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Causes of Variability: Disentangling Nature and Nurture*

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Was du ererbt von deinem Vätern hast, Erwirb es, um es zu besitzen.

(Goethe, *Faust*, Part I, Scene 1)[1]

1. OBJECTIVES

People differ. From the very beginning of life even the standard forms of behavior are plastic, varying not only in their quality but also in the schedule of their growth. For example, developmental studies (Bayley 1969) tell us that the age at which infants begin to make crawling movements can vary anywhere from three days to three months. Although infants usually respond to a voice by three weeks, that response may first occur in a range from one to eight weeks. When it comes to walking, taking the first few steps alone can happen anywhere from age nine months to seventeen months. Speech generally develops by fourteen months, at which time we may expect infants to say a couple of words, but this achievement can occur as early as ten or as late as twenty-three months. Thus even highly channeled behavior, like the development of motor skills or language acquisition, shows an enormous range and variability. The same is true for virtually all other items of human activity, including the many aspects of cognition and personality. Part of understanding human beings involves understanding the sources of individual differences that make up this variability.

Perennially, scientists and social thinkers have looked to two polar sources for this understanding; namely, to heredity and to the environment. The priority dispute that results from placing primary emphasis on one or another of these factors has come to be known as the nature-nurture controversy. There is no necessity, however, to regard these two sources as conflicting. For given the range of variability to be explained one could look for a compatibilist resolution and expect that both heredity and environment have contributions to make in

most cases. That is, one can expect that they each account for some portion of the total variability. (Indeed, one might anticipate that even when the contributions of nature and nurture are combined there will still be a significant amount of individual difference to be explained.) In the abstract, such a compatibilist strategy with regard to the nature-nurture controversy seems attractive and plausible. Whether it remains so in the field, however, depends on whether one can sort out the respective contributions of heredity and environment convincingly, and thereby effect a reconciliation.

When we describe heredity and the environment as sources of individual difference and when we propose to investigate the extent to which they can account for (or explain) behavior, we adopt a causal idiom and treat studies of human behavior as causal disciplines. This is certainly the idiom and attitude of dominant approaches to the human sciences, which makes these sciences an especially good testing ground for philosophical ideas concerning causality, its nature and implications.[2] The examination of the nature-nurture issue below uses this opportunity to examine the determinability of causes and the philosophical idea that causal analysis commits one to belief in the reality of the causal agents. Thus this essay has two objectives: to investigate the extent to which one can apportion individual differences to the effects of heredity or environment, and to investigate the extent to which the scientific techniques employed in this area support belief in a causal order and realism over the causes of behavior. These objectives are strongly connected.

2. NATURE AND NURTURE

In the 1970s public discussion of nature versus nurture centered on the IQ controversy that surrounded the writings of Jensen (1969), Herrnstein (1971) and Shockley (1972). Concern focused on the heritability of IQ and the implications for that of educational and social policy, and also for the conduct of scientific research (Tobach and Proshansky 1976). That controversy also generated reflections on the methodological problems involved in the nature-nurture issue; especially on the question posed above of whether for significant human behavioral traits we would ever be able to sort heredity from environment in a reasonably satisfactory way. One of the most thoughtful contributions to the public debate on this issue was made by David Layzer, who identified the conditions required for an adequate study.

> Nevertheless, heritability can still be defined as the purely genetic fraction of the phenotypic variation. The question now arises: Can this purely genetic contribution be estimated from the appropriate measurements of correlations between genetically related persons? The answer turns out to be yes—*provided that, in the sample population for which measurements are carried out, genotype and environment are statistically uncorrelated.* In experimental animal populations, this condition may be insured by placing newborn animals in random environments. In natural human

> populations, on the other hand, it is probably impossible to eliminate genotype-environment correlation. Genotype-environment correlation is always present when children are reared by biologically related person. For adopted children, the effects are smaller but not entirely absent. Thus it would seem a hopeless task to determine the heritability of a phenotypically plastic trait in a natural human population. The data theoretically available in these circumstances are simply not adequate to the task. (Layzer 1976, 69)[3]

Almost as these words were being written, in 1974, at the Institute for Behavioral Genetics at the University of Colorado, Robert Plomin and John C. DeFries began plans for the Colorado Adoption Project (CAP), a landmark adoption study designed to answer questions about genetic versus environmental influences on human development. Indeed, the study was designed to address precisely the questions concerning which Layzer and others (e.g., Lewontin 1975) were so pessimistic. In work that is still going on, the project planned to study its subjects from infancy through adolescence. The report of the project published over ten years later (Plomin and DeFries 1985) covers only the first two years of life. It involves data from 182 infants adopted approximately within a month of birth and 165 matched control infants (nonadopted), all of whom were tested at 12 and at 24 months of age in lengthy home visits. The test data involve infant measures for cognitive development, communication, personality-temperament, behavioral problems, motor development, and health. The testers also made videotaped observations of mother-infant interactions. Data on the physical, social, and emotional aspects of the environment (everything from toys, verbal stimulation, and the family structure to noise levels, lighting, and the quality of the neighborhood) was extracted from lengthy interviews with the mothers, self-reports, and observation-interview instruments. The parents were also given a battery of tests covering cognition, personality, interests and talents, behavioral problems, drug use, and demographic background. The results involve a massive amount of information of all sorts, leading to virtually endless parent-child comparisons that can be processed and examined in many different ways. Although not perfect, the variety of techniques and tools used to collect information and the sensible way the information was handled is very impressive. A study on this scale, and executed with such care and good sense, is just the place to look if one wants to understand the possibilities of sorting nature from nurture.

The theory behind the study follows the random sorting paradigm alluded to by Layzer, above. Biological parents and their adopted-away infants share heredity but not environment. When the data from an adoption study is sorted for this combination the study approximates the ideal of randomizing family environment to reveal the effects of heredity on genetically related individuals. Adopter parents and their adopted infants share environment but not heredity. When the data is sorted *this* way the study approximates the ideal of randomizing heredity to reveal the effects of family environment on non-genetically

related individuals. Thus adoption studies involve a powerful twofold design that seems, in principle, capable of disentangling the effects of heredity and environment, effects that covary in the nonadoptive family.[4] But they can do even more. For by adding a control group of nonadoptive families one can use this covariance to support further inferences by comparing the correlation of traits in the control, where the effects of heredity and environment are compounded, with the correlation of traits in the adoptive families where these effects, presumably, are not. Thus, for example, if one takes height to be a heritable trait then one would expect a greater correlation between parent height and child height among the control families than among the adoptive families. The same would be true for features of cognition, to the extent to which they are heritable. On the other hand if sociability, for example, were determined by environmental factors alone, then it seems that one would expect no difference in the correlation of parent sociability with infant sociability between the control and adoptive families, since heredity would not enter in.

3. CAUSAL ANALYSIS AND CAP

CAP uses path analysis and causal models to make the inferences sketched above quantitative and rigorous. The models employ directed arrows to indicate the path from cause to effect, and the techniques of partial regression (or "path coefficients") to quantify the causal influence and to compute expected correlations between the causal factors.[5] One can then check the model from the data in the normal hypothetical-deductive way. The makers of the study are quite clear-headed about the use of these models. There is, for example, no suggestion that one could extract causal conclusions from the correlational data alone. The causal conclusions that are drawn rely on the empirical goodness of fit of the causal models and on the network of causal hypotheses that go into their construction. There is, nevertheless, a special vulnerability in the modeling technique that shows up dramatically in this study (and in others in behavioral genetics); namely, the nonspecificity of the hypothetical causal factors. The study cannot identify exactly what genetic or environmental factors are at work nor describe how the putative causal effects are supposed to have been achieved. In short, there is no detailed causal story to tell. The crosschecks we normally expect for causal accounts, including independent information about the particulars of the supposed causes, are absent here. This is a failure of purely structural models in general, and one that makes it reasonable to hold off crediting their limited predictive success to the accuracy of their underspecified descriptive hypotheses, at least until some reliable, independent details are available. I take the critical point here to be very serious, but I shall not press it below for I want to look at other problems about the modeling in examining the causal conclusions of the study and how they are drawn.[6] Since the study is really too massive for any adequate survey here of its results, I will focus instead on just one of the conclusions, one that the authors themselves highlight, and concentrate the discussion there.

The conclusion I want to look at is one of the most intriguing results of the study; namely, that even where the environment clearly influences the development of a trait, that influence is also mediated by heredity. Thus, infant sociability seems to be influenced by the character of the home environment (i.e., open family styles make for more outgoing babies). But it would also appear that genotype controls the extent to which this environmental influence takes hold. The authors describe the evidence for this as the "most exciting" of several unexpected results of their analysis of environmental measures (Plomin and DeFries 1985, 340). The *New York Times* agrees and recently featured the finding in an article that began by referring to chromosomes as providing a "blueprint for personality" and then went on:

> The question today is no longer whether genetics influence personality, but rather how much and in what ways. The answers emerging in the last few years primarily from long-term studies of twins and adopted children, bring increasing clarity to the nature/nurture debate. While environmental forces *can* help shape temperament, it is apparently equally true that genes can dictate an individual's response to those environmental forces. (Franklin 1989, 36)

The CAP data showed a generally higher correlation between environment-infant characteristics among biological families than among adoptive families. In particular, the correlation between (roughly) family sociability and (roughly) infant sociability was more than twice as high (0.34) among the control families than it was among the adoptive ones (0.16) (Plomin and DeFries 1985, 219). Thus the authors infer that heredity mediates the effect of family environment on infant sociability. To support the inference the study makes use of causal models whose relevant features I reproduce below in a somewhat simplified form.

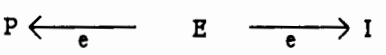

FIGURE 1a

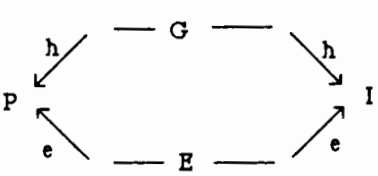

FIGURE 1b

Path models for parent (P)-infant (I) resemblance in (1a) adoptive families and (1b) biological families.

In these diagrams *E* represents shared family environmental influences and *G* (additive) genetic factors. The relevant parent and infant phenotypes are

given by P and I, respectively, which we associate with the relevant CAP measures for parent and infant sociability.[7] The proportion of the phenotypic variation due to E is just the contribution that the path through E makes to the correlation Corr(P, I) between P and I. The simple rules of path analysis tell us to compute that contribution by multiplying the causal factors (path coefficients) associated with each causal arrow in the path. Thus both in figures (1a) and (1b) we find that E is responsible for the same fraction of the phenotypic variation; namely, e^2. The IQ controversy centered on the heritability of IQ; that is, on the proportion of the variance of IQ that results from genetic factors. For diagram (1b) above, heritability (in what is sometimes called the "narrow" sense) is just the contribution the path through G makes to the correlation Corr(P, I); namely, h^2. Finally, to find the correlation between factors in a path diagram just add up the contributions from all the paths connecting them. So in figure (1a)

$$\text{Corr}(P, I) = e^2 \tag{1A}$$

Whereas in figure (1b),

$$\text{Corr}(P, I) = h^2 + e^2 \tag{1B}$$

My pictures here oversimplify the causal analysis in several respects. I have ignored the fact that in the biological families only half the genes are shared between parents and offspring (which would reduce h^2 in (1B) by a factor of 2, assuming random mating). I have left out factors corresponding to nonshared environmental influences and, for the adopting parents, nonshared genetic influences too. These would add some arrows pointed at P and I, but no new paths connecting them. Hence these influences would not affect Corr(P, I). There are various other factors that could be introduced into the models to make them more realistic, but whatever their complications the basic logic would be the same as that represented above: the control parents contribute genes and environment to their children, the adoptive parents environment only.[8]

Equations (1A) and (1B) imply that, unless sociability is not heritable at all (i.e., unless $h = 0$), Corr(P, I) should be greater in the control group than in the adoptive group. Since the data does show greater correlation between family sociability and infant sociability among the control families, it seems to support the conclusion that heredity is a causal factor in the transmission of sociability. One could even do more and use the quantitative features of the model (or a suitable refinement), together with the correlational data, to estimate the heritability of sociability, i.e., the proportion of the variance in sociability that is due to genetic factors. It turns out, however, that the data on sociability used in these considerations is not so straightforward, and that the report in the *Times* is seriously misleading in this regard. The study used two different kinds of instruments to measure characteristics of the family and features of infant behavior. One kind we might call "interpersonal." It involved home visits, on-site observations, and independent evaluation by third parties of the family, the child, and the family-child interactions. When family and infant sociability is measured by these interpersonal means *no significant differences occur between the*

biological families and the adoptive ones. There was, however, a different and "personal" measure as well. (See note 4.) It involved self-evaluation by the mothers of the family environment and of the child. It is only with respect to this personal measure that the correlations increase in the control group. What does this mean?

The higher sociability correlations in the control group are taken to support the conclusion that heredity is a causal factor. But it does not seem to be a causal factor in the genesis of infant sociability as such. If the conclusion is valid, then heredity is a causal factor in the genesis of the *mother's perception* of infant sociability. So it is not the case that insofar as open families make for more outgoing babies genes play a role. *The data do not support that inference at all.* Rather when the family environment is perceived as open and expressive, the child tends to be perceived as less shy. Genes (somehow) play a role in fostering that perception. So we are not talking about infant sociability here, but about parents' (more specifically, mothers') impressions of the temperament of their children. What the data suggest is that "heredity interfaces with the environment by altering perceptions of environment and experience" (Plomin and DeFries 1985, 239). If this is correct there ought to be features of the parents that are related genetically to their perceptions of family and child. The authors consider whether this genetic link to perceptions consists in personality traits of the parents, perhaps in combination with IQ. But the data rule this out (Plomin and DeFries 1985, 220–22). We are left with the finding that there is a genetically based tendency to see our children as sociable when we see the family as relatively open.

4. MODEL BUILDING

I have emphasized how that conclusion depends on using the correlational data in conjunction with a causal model. The inference to heredity as a causal factor in the mediation of family-infant perceptions is highly sensitive to this dependence. It only emerges when the causal arrows are placed just so. Consider, for example, the following models of the way heredity can mediate between parents' perception of the family (*P*) and their perception of the temperament of the infant (*I*).

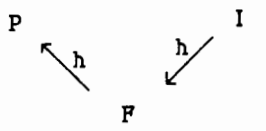

Figure 2a

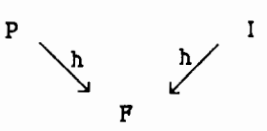

Figure 2b

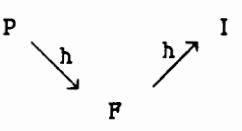

Figure 2c

In figure (2a) we might suppose that there is some genetic factor *F* such that *perception* of infant sociability (*I*) causally activates *F* (whose expected value changes with respect to *I* at rate *h*), and *F* in turn causally influences

perception (*P*) of family sociability (at the same rate).[9] If this causal network were the operative one, from the point of view of heredity, then we could replace the *G*-path on top of figure (1b) by (2a). Equations (1) would still hold, and the CAP correlational data would support this revised model just as well as they do the original. But in the revised model that incorporates (2a) genetic factors are not a common cause of *P* and *I*, as in (1b), but rather a transmitting or intermediate cause. Indeed the supposition that (2a) incorporates is just this: that the way a mother perceives her biological child tends to trigger genetically based responses that influence the way she perceives the family unit. This is certainly a sensible suggestion, or at least not less sensible than the conjecture raised in the study that genes alter perceptions of both the family environment and the experience of the child. Figure (2c) represents a suggestion similar to that of (2a), but with the causal flow reversed. In (2c) the causal chain starts with the perception of the family, which influences the perception of one's biological child via some genetic intermediaries.

Figure (2b) represents something different. Here, presumably, there is some genetically related factor *F* that is the common causal *result* of the perception of infant sociability and of the perception of family sociability. What could *F* be? Perhaps one possibility relates to self-image. One tends to shape a self-image in terms of the way one experiences significant people. In the case of a biological child, perception of the child's temperament in conjunction with experience of the family might well influence how the mother conceives of herself. If the mechanism of this image formation were genetically tuned and sensitive to the biological connection between mother and child (so that the image of myself as mother is linked genetically to the fact that I actually gave birth to a child like *that*) then its development in the case of an adopted child would be negligible. The causal path represented by (2b) would then occur in the biological families but not the adoptive ones, and equations (1) could govern the correlations, in accord with the CAP data.[10]

The suggestion just raised is worth considering further. In building causal models there is a tendency to look for mediating factors that are "objective"; for example, factors like personality traits or IQ, which the study considered and rejected. But one should not overlook the possibility that causes and effects may well be mainly at the level of beliefs and ideas; that they are, as it were, in the head. Thus even in (2a) and (2b) we might look for causal intermediaries that are subjective and which would link children to their biological parents, but not to their adoptive ones. Here again the idea of self-image is suggestive. If there were a special kind of self-image that the mother forms from the experience of her own child *just because she knows that it is hers,* then that might in turn influence her perception of the family as in (2a). Since the causal path would be open only for biological families, equations (1) would obtain. Conversely, a self-image derived from perceptions of the family might selectively influence how a mother views her own child, again, *just because she knows that it is hers.* In that case the causal path represented by (2c) would only be operative for a biological family and equations (1) would once more result. One could even go back to the

CAP model with this idea of a subjective intermediary and note that the only requirement for the model is that the top *G*-path in the biological case of (1b) not be available in the adoptive case of (1a). A self-image selectively tuned to the idea that the child is one's own satisfies this requirement. Hence the common cause *G* in (1b) might itself be just such an image. That image, moreover, could be a cultural and social product. It might, for example, be constructed from the idea that there is an intimate connection between the sort of person I am and the sort of person my child is. The transmission could be entirely cultural. No genes and no heredity need be involved.

So far one might characterize the discussion as an exercise in underdetermination. The CAP project provides data in the form of correlations (or multiple regressions). To interpret the data we identify the range of factors we think might be involved in terms of culture or environment and in terms of heredity. We arrange these factors in a reasonable causal model, fit the data to the causal paths of the model and then examine the extent to which the data satisfy further constraints that the model requires (as in equations (1)). To the extent to which the satisfaction is good, we count that as confirmation of the causal hypotheses of the model, which we then hold to be the "findings" or "implications" of the study. The suggestion in offering the models in figures (2a, b, c) is that there are alternative ways of arranging the causal arrows that fit the correlational data equally well. Proliferation of this sort is a well-known feature of causal modeling, one that Woodward (1988) refers to as its "central methodological problem" (n. 6, p. 260). The usual tactic in general discussions is to suggest that we can cut down on the array of possible models in real applications because in real life not all possible rearrangements of causes make sense.[11]

The discussion above shows just the opposite. Precisely in the context of the CAP study and the personal measures of family and infant that give rise there to the question of the role of heredity in the transmission of perceptions, each of the possible causal paths does indeed make sense. In this instance, contrary to the general methodological maxim, in real life nothing is ruled out. Hence the conclusion that "heredity interfaces with the environment by altering perceptions of environment and experience" is just one of several equally well-supported causal interpretations of the data, ones that do not give hereditary factors a primary causal role at all. The inference to a causal role for genetic factors was based on stronger parent-child correlations among biological families than among adoptive ones. The existence of sensible alternative causal models consistent with these correlations shows that neither the data alone nor their combination with sound methodological maxims allow one to draw the conclusion about the influence of heredity. Moreover, the discussion suggests that where causal models involving genetic factors fit the data there are plausible competing interpretations of the models in terms of cultural mediation of beliefs that involve no factors related to heredity at all. Thus both the pattern of causal influences and the character of the causal factors are severely underdetermined by the CAP data. So, to make inferences from correlations to causes, even in the presence of well-fitting causal structures, is to put oneself on very shaky ground.[12]

The usual remedy for this prescribed by the causal theorists is to supplement the assumptions about what causal relations are sensible with even stronger assumptions about the completeness of the causal analysis. This is, for example, the sort of program that Cartwright (1989) develops, relying on the common cause principle (see section 5, below) and even more general causal considerations. The difficulty is that although strong causal assumptions can indeed imply causal conclusions, one needs to investigate the soundness of the assumptions before those conclusions are detachable. Insofar as this is an empirical question it is a matter of gathering further correlational data which, once again, will be liable to various causal interpretations. Further causal assumptions will be needed to sort the new data. So as we go down the line we will need to make more causal assumptions, not less. This is not a procedure for narrowing down the class of models, but rather for expanding it. The remedy appears to be worse than the disease.

Let me give just one illustration of the problem. One might try to sort out the differences in causal order between (1b) and (2) by making a strong causal assumption to the effect that heredity is the origin of social perceptions in general. Thus genotype would determine a selective framework that would affect how we experience the world socially. One might call such a view Social Kantianism. This would pick out the common cause arrangement of (1b) as the right connection between the mother's perception of the family as a social unit and of the child as a social being. But Social Kantianism has further consequences. For instance, if twins were raised apart in different family environments Social Kantianism would suggest that insofar as they share a selective genetic framework they would tend to see their different families as socially similar. To investigate this we could get older separated twins to do the same sort of family social ranking as was done by the CAP mothers and then look at the correlation between their assessments of family styles. In fact the study has been done (reported in Franklin 1989), and with the expected result; namely, that twins tend to rank their families similarly. Of course proponents of the different placement of arrows in figure (2) might also take to the road of high-level causal assumptions. They could suggest that heredity is not the causal origin of but only plays a role in transmitting social perceptions, in accord with (2a, c). In the family context this would imply a feedback mechanism: perceptions of the family would cause some genetically mediated responses in a child which would in turn influence further perceptions of the family, and so on. Even so, one would expect that since twins share the same genes, the repeated operation of the genetic feedback loop would tend to wash out differences in perception and to exaggerate similarities. Thus the study of separated twins and their family perceptions will not distinguish Social Kantianism from the feedback hypothesis. There are, moreover, other explanations for the similarity of separated twins' social perceptions that have little to do with either hypothesis, while adding further to the causal proliferation. For example, if genes influenced personality so that twins tended to be similar socially, then it would not be surprising to find that different families responded similarly to socially similar children, and that the children then

perceived the families as similar. Since the assumption of genetic influence on personality runs counter to the CAP findings, however, maybe it is just that families tend to respond similarly to infants insofar as they look alike, as twins do, and the twins' perceptions of the families reflect that response. This raises an even further possibility. For maybe we tend to respond similarly to people who look alike because we *believe* that they *are* alike in terms of personality. (Probably we can all recall the experience of taking to new acquaintances right away just because they look like people we have known and cared for.) That tendency to respond to appearances may trade on beliefs about the relation of appearance to personality and to character, beliefs that may well be primarily social in origin and not genetic. What we see in all this is that checking on causal hypotheses simply generates more causal hypotheses. We are not eliminating the alternatives in figure (2) or the alternative social interpretations. We are not getting closer to the CAP conclusions, but moving farther away.

The situation, in fact, is even worse, as we can see by considering the model in figure (3) below.

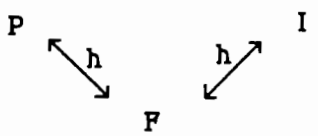

Figure 3

In accord with the conventions of path analysis, double-headed arrows signify correlations and not causal connections. Thus figure (3) represents a synthesis of figures (2) and the *G*-path of (1b), where all directed causal arrows, in whatever direction, are replaced by double arrows; that is, where all causal connections are replaced by correlations. Indeed it might be best to think of things the other way around. Since the CAP data are correlational to begin with, one might suppose that we start model building by arranging the data in a purely correlational representation as in figure (3). Notice that if we replace the *G*-path of (1b) with (3) then Corr(*P,I*) in the revised model will be exactly as in equation (1B). Thus when we represent the correlational data by correlational structures, we come out with a perfect fit. These structures, of course, do not permit causal inferences, but only inferences as to correlations.

Using the correlational structure of figure (3) one could not say that genetic mechanisms interface with the environment by *influencing* (or by *transmitting*) the perception of the family and the child. We could only say that genetic mechanisms interface with the environment in a way that shows a correlation with these perceptions. In stepping away from causal models in favor of correlational ones, however, we are still able to describe the central findings of the study and assess their implications for practice. For example, perhaps the most important finding about adoption was a negative one. The study found no important developmental differences between the CAP adopted and nonadopted

children in infancy. This is a correlational finding, well represented by structures like figure (3), and not a causal one. We might gloss it causally by saying that, according to the study, adoption is not harmful to the adopted infants. Whether we choose to report it this way or not, the finding itself is still useful in making adoption decisions, and comforting to both the biological and adoptive mothers concerned with the welfare of their children. Similarly, those interested in gender might be surprised to learn that no significant gender differences show up in the analysis of environment-infant correlations for the 185 relations between environmental factors and infant development in the CAP and in a comparison of mother-infant correlations in the control and adoptive groups. The authors report this as showing that girls and boys are equally susceptible to environmental influences. Again, whether or not one likes that causal reading, the correlational structure that corresponds to the data (assuming, as the study does, that it is reliable for predictions) certainly gives no support to child-rearing practices that involve treating girls and boys differently because one supposes that, constitutionally, they require it.

Of course if one were to stick only with correlational structures to model the data there would be no point in looking to the CAP, or other such studies, to sort out the relative influences of heredity and environment on child development. From the point of view of purely correlational structures heredity and environment are not "influences," they are just intermediate nodes in the correlational paths. They mediate the statistical relationships between various determinable factors or traits. As illustrated above, this gives us a great deal of useful information, but not about causes. Nevertheless, it does enable one to divide the covariance in behavioral traits into the proportion mediated by heredity and the proportion mediated by the environment, and to see what is left over. Working with correlational structures also enables one to add further factors to a model, to experiment with different configurations and different interpretations. Indeed these structures offer the same advantages of model building that causal models do, and they have one less disadvantage. Whereas causal models for a given data set with fixed factors are numerous and highly undetermined by the data, the correlational structure is unique, being the unique substratum out of which causal models arise by imposing one-way paths on the two-way correlational traffic. Thus correlational structures escape one of the two sources of underdetermination described above. The other source, the possibility of reinterpreting the nodes in the structure, of course, remains. Still, reducing the range of possible models is an advantage to correlational structures that should not be overlooked. It is the dividend we can earn by foregoing talk of "influences" and the like.

Nevertheless I do not propose that we abandon causal talk and the causal analysis of behavior. Rather I highlight the utility of purely correlational structures in order to emphasize that there is a generically different and yet reasonable way to process information from a study like CAP and to report its findings. The point is not just to repeat that we only get causes out of the data if we put causes in (i.e., if we use causal models and auxiliary assumptions). It is

especially to underscore that *with respect to each path* there is always a genuine choice of whether to treat it causally or not. The data do not determine which way to go. Real applications always present us with decisions about what to treat causally and what to treat as correlations only. Indeed, most working models do contain residual correlations: i.e., factors connected only by double arrows. Generally the choice of what connections to treat causally relates to the aims of the investigation (e.g., being set on determining the "influence" of heredity on IQ), the conception of the subject matter ("Surely," we say, "the way children are raised affects their character"), or other general causal convictions. Whatever the basis, there is a choice to be made. That means we have to recognize that one way of describing the relation of factors along a putative causal path is that they merely correlate with one another. This is always an option, and it might sometimes be a good one to take.[13]

5. CORRELATIONS AND CAUSES

There is a philosophical thesis, however, that runs counter to the liberal view advocated above; namely, Reichenbach's (1956) so-called principle of the common cause. That principle directs us to explain all correlations between factors as due either to direct causal connections between the factors or to common causes affecting both factors. It implies that in a *complete* causal model; that is, one in which all contributing (and inhibiting) causes are included, there will be no residual correlations; i.e., no double arrows. So we could rephrase the principle as requiring that a complete causal analysis leave no correlations unexplained. Why? Why must explanation be causal so that correlations are only explicable by means of causes? Why are correlations singled out in a completely context-free way so that they always need to be explained regardless of context or content? And why, finally, is explanation promoted as the primary mode in our approach to understanding nature?

The emphasis on explanation seems to me typical of an intellectualization of science that fails to appreciate the role played by experimental, practical, and policy considerations. The focus on explanations loses sight of how these practical concerns enter substantively into scientific inquiry. Like politics, science, too, is the art of the doable. Good scientific questions are the ones that we can get a handle on and investigate. The answers we get depend on the results of the investigation, which are a function of when we take the investigation to be over. Often enough that judgment depends critically on what resources and techniques are available, and whether we consider further effort likely to be relevant, from a practical or policy point of view. Thus experimental, practical, and policy considerations function crucially in determining what counts as a good question and what counts as an acceptable answer. Since scientific understanding accumulates with the accumulation of answers, explanation turns out to be just one element in understanding nature. This fact of scientific life suggests that sometimes the accumulation of reliable correlations that can be turned to a practical use may itself be counted as a suitable form of understanding. It is important to

bear in mind that the unmasking impulse, which pushes one always to get "behind" the phenomena, may itself not always be appropriate or reasonable.

Thus the global character of the common cause principle, that correlations always require causal explanation, betrays its distance from scientific life. While in the social sciences especially, the maxim to look for causes is a useful heuristic principle it is not more than that. It is not a scientific Golden Rule representing the Ideal Form of investigation, one whose transgression consigns the investigator to a realm of inferior beings (e.g., empiricists or other philistines). Rather, in the search for causes, as in all areas of science, one has to judge in the context of a specific inquiry whether it is reasonable to go on. That means one will sometimes judge correctly that it is not. I believe there are no set rules or principles for the operation of such judgments. They are tempered mostly by the history of an investigation, the immersion in the details of the subject, and one's sense, given the subject and its history, of what seems fruitful to pursue and what not. Reasonable people may disagree on what constitutes a fruitful course of action, and further investigation may either vindicate a party or show them to have been wrong.

Finally it is a mistake, I think, to hold up causal explanation as the only suitable kind. For surely the form of understanding provided by a scientific discipline relates to the content of the covering stories (the theories or models or whatever) that the discipline provides in context. In the quantum theory, for example, one acquires considerable understanding from the capacity to make very accurate predictions of the outcomes of certain well-defined experiments, although the theory does not provide much by way of causal accounts of the details concerning the production of the experimental outcomes. This is not because the theory is probabilistic, but because the theory works in terms of certain general descriptions of the phenomena that are structural and not causal at all. Of course we all know that quantum theory is peculiar. But relativity, which respects causality, is not. Yet if we ask why light travels with a constant velocity or why no causal signal can be transmitted with a speed faster than light, there is no causal answer. The explanation is once again structural. The very structure of space-time requires (or perhaps even embodies) these features. A similar situation prevails in Freudian psychoanalytic theory, which is also strongly causal. Psychoanalytic method relies on a correspondence between certain chains of associations and causal relations that connect the associated items, or their surrogates, psychologically. If we ask why this correspondence obtains, why one tends to produce the psychological causal order by following the chain of associations, however, the theory has no causal answer.[14] The fundamental theoretical structure is defined by a group of permissible transformations, just as in space-time theories. Transformation by association is one of the permitted ones. So, if you like, the structure of the mind requires that associations of a certain kind correspond to the psychological causal order. The point is that where we have a good causal story to tell, we can sometimes tell it to explanatory advantage. But whether we have a causal story at all depends on the particular case at hand. Thus to demand causal explanation, as the common cause

principle does, is to place a constraint on the form of a story independently of the subject matter. Since form and content are not dual, it is not the case that all correlations, or anything else, require causal explanation.[15]

6. CAUSAL CHOICE

Rejecting the common cause principle opens the door to incorporating some double arrows in causal models in a fundamental way; that is, without having to suppose that as knowledge develops in the future the double arrows will become single or become the terminus of some common causal arrows. It allows the possibility that even in a complete causal account, when all the causal arrows are in place, double-arrowed paths like figure (3) will still occur. As discussed in section 5, figure (3) signifies that heredity is not a causal factor in mediating perceptions of sociability of the family and the child. If data from the CAP do not discriminate between the *G*-path of (1b) and figure (3) then they do not determine whether to count heredity as a causal factor at all. Hence in rejecting the common cause principle we add to the query raised by figure (2), concerning what the causal connections are between heredity and perceptions of the family and infant, a further question as to whether those perceptions are connected to one another and to hereditary factors in a causal way at all.

If the argument concerning the possibilities raised by figure (2) is correct, then causes do not supervene on correlations, and the statistical data from investigations like the CAP underdetermine the causal order. Hence to place the various factors in a particular causal arrangement involves a judgment that goes beyond the information provided by the study. It represents a meta-empirical commitment to a particular causal flow. Although it may be reasonable to make such a commitment, it may also be reasonable to withhold it, or to place the causal order differently. I would say the same for the further question of whether, with regard to particular factors, one should be committed to connecting them causally. Since the findings of a study like CAP can be modeled in a number of different ways, it seems fair to urge caution over commitment to the details of any special representation, whether causal or not. Such caution, while not endorsing causal realism, does not amount to instrumentalism over causes either.

In general, instrumentalism cautions agnosticism with regard to the more theoretical elements (sometimes the "unobservables") of science. Instrumentalism correctly points out that we can use a theoretical structure, and take it seriously, without believing it to be literally true and so without believing in the reality of the entities that it employs (or to which it putatively refers). Agnosticism results when instrumentalism adds a recommendation that one tailor one's beliefs to accord with the following empiricist precept: do not believe what no empirical evidence can compel one to believe. Applied to the preceding discussion, instrumentalism would have us withhold belief in any particular causal order and in any particular causal connections. In short instrumentalism does not believe that causes are real.[16]

The position I have been trying to shape here is different. It does not begin with some entrenched scientific structure and recommend that we water down our beliefs about the structure to bring them in line with certain belief-warranting principles. That is bad belief management, requiring us to trade in scientific beliefs to pay for epistemological ones, regardless of context or content. Rather I have emphasized what all makers of causal models for data from a study like the CAP are well aware of; namely, that causal conclusions depend on a decision to employ models with particular causal arrangements, and that other models might equally well have been employed so far as the data go. I see no basis here for general agnosticism about causes. In particular, no special epistemological principles are imported to mold beliefs. Indeed, I recognize that there may be good reasons to impose models with certain causal connections and excellent reasons to accept such models, perhaps because of their fertility and the way they fit with other models or considerations that emerge from related investigations. I do not endorse any general epistemological strategy of restraint with regard to commitments or belief. Nothing of the sort follows from the fact that it is possible to believe less without suffering empirical loss. On the other hand, I point out that just because one *can* believe less the question of whether to do so may be an open one scientifically, and that withholding certain commitments, while not mandated, is possible and may be just as reasonable as the opposite, or maybe even more so. The situation in causal modeling presents us with the necessity to make judgments about how to proceed which are not forced on us by the data in the field. Such judgments, I would urge, are always to be found at the center of scientific life if we care to look with an open mind. The fact that we have considerable latitude in deciding whether to use causal models, and which factors to connect causally, does not support instrumentalism over causes. It does not support realism either.

The scientists involved in the CAP conclude that heredity is a significant causal factor in shaping our view of the social climate of the family and the temperament of our children. If we agree, then we accept genetic factors as causes. It is important to understand that we could do that instrumentally, merely accepting the genetic idiom as useful but without actually committing ourselves to believing that genetic factors are really there, acting as causes. For *acceptance* is a specification-hungry concept.[17] One accepts for a purpose, or with certain reservations, or under a certain description, and so on. We could also accept the conclusion realistically, admitting belief in some genetic causes whose exact specifications and mode of operation, as the authors suggest, are yet to be determined. But we may also just disagree with the conclusion drawn about heredity. The statistical inference to genetic causes that is based on higher correlations in the control group is not sound (section 4). Hence we might think it premature to put a causal gloss on the heritability factor at all. We might think that other causal models, ones that assign a different causal role to heredity, will stand the test of time better. We might raise the question of interpretation of the path diagrams. For example, we might believe that reinterpreting the *G*-nodes in terms of certain culturally transmitted factors, as suggested in section 4, will

turn out to be a better construal than that of heredity when all the study data are taken into account, not just the material on perceptions of sociability. Each of these options (and certainly there are others as well) offers a reasonable basis for assessing the results of the study using desiderata that scientists in the field would certainly recognize as relevant. No specifically philosophical doctrines are required for that purpose, neither realism, nor instrumentalism, nor general skepticism.[18] The multitude of options plainly shows that the data do not interpret themselves and that the conclusions drawn by the study are not the only reasonable ones to draw.

We interpret and we choose. But not in a vacuum. Reflections about nature and nurture are part of a continuing cultural dialogue. It is a reasoned activity that engages social and educational policy, affecting what kind of creatures we take ourselves to be and, hence, what kind of people we become. The polar categories themselves reflect our social conditions and aspirations and draw their force from other polarities common in the culture. For example, they seem to relate to fundamental dualities connected with our bodies, the inner and the outer, which in turn relate to mobility and to what we can control and accomplish, and what not. Despite this entrenchment, I would argue (but not here!) that none of these dualities is necessary in the sense that we need to employ the posited categories in order to achieve an adequate understanding of ourselves or perhaps of life more generally. In particular, it may be better to think about ourselves without imposing the categories of nature or nurture with all their associations. Nevertheless, these alternatives constitute the categorial framework from which work like the CAP study draws its stock of causal factors. Because we have a choice about what to learn from such studies, the conclusions we accept are bound to involve our feelings about the background framework and the issues that relate to it for otherwise there would be no basis for choice at all. This entanglement with features of culture at large is always present in science, in physics no less than in sociology, for science always requires evidential and interpretive choice that must involve ordinary commonsense judgments; that is, judgments drawn from concepts common in our culture.

Given the data produced in studies like CAP, behavioral genetics will be inclined to credit heredity with considerable causal influence, if not on temperament itself, then on our perceptions of temperament. They have their reasons. That, after all, is what the factors drawn from the categories they employ put them on the lookout for. They frame their models for just that purpose. When the data are favorable, they are all set to go with their models. On the other hand, one may wonder about the framework of nature and nurture, or of environment and heredity, and therefore wonder whether it would not be better for social thinking and scientific talents to be engaged with different concepts. As explored above, there are also questions to be settled about the evidence for these particular models and the necessity for particular paths being causal at all. Then there are the problems of independence and underspecification that are serious for the credibility of any such structural undertaking. All in all, I am not inclined to credit heredity with causal influence. I have my reasons too.

NOTES

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1. "What you have inherited from your fathers, acquire it to make it your own." Quoted by Freud (1949, 64) who holds the id to represent influences of heredity that we "make our own" through their interactions with family and cultural environment, represented by the development of the ego and superego.

2. I mean to include constructivist, hermeneutic, phenomenological, and ethnomethodological approaches in this characterization, for they use modalities such as "making," "constructing," "intending," "interpreting," "bracketing," "moving," "responding," and so forth, all of which are causal. The dichotomy that is sometimes drawn between these "interpretive" approaches and causal ones seems to me false. Both approaches are intermingled in all the sciences. To be sure, the formal techniques of causal analysis that are the focus of this essay may not be universal. The problems I develop about causal inference, however, are.

3. Note the callous attitude expressed here toward the separation of animal families for experimentation, an attitude more prevalent in the 1970s than it is now—or so I hope.

4. In adoption studies one has to worry about selective placement (i.e., matching adoptive to biological parents), whose effects amount to some covariance between gene and environment. However, the adoption agencies involved in CAP did not attempt to match adoptees to adopters, and other measurable sources of selectivity turn out to have been minimal. Still, the placement of adopted infants is far from random, and this is a possible weakness in the study to which critics, pursuing Layzer's line, could attend.

5. Kempthorne (1957) contains a standard introduction to causal path analysis. See note 9 for more on the path coefficients.

6. The problem about underspecification and the need for independent crosschecks in causal modeling is well illustrated by Horan (1989) and Lloyd (1989) in their discussion of biological models. Although they disagree about what can be pinned on or credited to the semantic view of theories, they do not disagree about the need for specificity and independence in taking predictive success to count in favor of the credibility of the model.

7. The study used a derived measure of family style called the FES (Family Environment Scales) personal growth factor, which we can take for P in the diagrams. This factor involves a self-rating by parents with regard to their being intellectual, active, permissive, and expressive. For I the study used the midparent CCTI (Colorado Childhood Temperament Inventory) measure for sociability. This measure is a rating by parents that relates to infant shyness, conceived of as a combination of emotionality-fearfulness and low sociability, rather than gregariousness. See Plomin and DeFries (1985), chap. 4.

8. For a detailed discussion of more complex causal models for the CAP data, see Fulker and DeFries (1983).

9. The path coefficient is derived from partial linear regression of the cause factor on the effect factor. It is best understood as an approximation to how much the average or expected value of the effect variable changes in the population as a result of a unit change in the variable representing the cause, other causes being held fixed.

10. I really have to emphasize the *could* here, for (2b) is different from the other path diagrams in that one cannot actually compute the correlation between P and I from it without additional assumptions. One could make such assumptions, however, or just put in the correlation "by hand" in a way that is compatible with the data.

11. E.g., see Blalock (1979).

12. Of course in some particular application it might happen that competing causal models and competing interpretations of the causal structures are ruled out by features of the context. The likelihood of such circumstances is pretty slim, but even so it would be

compatible with my conclusion here that inferences from correlations to causes, even in conjunction with causal models, are not reliable as such.

13. My (1989) argues that basic, residual correlations are a natural feature in an indeterminist worldview; for example, the worldview usually associated with the quantum theory.

14. Here may be one of the sources of Grünbaum's (1984) reservations about this aspect of psychoanalytic method. For a criticism of Grünbaum on this point, see Forbes and Fine (1986).

15. In this section I have concentrated on general considerations that lead one to reject the search for common causes as a sound requirement on theory construction. But there are more specific reasons, drawn from areas of biology and physics, where it appears that the common cause conditions simply cannot be met. For biology see Sober (1984), and for physics see van Fraassen (1985 and 1989), and my (1989). Cartwright (1989, chap. 6) makes an interesting suggestion for how to restore common causes in the physics' case, but acknowledges that her causal reconstruction has peculiarities of its own.

16. This is probably too broad. But it does come close to the point of view expressed by van Fraassen (1989, 109).

17. My (1990) develops this point about acceptance.

18. My (1986) explores the "no additives" theme touched on here and the nonskeptical reasons that lead one away from realism and instrumentalism more generally.

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