and Methods in Quantitative Genetics", in J. Hirsch (ed.), *Behavior-Genetic Analysis*. New York: McGraw-Hill.
Rowe, David C. (1994), *The Limits of Family Influence*. New York: The Guilford Press.
Sober, Elliott (2000), "The Meaning of Genetic Causation", in A. Buchanan et al. (eds.), *From Chance to Choice*. Cambridge: Cambridge University Press.
——— (2001), "Separating Nature and Nurture", in Wasserman and Wachbroit (2001).
Taylor, Kenneth A. (2001), "On the Explanatory Limits of Behavioral Genetics", in Wasserman and Wachbroit (2001).
Wachbroit, Robert (2001), "Understanding the Genetics-of-Violence Controversy", in Wasserman and Wachbroit (2001).
Wasserman, David, and Robert Wachbroit (eds.) (2001), *Genetics and Criminal Behavior*. Cambridge: Cambridge University Press.

https://doi.org/10.1086/377384 Published online by Cambridge University Press