Commentary

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The Blind Psychological Scientists and the Elephant: Reply to Sherlock and Zietsch

Robert Waldinger[1] and Marc Schulz[2]
[1]Department of Psychiatry, Massachusetts General Hospital, Boston, Massachusetts, and [2]Department of Psychology, Bryn Mawr College

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The Commentary by Sherlock and Zietsch (2018) raises important questions about how the science of human psychological development moves forward as researchers make dramatic strides in understanding the role of genetics. In this response, we return to these larger questions, but we begin by addressing Sherlock and Zietsch's specific argument that "genetic contributions should almost always be considered when dealing with associations between parents' behavior and their children's behavior" (p. 156). In our 60-year study of the association between warmth of the childhood family environment and late-life security of attachment (Waldinger & Schulz, 2016), we did not mention the potential influence of genetic factors, and we regret this omission. Moreover, we acknowledge that in some places in the article, we used causal language that was not justified by the design of the study. Although we explicitly stated that the association between warmth of the childhood family environment and late-life security of attachment is correlational and not causal, we went on to interpret these findings as indications of support for the influence of childhood environment on well-being in adulthood. Although this is a plausible interpretation, Sherlock and Zietsch are justified in arguing that genetic influences could contribute to this link and that our article should have noted this. The editorial review process encourages investigators to highlight the implications of their work, and in our enthusiasm to convey the importance of an association that spans six decades, we did not adequately address possible alternative explanations for this association.

Evidence for Genetic and Environmental Influences on Romantic Attachment Across the Life Span

Sherlock and Zietsch's Commentary prompts us to consider two sets of issues: those particular to the field of attachment research and broader concerns about how researchers make sense of genetic and environmental influences in human development. Sherlock and Zietsch raise important questions about the role of genes and the environment in shaping attachment phenomena. The twin studies they cite consistently found genetic influences on attachment styles and behavior. These studies (Brussoni, Jang, Livesley, & Macbeth, 2000; Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014; Picardi, Fagnani, Nisticò, & Stazi, 2011) estimated genetic influences as accounting for 25 to 45% of the variance in attachment outcomes; thus, the larger proportion of variance was accounted for by shared and nonshared environmental influences. Low estimates of shared environmental influence in some twin studies (e.g., Fearon et al., 2014) are often cited as evidence that parents do not significantly influence their children's security of attachment. This interpretation involves reducing the influences of family upbringing to shared environment. However, each child within a given family has different experiences, as each child—even a twin—has a unique relationship with his or her parents and plays a unique set of family roles. Parents are thus likely to contribute to the nonshared as well as the shared environment that affects each child.

With respect to the determinants of attachment across the life span, it is critical to recognize that attachment is not a unitary construct. There is ample empirical evidence that elements of attachment vary in important ways across developmental epochs and types of relationships (e.g., parents vs. romantic partners), and that the mode of assessment (interview vs. self-report vs. observation) shapes the nature of the

Corresponding Author:
Robert Waldinger, Department of Psychiatry, Massachusetts General Hospital, 151 Merrimac St., 2nd Floor, Boston MA 02114
E-mail: rwaldinger@partners.org

construct being assessed (Roisman et al., 2007). None of the research cited by Sherlock and Zietsch examined predictors of late-life romantic attachment, which was the focus of our study. Instead, all focused on infancy, childhood, and young adulthood. Moreover, the studies by Brussoni et al. (2000) and Picardi et al. (2011) used self-report measures of attachment. There is evidence that the interview-based measure used in our work assesses aspects of attachment that may not be captured by traditional self-report approaches (Roisman et al., 2007). Therefore, there exists little empirical basis for estimating what portions of variance in late-life attachment—particularly as assessed by the Current Relationship Interview—are accounted for by genes, shared environment, and nonshared environment. It is possible, and even likely, that there is a genetic component to late-life romantic attachment, but the studies cited by Sherlock and Zietsch do not provide any specific evidence that this is the case. Employing the Current Relationship Interview in a genetically informative study could greatly enhance understanding of the causal role of genes and environment in late-life attachment.

Rigorous experimental work with animals has provided persuasive evidence for the influence that parenting and early environments can have on development, including the development of relationship behaviors relevant to attachment (e.g., Harlow, Dodsworth, & Harlow, 1965; Suomi, 1987; Weaver, Meaney, & Szyf, 2006). Opportunities to trace connections from childhood to late-life attachment across six decades are exceedingly rare, and the challenge is to understand the mechanisms that are responsible for such long-term links. The field would benefit from continued research with animals and humans that can help shed light on the mechanisms by which both environments and genes contribute to long-term developmental outcomes.

Illuminating the Complexity of Causal Pathways

The broader questions for our field concern the underlying goals of psychological science and the best pathways toward unearthing causal mechanisms that underlie psychological traits. Part of the wisdom in Sherlock and Zietsch's Commentary is their caution about oversimplifying complex phenomena. Determining the degree to which a particular behavioral phenomenon is shaped by genes and environmental processes is complicated. There is disagreement about how heritability estimates derived from twin studies should be interpreted and about their potential limitations and utility (Turkheimer & Waldron, 2000). One danger of focusing too narrowly on estimates of the strength of heritability is that such emphasis might discourage investigators from investigating nonheritable influences. For example, the fact that height is estimated to be highly heritable across human populations does not preclude the profound environmental impact of preventable factors, such as malnutrition and disease (Silventoinen et al., 2003).

The notion that genes and environment make identifiably separate contributions to human psychological development engenders a dichotomy that runs counter to much of what is known about biology (Gottlieb, 2007). Heritability studies may inadvertently fuel this dichotomous thinking by parsing variance into discrete packets. Although some phenotypes (e.g., cystic fibrosis) can be traced to specific genes and are predominantly genetic in origin, the origins of most phenotypes are decidedly mixed because genes and environment interact in every moment of development. DNA codes for varieties of RNA, which in turn code for proteins, which build cells that form complex structures that underlie psychological traits. At every juncture, there is opportunity for gene expression to be shaped by feedback from and interaction with the environment and other critical biological systems (e.g., neural, hormonal). Thus, a given DNA sequence operating in different environments can generate different products in different amounts at the cellular and phenotypic levels. This interactionist emphasis, of course, also implies that the same environment operating on people with different genetic makeups can generate different outcomes. Pinker (2004) noted that the extremes of both rigid interactionism and rigid genetic or environmental determinism fail to account for the complexity of most behavioral and developmental phenotypes.

As psychological scientists, we endeavor both to discover new knowledge and to identify possible targets for improving the human condition. Three decades ago, Rutter (1987) wisely pointed out that prediction alone is not sufficient; understanding mechanisms is critical to this endeavor. A key aspect of our study involved examining the adaptiveness of midlife emotion-regulatory styles as a possible mechanism by which nurturance during childhood might result in secure late-life attachment. The finding that adaptive emotion-regulatory styles did, in fact, partially mediate the link between childhood experience and late-life functioning points to a potential target for intervention to help individuals who did not experience a warm and nurturing childhood environment achieve more stable and adaptive intimate relationships. The findings do not prove causation and clearly require replication, but they point toward what may be a helpful avenue for intervention.

In an era when research is based increasingly on technical expertise that is not shared across the field, integration of methods and findings is challenging but

essential. Both experimental and nonexperimental research have important limitations. The benefits to internal validity that are the centerpiece of experimental research derive in large part from the isolated study of one or two mechanisms. Nonexperimental research—whether genetic or psychosocial—is hindered by difficulties ruling out potential confounds and can also be simplified in ways that do not match the complexity of the phenomena under investigation. Our psychosocial, nonexperimental study is but one small contribution to a rich body of empirical investigation linking parent-child relationships to later functioning. Spurred by enthusiasm for our points of view, the temptation to overstate afflicts us all. Humility and appreciation of the limits of any one perspective are of paramount importance in presenting our work. We all view the world through the lenses of our particular intellectual traditions. In this respect, to paraphrase an ancient Indian parable, we are all blind men and women trying our best to understand this infinitely complex elephant that is human psychological development.

Action Editor

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Author Contributions

R. Waldinger and M. Schulz jointly developed and wrote this Commentary.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

References

Brussoni, M. J., Jang, K. L., Livesley, W. J., & Macbeth, T. M. (2000). Genetic and environmental influences on adult attachment styles. *Personal Relationships*, *7*, 283–289. doi:10.1111/j.1475-6811.2000.tb00017.x

Fearon, P., Shmueli-Goetz, Y., Viding, E., Fonagy, P., & Plomin, R. (2014). Genetic and environmental influences on adolescent attachment. *Journal of Child Psychology and Psychiatry*, *55*, 1033–1041. doi:10.1111/jcpp.12171

Gottlieb, G. (2007). Probabilistic epigenesis. *Developmental Science*, *10*, 1–11.

Harlow, H. F., Dodsworth, R. O., & Harlow, M. K. (1965). Total social isolation in monkeys. *Proceedings of the National Academy of Sciences, USA*, *54*, 90–97.

Picardi, A., Fagnani, C., Nisticò, L., & Stazi, M. A. (2011). A twin study of attachment style in young adults. *Journal of Personality*, *79*, 965–992. doi:10.1111/j.1467-6494.2010.00707.x

Pinker, S. (2004). Why nature & nurture won't go away. *Daedalus*, *133*(4), 5–17. doi:10.1162/0011526042365591

Roisman, G. I., Holland, A., Fortuna, K., Fraley, R. C., Clausell, E., & Clarke, A. (2007). The Adult Attachment Interview and self-reports of attachment style: An empirical rapprochement. *Journal of Personality and Social Psychology*, *92*, 678–697.

Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry*, *57*, 316–331.

Sherlock, J., & Zietsch, B. (2018). Longitudinal relationships between parents' and children's behavior need not implicate the influence of parental behavior and may reflect genetics: Comment on Waldinger and Schulz (2016). *Psychological Science*, *29*, 154–158.

Silventoinen, K., Sammalisto, S., Perola, M., Boomsma, D. I., Cornes, B. K., Davis, C., . . . Kaprio, J. (2003). Heritability of adult body height: A comparative study of twin cohorts in eight countries. *Twin Research*, *6*, 399–408.

Suomi, S. J. (1987). Genetic and maternal contributions to individual differences in rhesus monkey biobehavioral development. In N. Krasnegor, E. Blass, M. Hofer, & W. Smotherman (Eds.), *Perinatal development: A psychobiological perspective* (pp. 397–420). New York, NY: Academic Press.

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

Waldinger, R. J., & Schulz, M. S. (2016). The long reach of nurturing family environments: Links with midlife emotion-regulatory styles and late-life security in intimate relationships. *Psychological Science*, *27*, 1443–1450. doi:10.1177/0956797616661556

Weaver, I. C. G., Meaney, M. J., & Szyf, M. (2006). Maternal care effects on the hippocampal transcriptome and anxiety-mediated behaviors in the offspring that are reversible in adulthood. *Proceedings of the National Academy of Sciences, USA*, *103*, 3480–3485. doi:10.1073/pnas.0507526103