Commentary

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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)

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Waldinger and Schulz (2016) provided evidence that individual differences in warmth of the parent-offspring relationship during adolescence are associated with the quality of the offspring's romantic attachments 60 years later. Although the reported association was only a longitudinal correlation, Waldinger and Schulz interpreted it in causal terms: For example, they stated, "This study captured the long reach of warm parent-child relationships and nurturing family environment in shaping key aspects of functioning later in life" (p. 1449) and "The findings underscore the far-reaching influence of childhood environment on well-being in adulthood" (p. 1443). Unfortunately, genetic influences on attachment styles throughout the life span create a confound in such studies that calls into question causal interpretations. Indeed, similar errors are common in the broader literature on the effects of parenting (as we discuss later). Causal inferences about parental environment must be backed by appropriate evidence, and, at the very least, the possibility of genetic confounding should be discussed to avoid misleading other scientists and the media.

Studies of Attachment

The evidence that genetic factors influence attachment styles is overwhelming. Much of this evidence comes from the natural experiment provided by identical (i.e., 100% genetic similarity) and nonidentical (i.e., 50% genetic similarity) twin pairs. By comparing trait similarity of sibling pairs with different degrees of genetic relatedness, twin studies provide estimates of the genetic and environmental influences on a trait. Twin studies have documented a significant genetic component in attachment style from adolescence onward, and little influence of family environment. Although no published twin studies have used the Current Relationship Interview used by Waldinger and Schulz, every studied measure of attachment has shown substantial genetic effects. Using data from 551 twin pairs who completed the Child Attachment Interview at age 15, Fearon, Shmueli-Goetz, Viding, Fonagy, and Plomin (2014) estimated that genetic influences accounted for 40% of the variance in twins' responses, whereas the influence of the shared environment (i.e., family of upbringing) was negligible. Similarly Picardi, Fagnani, Nisticò, and Stazi (2011) found that genetic influences accounted for 45% of the variation in young adult twins' attachment-related anxiety and 36% of the variation in their avoidance, as assessed by the Experiences in Close Relationships Scale; again, no influence of the shared environment was found. Brussoni, Jang, Livesley, and Macbeth (2000) obtained comparable results using the Relationship Scales Questionnaire: The shared environment did not contribute to adult twins' attachment styles, with the exception that dismissing attachment style showed moderate shared environmental effects accounting for 29% of the variance. Genetic effects accounted for between 25% and 43% of the variance in secure, fearful, and preoccupied attachment styles.

Because parents provide both genes and environment to their children, studies (such as Waldinger and Schulz's) that investigate the latter while ignoring the

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former are inherently confounded (McAdams et al., 2014). For example, parents with a genetic predisposition for coldness in close relationships will tend to treat their children coldly, and their children will tend to have relatively low-quality intimate relationships when they grow up. This association will emerge even if there is absolutely no causal influence of the parents' cold behavior, as the parents will have given their children the genetic predisposition for coldness. Attachment traits might be affected by parenting behavior (as in the case of dismissing attachment), but that cannot be established without controlling for genetic influences, usually with a family-based design.

Waldinger and Schulz did not measure or control for genetic effects, so no conclusions about the effects of parenting on later adult attachment should be drawn from their data. Waldinger and Schulz acknowledged that "causal conclusions cannot be drawn given the study's nonexperimental nature" (p. 1449), but this caveat is undermined by the strong causal language used elsewhere in the article, including shortly after that statement. Moreover, the authors never mentioned the probable genetic confound in their study.

Gene-Environment Confounds in Other Areas of Psychology

This issue is not unique to Waldinger and Schulz's article, as it speaks to a broader problem in psychology: Researchers often fail to consider the potential role of genetics in the relationship between parents' and offspring's behavior. By way of example, we briefly describe three recent, typical examples. First, in an article published in *Prevention Science*, Donaldson, Handren, and Crano (2016) interpreted an association between parental alcohol consumption and offspring's later drinking as causal. The article did not mention the possibility that the association could be driven entirely by offspring's inheritance of their parents' genetic predisposition to drink alcohol, despite the extensive evidence that alcohol consumption is heritable (Verhulst, Neale, & Kendler, 2015). In another case, in an article published in *Developmental Psychology*, Prenoveau et al. (2017) interpreted an association between maternal postnatal depression and infants' emotional negativity at 24 months of age as causal. Again, the possibility that a heritable disposition might underlie this association was not mentioned, even though it is well established that children's negative affect (Saudino, 2005) and perinatal (including pre- and postnatal) depression (Viktorin et al., 2016) are substantially heritable.

Even molecular genetic studies are susceptible to this confound. For instance, an article published this year in *Social Psychological and Personality Science* (Stanton et al., 2017) reported associations between maternal attachment anxiety and avoidance and offspring's expression of a gene thought to be involved in stress regulation (*NR3C1*). The authors interpreted these associations as causal: "Our results suggest that mothers' adult attachment orientations influence children's expression of a gene relevant to both adaptive stress regulation and the inflammatory response" (p. 7). However, attachment orientation (as mentioned earlier) and *NR3C1* expression (Wright et al., 2014) are both substantially heritable, which creates the familiar potential for genetic confounding. The researchers acknowledged the possibility of these confounds, but interpreted them as "additional mechanism[s] by which children respond to parental behavior" (p. 7), which misses the point that the results might not reflect children responding to parental behavior at all.

A danger of reports such as these, beyond misleading other researchers, is that unjustified interpretations can be picked up in the broader culture, leading to misplaced blame on parents for negative outcomes in their children. Waldinger and Schulz's study was reported by high-profile media outlets, such as *Scientific American* (Caruso, 2016), where the findings were said to show that "how we take care of children is just so vitally important" (final paragraph). This type of reporting continues a long history of placing unjustified blame on parents for their children's outcomes. For example, the term "refrigerator mothers" was coined as a label for mothers whose lack of maternal warmth was said to cause autism (Kanner, 1949). This idea persisted for decades, even though it is now well established that autism is highly genetic and is not caused by lack of maternal warmth (Tick, Bolton, Happé, Rutter, & Rijsdijk, 2016). Parents have also been blamed for their children's schizophrenia (Hooley, 1985) and obesity (Kokkonen, 2009), whereas both disorders are primarily genetically transmitted and do not appear to be substantially influenced by parental behavior (Gejman, Sanders, & Duan, 2010; Wardle, Carnell, Haworth, & Plomin, 2008). Such reporting compounds the effects of negative childhood outcomes by adding guilt to the grief parents are already feeling for their children's suffering.

To avoid such harmful ideas arising from data that do not justify them, researchers and journal editors should consider genes, just as they consider any other possible confound, when interpreting associations between variables. Whenever genetic factors might influence the variables of interest (e.g., a trait or behavior of parents and a trait or behavior of children), even a small overlap in the genetic influences on the two variables can cause significant confounding (Barbaro, Boutwell, Barnes, & Shackelford, 2017). When potential

for genetic confounding exists, researchers must demonstrate that genetic effects are not responsible for an observed association if they wish to posit a causal interpretation. McAdams et al. (2014) have provided a review of genetically controlled methodologies and examples of studies that revealed previously identified behavioral correlations as spurious. When appropriate study designs are not possible, consideration should be given to all plausible explanations, and a balanced accounting of evidence relating to each should be provided. Given that almost every studied trait is heritable to some degree (Polderman et al., 2015), genetic contributions should almost always be considered when dealing with associations between parents' behavior and their children's behavior. Researchers, reviewers, and journal editors are all responsible for insisting on appropriate standards of evidence for causal interpretations of such associations, even when the longitudinal relationship rules out reverse causality.

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

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