Three Laws of Behavior Genetics and What They Mean

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Abstract

Behavior genetics has demonstrated that genetic variance is an important component of variation for all behavioral outcomes, but variation among families is not. These results have led some critics of behavior genetics to conclude that heritability is so ubiquitous as to have few consequences for scientific understanding of development, while some behavior genetic partisans have concluded that family environment is not an important cause of developmental outcomes. Both views are incorrect. Genotype is in fact a more systematic source of variability than environment, but for reasons that are methodological rather than substantive. Development is fundamentally nonlinear, interactive, and difficult to control experimentally. Twin studies offer a useful methodological shortcut, but do not show that genes are more fundamental than environments.

Keywords

genes; environment; development; behavior genetics

The nature-nurture debate is over. The bottom line is that everything is heritable, an outcome that has taken all sides of the nature-nurture debate by surprise. Irving Gottesman and I have suggested that the universal influence of genes on behavior be enshrined as the first law of behavior genetics (Turkheimer & Gottesman, 1991), and at the risk of naming laws that I can take no credit for discovering, it is worth stating the nearly unanimous results of behavior genetics in a more formal manner.

- *First Law*. All human behavioral traits are heritable.
- *Second Law*. The effect of being raised in the same family is smaller than the effect of genes.
- *Third Law*. A substantial portion of the variation in complex human behavioral traits is not accounted for by the effects of genes or families.

It is not my purpose in this brief article to defend these three laws against the many exceptions that might be claimed. The point is that now that the empirical facts are in and no longer a matter of serious controversy, it is time to turn attention to what the three laws mean, to the implications of the genetics of behavior for an understanding of complex human behavior and its development.

VARIANCE AND CAUSATION IN BEHAVIORAL DEVELOPMENT

If the first two laws are taken literally, they seem to herald a great victory for the nature side of the old debate: Genes matter, families do not. To understand why such views are at best an oversimplification of a complex reality, it is necessary to consider the newest wave of opposition that behavior genetics has generated. These new critics, whose most articulate spokesman is Gilbert Gottlieb (1991, 1992, 1995), claim that the goal of developmental psychology is to specify the actual developmental processes that lead to complex outcomes. In lower animals, whose breeding and environment can be brought under the control of the scientist, it is possible to document such developmental processes in exquisite detail. The critics draw an unfavorable comparison between these detailed animal studies and twin studies of behavior genetics, which produce only statistical conclusions about the relative importance of genes and environment in development.

The greatest virtue of the new challenge is that it abandons the

implausible environmentalist contention that important aspects of behavior will be without genetic influence. Gottlieb (1992) stated, "The present . . . viewpoint holds that genes are an inextricable component of any developmental system, and thus *genes are involved in all traits*" (p. 147). Unlike earlier critics who deplored the reductionism they attributed to behavior genetic theories of behavior, the developmental biologists take behavior genetics to task for not being mechanistic *enough*. Once vilified as the paragon of determinist accounts of human behavior, behavior genetics is now chastised for offering vague and inconclusive models of development (Gottlieb, 1995; Turkheimer, Goldsmith, & Gottesman, 1995), and judged by the standards of developmental psychobiology in lower animals, it is true enough that behavior genetic theories of complex human behavior seem woefully poorly specified. But ultimately the charge is unfair, because there is no equivalent in developmental psychobiology to the behavior genetic study of marital status or school performance. The great preponderance of the exquisite experimental science that goes into animal psychobiology is quite simply impossible to conduct in humans.

Human developmental social science is difficult—equally so for the genetically and environmentally inclined—because of the (methodologically vexing, humanistically pleasing) confluence of two conditions: (a) Behavior emerges out of complex, nonlinear developmental processes, and (b) ethical considerations prevent us from bringing most human developmental processes under effective experimental control. Figure 1 is a schematic illustration of the problem. Individual genes (Genes 1, 2, and 3) and their environments (which include other genes) interact to initiate a complex developmental process that determines adult personality. Most characteristic of this process is its interactivity: Subsequent environments to which the organism is exposed depend on its earlier states, and each new environment changes the developmental trajectory, which affects future expression of genes, and so forth. Everything is interactive, in the sense that no arrows proceed uninterrupted from cause to effect; any individual gene or environmental event produces an effect only by interacting with other genes and environments.

For the behavior geneticist,

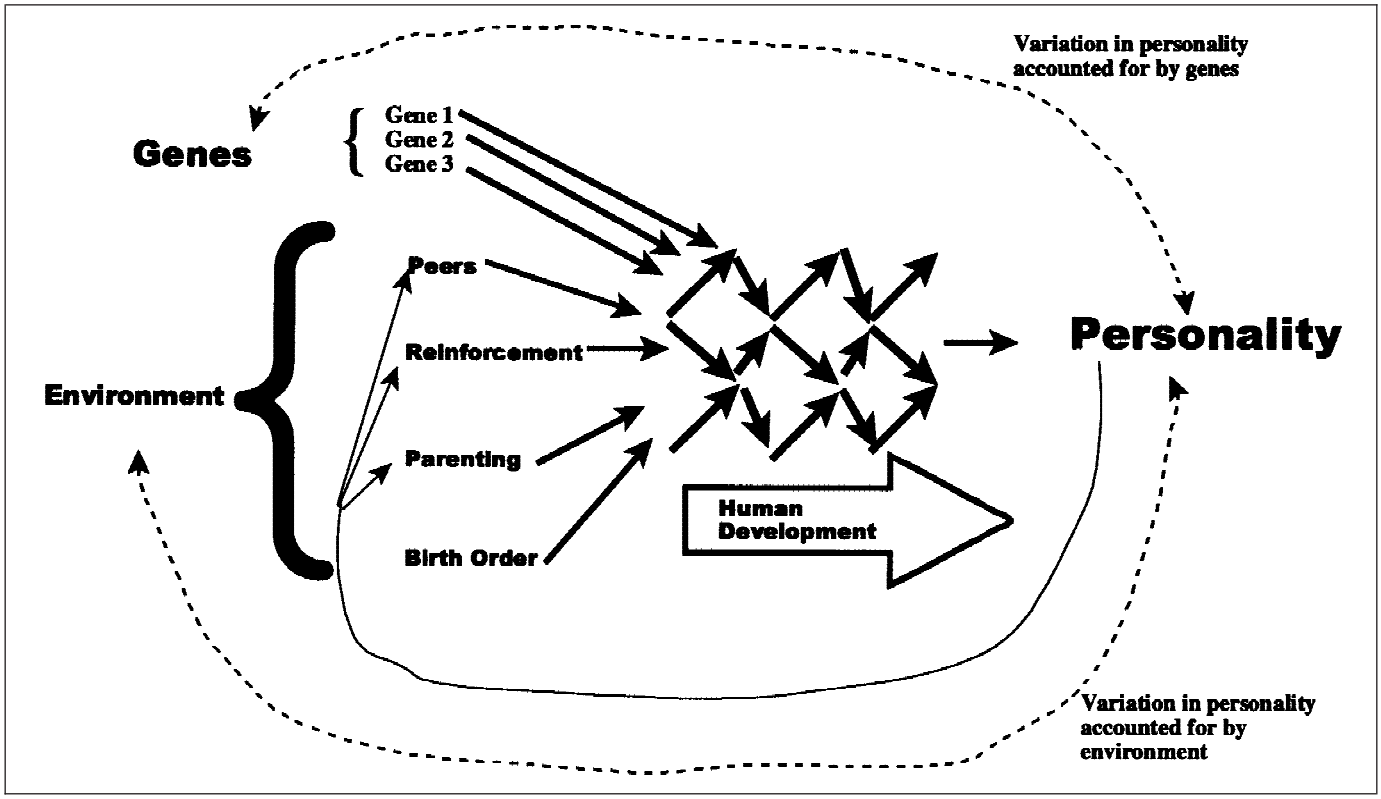

Fig. 1. Schematic diagram of contrasting roles of genes and environment in development of personality. One-headed arrows link causes to effects; two-headed arrows indicate correlations. Genes and environments are both causal inputs into an interactive developmental system (represented by the network of arrows in the center of the figure), but because people select and shape their own environments (as represented by lighter one-headed arrows from personality to environments), correlations across the developmental system (dotted two-headed arrows) are easier to detect for genes than for environments.

however, the quasi-experimental gift of genetically identical and nonidentical twins offers a remarkable, if deceptively simple, method to span this daunting interactive complexity. Thanks to the fact that identical twins are on average exactly twice as similar genetically as nonidentical twins, one can use straightforward statistical procedures to estimate the proportion of variability in complex outcomes that is associated with causally distant genes, all the while maintaining a state of near-perfect ignorance about the actual causal processes that connect genes to behavior. This methodological shortcut is not available to rivals of behavior genetics who seek to measure the effects of families on behavior. How similar was my rearing environment to that of my siblings? And how similar was it to the environment of my adopted sibling, if I have one, or to the environment of my biological sibling who was raised by someone else? The apparent victory of nature over nurture suggested by the first two laws is thus seen to be more methodological than substantive. In a world in which there were occasional occurrences of "identical environmental twins," whose experiences were exactly the same, moment by moment, and another variety who shared exactly (but randomly) 50% of their experiences, environmentalists could reproduce the precision of their rivals, and like the behavior geneticists could measure with great precision the total contribution of the environment while knowing almost nothing about the developmental processes that underlie it.

The old-fashioned nature-nurture debate was about whether or not genes influence complex behavioral outcomes, and that question has been decisively answered in the affirmative. The new question is how we can proceed from partitioning sources of variance to specifying concrete developmental processes (Turkheimer, 1998), and although critics like Gottlieb are correct that heritability per se has few implications for a scientific understanding of development, they have failed to emphasize two crucial points. First, heritability does have one certain consequence: It is no longer possible to interpret correlations among biologically related family members as prima facie evidence of sociocultural causal mechanisms. If the children of depressed mothers grow up to be depressed themselves, it does not necessarily demonstrate that being raised by a depressed mother is itself depressing. The children might have grown up equally depressed if they had been adopted and raised by different mothers, under the influence of their biological mother's genes. For every behavior geneticist who continues to report moderate heritabilities as though they were news, there is an environmentalist who reports causally ambiguous correlations between genetically related parents and children. Second, the problem the critics have uncovered extends well beyond behavior genetics: It is a rare environmentalist who has never used statistical methods to predict behavioral outcomes from earlier events, in the hope that the specific developmental mechanisms can be filled in later. The disconnect between the analysis of variance and the analysis of causes, to use Lewontin's (1974) phrase, is not a proprietary flaw in behavior genetic methodology; in fact, it is the bedrock methodological problem of contemporary social science.

NONSHARED ENVIRONMENT AND THE GLOOMY PROSPECT

Even after the effects of genes and the shared effects of families have been accounted for, around 50% of the differences among siblings is left unexplained. In recent years, scientists interested in the genetics of behavior have come to call this unexplained portion the "nonshared environment." Although according to the second law shared environment accounts for a small proportion of the variability in behavioral outcomes, according to the third law, nonshared environment usually accounts for a substantial portion. So perhaps the appropriate conclusion is not so much that the family environment does not matter for development, but rather that the part of the family environment that is shared by siblings does not matter. What does matter is the individual environments of children, their peers, and the aspects of their parenting that they do not share. Plomin and Daniels (1987) reviewed evidence of the predominance of nonshared environmental variance and posed a seminal question: Why are children in the same family so different? They proposed that siblings are different because nonshared environmental events are more potent causes of developmental outcomes than the shared environmental variables, like socioeconomic status, that have formed the traditional basis of sociocultural developmental psychology.

Plomin and Daniels's explanation involves a subtle conceptual shift, best described in terms of a distinction between the objective and effective environment (Goldsmith, 1993; Turkheimer & Waldron, 2000). What qualifies an environmental event as nonshared? There are two possibilities. The first is objective: An event is nonshared if it is experienced by only one sibling in a family, regardless of the consequences it produces. The other possibility is effective: An environmental event is nonshared if it makes siblings different

rather than similar, regardless of whether it was experienced by one or both of them. Plomin and Daniels's proposal, then, is that the nonshared environment as an effectively defined variance component can be explained by objectively nonshared environmental events. The question, "Why are children in the same family so different?" is answered, "Because measurable differences in their environments make them that way."

This proposal has been enormously influential, spawning an entire area of empirical inquiry into the consequences of measured environmental differences among siblings. Ironically, that same literature has quite decisively demonstrated that the conjecture is false. A review of 43 studies that measured differences in the environments of siblings and related them to differences in the siblings' developmental outcomes (Turkheimer & Waldron, 2000) has shown that although upwards of 50% of the variance in behavioral outcomes is accounted for by the effectively defined variance component called nonshared environment, the median percentage accounted for by objectively defined nonshared events is less than 2%. What could be going on?

Plomin and Daniels (1987) almost identified the answer to this question, but dismissed it as too pessimistic:

> One gloomy prospect is that the salient environment might be unsystematic, idiosyncratic, or serendipitous events such as accidents, illnesses, or other traumas .... Such capricious events, however, are likely to prove a dead end for research. More interesting heuristically are possible systematic sources of differences between families. (p. 8)

The gloomy prospect is true. Nonshared environmental variability predominates not because of the systematic effects of environmental events that are not shared among siblings, but rather because of the unsystematic effects of all environmental events, compounded by the equally unsystematic processes that expose us to environmental events in the first place (Turkheimer & Gottesman, 1996).

A model of nonshared variability based on the gloomy prospect is radically different from the Plomin model based on systematic consequences of environmental differences among siblings. Most important, the two models suggest very different prospects for a genetically informed developmental psychology. Again and again, Plomin and his colleagues have emphasized that the importance of nonshared environment implies that it is time to abandon shared environmental variables as possible explanations of developmental outcomes. And although modern environmentalists might not miss coarse measures like socioeconomic status, it is quite another thing to give up on the causal efficaciousness of normal families, as Scarr (1992), Rowe (1994), and Harris (1998) have urged. If, however, nonshared environmental variability in outcome is the result of the unsystematic consequences of both shared and nonshared environmental events, the field faces formidable methodological problems—Plomin and Daniels's gloomy prospect—but need not conclude that aspects of families children share with siblings are of no causal importance.

CONCLUSION: ANTICIPATING THE GENOME PROJECT

It is now possible for behavior genetics to move beyond statistical analyses of differences between identical and nonidentical twins and identify individual genes that are related to behavioral outcomes. What should we expect from this endeavor? Behavior geneticists anticipate vindication: At long last, statistical variance components will be rooted in the actual causal consequences of actual genes. Critics of behavior genetics expect the opposite, pointing to the repeated failures to replicate associations between genes and behavior as evidence of the shaky theoretical underpinnings of which they have so long complained.

There is an interesting parallel between the search for individual genes that influence behavior and the failed attempt to specify the nonshared environment in terms of measured environmental variables. In each case, investigators began with statistically reliable but causally vague sources of variance, and set out to discover the actual causal processes that produced them. The quest for the nonshared environment, as we have seen, got stuck in the gloomy prospect. Although individual environmental events influence outcomes in the most general sense, they do not do so in a systematic way. One can detect their effects only by accumulating them statistically, using twins or adoptees.

If the underlying causal structure of human development is highly complex, as illustrated in Figure 1, the relatively simple statistical procedures employed by developmental psychologists, geneticists, and environmentalists alike are being badly misapplied. But misapplied statistical procedures still produce what appear to be results. Small relations would still be found between predictors and outcomes, but the underlying complex causal processes would cause the apparent results to be small, and to change unpredictably from one experiment to the next. So individual investigators would obtain "results," which would then fail to replicate and accumulate into a coherent theory because the

simple statistical model did not fit the complex developmental process to which it was being applied. Much social science conducted in the shadow of the gloomy prospect has exactly this flavor (e.g., Meehl, 1978).

The gloomy prospect looms larger for the genome project than is generally acknowledged. The question is not whether there are correlations to be found between individual genes and complex behavior—of course there are—but instead whether there are domains of genetic causation in which the gloomy prospect does not prevail, allowing the little bits of correlational evidence to cohere into replicable and cumulative genetic models of development. My own prediction is that such domains will prove rare indeed, and that the likelihood of discovering them will be inversely related to the complexity of the behavior under study.

Finally, it must be remembered that the gloomy prospect is gloomy only from the point of view of the working social scientist. Although frustrated developmental psychologists may be tempted to favor methodologically tractable heuristics over chaotic psychological reality, it is a devil's choice: In the long run, the gloomy prospect always wins, and no one would want to live in a world where it did not. Psychology is at least one good paradigm shift away from an empirical answer to the gloomy prospect, but the philosophical response is becoming clear: The additive effect of genes may constitute what is predictable about human development, but what is predictable about human development is also what is least interesting about it. The gloomy prospect isn't.

Recommended Reading

Gottlieb, G. (1992). (See References)
Lewontin, R.C. (1974). (See References)
Meehl, P.E. (1978). (See References)
Plomin, R., & Daniels, D. (1987). (See References)

Note

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References

Goldsmith, H. (1993). Nature-nurture issues in the behavioral genetic context: Overcoming barriers to communication. In R. Plomin & G. McClearn (Eds.), *Nature, nurture and psychology* (pp. 325–339). Washington, DC: American Psychological Association.

Gottlieb, G. (1991). Experiential canalization of behavioral development: Theory. *Developmental Psychology*, *27*, 4–13.

Gottlieb, G. (1992). *Individual development and evolution*. New York: Oxford University Press.

Gottlieb, G. (1995). Some conceptual deficiencies in "developmental" behavior genetics. *Human Development*, *38*, 131–141.

Harris, J.R. (1998). *The nurture assumption: Why children turn out the way they do*. New York: Free Press.

Lewontin, R.C. (1974). The analysis of variance and the analysis of causes. *American Journal of Human Genetics*, *26*, 400–411.

Meehl, P.E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, *46*, 806–834.

Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences*, *10*, 1–60.

Rowe, D.C. (1994). *The limits of family influence: Genes, experience, and behavior*. New York: Guilford Press.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development, 63*, 1–19.

Turkheimer, E. (1998). Heritability and biological explanation. *Psychological Review*, *105*, 782–791.

Turkheimer, E., Goldsmith, H.H., & Gottesman, I.I. (1995). Commentary. *Human Development*, *38*, 142–153.

Turkheimer, E., & Gottesman, I.I. (1991). Is $H^2 = 0$ a null hypothesis anymore? *Behavioral and Brain Sciences*, *14*, 410–411.

Turkheimer, E., & Gottesman, I.I. (1996). Simulating the dynamics of genes and environment in development. *Development and Psychopathology*, *8*, 667–677.

Turkheimer, E., & Waldron, M.C. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin*, *126*, 78–108.