HERITABILITY AND CAUSALITY*

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The critics of "hereditarianism" often claim that any attempt to explain human behavior by invoking genes is confronted with insurmountable methodological difficulties. They reject the idea that heritability estimates could lead to genetic explanations by pointing out that these estimates are strictly valid only for a given population and that they are exposed to the irremovable confounding effects of genotype-environment interaction and genotype-environment correlation. I argue that these difficulties are greatly exaggerated, and that we would be wrong to regard them as presenting a fundamental obstacle to the search for genetic explanations. I also show that, to the extent they are cogent, these objections may prove to be even more damaging to the "environmentalist" standpoint.

1. Nature vs. Nurture Once Again. The basic point of disagreement between "environmentalists" and "hereditarians" is located in the environmentalists' claim that any interesting explanation of human behavioral tendencies invoking genes is presently confronted with insurmountable methodological difficulties that make all such theorizing nonsensical or premature. Accordingly, environmentalists are distinguished by their global and principled doubt that the behavioral sphere can be illuminated by information derived from genetics.

This view of the nature-nurture debate has two virtues. First, the environmentalist standpoint is in this way given content without being reduced to a caricature denying genes any causal power. Genes *are* causally operative and they are seen as necessary in the sense of the interactionist truism ("No organism without either genes or environment"); they simply do not figure in *interesting* explanations. The qualification "interesting" is required because environmentalists usually find no fault with such boring genetic explanations as that "genes . . . specify that we are large enough to live in a world of gravitational forces, need to rest our bodies

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by sleeping, and do not photosynthesize" (Gould 1977, 253) or that "[t]he range of behaviors which animals (including humans) can emit is, of course, genetically constrained [in that] [h]umans will never fly without the aid of mechanical devices" (Burian 1978, 392).

Second, we can now more clearly separate two lines of attack on hereditarianism. By "hereditarianism" I mean the view that, contrary to the environmentalist claim, interesting genetic explanations of human behavioral tendencies are not ruled out on general grounds, *plus* the belief that there is also substantial empirical evidence for such explanations in some specific area. The formulation admits objections to hereditarianism from two sources: (1) global methodological arguments, in the hope that this first step will itself decide the matter (wherefore the whole discussion of concrete empirical evidence adduced in favor of hereditarianism would be beside the point); and (2) strictly on hereditarianism's empirical merits, finding nothing *generally* objectionable in the idea.

In this paper, I address the first line of criticism. Although, undoubtedly, the distinction between methodological and empirical criticism is not sharp, it might be useful to bring into focus those arguments against hereditarianism which are meant to have general import and which, due to their relative independence of empirical material, have been brought to bear on a wide range of issues.

The two strategies of attacking hereditarianism are sometimes incoherently run together. For instance, S. Rose writes:

> I have often been asked whether I am opposed to work on 'the genetics of average race differences in IQ' on ethical grounds. My response is that I am opposed to it on the same grounds that I am opposed to research on whether the backside of the moon is made of gorgonzola or of stilton. That is, it is a silly question, incapable of scientific answer and actually, *sensu strictu* [*sic*], meaningless. The question makes grammatical, but not scientific, sense. . . . (1986, 35)

Is Rose condemning claims about heritability because they are meaningless (as explicitly stated) or merely because they are grotesquely false (as suggested by the analogy with "the backside of the moon is made of gorgonzola or of stilton")? This is no mere slip of the pen, for it displays a characteristic indecisiveness in the writings of some environmentalists; they cannot make up their minds whether to attack the hereditarian claim with methodological weapons, trying to destroy it as nonsensical at the very level of formulation (before the question of empirical evidence is raised at all), or to meet it on empirical grounds and argue that it lacks any evidential support. (For similar environmentalist vacillation see Layzer 1976, 201 and Taylor 1980, 9, 111, 205–206.)

Having himself noted this peculiar two-track strategy of environmentalists, T. J. Bouchard has gone so far as to conclude that methodological arguments against hereditarianism scarcely deserve attention:

> We need not dwell on these arguments for long. If they were at all persuasive there would be little, if any, need to attack the evidence underlying the hereditarian viewpoint. It would fall of its own weight. The massive, and vituperous, attacks on hereditarian findings clearly signal how seriously the environmental program is challenged by this evidence. (1987, 58)

Since, however, the methodological arguments of environmentalists still inform biological discussions, and even more the opinion of the general public on these matters, we should take them seriously. Scrutinizing and evaluating these arguments may be regarded as an appropriate assignment for Locke's "underlabourer", whose modest ambition is "to clear the ground a little" for those working in the science proper.

2. Heritability under Attack. The total phenotypic variance ($\mathbf{V_P}$) of a trait in a population is in the simplest case broken up into two components: the one ($\mathbf{V_G}$) due to genetic differences (genetic variance) and the other ($\mathbf{V_E}$) due to environmental differences (environmental variance):

> The corresponding partition of the variance into genotypic and environmental components formulates the problem of "heredity versus environment" or "nature and nurture"; or, to put the question more precisely, the relative importance of genotype and environment in determining the phenotypic value. (Falconer 1964, 130)

This decomposition finds its standard representation in the following formula:

$$\mathbf{V_P = V_G + V_E}.$$

Genetic variance ($\mathbf{V_G}$) can itself be further partitioned into additive variance ($\mathbf{V_A}$), dominance variance ($\mathbf{V_D}$), epistatic variance ($\mathbf{V_{EP}}$) and variance due to assortative mating ($\mathbf{V_{AM}}$). Total genetic variance can then be expressed as the sum:

$$\mathbf{V_G = V_A + V_D + V_{EP} + V_{AM}}.$$

The ratio of total genetic variance to phenotypic variance ($\mathbf{V_G/V_P}$) is called "heritability in the broad sense" or "coefficient of genetic determination", and is symbolized by "h^2". The so-called "heritability in the narrow sense" is the ratio of additive genetic variance to phenotypic variance ($\mathbf{V_A/V_P}$). Since the opinion prevails (see Crow and Kimura 1970, 124; Loehlin, Lindzey and Spuhler 1975, 81; Jensen 1976, 88) that of

the two concepts broad heritability is of greater general interest (for it is this notion that reveals how strong the relative influences of genotype and environment are on phenotypic differences[1]) the term "heritability" will in the following be understood as *broad* heritability.

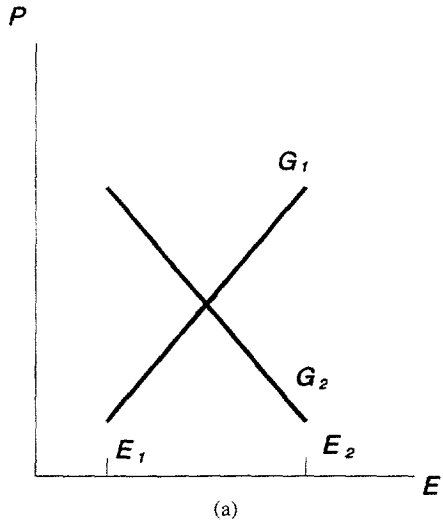
The heritability claims have often been regarded as important primarily on the expectation that they would furnish valuable information about the *causal* strength of genetic influence on phenotypic differences. Recall that when the term "heritability" was introduced for the first time in 1940 (by J. L. Lush) it was defined "as the fraction of the observed variance which was *caused* by differences in heredity" (quoted in DeFries 1967, 324; emphasis added). That the major aim of heritability estimates is the search for causality has been still more forcefully emphasized by R. C. Roberts, "We need to know how much of the total variation [in a population] is due to various genetic causes, for it is axiomatic that the importance of variation is proportional to the contribution it makes to the total variation" (1967, 217).

The idea that heritability reflects the causal strength of genetic influence on phenotypic differences has been consistently opposed by a number of authors. It has been said, for example, that heritability estimates are "both deceptive and trivial" (Hirsch 1976, 168); that they are "nearly equivalent to no information at all for any serious problem of human genetics" (Feldman and Lewontin 1975, 1168); that it is dubious whether a clear meaning can be given to "genetic determination of traits" (Burian 1981–1982, 51); that inferences about genetic determination of traits should be "disavowed once and for all" (Kitcher 1990, 97); that "mathematical estimates of heritability tell us almost nothing about anything important" (Jencks et al. 1975, 76); that the attempt quantitatively to determine the part of the phenotypic variance due to genetic causes is "biological nonsense" (Lewontin 1982, 14–15); and so forth.

This widespread conviction that heritability claims are devoid of interesting content is sustained by the argument from *statistical interaction* of genes and environment. (Statistical interaction should not be confused with *causal* interaction. To avoid misunderstandings, Waddington 1957, 94, and Bouchard and Segal 1985, 393, have suggested that the term "interaction" be used to refer only to the statistical concept; but this suggestion has been ignored.)

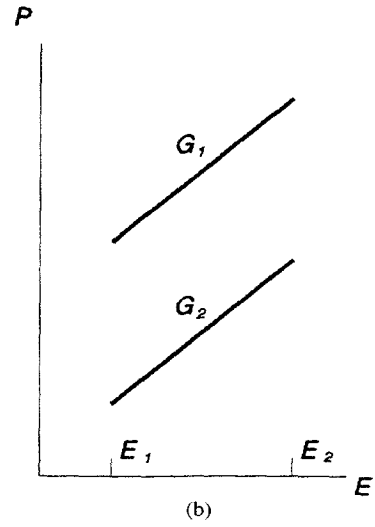
3. Statistical Interaction. An antihereditarian argument based on statistical interaction of genes and environment is most clearly and forcefully presented by R. Lewontin (1976) in his classic paper, still quoted with

[1]For a dissenting view, however, see Fuller (1972, 17–18).



(a)

Figure 3.1a. Statistical interaction: no main effects.



(b)

Figure 3.1b. Main effects: no statistical interaction.

approval by leading philosophers of biology (see, e.g., Sober 1984, 107–108, 266).

Lewontin wants to show that, contrary to widespread opinion, the analysis of variance cannot bring us any nearer to the thing we are really interested in: the extent to which genes determine phenotypic differences.

Take a population of organisms with two different genotypes[2] G_1 and G_2 that are with equal frequency distributed in two different environments E_1 and E_2. Suppose also that (as illustrated in figure 3.1a) organisms with genotype G_2 in environment E_1 have higher phenotypic value (with respect to some trait P) than organisms with genotype G_1; alternatively, in environment E_2, G_1 organisms have higher P value. Now, should the phenotypic differences in that population be ascribed to genes or to environment? Either answer is plainly wrong. Assuming that genotypes and environments are uniformly distributed, neither the genetic difference (between G_1 and G_2) nor the environmental difference (between E_1 and E_2) has a net effect on P. Their average effect is zero since the effect of genotype depends crucially on environment, and the effect of environment equally depends on genotype. Put differently, phenotypic variance is here due neither to genes nor to environment alone: It is the outcome of statistical interaction between genes and environments.

[2]In speaking about organisms having the same (different) genotype I follow the standard genetic terminology, meaning that they possess (do not possess) the same relevant allele(s).

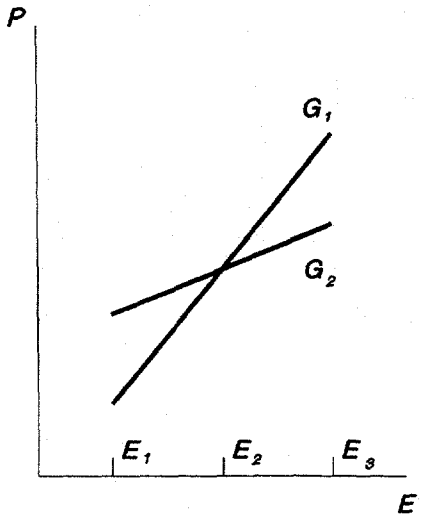


Figure 3.2. Main effects? Yes and no.

Generally speaking, two factors A and B are said to interact statistically if the influence of factor A changes its magnitude with the changing values of the other factor B. This condition is clearly satisfied in our simple example: The change in factor G from G_1 to G_2 increases phenotypic value P if factor E has value E_2, whereas it has negative effect otherwise (if E is fixed on E_1). Under these conditions it hardly makes sense to ask what is the effect of G *tout court*. It depends.

In the case pictured in figure 3.1b, however, we can easily ascertain how much phenotypic variation is due to genetic and how much to environmental causes. Moving from one environment to the other brings about the same phenotypic change, irrespective of genotype. Similarly, shifting from one genotype to another has always the same phenotypic effect (in both environments). Environmental and genetic contributions to variance add up to total variance: $\mathbf{V_P} = \mathbf{V_G} + \mathbf{V_E}$. This neat solution is in marked contrast to the intractable case (from figure 3.1a) where norms of reaction are not parallel and where the effects of genes and environment have become hopelessly jumbled.

Lewontin makes much of the following consequence of gene-environment interaction. In the presence of interaction, it may happen that the amount of genetic variance in a population will depend on how the environments are distributed. For example, as shown in figure 3.2 (adapted from Lewontin's figure), if environments were heavily weighted to the left,

> then G_1 would be consistently superior to G_2, and an analysis of variance would show a strong effect of genotype as well as of environment, but very little genotype-environment interaction. Thus the analysis of variance would reflect the particular environmental circumstances and give a completely incorrect picture of the general relationship between cause and effect here, where there is overall no effect of genotype but a strong genotype-environment interaction. (Lewontin 1976, 186; the same argument is advanced in Feldman and Lewontin 1975, 1166, and in Block and Dworkin 1976, 484)

Lewontin's final conclusion leaves no doubt about how radical and far-reaching are the consequences he ascribes to his argument, "The simple analysis of variance is useless for these purposes [for the search for causes] and indeed it has no use at all. In view of the terrible mischief that has been done by confusing the spatiotemporally local analysis of variance with the global analysis of causes, I suggest that we stop the endless search for better methods of estimating useless quantities" (1976, 192). Note that the hope that heritability might disclose some causal relations is not criticized here as being wrong from the narrow standpoint of genetics: The whole idea is rejected as being flawed on general methodological grounds.

Lewontin's main argument against ANOVA's (analysis of variance) causal pretensions is the "locality" objection: The effects inferred strongly depend on the actual distribution of environments and genotypes in our sample. As we have already seen in figure 3.2, we will come to notably different conclusions, depending on whether the environments are "heavily weighted" to the left, or whether they are, alternatively, evenly spread out over the entire range E_1–E_3. In the first case we infer a strong main effect of genotype, whereas in the second case this effect is virtually nonexistent.

Is this kind of unreliability really a sufficient ground to condemn ANOVA as "having no use at all" in the search for causal connections? I think not. I argue that, contrary to Lewontin's central claim, ANOVA does have resources to distinguish between two crucially different cases: (a) genetic variance expressing general, functional relations between genes and phenotype; and (b) genetic variance reflecting only local, spatiotemporal conditions.

Returning to figure 3.2, let us take a closer look at the situation where environments are heavily weighted to the left, that is, where G_1 and G_2 organisms live far more often in environments near E_1 than in others. True, we are led here to the conclusion that phenotypic differences are in great measure caused by genetic differences. But this is as it should be; for, under the strong prevalence of environments near E_1, genetic

differences will tend to manifest themselves consistently in phenotypic differences (G_1 organisms will be systematically superior to G_2).

It is, however, wrong to suggest (as Lewontin does) that by making this causal inference one is committed also to a "general", "functional" relation between G and P *over the whole environmental range* E_1–E_3. On the contrary, even after accepting the causal conclusion about "local" origins of phenotypic differences, we have a simple way to check whether the more general relationship between G and P holds as well. We need only to compare phenotypic differences between G_1 and G_2 organisms in the rarely encountered environments from E_2 to E_3 in order to realize that G_2 is now systematically superior to G_1, and that due to the mutual cancelation of these diametrically opposite genetic influences there is *on the whole* no effect of G on P.

Thus, so long as we have data about what happens in the thinned-out environmental range on the right, ANOVA *can* discriminate between the genetic influence on phenotype reflecting a *general* relation between two variables (constant across all environments) and genetic influence being, *locally*, a mere consequence of the uneven distribution of environments.

4. Heritability and Locality. What to say, though, when the data we have are restricted to a given environmental range (as it most frequently will be the case in nonexperimental research)? Critics of hereditarianism like to point out that heritability is applicable solely to the environments for which it has been calculated, and that we have therefore no right to generalize it to new, unexamined domains. The *global* causal relations between genes and phenotype again seem to escape us, for they are contained in total norms of reactions, and not in their partial segments (which are, as a rule, all we happen to know).

Is it, then, permissible to project norms of reaction outside the scope within which they were confirmed? Although one should be cautious with such inferences, I argue that it is an exaggeration to view any extrapolation here as being always as good as any other. Generalization may be extremely risky but it cannot be ruled out as in principle illegitimate.

In a certain sense, heritability estimates are local. When it is, for instance, discovered that in a given population the difference between organisms with genotype G_1 and those with genotype G_2 accounts for most of the differences in phenotypic trait P, biologists usually do not assume that a scientific law has been discovered according to which G_1 causes the increase in P. They are aware that they are still too much in the dark about the way G influences P, and that it would be therefore inappropriate to honor this discovery with the term "law". A law requires, at the very least, that something be also known about the conditions under which G causes P. But this requirement is not satisfied here: Apart from knowl-

edge that G causally influences P in *this very* situation, it is unclear what *altered* conditions would continue to sustain this causal connection. It is known only (a) that *in a given situation* genetic differences are strongly reflected in phenotypic differences; and (b) that, very probably, the hidden underlying mechanism by which G influences P is so complex that it is uncertain what would happen under *changed* circumstances. Not knowing which factors are relevant and in what way, we simply cannot be sure whether our locally discovered causal relations would reappear even under just slightly altered conditions. Clearly, at issue is not merely the general uncertainty of inductive generalizations; the special difficulty here is that we are in this case trying to project something *less* than a law.

Granting all this, however, by no means justifies the extreme thesis that a heritability estimate obtained for a given population has no informative value for other populations. On the contrary, we can concede the point that heritability extrapolations may be highly sensitive to small environmental perturbations, but still assert that the less a new population differs from the original one, the more reasonable it would be to expect similar heritability values.

The limitation of ANOVA and its dependence on local conditions can be dramatized by examples like the following. By (correctly) applying the same ANOVA techniques in epidemiological studies it was discovered that a certain cancer was environmentally caused in Sweden and genetically caused in Switzerland. (The explanation is that a pollutant was differentially distributed in Sweden while in Switzerland a gene was responsible for susceptibility.[3]) What does this fact reveal?

It shows that ANOVA cannot provide the *full* causal story about how, and under what conditions, the cancer develops. But it does not show that *no* important causal knowledge has been thereby obtained. On the contrary, the studies of Swiss and Swedish populations have indicated the presence of two *causal* factors (genetic and environmental, respectively) in the genesis of cancer. Furthermore, it is possible that *both* factors are *causally necessary* for producing cancer, and that in each situation we pick out one of them as *the* cause simply because of greater interest in the factor which accounts for more variance. (If the pollutant is omnipresent or at least uniformly distributed, the presence of the genetic factor explains why some people get cancer and others do not. In a different situation, the pollutant might become a factor which makes a greater difference.)

The less information we possess about the precise causal mechanism

[3]I owe this example to an anonymous referee for *Philosophy of Science*. A similar illustration with tuberculosis can be found in Jensen (1969, 45).

that brings about a given effect, the less reliable will be extrapolations of such a fragmentary causal knowledge to new situations. Nevertheless, causal mechanisms are rarely discovered without exploring, preliminarily and with the help of statistics, which variables are causally relevant. Therefore, the claim that ANOVA is "useless" or intrinsically suspect as an instrument in the search for causes is tantamount to condemning a causal inference because of its questionable generalizability, although it is a crucial and indispensable first step in most etiological investigations.

A. Jensen once said that "a heritability study may be regarded as a Geiger counter with which one scans the territory in order to find the spot one can most profitably begin to dig for ore" (1972, 243). That Jensen's advice as to how to look upon heritability is merely an application of a standard general procedure in causal reasoning is confirmed by the following observation from an introduction to causal analysis, "[T]he decomposition of statistical associations represents a first step. The results indicate which effects are important and which may be safely ignored, that is, where we ought to start digging in order to uncover the nature of the causal mechanisms producing association between our variables" (Hellevik 1984, 149). High heritability of a trait (in a given population) often signals the worth of digging further in the sense that an important genetic mechanism may thus be uncovered. High heritability indicates that genes are strongly implicated simply by telling us that the other causal factor (environment) is, under the circumstances, relatively unimportant for the phenotypic variation. Manipulating the environments *in the environmental range where high heritability prevails* is unlikely to have an appreciable effect on the existing phenotypic differences. (For an argument to this effect, see Scarr-Salapatek 1976, 127.)

One might object here by saying that heritability, and ANOVA approach in general, cannot help us locate causally relevant variables but only variables that are "candidates" for causal relevance. Indeed, it may be true that hypotheses advanced in the early explorations tend to be epistemologically insecure, so that in these phases we are well-advised to be on special guard with our causal claims. But our fallibilism, it seems, should not lead us so far as to deny that we often do locate causally relevant variables *even before we know anything about the causal mechanisms through which they operate.*

It is, of course, possible that an effect that is, in a given set of environments, purely genetic ceases to be so when novel environments are introduced, and that it becomes responsive to these new environmental influences. The case in point is the human disease PKU which was once classified as a genetically caused disorder; but after the discovery that its pathogenic manifestations can be avoided by taking an appropriate diet, it is now better regarded as the result of gene-environment interaction.

All this proves (what needs no proof) that with new knowledge a previously adopted causal picture has often to be modified or abandoned. But, surely, it does not mean that we should give up our current causal beliefs because of a *mere possibility* of their being refuted by some future knowledge. Therefore, if "locally" available evidence supports a hypothesis that a given phenotypic difference is under strong genetic influence, one cannot call that hypothesis in question by pointing out that, *maybe*, this effect disappears in some yet uninvestigated environments.

The example of PKU is usually introduced in order to illustrate the incompleteness of causal information derivable from heritability estimates and the importance of knowledge of causal mechanisms. But another lesson is frequently forgotten: that the discovery of some causal mechanisms was only possible because the earlier hypothesis about genetic causal origin of phenotypic differences was taken seriously.

5. The Norm of Reaction: A Misleading Concept. Those who dismiss the idea that heritability might have anything interesting to say about the relation between genes, environment and phenotype tend to argue for using the concept of norm of reaction instead:

> High or low heritability tells us absolutely nothing about how a given individual might have developed under conditions different from those in which he actually developed. Heritability provides no information about norm of reaction. . . . Norm of reaction is a developmental reality of biology in plants, animals, and people. (Hirsch 1976, 172–173)

Merits of the norm-of-reaction approach are also defended by Lewontin, Gould, Burian, Kitcher, Block and Dworkin, among others. It is dubious, though, whether our understanding of the heredity-environment issue is significantly advanced with the introduction of this concept.

The norm of reaction is introduced to compare *ranges of reaction* of various genotypes to different environments. To insist on retaining the norm-of-reaction perspective would appear to make little sense if all these ranges were equal, or equivalently, if the norms of reaction were mutually parallel (or, again equivalently, if there were no gene-environment interaction).[4] Even Lewontin (1976, 189), the most uncompromising opponent of heritability, agrees that in the case of no interaction the analysis of variance gives entirely satisfactory results, and that nothing would be gained by adopting the alternative approach.

It follows that if the gene-environment interaction were relatively rare

[4] "If there is [gene-environment] interaction, it is useful to think of the *norm of reaction*" (Maynard Smith 1989, 95).

the norm of reaction would be of no general explanatory use. But how do we know that it is not so? One should be aware of a tendency to try to force the belief in the omnipresence of interaction by falsely representing it as the matter of scientific consensus. See, for example, Lewontin (1983, 277); Block and Dworkin (1976, 483–484); Layzer (1976, 209); Lewontin et al. (1984, 269); Layzer (1974, 1259); Sultan (1987, 133); Stearns (1989, 443).

Contrary to this picture of general agreement, many scientists adopt a "less than optimistic view of interactions [and think that] nonadditive interactions rarely account for a significant portion of variance" (Plomin et al. 1988, 228–229), or that, "so far, it has certainly been easier to talk about genotype-environment interactions than it has been to find them" (Plomin 1986, 108). Others assert that the only evidence for *G-E* interaction comes from research on rats (presumably the frequently cited experiment of Cooper and Zubek) and that "[n]othing like it has yet been found in human mental ability" (Jensen 1981, 124). D. Futuyma writes, "We will proceed, *as most workers in the field do*, by *ignoring* the genotype x environment interaction, which in practice is often included in the term V_E" (Futuyma 1986, 197; emphasis added).[5] It is also said that "the simpler additive model *in most cases* comes close to fitting the expectancies" (Cattell 1982, 66; emphasis in the original), and that "there is very little empirical support for [the] existence [of genotype-environment interactions] in the behavioral domain" (McGue 1989, 507). Concerning the heritability of intelligence, R. Herrnstein states that "the data from the twins reveal no interaction (in the technical sense) of heredity and environment" (1973, 180). Even C. Jencks who cannot be accused of hereditarianism, has said that "virtually all analysts assume [that] the effects of genotype and environment are additive" (1980, 732). The geneticist J. Crow argues that "[i]f interactions were rampant, evolution (at least in sexual species) would be impossible [and that a] certain amount of additivity is a prerequisite for evolution" (1990, 127).

The relative lack of empirical evidence for interactions, however, may be due to difficulties in designing the tests that could detect them. But since this cannot vindicate the dogmatic belief in the omnipresence of interactions, it seems reasonable to conclude that the more uncertain the frequency of *G-E* interactions, the more dubious is the alleged usefulness of the norm-of-reaction approach, which was specifically introduced in order to take account of this phenomenon.

Even if interactions were ubiquitous, we would not be compelled to abandon the factorial approach. Interactions might prove to be removable by mathematical transformation (see Box et al. 1978, 218–219) or they

[5]This procedure cannot be warranted unless interaction is relatively small.

might simply not be of such a magnitude as to make (partially) additive models unemployable. To despair over the complexity and intractability of fully interaction-ridden situations without first exploring the limits of application of simpler methods would be in effect "asking us to run before we can walk" (Cattell 1982, 66).

But our criticism should strike at a deeper level. Let us therefore raise the following question: Assuming that strong and nonremovable *G-E* interactions are very frequent, would the description in terms of norm of reaction prove superior to other approaches in some important theoretical respect? That is, would we lose anything if we decided to drop the norm-of-reaction talk altogether,[6] adhering simply to the precisely defined concept of statistical interaction? R. Plomin argues that we would thereby only free ourselves from many vague connotations which the term "norm of reaction" drags in its wake:

> Indeed, the connotations of the phrase may be responsible for its popularity. Reaction range suggests the notion that, regardless of genotype, an individual can grow up to be a pauper or a prince depending on environmental circumstances. Although such plots make good fairy tales, all we can assess is environmental variance, genetic variance, and variance due to genotype-environment interaction. The term *reaction range* adds nothing to these concepts. (1986, 95)

Plomin's "fairy tale" irony is pertinent, for the norm of reaction (as used by some authors) does tend to divert our attention from the manageable empirical data over which we have some control, and to direct it to the question of what might have been the case in different and ill-defined circumstances. Speculation about these remotely conceivable situations may then dominate the picture so much that we witness the curious triumph of the *possible* over the *actual*.

Dissatisfaction with the "too local" nature of heritability estimates leads too easily to asking, "How would organisms with different genotypes differ across *all possible* environments?" Here the norm of reaction comes in, "Complete knowledge of a norm of reaction would require placing the carriers of a given genotype in *all possible* environments, and observing the phenotypes that develop" (Dobzhansky 1955, 75; emphasis added). However, we simply cannot know what would happen in *all possible* environments. And, indeed, Dobzhansky adds, "The performance of a genotype cannot be tested in all possible environments, because the latter are infinitely variable" (ibid., 77).

[6]Notably, when Schiff and Lewontin (1986, 188–189) analyzed 11 genetics textbooks written by prominent scientists in the field, not one of them contained a reference to the norm of reaction!

For Dobzhansky, the norm of reaction was an idealization, useful for some expository purposes, but not *the* correct account of the relations between genes, environment and phenotype. Therefore he could well afford not to be bothered much by the fact that norms of reaction are, strictly speaking, unknowable. But this should seriously worry those who regard norm of reaction as "the basic concept for a correct understanding of gene and organism" (Lewontin 1982, 21), as "the real object of study for both programmatic and theoretical purposes" (1976, 184), or as "a developmental reality of biology" (Hirsch 1976, 173).

To suggest that if hereditarians want to prove their point, they are under an obligation to show that genes have a major influence in *all possible* environments seems absurd. After all, even with respect to genuine causal laws expressed by the statement, "Events of type *A* cause events of type *B*" (which are, one should remember, stronger claims than heritability estimates aspire to be), we usually know in advance that there are circumstances ("environments") where events of type *A* do *not* cause events of type *B*! This partial failure of application is, moreover, sometimes built into the concept of causal law itself (see Stinchcombe 1968, 31).

True, hereditarians may now and again predict, rashly, that nothing in the empirically observed relations would be changed even under a radically different environmental regime. But such unsupported assertions should not be regarded as belonging to the hard core of the hereditarian standpoint; otherwise, one could pari passu take as the mark of environmentalism the equally unjustified and occasionally advanced assurance that environments which eradicate all differences of genetic origin are just around the corner. In fact, neither position in the debate is committed to prophesying what would happen in some distant and at present inaccessible possible worlds.

The weaknesses of this "possible-worlds" approach to the heredity-environment controversy are best recognized and most explicitly formulated in Kitcher's attempt at reconstruction. He starts by supposing, wisely, that each camp misrepresents the opposite standpoint by understanding it to claim that only genes or only environment determine phenotype: Too facile a victory is won by attacking these men of straw. Kitcher then argues that there must be a point of real and rational disagreement between two sides, even after they acknowledge the "interactionist truism"—the fact that *both* genes *and* environment determine phenotype. His proposed solution is to burden the "genetic determinists" with the claim about what happens in *all possible* environments (Kitcher 1985, 25–29; 1987, 66). Accordingly, genetically determined phenotypic differences are identified, roughly, as those that cannot be eliminated by *any* environmental changes. Kitcher believes that this reinterpretation is fair to hereditarianism in that it avoids making it either simplistic ("*only*

genes determine phenotype") or unexciting ("genes have *some* influence on phenotype").

Real hereditarians will not be made happy with such a division of roles. Agreeing to play the part assigned to them in the debate by Kitcher would probably be regarded as equivalent to admitting defeat. For the one who takes on the Herculean task of proving that something holds literally *under all circumstances* should be aware that no defense is available against Kitcher's correct claims about "the immensity of our ignorance about the environmental influences on human behavior" (1985, 29) and about "our [profound] ignorance of the shapes of the norms of reaction" (1987, 66).

It would be much fairer if the heredity-environment discussion were reconstructed so that it dealt with our real world, not with uncontrollable "might-have-beens" and counterfactuals gone wild. But then we would not get very far with the "norm of reaction" interpreted in such a way that it only encourages flight into speculation. Rather, we should aim at discovering the connections that hold locally, the only piece of knowledge we can hope to obtain under the circumstances.

In fact, the *actually existing* relationships between genes, organisms and phenotypes are interesting not only from the standpoint of scientific knowledge; notwithstanding frequent environmentalist claims to the contrary, these relationships are also of great importance in political considerations; they can even offer useful guidance in educational reforms:

> [T]he great majority of immediate policy decisions revolve around just that set of environments for which heritability estimates have the most relevance: the existing set. Most proposed policy changes involve minor redistributions of environments within the existing range, and it is precisely regarding such changes that a heritability estimate has its maximum predictive value. For instance, one message that a high heritability coefficient can convey is that minor fiddling around with environmental factors that already vary widely within the population has poor odds of paying off in phenotypic change—and thus new ideas about environments need to be tried. Surely, this is a message of enough social and practical implication to justify continued interest in heritability and its estimation. (Loehlin et al. 1975, 99)

Incidentally, this was also one of Jensen's (1969) main points which passed almost unnoticed amidst united attacks on his "hereditarianism" and "racism".

6. The Hereditarian Strikes Back. The influences of genes and environment are inseparable epistemologically: We cannot know one without the other. For this reason, regarding the interest in heritability as a symptom of the obsession with genes is incorrect. By knowing heritability one

knows ipso facto the complementary contribution of environment to phenotypic variation—called "coefficient of environmental determination" (DeFries 1972, 12) or "environmentality" (Plomin et al. 1980, 225). Heritability and environmentality are simply two ways of conveying the same information, just as saying that a glass is two-thirds full is the same as saying that it is one-third empty.

It is hence inconsistent for environmentalists to attack heritability, for if their criticism succeeds in making the concept methodologically suspect or scientifically useless, they thereby pull the rug from under their own feet: They have then no right to speak about the environmental influence on phenotypic variation, either. (A concept complementary to a meaningless concept is itself meaningless.)

Let us see, more concretely, how two major environmentalist objections to heritability backfire.

1. Take first the (statistical) interaction objection. It is well known that when two independent variables strongly interact, then their main effects, although obtainable by calculation, may lend themselves to no meaningful interpretation (see Hays 1973, 497–498; and Blalock 1979, 365–366). On this basis, environmentalists sometimes argue that the impact of a genetic difference on phenotype may vary so greatly across different environments that it makes no sense to speak about *the* genetic influence. It is not always realized that if this turns out to be the case, it will be equally senseless to talk about *the* environmental influence. (This follows from the symmetry of statistical interaction.) Or, to spell it out, if genetically dissimilar organisms reacted differently to the same environmental change, there would obviously be no *general* effect of that environmental change.

In point of fact, the more the heritability of a trait is made indeterminate through interaction, the more important it becomes, for educational and political purposes, to know how the genes influence it. For only so long as the effects of genes and environment are separable can one hope to improve the situation by global and massive environmental reforms. If, however, these effects are lost in interactions, we will know in advance that no environment is best for all, independently of genotype; the task of reform would in these circumstances be the arduous (and often impossible) one of finding different optimal environments, tailor-made for individuals of different genetic constitutions.

2. Another environmentalists' objection to heritability that also turns against them relies on the statistical *correlation* of genes and environments. Genotype and environment are correlated if organisms with a given genotype are more often exposed to a specific environment than would be expected according to a random distribution. This may easily give rise to incorrect causal conclusions. For instance, organisms with genotype

G_1 might be superior in phenotypic trait P *not* because G_1 causes the increase in P, but merely because organisms with G_1 happen more frequently to live in favorable environments E_1.

Especially elusive are so-called "active" and "reactive" *G-E* correlations (these terms were introduced in Plomin et al. 1977) where genes have causal impact on phenotype first through modifying the environment, which then has a phenotypic influence of its own. For example, if intelligence were (partially) inherited, the more intelligent children would tend to create for themselves more intellectually stimulating environments, whereby their level of intelligence would be raised even more. The problem would then arise of how to disentangle the *direct* effect of genotype from its *indirect* path of influence in which environment plays a major role.

One suggestion is simply to include the effects of *G-E* correlation in genetic variance. This move is justified by saying that genetic variance should comprise everything that is caused by genes, irrespective of the way they operate causally. Or, in the often quoted words of 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" (1967, 218).

In the opinion of a number of authors, however, two ways in which genes can cause phenotype must be distinguished: (1) when proximate causes are purely biological; and (2) when they are social, that is, when "the causal path from gene to characteristic passes through the behavior of *other* persons" (Block and Dworkin 1976, 481). The distinction is easy to make in theory but, so the argument goes (Layzer 1974; Feldman and Lewontin 1975, 1164; Block and Dworkin 1976, 482; Kitcher 1985, 247–248), the two influences cannot be separated empirically: If organisms with similar genotypes "select" similar social environments, we cannot know how much their phenotypic similarity is due to their common genes and how much to a shared environment.

The difficulty here may not be insoluble. To conclude, on account of present obstacles, that it is *forever* and *absolutely* impossible to uncouple the two causal influences seems rash. For example, we might argue that the more rigidly a genotype is connected with an environment, the more we are entitled to regard the final effect as being caused by the genotype. It is a familiar fact about causality that the more invariably and inflexibly A produces B, which then produces C, the more justified it is to leave out the intermediate link B and to say, simply, that A causes C. Applied to our case this means, unexpectedly, that the more *G-E* correlation resists our efforts to estimate it, the greater our right not to regard it as an independent contribution to variance, and to include it in a heritability coefficient. Moreover, we could rely on Jensen's (1976) proof that *G-E*

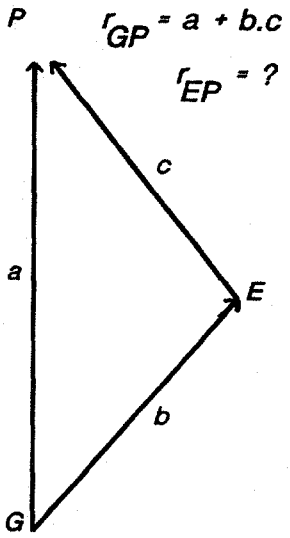


Figure 6.1. From correlations to causes.

correlation has an influence of importance only when heritability and environmentality are substantial too; on that basis we could then defend the view that, even when the precise magnitudes of different sources of variance were not individually ascertainable, it would be safe to predict that heritability is nonnegligible.

Let us, however, show in conclusion how environmentalists are again trapped by their own argument. Expressed in the terminology of path analysis, their objection to hereditarianism amounts to the claim that it is impossible to decompose an empirically found correlation between genotype and phenotype into two causal paths (the direct and the indirect) connecting the two variables: With reference to figure 6.1, it is asserted that, despite the coefficient of correlation between G and P being known, quantities a, b and c (that is, path coefficients or strengths of causal influence along different paths) cannot be derived from it.

Although directed against hereditarians, this kind of causal indeterminacy is more troublesome for environmentalists themselves. By comparing the two cases, I hope to prove that mere *statistical* information about correlations (together with some background causal assumptions) allows important conclusions about the *causal* influence of genes, whereas a similar inference from statistical data to the causal impact of environment is in general impermissible.

Genes (G) can influence phenotype (P) directly or indirectly, through environment (E). The strength of genetic influence on the first leg ($G \rightarrow$

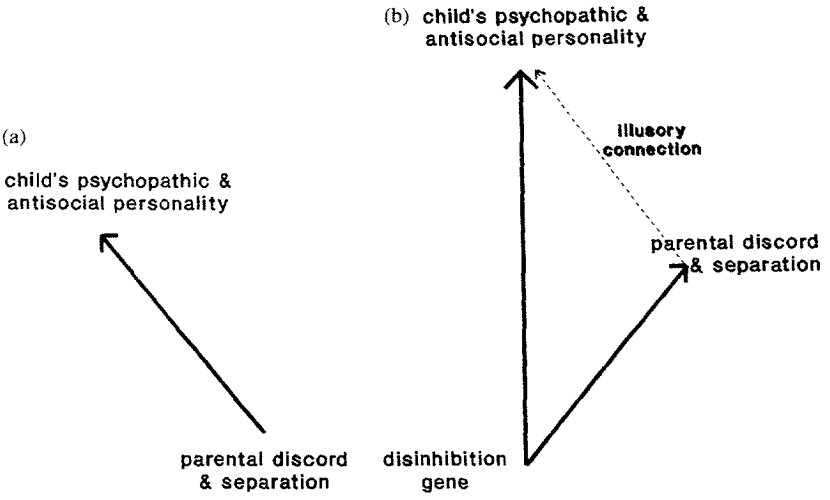


Figure 6.2a. Environmentalist hypothesis. Figure 6.2b. Hereditarian hypothesis.

P) is a, while on the other leg ($G \rightarrow E \rightarrow P$) it is $b.c$. The second value is obtained with the help of the so-called "chain rule" (see Heise 1975, 52), which states that the strength of causal influence along the compound path equals the product of path coefficients of all individual links. Furthermore, and this is the most important piece of information here, path analysis tells us also that the coefficient of correlation between G and P is equal to the *total* causal impact of G on P. That is, $r_{GP} = a + b.c$. In other words, r_{GP} provides us with information about how strongly G determines P, although we cannot (from these data alone) learn more precisely to what extent G exercises its influence directly and to what extent it does this via E. What differentiates the method of path analysis from the elementary "correlation-to-causation" fallacy is that, as its discoverer S. Wright said, "[T]he method of path coefficients is *not intended to accomplish the impossible task of deducing causal relations from the values of the correlation coefficients*. . . . The method depends on the *combination* of knowledge of the degrees of correlation among the variables in a system with such knowledge as may be possessed of the causal relations" (quoted in Duncan 1971, 135; emphasis added). So, in our example too, the background causal assumptions built into figure 6.1 are (a) that there are no causal loops (nonrecursiveness); (b) that there is no causal influence of environment upon genes; and (c) that there is no G-E statistical interaction. The last assumption is justified in the view of the fact that we are here interested in methodological obstacles created specifically by G-E correlation itself.

What about the knowledge of environmental causality under the conditions represented in figure 6.1? The environment acts on P only directly, and the strength of its causal influence is represented by c. In contrast to the previous case, however, it is now impossible to deduce any causal information from knowledge about how strongly E and P are correlated: r_{EP} does not authorize any inference about c. To see this, one need only note that r_{EP} can have the maximal value 1 and c can still be zero—if both P and E are directly and totally determined by G. Whatever the strength of correlation, *no* causal connection is guaranteed.

The crucial asymmetry can be summarized as follows. From the statistical fact "Similar genes—similar phenotypes" (together with some background causal assumptions), hereditarians are in a position to infer correctly the *total* causal impact of genes on phenotype. It is only when they attempt to refine this knowledge and to divide the total causal strength over the two paths that they are exposed to error: They can mistake a *direct* cause for the *indirect* one, or vice versa. Environmentalists fare much worse. Starting with the statistical fact "Similar environments—similar phenotypes", they cannot squeeze out any *causal* claim. The error to which they are susceptible, when they hazard a guess, is much more serious: They can mistake a *spurious* cause for the *real* one.[7]

To flesh out this abstract possibility of error in our present field of interest, let us borrow an example from a working geneticist. D. E. Comings cites a psychological hypothesis according to which "the psychopathic or antisocial personality was due to separation of the child from its parents at an early age" (1989, 457). The hypothesis is represented in figure 6.2a. He then considers an alternative *genetic* explanation which would unmask the environmental influence as entirely spurious. Perhaps there is a common disinhibition-disorder gene that causes in a parent marital chaos and separation, and in the child psychopathic and antisocial personality. As shown in figure 6.2b (adapted from Comings), despite a significant correlation between parental separation and the child's psychopathic personality there is no causal connection between the two. On this account, "the problem is not something the parents did to their children but rather something they gave to their children—their genes" (ibid., 458).

It matters little which of the two hypotheses is actually true. The point of the example was merely to exhibit the important causal asymmetry in the light of which it must seem ironic that some environmentalists used difficulties in inferring causality from statistical correlation as a *special*

[7] "Ordinarily we would consider a failure to control for intervening variables as a less serious error than shortcomings in the control for prior variables. Clearly, the distinction between spurious and causal effect is of greater importance than the distinction between the two kinds of causal effect" (Hellevik 1984, 27–28).

problem for hereditarianism. For two other examples which, by possessing the same causal structure, can also illustrate our general point, see Mednick et al. (1987, 85) and Erlenmeyer-Kimling (1972, 187).

7. Conclusion. The use of ANOVA in behavioral genetics (and elsewhere) requires caution. Applying the technique uncritically can lead to inferences that distort the picture of actual causal connections. Therefore, it is perhaps good from time to time to be reminded, as it is again done most recently (Wahlsten 1990, 152), that a large sample size is necessary for the test of statistical interactions; that if the functional relationship of two factors is nonadditive, then it makes no sense to speak of mutually exclusive and independent causes; that ANOVA is appropriate in some situations but not in others; and so forth. But pointing to all these possible pitfalls and limitations cannot support the strong claim that we should abandon the goal of partitioning variance among mutually exclusive causes and of calculating heritability coefficients (ibid., 109), or that the ANOVA approach is somehow irremediably and intrinsically defective as an instrument in the search for causes. An occasionally misused method is not a useless method.

REFERENCES

Blalock, H. M., Jr. (1979), *Social Statistics*. 2d ed. New York: McGraw-Hill.

Block, N. J. and Dworkin, G. (1976), "IQ, Heritability and Inequality", in N. J. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House, pp. 410–540.

Bouchard, T. J., Jr. (1987), "The Hereditarian Research Program: Triumphs and Tribulations", in S. Modgil and C. Modgil, (eds.), *Arthur Jensen: Consensus and Controversy*. New York: The Falmer Press.

Bouchard, T. J., Jr. and Segal, N. L. (1985), "Environment and IQ", in B. B. Wolman (ed.), *Handbook of Intelligence: Theories, Measurements, and Applications*. New York: Wiley, pp. 391–464.

Box, G. E. P.; Hunter, W. G.; and Hunter, J. S. (1978), *Statistics for Experimenters: An Introduction to Design, Data Analysis and Model Building*. New York: Wiley.

Burian, R. M. (1978), "A Methodological Critique of Sociobiology", in A. L. Caplan (ed.), *The Sociobiology Debate*. New York: Harper & Row, pp. 376–395.

———. (1981–1982), "Human Sociobiology and Genetic Determinism", *The Philosophical Forum 13*: 43–66.

Cattell, R. B. (1982), *The Inheritance of Personality and Ability*. New York: Academic Press.

Comings, D. E. (1989), "Presidential Address: The Genetics of Human Behavior—Lessons for Two Societies", *American Journal of Human Genetics 44*: 452–460.

Crow, J. F. (1990), "How Important is Detecting Interaction?", *Behavioral and Brain Sciences 13*: 126–127.

Crow, J. F. and Kimura, M. (1970), *Introduction to Population Genetics*. New York: Harper & Row.

DeFries, J. C. (1967), "Quantitative Genetics and Behavior: Overview and Perspective", in J. Hirsch (ed.), *Behavior-Genetic Analysis*. New York: McGraw-Hill, pp. 322–339.

———. (1972), "Quantitative Aspects of Genetics and Environment in the Determination

of Behavior", in L. Ehrman, G. S. Omenn and E. Caspari (eds.), *Genetics, Environment and Behavior*. New York: Academic Press, pp. 6–16.
Dobzhansky, T. (1955), *Evolution, Genetics, and Man*. New York: Wiley.
Duncan, O. D. (1971), "Path Analysis: Sociological Examples", in H. M. Blalock, Jr. (ed.), *Causal Models in the Social Sciences*. Chicago: Aldine, pp. 115–138.
Erlenmeyer-Kimling, L. (1972), "Gene-Environment Interaction and the Variability of Behavior", in L. Ehrman, G. S. Omenn and E. Caspari (eds.), *Genetics, Environment and Behavior*. New York: Academic Press, pp. 181–208.
Falconer, D. S. (1964), *Introduction to Quantitative Genetics*. Edinburgh: Oliver & Boyd.
Feldman, M. W. and Lewontin, R. C. (1975), "The Heritability Hang-up", *Science 190*: 1163–1168.
Fuller, J. L. (1972), "Discussion", in L. Ehrman, G. S. Omenn and E. Caspari (eds.), *Genetics, Environment and Behavior*. New York: Academic Press, pp. 17–21.
Futuyma, D. J. (1986), *Evolutionary Biology*. Sunderland, MA: Sinauer Associates.
Gould, S. J. (1977), "Biological Potential vs. Biological Determinism", in *Ever Since Darwin*. Harmondsworth: Penguin, pp. 251–259.
Hays, W. L. (1973), *Statistics for the Social Sciences*. 2d ed. New York: Holt, Rinehart & Winston.
Heise, D. R. (1975), *Causal Analysis*. New York: Wiley.
Hellevik, O. (1984), *Introduction to Causal Analysis: Exploring Survey Data by Crosstabulation*. London: George Allen & Unwin.
Herrnstein, R. J. (1973), *I.Q. in the Meritocracy*. Boston: Atlantic Monthly Press.
Hirsch, J. (1976), "Behavior-Genetic Analysis and Its Biosocial Consequences", in N. J. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House, pp. 156–178.
Jencks, C. (1980), "Heredity, Environment, and Human Policy Reconsidered", *American Sociological Review 45*: 723–736.
Jencks, C. et al. (1975), *Inequality*. Harmondsworth: Penguin.
Jensen, A. R. (1969), "How Much Can We Boost IQ and Scholastic Achievement?", *Harvard Educational Review 39*: 1–123.
———. (1972), "Discussion", in L. Ehrman, G. S. Omenn and E. Caspari (eds.), *Genetics, Environment and Behavior*. New York: Academic Press, pp. 240–246.
———. (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.
———. (1981), *Straight Talk about Mental Tests*. London: Methuen.
Kitcher, P. (1985), *Vaulting Ambition: Sociobiology and the Quest For Human Nature*. Cambridge, MA: The MIT Press.
———. (1987), "The Transformation of Human Sociobiology", in A. Fine and P. Machamer (eds.), *PSA 1986*, vol. 2. East Lansing: Philosophy of Science Association, pp. 63–74.
———. (1990), "Developmental Decomposition and the Future of Human Behavioral Ecology", *Philosophy of Science 57*: 96–117.
Layzer, D. (1974), "Heritability Analyses of IQ Scores: Science or Numerology", *Science 183*: 1259–1266.
———. (1976), "Science or Superstition? A Physical Scientist Looks at the IQ Controversy", in N. J. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House, pp. 194–241.
Lewontin, R. C. (1976), "The Analysis of Variance and the Analysis of Causes", in N. J. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House, pp. 179–193.
———. (1982), *Human Diversity*. New York: Scientific American Books.
———. (1983), "Gene, Organism and Environment", in D. S. Bendall (ed.), *Evolution from Molecules to Men*. Cambridge, England: Cambridge University Press, pp. 273–285.
Lewontin, R. C.; Rose, S.; and Kamin, L. (1984), *Not in Our Genes*. Harmondsworth: Penguin.

Loehlin, J. C.; Lindzey, G.; and Spuhler, J. N. (1975), *Race Differences in Intelligence*. San Francisco: W. H. Freeman.

Maynard Smith, J. (1989), *Evolutionary Genetics*. Oxford: Oxford University Press.

McGue, M. (1989), "Nature-Nurture and Intelligence", *Nature 340*: 507–508.

Mednick, S. A.; Gabrielli, W. F.; and Hutchings, B. (1987), "Genetic Factors in the Etiology of Criminal Behavior", in S. A. Mednick, T. E. Moffitt and S. A. Stack (eds.), *The Causes of Crime*. Cambridge, England: Cambridge University Press, pp. 74–91.

Plomin, R. (1986), *Development, Genetics, and Psychology*. Hillsdale, NJ: Lawrence Erlbaum.

Plomin, R.; DeFries, J. C.; and Fulker, D. W. (1988), *Nature and Nurture During Infancy and Early Childhood*. Cambridge, England: Cambridge University Press.

Plomin, R.; DeFries, J. C.; and Loehlin, J. C. (1977), "Genotype-Environment Interaction and Correlation in the Analysis of Human Behavior", *Psychological Bulletin 84*: 309–322.

Plomin, R.; DeFries, J. C.; and McClearn, G. E. (1980), *Behavior Genetics: A Primer*. San Francisco: W. H. Freeman.

Roberts, R. C. (1967), "Some Concepts and Methods in Quantitative Genetics", in J. Hirsch (ed.), *Behavior-Genetic Analysis*. New York: McGraw-Hill, pp. 212–219.

Rose, S. (1986), "The Limits to Science", in S. Rose and L. Appignanesi (eds.), *Science and Beyond*. Oxford: Blackwell, pp. 26–37.

Scarr-Salapatek, S. (1976), "Unknown in the IQ Equation", in N. J. Block and G. Dworkin (eds.), *The IQ Controversy*. New York: Random House, pp. 113–130.

Schiff, M. and Lewontin, R. (1986), *Education and Class: The Irrelevance of IQ Genetic Studies*. Oxford: Clarendon Press.

Sober, E. (1984), *The Nature of Selection*. Cambridge, MA: The MIT Press.

Stearns, S. C. (1989), "The Evolutionary Significance of Evolutionary Plasticity", *BioScience 39*: 436–445.

Stinchcombe, A. (1968), *Constructing Social Theories*. New York: Harcourt, Brace & World.

Sultan, S. E. (1987), "Evolutionary Implications of Phenotypic Plasticity in Plants", in M. K. Hecht, B. Wallace and G. T. Prance (eds.), *Evolutionary Biology 21*. New York: Plenum Press, pp. 127–178.

Taylor, H. F. (1980), *The IQ Game: A Methodological Inquiry into the Heredity-Environment Controversy*. Brighton: Harvester Press.

Waddington, C. H. (1957), *The Strategy of the Genes*. London: George Allen & Unwin.

Wahlsten, D. (1990), "Insensitivity of the Analysis of Variance to Heredity-Environment Interaction", *Behavioral and Brain Sciences 13*: 109–120, 146–155.