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

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Longitudinal relationships between parent and child behavior need not implicate the influence of parental behavior and may reflect genetics: Comment on Waldinger and Schulz

Waldinger and Schulz (2016) [W&S] provide evidence that individual differences in parent-offspring relationship warmth during adolescence are associated with the quality of the offspring's romantic attachments sixty years later. Although this association is only a longitudinal correlation, W&S interpret it in causal terms: for example, "This study captured the long reach of warm parent-child relationships and nurturing family environment in shaping key aspects of functioning later in life" and "The findings underscore the far-reaching influence of childhood environment on well-being in adulthood." Unfortunately, genetic influences on attachment styles throughout the lifespan create a confound in such studies that call into question causal interpretations. Indeed, similar errors are common in the broader literature on the effects of parenting (see below). 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.

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 to genetic relatedness between siblings, twin studies provide estimates of the impact of genetic and environmental variation in a trait. Twin studies have documented a significant genetic component in attachment style from adolescence onwards, and little influence of family environment. Although there are no published twin studies using the Current Relationship Interview used by W&S, 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, while 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 attachment-related anxiety and 36% of the variation in avoidance in young adults' responses to the Experiences in Close Relationships questionnaire, again with no influence of the shared environment. Comparable results have been observed using adult twin data on the Relationship Scales Questionnaire, whereby the shared environment did not contribute to attachment styles, with the exception of dismissing attachment style which showed moderate shared environmental effects, accounting for 29% of variance. Genetic effects accounted for between 25% and 43% of the variance in secure, fearful, and pre-occupied attachment styles (Brussoni, Jang, Livesley, & Macbeth, 2000).

Because parents provide both genes and environment to their children, studies that investigate the latter while ignoring the former, like W&S, 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 lower quality intimate relationships when they grow up. This relationship will emerge even if there is absolutely no causal influence of the parent's cold behaviour, as the parents will have given their children the genetic predisposition for coldness. Attachment traits might be affected by parenting behaviour (as in the case of dismissiveness), but that cannot be established without controlling for genetic influences, usually with a family-based design.

W&S's design does not measure or control for genetic effects, so *no conclusions should be drawn about the effects of parenting on later adult attachment*. W&S acknowledge that "causal conclusions cannot be drawn given the study's nonexperimental nature", but this caveat is undermined by the strong causal language used elsewhere, including shortly after that statement. Moreover, the probable genetic confound is never mentioned.

This issue is not unique to W&S, as it speaks to a broader problem in psychology whereby researchers often fail to consider the potential role of genetics in the relationship between parent and offspring behavior. By way of example, we briefly describe three recent, typical examples. First, a paper published in *Prevention Science* interpreted an association between parental alcohol consumption and offspring's later drinking as causal (Donaldson,

Handren, & Crano). The paper does not mention the possibility that the association could be driven entirely by the offspring's inheritance of their parents' genetic predisposition to drink alcohol, despite the extensive evidence that alcohol consumption is heritable (Verhulst, Neale, & Kendler, 2015). Another study, in *Developmental Psychology* (Prenoveau et al., 2017), interpreted an association between maternal postnatal depression and infant 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 substantial heritability of child negative affect (Saudino, 2005) and perinatal (including pre- and post-natal) depression (Viktorin et al., 2016) are well-established.

Even molecular genetic studies are susceptible to this confound. For instance, a paper published this year in *Social Psychological and Personality Science* (Stanton et al., 2017) found associations between maternal attachment anxiety and avoidance and offspring 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". However, attachment orientation (see above) and NR3C1 expression (Wright et al., 2014) are both substantially heritable, creating the familiar potential for genetic confounding. The researchers acknowledge the possibility of these confounds, but interpret them as "additional mechanism[s] by which children respond to parental behavior", which misses the point that the results might not reflect children responding to parental behaviour 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. W&S'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". This type of reporting continues a long history of unjustified blame on parents for 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 schizophrenia (Hooley, 1985) and obesity (Kokkonen, 2009), whereas both disorders are primarily genetically transmitted and do not appear 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 parental guilt to the grief they 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 any other possible confound, when considering associations between variables. Whenever genetic factors might influence the variables of interest (e.g., a parental trait or behavior and their child's trait or behavior), even a small overlap in the genetic influences on the two variables can cause significant confounding (Barbaro, Boutwell, Barnes, & Shackelford, 2017). Where potential for genetic confounding exists, researchers must demonstrate that genetic effects are not responsible for the association if they wish to posit a causal interpretation. McAdams et al. (2014) provide a review of genetically controlled methodologies, and give examples of studies that reveal previously identified behavioral correlations as spurious. When appropriate study designs are not possible, consideration should be given to all plausible explanations, with a balanced accounting of evidence relating to each. 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 parent and child 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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