Lewontin’s verdict does not lack clarity: he thinks that heritability analysis is not helpful in explaining differences between means for different human groups or generations. Biol Theory Integr Dev Evol Cognit 2006;1 (In Press).


analysis of variance it is possible to estimate the amount of variation in behaviour that can be ascribed to genetic factors. This is captured in the symbol of $h^2$. If $h^2 = 0.8$ this means that 80% of the variation in behaviour can be explained by genetic factors.

It is crucial to note that a heritability analysis will be of little interest if it does not provide information about causes of behaviour in one way or another. If research shows that the heritability of IQ is 80%, this result has value only when it can be maintained that somehow IQ is to a large extent under the control of genes or that genes have at least an indirect impact on IQ. It is often claimed that this type of result says something only about populations and, hence, is not informative with regard to individuals. This is a mistake. The translation of information about populations to individual cases is fairly straightforward. If heritability of IQ is 80%, this result has value only when it can somehow be inferred that such an estimate is a causal relationship or whether the results should be interpreted more cautiously.

However, Lewontin, and many others after him, questions the possibility that an analysis of variance can be translated into a causal story.

The locality objection

Lewontin points out that heritability analysis is, by definition, a spatiotemporally local analysis. Heritability figures represent ‘a unique function of the present distribution of environment and genotypes’. As such it does not ‘provide us with the basic knowledge we require for correct schemes of environmental modification and intervention’. Such an analysis is different from the global analysis of causes. And furthermore it is impossible to make the step from an analysis of variance to a conclusion about causes. Consequently the analysis of variance boils down to a method ‘of estimating useless quantities’ [p. 410 in Ref. (1)].

Lewontin’s arguments to dismiss a causal interpretation of heritability figures on the basis of the locality objection do not seem sufficient. It is true that a heritability estimate does not give any clues as to how we can boost, for instance, the IQ of a population. The important question, however, is whether heritability figures can be taken to mean that genetic factors are, to a certain extent, responsible for the IQ measured. If we accept that a particular study provides a proper picture of the role of genes vs environment, it is not important whether the result is local. If it is local, this simply means that in other populations one may find different results. It does not follow that the local information is not relevant for understanding causes. The locality objection, then, is not an objection to the possibility of a causal interpretation of variation but relates to the generalizability of particular research findings. This is an empirical issue (cf. Sesardic). In this respect, it would surely be possible that a large number of studies on the determinants of IQ would provide information about the role of genetic factors in IQ in general.

Lewontin’s objection, however, also implies that a heritability analysis only provides a limited look at possible genotype–environment associated with phenotypes. This point is fully brought home in the objection, which we will now discuss.

Which model is appropriate?

In his paper Lewontin maintains that the proper study of causes pertains to relations between phenotype and genotype–environment combinations. The notion of ‘norm of reaction’ specifies the relations between those entities. It charts the different types of phenotypic outcomes of a given genotype as a result of various genotype–environment combinations. For complex human traits the actual study of the ‘norm of reaction’ of given genotypes is impossible ‘because the same genotype cannot be tested in a variety of environments’ (p. 190). The notion of ‘norm of reaction’, however, does provide a theoretical model that Lewontin utilizes to examine the analysis of variance. As this model purports to provide ‘the proper study’ of causes, it can be seen as a ‘reality model’. It provides a standard against which a heritability analysis can be properly assessed. After inspecting different reaction norms, Lewontin concludes that heritability analysis provides a false image of the causal relation between genotype, environment, and phenotype (p. 407).

Lewontin’s critique implies that an analysis of variance might very well show that genotype and high IQ correlate highly, but it does not necessarily follow that genetic factors have a large say in intellectual development (or vice versa). As a matter of fact, nothing follows!

Two questions are now of importance. First: does the concept of norm of reaction provide the right standard for judging heritability analysis? And second: is Lewontin’s judgment of the heritability analysis fair?

With regard to the first question, it must be stressed that although the norm of reaction concept can be seen as an alternative to heritability, it is a theoretical model that is in the same league as the analysis of variance. Both approaches assume the same basic and distinguishable entities: genotypes, behaviour (phenotypes), and environment. Of course, norm of reaction research requires that genotypes and environmental differences are known in detail. It is precisely this knowledge (which is unattainable with regard to human traits) that allows us to judge the heritability analysis. Lewontin, then, does not use an improper model in his judgement of the heritability analysis (this does not imply that the model itself is adequate;
that is a separate issue, which I will not address here). The way in which Lewontin employs this method is, however, not completely fair.

Lewontin asks the following question: given particular situations in which we possess knowledge about actual causes (by means of reaction norms), what would an (hypothetical) analysis of variance show? Well, an analysis of variance is necessarily based on outcome data, so has, by definition, much room for speculating about the processes (causes) that have led to the outcomes. This means that it is (nearly) always possible that outcome data are compatible with various processes, even ones that seem to contradict the outcomes. For example, we find high heritability, yet the behaviour at issue is actually the result of the impact of the environment (or of a specific genotype–environment combination).

This type of critique of the heritability analysis is rather common (I have used it myself). It does show, certainly, that an analysis of variance does not automatically suggest the correct causal story and, because this is so, it follows that an analysis of variance does not provide an adequate basis for a straightforward translation into causal terms.

Lewontin also holds that an analysis of variance provides misleading results, because it does not address the full range of reactions of a given genotype. An analysis of variance is restricted to a limited set of genotype–environment combinations and consequently cannot provide information in a general way about the effect of a genotype with respect to a set of environments. Consequently the result of an analysis of variance is restricted to a small set of genotype–environment combinations. Hence, the objection to the choice of model also supports the locality objection.

Though this critique of the analysis of variance is, strictly speaking, correct, it is nonetheless unfair. The unfairness lies in the fact that behavioural geneticists are denied the use of background theories and interpretative devices. A mundane example will illustrate my point. Research on height clearly shows that this is a heritable trait. An analysis of variance is restricted to a well-defined dataset.

An analysis of variance can capture interaction in the wider sense meaning that it can deal with the combination of two broadly defined variables (genotypes and environments) that taken together produce a result (behaviour). The analysis of variance can also deal with interactions in a statistical sense, albeit with some ramifications, as Wahlsten has shown.

Lewontin, then, probably does not think of ‘interaction’ in these terms. Indeed, Lewontin talks about ‘interaction’ in terms of ‘the joint operation of causative chains’. Various authors have elaborated this notion of interaction further, including Gottlieb. Gottlieb maintains that human development is a complex and dynamic process in which genetic factors, in combination with other biological and environmental factors, yield behavioural outcomes. Like Lewontin, Gottlieb argues that studying the correlations in a population cannot illuminate this process. Notice that this argument introduces a model—in this case a kind of dynamic system model. Because ‘reality’ (which is captured by that model) is interactive, it is not possible to say anything sensible about the separate impact of genes or environments. Consequently a method that is designed to single out the separate impact of genetic or environmental influences, cannot yield meaningful results.

There is no doubt that human behaviour is the result of complex interactions at various levels. As stated before, however, one should not use models to simply dismiss outcomes of research that start from different premises. These models can have a more constructive theoretical use. The challenge, as I see it, is one of explaining the outcomes of the heritability analysis within the parameters of an adequate model. This task has yet to be started. Its execution demands that the principled models not only allow for various levels of interaction, but also for various levels of descriptions of interactions. An analysis of variance operates on the level of overall effects; these may be quite different from the multitude of interactions which might be found at lower levels. The benefits of using this kind of model are that they (re)direct
research questions and might stimulate new explanations within behavioural genetics.

Conclusion

Let me conclude by sketching the awkward state that the research on the basic determinants of behaviour is in. On the one hand, there is a group of researchers, the behavioural geneticists, who use (advanced) statistical machinery in order to estimate the relative weight of genes vs environment on all sorts of human behaviour and behavioural traits. They continue to claim that heritability analysis provides important insight into the causes of all sorts of behaviour. On the other hand we have a more heterogeneous group of researchers and theorists, who can be labelled as ‘interactionists’. Interactionists, generally speaking, view the development of human behaviour and human traits as the result of a multilevelled process of interaction, which cannot be understood in terms of the impact of separate factors, be they genetic or environmental. Consequently, interactionists do not attach any weight to the hard won data of behavioural geneticists. They rather look at experimental (often animal) research for causal knowledge, and tend to produce more advanced models rather then deliver new insights. There is, I think, much to be gained by figuring out in what ways heritability analysis would fit with the optimal model. In that way the analysis of variance is still an enterprise that differs from the analysis of causes, but it would nonetheless contribute to it.

References