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Beyond Heritability: Twin Studies in Behavioral Research

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Abstract

The heritability of human behavioral traits is now well established, due in large measure to classical twin studies. We see little need for further studies of the heritability of individual traits in behavioral science, but the twin study is far from having outlived its usefulness. The existence of pervasive familial influences on behavior means that selection bias is always a concern in any study of the causal effects of environmental circumstances. Twin samples continue to provide new opportunities to identify causal effects with appropriate genetic and shared environmental controls. We discuss environmental studies of discordant twin pairs and twin studies of genetic and environmental transactions in this context.

Keywords

heritability; twin study; genetic and environmental influences; discordant twin pairs; gene–environment transactions; epigenetics

In his characteristically colorful language, twin researcher David Lykken occasionally remarked that “Behavior genetics rearranged the furniture in psychology’s house” by showing again and again that virtually every trait, from social attitudes to psychopathology, shows genetic influence. It was no Saturday afternoon whim arising out of boredom when behavior genetics moved the behavioral chair over near the genetic lamp. The new arrangement was more accurate: The chair was better placed for scientific reading. By now we have a fundamental understanding that genetic influences are involved in all aspects of psychology and behavior. Turkheimer (2000) even enshrined this as the First Law of Behavioral Genetics, and the law actually underlies all of behavioral science.

Classic twin studies carried out by literally hundreds of researchers have provided an abundance of evidence for this. Throughout the world, perhaps 800,000 pairs of twins have been collected into more than 50 different study samples. Many of these samples would be of considerable epidemiological value even if they did not contain twins, because they are closely representative of the populations from which they have been drawn, having been recruited from birth and other systematic records. Like all experiments, these experiments of nature are not perfect: Differences between twins and singletons, differences between

monozygotic (MZ) and dizygotic (DZ) twins, and methodological biases and limitations could be distorting estimates of the extent of genetic influence. But the sheer volume of evidence from twin studies and the corroborating evidence from adoption studies and studies of other combinations of relatives makes it unreasonable to deny the presence of genetic influences on behavior. And because the genotype pre-exists all behavior, these genetic influences have to be considered causal at some level.

BEYOND ESTIMATES OF GENETIC INFLUENCES

The word “causal” above is important. Ultimately, behavioral science is about understanding what causes behavior, psychopathology, disease, and whether particular kinds of circumstances such as substance abuse, poverty, or specific clinical interventions have causal effects on important life outcomes. But studies that can establish causal effects conclusively are rare because of ethical limitations on experimentation in humans, artificiality of laboratory conditions, and uncertainties of extrapolating from experiments with nonhuman animals to humans. Ironically, once we acknowledge the presence of genetic influences on behavior, the value of twin studies shifts from their ability to demonstrate genetic influences to their ability to illuminate causal environmental influences.

To understand why heritability estimates are no longer important, it is necessary to understand that they are completely dependent on the specifics of the samples and environmental conditions from which they are taken. When environments are homogeneous for all, all individual differences become heritable. When there are both genetic and environmental differences, most of the mechanisms through which genes exert their causal influences on behavior are not the straightforward one gene—one (bit of) trait association one learns about in high-school biology. Instead, much gene expression is contingent on the presence of other gene products, environmental circumstances, and prior levels of gene expression, sometimes even in prior generations. Causal genetic influences are thus intimately bound to causal environmental circumstances (Johnson, 2007). People tend to think of heritability estimates as relatively consistent—for example, in adulthood general intelligence is 50% heritable, personality is 40%, and height is 80%—because the traits are broad, continuous, and clearly polygenic, which alone produce stability in the estimates. Moreover, we tend to average these estimates in our heads. In reality, estimates of heritability of general intelligence commonly range from 50 to 80%, personality from 20 to 50%, and even height from 70 to 95%. Such ranges can be demonstrated even within samples (e.g., Krueger, South, Johnson, & Iacono, 2008). We are only beginning to understand how inaccuracy of measurement, population-genetic differences, and environmental circumstances may transact to produce these differences in heritabilities. Moreover, even highly heritable traits can be strongly manipulated by the environment, so heritability has little if anything to do with controllability. For example, height is on the order of 90% heritable, yet North and South Koreans, who come from the same genetic background, presently differ in average height by a full 6 inches (Pak, 2004; Schwekendiek, 2008).

This means that little can be gleaned from any particular heritability estimate and there is little need for further twin studies investigating the presence and magnitude of genetic influences on behavior. As in psychology 30 years ago, heritability studies do continue to have some importance in areas of the social sciences in which genetic influences have not been acknowledged. For example, Fowler, Baker, and Dawes (2008) demonstrated that more than half of the variation in voting behavior was under genetic influence. In political science, theoretical and empirical models have been able to account only for perhaps a third of the variance (e.g., Plutzer, 2002), so the result that more than half the variance could be attributed to genetic influences seems stunning to social scientists used to thinking of genetic

influences as deterministic and behavior as the product of circumstantial influences. These scientists have the advantage, however, of realizing the role of genetics in their phenomena of interest in an era when the dependence of genetic expression on environmental circumstances, and thus the limitations of the causal inferences that can be drawn from any estimate of heritability, are becoming increasingly clear.

The presence of genetic influences does not mean that genes “cause” behavior in any preordained way. Rather, it means that genes predispose toward some (tacit or active) ongoing series of individual behavioral choices. These choices have effects on later circumstances that affect later options for genetically influenced behavioral choices, and so on. This causal chain is captured in the behavior-genetic literature through the concept of gene–environment correlation, or genetically influenced differences in environmental exposure. Gene–environment correlation may be passive, active, or evocative (Plomin, DeFries, McClearn, & McGuffin, 2008). It is passive when, for example, children receive both genes influencing antisocial behavior and abusive treatment from their parents and model the behavior they receive. It is active when people genetically inclined to be social seek social groups in which to participate and avoid spending long periods of time alone. It is evocative when children with genetically influenced difficulties with emotional control throw temper tantrums that generate angry responses from their parents. Gene–environment correlation is described in the developmental psychopathology literature as social selection, in which the association between risk and outcome reflects origin of risk in the individual rather than the effects of risk. For example, poverty may be a risk factor for schizophrenia because people at genetic risk for schizophrenia tend to drift into poverty because of inability to maintain educational and occupational performance, as well as because poverty provides the disease-triggering stress. In the epidemiological literature, gene–environment correlation is described as reverse causation or confounding, in which some to all of the association between an experience and an outcome reflects the effects of people on their environments rather than the effects of their environments on them.

USING TWIN STUDIES TO DISTINGUISH SELECTION FROM ENVIRONMENTAL CAUSATION

This is where twin studies have particular value. Whatever the label, the First Law of Behavioral Genetics implies that it is always possible that common genetic influences creating selection bias underlie any apparently causal, naturally occurring association between environmental circumstance and outcome. But it is not just common genetic influences that create confounds of this type. If poorer children attend inferior schools, live in more poorly lit homes, and do more poorly in third grade than children from wealthier families, there will be an association between home lighting and school performance. If home lighting has no direct effect on school performance, this association will be due to environmental influences shared by members of some families but not by members of other families. The twin design makes possible the control of both genetic and shared environmental background without specifying all the particular mechanisms involved, and thus makes it possible to isolate and test for the presence of the environmental effects of interest. In other words, given that genetic influences are routinely involved in behavior, the importance of twin studies lies not in their ability to estimate those genetic influences but rather because they can be used as quasi-experimental tests of environmental explanations.

To return to voting behavior, there are many reasons that people vote or do not, but some are likely to be related to personality characteristics like dutifulness, to attitudes like belief in democracy, and to environmental circumstances like access to information and ability to reach the polls. These are all transmitted in part both genetically and through culture/shared environment by parents, but exactly how is far from clear. It does not matter, however, if

what we want to know is whether belief in democracy, for example, has a direct effect on voting behavior. Once we accept the presence of some lump of genetic and shared-environmental influences on both belief in democracy and voting behavior, it becomes apparent that, in MZ twins who differ in belief in democracy, any difference in their voting behavior cannot be attributed to genetic confounds nor to any aspect of the familial environment that they share, such as sociodemography, parental attitudes toward democracy, or parental voting record. Twin studies not only control for genetic selection; they also control for shared-environmental selection, without identifying either the specific genes or the specific shared-environmental influences involved. Such studies therefore provide tough tests of causal associations. The tests are not completely rigorous, of course: The within-twin pair correlations are still confounded by any other relevant variables that differ between the twins, and there could be relevant ways that life with a co-twin differs from life as a singleton. Still, in the human social sciences, where experimentation is restricted, this kind of quasiexperimental control is important in differentiating potentially causal associations from selection processes.

A SEXY EXAMPLE OF BEHAVIOR GENETICS AT WORK

Sometimes the results of applying such controls also call attention to unstated assumptions in psychological research. For example, Armour and Haynie (2007) investigated the relation between timing of first sex in adolescents and later delinquency, expressing concern that premarital sex was likely to contribute to untoward consequences for adolescents. As they had anticipated, they found a significant association, which they interpreted causally: The experience of early sexual activity contributed to later delinquent behavior. Of course, another possibility is that some other variable, either genetic or environmental, was responsible for the association. Harden, Mendle, Hill, Turkheimer, and Emery (2008) used twins from the same data set to evaluate the association after controlling for genetic and shared-environmental influences on age of first sex and contemporaneous delinquency. Even MZ twins differed considerably in age of first sex, but of course age of first sex was confounded with genetic and shared environmental influences. In contrast to Armour and Haynie's observation, however, Harden et al. found that, within twin pairs, with genetic and shared environmental confounds controlled, the twins with earlier age of first sex actually showed *lower* levels of delinquency than the co-twins who initiated sexual activity later. The overall association Armour and Haynie had observed was due to selection on genetic and shared environmental influences contributing to both age at first sex and delinquency rather than to age at first sex alone. These results have clear implications for understanding the role of sexual behavior in adolescent development as well as for the development of public health and sex-education programs for adolescents.

In a related vein, intelligence and education are often associated with healthier lifestyle choices (Deary, Whalley, Batty, & Starr, 2006; Gottfredson, 2004). Controlling for genetic and shared-environmental influences, however, Johnson, Hicks, McGue, & Iacono (in press) found that both higher IQ and greater education were associated with more alcohol and nicotine use in both sexes at age 24. This suggests that the generally lower substance use among brighter and better-educated young adults results from selection on genetic and family influences contributing to an environment emphasizing both educational attainment and reduced substance use, rather than from direct application of intelligence and/or education in substance-use choices.

UNPACKING THE GENETIC AND SHARED-ENVIRONMENTAL “LUMP”

Ultimately, we want not just to identify purely environmental associations but also to understand how genetic and shared-environmental influences come together to create

selection processes. Again, twin studies can help, but not through estimates of heritability. Rather, they can do so through studies of gene–environment transactions, or changes in the patterns of genetic and environmental variances with changes in environmental circumstances. In contrast to studies of discordant twin pairs, studies of gene–environment transactions rely on patterns of relative similarity in MZ and DZ twins to examine population changes in magnitudes of genetic and environmental influences of one trait with levels of another. The models used in these studies still need refinement, but they can also allow for estimation of the extent to which genetic and environmental influences on one trait are also involved in the other. When such overlap exists, changes in magnitudes of genetic and environmental influences are associated with changes in degree of overlap in regular ways. Because overlap on a trait and a putative environmental circumstance is an indication of selection bias, the models reveal patterns of both differences in genetic expression and selection effects.

For example, there is a well-known and robust negative association between income and/or socioeconomic status and physical health problems. Less affluent people tend to have poorer health and to die younger. Johnson and Krueger (2005) used a gene–environment transaction study to understand this association. There was more genetic variance in physical health problems among those with low income and/or low perceived personal control. One interpretation of this result is that more stressful living conditions associated with lower income or personal control trigger greater expression of genetic vulnerabilities to health problems. Put another way, more affluent people and those with greater perceived control are freer from “genetic destiny.”

In addition, at higher levels of income and perceived control, genetic influences on physical health problems largely overlapped those on income and perceived control. When income and perceived control were low, however, there was much less overlap. The possible interpretation? Genetically influenced personal characteristics involved in earning greater income and maintaining control of one's life are also characteristics that can be used to prevent the development of physical health problems. At the same time, the poorer health of people in lower income/perceived-control situations occurs because the environmental stress they experience causes greater expression of genetic vulnerabilities unrelated to those personal characteristics. Studies of biological responses to stress increasingly support this view. This interpretation does not depend on any specific genetic polymorphisms: The definition of physical health problems was intentionally broad and included a variety of medical conditions.

CONCLUSION

The discovery that all behavior is partially heritable transformed psychology, but, ironically, it also transformed behavior genetics. Once we accept that basically everything—not only schizophrenia and intelligence, but also marital status and television watching—is heritable, it becomes clear that specific estimates of heritability are not very important. The omnipresence of genetic influences does not demonstrate that behavior is “less psychological” or “more biologically determined” than had originally been thought; rather it shows that behavior arises from factors intrinsic as well as extrinsic to the individual. The real implications of heritability lie not in questions of relative biological determinism but in revealing the need to understand both the mechanisms through which the individual, whether consciously or not, directs his or her own life course and his or her power to do so. In psychology, where it is not ethically possible to conduct randomized experiments on life outcomes, the natural experiment provided by the twin study can be most helpful in addressing these issues.

Recommended Reading

- Bouchard TJ Jr. Experience Producing Drive Theory: How genes drive experience and shape personality. *Acta Paediatrica, Supplement*. 1997; 422:60–64. [PubMed: 9298795] An outline of a theory explaining how selection processes develop.
- Johnson, W. 2007. (See References). An exploration of the quantitative measurement of interplay between genetic and environmental influences as they are manifested in population-level social forces.
- Rutter M. Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science*. 2007; 2:377–395. A discussion of the use of twin studies and other situations with naturally occurring quasiexperimental controls to examine causality.
- Turkheimer E. Heritability and biological explanation. *Psychological Review*. 1998; 105:782–791. [PubMed: 9830379] An exploration of the meaning of heritability and biology to psychology.
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