

Mobiles

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My colleagues and I (Turkheimer, Haley, D’Onofrio, Waldron & Gottesman, 2003) recently published a paper purporting to demonstrate that in relatively poor seven year-old twins, family environment is far more important than genes in the determination of individual differences in IQ. (In the portion of the sample raised in middle class or better environments, genes predominated, as they usually have in earlier twin studies.) We were fortunate enough to get the attention of Rick Weiss, the science reporter for the Washington Post, who wrote a story describing our results. As a result I had the opportunity to answer a great many questions about our findings from the lay public, interested government agencies, and other social scientists. By far the most common was this: Now that we can show that impoverished environments make a difference, what can be said about what the important environmental ingredients are?

This is a serious and well-intentioned question, and it was certainly tempting to speculate about schools, parenting, nutrition, peer groups, or perinatal care. But I didn’t, and not only for reasons of scientific reticence in the absence of good data. The honest answer to the question is that I don’t think there is anything in particular about the environment that is responsible for the effects of poverty, nothing that will set a fruitful course for future research, nothing that would make a particularly good intervention in impoverished homes, none of the things that one would like to be able to say. Yet I do believe in our finding: Differences in family environment are responsible for the majority of IQ differences among impoverished twins. The difficulty arises in the contrast between the idea of “environment” as it represented in twin studies, and the ways in which actual environments cause actual outcomes in actual children. As I explore these relationships in the essay that follows, I hope to illuminate what we can and can’t

learn about environmental effects on behavior, and by way of analogy say something about genetic effects on behavior as well.

Hopefully, readers of this volume have by now a basic understanding of the goal of twin studies, that is to partition variability of a trait into three parts: one part attributable to differences in genes, one part (the shared environment) attributable to differences among family environments, and a third (the nonshared environment) attributable to environmental differences among children in the same family. Part of the appeal of basic twin studies is that once you have found the twins, twin studies are very easy to conduct. In fact, it is quite possible to conduct a twin study and obtain estimates of the effects of “genes” and “environment” without measuring anything about the environment at all. For that matter, it isn’t necessary to measure anything about the genes either. All you do is measure a trait in twin pairs, and compute the twin correlations separately for identical (MZ) and fraternal (DZ) twins. The twin correlations are a measure of how similar twins are for the trait, so a comparison of the correlations for genetically identical MZ twins and the correlations for 50% genetically identical DZ twins indexes the contribution of genes to differences in the trait: double the difference between the correlations, and you get the genetic proportion of variance; double the DZ correlation and subtract the MZ, and you get the estimate for the family environment; subtract the sum of these estimates from 1.0, and you get the nonshared environmental portion.

The tendency of behavioral geneticists to reach conclusions about the environment without taking the trouble to measure anything about it has always rankled with environmentally oriented researchers (Wachs, 1983). Even when the environment

was actually measured in traditional twin studies, it was specified not in any detail, consisting (as was the case in our IQ study) of broad sociological measures of parental education, income, and occupational ratings, hardly the stuff of good environmental analysis. But once more finely-grained environmental measures were finally included in behavior genetic research, the payoff was significantly less than might have been hoped. Detailed measures of the family environment turn out to be substantially heritable themselves (Plomin & Bergeman, 1991, McGuire, 2003), and when relations between particular environmental variables and child outcomes are studied in contexts that allow researchers to estimate the specific environmental effects of one variable on another, there often seems to be nothing there (Reiss, Neiderhiser, Hetherington & Plomin, 2000).

In the kind of study I am describing, a researcher might record the number of negative communications between a mother and each of her two twin children, and the number of delinquent acts in which each of the children engage. The goal of such research is to show that the twin who is the target of more negative communications (an example of nonshared environment, since two twins raised by the same mother receive different environmental inputs) is also the twin who engages in more delinquent acts. Mary Waldron and I have conducted a meta-analysis that summarized the effects of all studies then available that used this design (Turkheimer & Waldron, 2000). We found that specific measures of the environment account for no more than a couple of percent of the total variability in outcome, despite the fact that the unspecified variance component called “environment” continued to explain most of the variability in outcome.

So following our example of adolescent delinquency, twin studies show that much of the variability in delinquency is “environmental,” especially the nonshared

variety that makes children raised in the same family different from each other. But what is it about the environment? Parents who make more negative comments to one sibling than the other? Peer groups? Schools? Our meta-analysis showed that studies of the relation between any of these particular measures and outcomes in children yielded very little. After almost two decades of research we know little more about the ingredients of the nonshared environment than we did when we started.

What is going on here? Finding a way out of the apparent contradiction between the predominance of the environment when it is studied broadly, and subsequent disappearance of those same effects when they are studied specifically, requires a realistic account of how environment might effect behavior. Fortunately, such a model is thoroughly intuitive and immediately at hand. I will prime your intuitions about the details of such a model by working through some others that turn out to be too simple for the task.

The simplest model of how the environment might effect behavior is predicated on individual environmental events with large, systematic effects on behavior. This model, illustrated in Figure 1, which might be called the one-environment-one-effect (OEOE) model, often arises in the early, irrationally exuberant phases of psychological theory development. Early versions of psychoanalysis are an example, in which it once seemed plausible to propose that particular forms of parent-child interaction led to particular patterns and pathologies in development later on. The occasional waves of enthusiasm for birth-order as a potent explanation of behavior are another example.

Needless to say, OEOE explanations, psychoanalytic or otherwise, have not fared very well as scientific explanations of behavior. Recognizing that it is probably futile to

expect an OEOE model to describe the environmental causes of actual behavioral outcomes, most social scientists turned to a model in which each of many environmental causes is posited to have a small effect on behavior, which can then be added up over many causes to achieve a reasonable level of explanation: that model, illustrated in Figure 2, might be called a quantitative environmental effect (QEE) model. QEE models are best characterized by the statistical procedure that is commonly used to investigate them: multiple regression, in which each of several correlated predictors is allowed to have an independent, (usually) small, and (usually) additive effect on outcome. So, for example, a researcher who is interested in why some children engage in more delinquent acts than others might obtain a variety of information about parental education and childrearing practices, school quality, neighborhood and peer-group characteristics, and the like, and then use multiple regression to find out how the several variables can be optimally weighted to predict delinquency scores.

Social scientists have become so accustomed to this kind of statistical analysis and its various routine justifications that it has become difficult to bear in mind that that such analyses might actually be intended to explain the causes of a real phenomenon. Instead, researchers either talk of “predicting” an outcome, although it is the rare application that is actually designed for the purpose of predicting anything, or disguise their causal intentions in circumspect language about outcomes that are “linked to” or “influenced by” the predictors. But if taken seriously, regression-based data analysis procedures specifically imply a QEE causal model. The causal implications of statistical prediction models are routinely ignored precisely because models of this kind never actually end up explaining anything, beyond enumerating lists of variables that appear to

have non-zero associations with outcome while controlling for an incomplete and arbitrary list of other variables.

Social scientists don't like to say it, but the reason the QEE model has been a failure in terms of causal explanation is glaringly obvious: no complex behaviors in free-ranging humans are caused by a linear and additive set of causes. Any important outcome, like adolescent delinquent behavior, has a myriad of interrelated causes, and each of these causes has a myriad of potential effects, inducing a squared-myrriad of environmental complexity even before one gets to the certainty that the environmental effects co-determine each other, or that the whole package interacts with the just-as-myrriad effects of genes (Figure 3). Following Plomin and Daniels, 1987, I have referred to this situation, with its discouraging implications for successful social science, as the gloomy prospect. (I recently attended a talk by Stevan Hyman, the former head of the National Institute of Health, in which he referred to a "nightmare," in which the causes of psychological outcomes [he was speaking about mental illness] are broken up into so many tiny nonlinear pieces that scientists can never get a handle on them. Plomin's gloomy prospect might also be known as Hyman's nightmare.)

As a social scientist, one can respond to the gloomy prospect in any number of ways. Plomin characteristically tries to wish it away, returning always to the conclusion that the failure to find something is not evidence that it isn't there; instead it is evidence that we need to look harder (Plomin, Asbury & Dunn, 2001). Although I can't hide my skepticism about these ongoing efforts, neither do I object to them: that's the way science works, and the day someone finds a specific within-family environmental variable or an individual genetic locus with a substantial, non-contingent, and

reproducible causal effect on a psychological outcome (Not just a significant correlation or linkage! These exist, obviously.) he will be right and I will be wrong. Alternatively, one could shrug at all the overwhelming complexity of environmental causation and contemplate abandoning the effort, as I may sometimes been guilty of doing myself. Far better, of course, is to get serious about the prospects of doing the best one can at understanding complex behavioral phenomena, at least around the edges. Rutter, Pickles, Murray & Eaves (2001) have produced a formidable catalog of methods to this end.

One example of doing the best one can is the point of this essay. Faced with the squared-myriad of ways that an impoverished environment might inhibit intellectual ability, one imperfect but potentially useful technique is to find a way to add them all together, willy-nilly. Bad schools, inferior nutrition, indifferent parenting, dangerous neighborhoods, anti-social peers, childhood diseases, environmental toxins, you name it, all rolled together into one variable called “socioeconomic status.” Socioeconomic status is a coarse, normative, *ceteris paribus* type of variable, and it has been much vilified by environmentalists put on the defensive by behavioral genetics. But the very coarseness of socioeconomic status can make it a blunt instrument for the detection of environmental effects that on their own are too small, uncontrolled, and nonlinear to be detected reliably, which is the role it played in our study of the genetics of intelligence.

So while I don't doubt that the socioeconomic interaction we reported must be composed of something, I fear that any effort to specify its composition will lead only to gloomily unsatisfying social science. Fortunately, the finding still has real consequences. Improving individual aspects of the conditions of impoverished children will probably have small positive effects, none reliably larger than the others. Removing

children from poverty entirely will probably have large positive effects, a prediction that has content even without specifying exactly what “poverty” entails. Start by improving the constituents of the socioeconomic index-- family income and the education and occupations of parents—and most of the rest will follow along.

I promised to conclude with an extension of my argument to the genetics of behavior. It might seem as though nothing could be farther from the gloomy muddle of environmental behavioral science than the gleaming modern science of genetics. Genetics, especially molecular genetics and its technological cousin neuroscience, are the latest in a long line of scientific white knights that have come to wake social science from its gloomy nightmare.

When I traced models of environmental causation from the simplicity of OEOE, through the regression-based QEE model, to the gloomy wilderness of Hyman’s nightmare, I deliberately followed a course set by the geneticists themselves. Corresponding to the OEOE model is what Plomin (1995) has called the one-gene-one-disorder (OGOD) model, useful enough for traditional medical genetics, but now universally recognized as inadequate for medical disorders as complex as diabetes or heart disease, to say nothing of schizophrenia or delinquency. In place of the OGOD model, Plomin proposes the QTL (quantitative trait locus) model on which so much of modern molecular behavioral genetics has pinned its hopes. In the QTL model, each of a great many environmental loci makes a small, but reliable, independent and additive contribution to an outcome.

The QTL model corresponds to the regression based QEE model of environmental effects, except that genetic models are rarely so multivariate as to

contemplate anything as sophisticated as multiple regression, the search for “genes for” schizophrenia or intelligence or delinquency generally proceeding one at a time. Yet somehow, what has come to seem almost pathetically futile in environmental social science—observing that parents who place mobiles over their children’s cribs have children who do better in third grade, and concluding that early visual stimulation plays an important causal role in school success—acquires a crisply technological, optimistically modern ring when exactly the same mistake is made in a genetic context. If a particular genetic locus occurs more frequently in a sample of very bright children than in a sample of borderline retarded children, it means either that the locus is a specific link in a direct causal chain leading from genes to neurons to brain function to intelligence, or it means that the locus is a mobile hanging in the vast, interactive, uncontrolled causal network that eventuates in some children performing better on IQ tests than others.

Which of these causal models you choose to believe in depends on the particular balance of scientific optimism vs. psychological realism you prefer. There is no proving that it is impossible to identify the additive set of QTLs that are jointly responsible for intelligence, which is why I do not object to the widespread efforts to sort it all out. Like mobiles over cribs, genes correlated with intelligence may not be causal in any straightforward sense, but neither are they irrelevant; and for now, the daunting network of correlations is all we have to go on. So as I have already said, the correct response is not to give up, but rather to change our scientific expectations in a direction that is potentially gloomy but refreshingly humanistic. The reductionist and anti-psychological expectation that we are going to identify a potent environmental ingredient that explains

the effects of poverty on IQ, or explain schizophrenia or delinquency via the genome project or the Decade of the Brain or whatever technological marvel comes next, will never be fulfilled. As scientists or consumers of science, we need to recognize that when technology from the natural sciences meets problems from the social sciences, the result is not social science exposed at last to the bright light of natural science, but rather technology that has become entangled in the prevailing social scientific gloom. Once we can accept this state of affairs, we will be ready to proceed with a humbler program of genetically and neurologically informed social science that may allow us to understand complex behavior a little better while we wait for the millennium of scientific psychology to arrive.

## References

- McGuire, S. (2003). The heritability of parenting. Parenting: Science & Practice, 3, 73-94.
- Plomin, R. (1995). Molecular genetics and psychology. Current Directions in Psychological Science, 4, 114-117.
- Plomin, R., Asbury, K. & Dunn, J. (2001). Why Are Children in the Same Family So Different? Nonshared Environment a Decade Later. Canadian Journal of Psychiatry, 46, 225-233.
- Plomin, R. & Bergeman, C. S. (1991). The nature of nurture: Genetic influence on "environmental" measures. Behavioral & Brain Sciences, 14, 373-427.
- Plomin, R. & Daniels, D. (1987). Why are children in the same family so different from one another? Behavioral & Brain Sciences, 10, 1-16.
- Reiss, D., Neiderhiser, J. M., Hetherington, E. M., & Plomin, R., (2000). The relationship code: Deciphering genetic and social influences on adolescent development. Cambridge, MA: Harvard University Press.
- Rutter, M., Pickles, A., Murray, R. & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. Psychological Bulletin, 127, 291-324.
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. Psychological Science, 14, 623-628.
- Turkheimer, E. & Waldron, M. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. Psychological Bulletin, 126, 78-108.

Wachs, T. D, (1983). The use and abuse of environment in behavior-genetic research. *Child Development*, 54, 396-407.

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