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

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

3
4 Waldinger and Schulz (2016) [W&S] provide evidence that individual differences in
5 parent-offspring relationship warmth during adolescence are associated with the quality of
6 the offspring's romantic attachments sixty years later. Although this association is only a
7 longitudinal correlation, W&S interpret it in causal terms: for example, "This study captured
8 the long reach of warm parent-child relationships and nurturing family environment in
9 shaping key aspects of functioning later in life" and "The findings underscore the far-reaching
10 influence of childhood environment on well-being in adulthood." Unfortunately, genetic
11 influences on attachment styles throughout the lifespan create a confound in such studies
12 that call into question causal interpretations. Indeed, similar errors are common in the
13 broader literature on the effects of parenting (see below). Causal inferences about parental
14 environment must be backed by appropriate evidence, and at the very least, the possibility of
15 genetic confounding should be discussed to avoid misleading other scientists and the media.

16 The evidence that genetic factors influence attachment styles is overwhelming. Much
17 of this evidence comes from the natural experiment provided by identical (i.e., 100% genetic
18 similarity) and nonidentical (i.e., 50% genetic similarity) twin pairs. By comparing trait
19 similarity to genetic relatedness between siblings, twin studies provide estimates of the
20 impact of genetic and environmental variation in a trait. Twin studies have documented a
21 significant genetic component in attachment style from adolescence onwards, and little
22 influence of family environment. Although there are no published twin studies using the
23 Current Relationship Interview used by W&S, every studied measure of attachment has
24 shown substantial genetic effects. Using data from 551 twin pairs who completed the Child
25 Attachment interview at age 15, Fearon, Shmueli-Goetz, Viding, Fonagy, and Plomin (2014)
26 estimated that genetic influences accounted for 40% of the variance in twins' responses,
27 while the influence of the shared environment (i.e., family of upbringing) was negligible.

1 Similarly Picardi, Fagnani, Nisticò, and Stazi (2011) found that genetic influences accounted
2 for 45% of the variation in attachment-related anxiety and 36% of the variation in avoidance
3 in young adults' responses to the Experiences in Close Relationships questionnaire, again
4 with no influence of the shared environment. Comparable results have been observed using
5 adult twin data on the Relationship Scales Questionnaire, whereby the shared environment
6 did not contribute to attachment styles, with the exception of dismissing attachment style
7 which showed moderate shared environmental effects, accounting for 29% of variance.
8 Genetic effects accounted for between 25% and 43% of the variance in secure, fearful, and
9 pre-occupied attachment styles (Brussoni, Jang, Livesley, & Macbeth, 2000).

10 Because parents provide both genes and environment to their children, studies that
11 investigate the latter while ignoring the former, like W&S, are inherently confounded
12 (McAdams et al., 2014). For example, parents with a genetic predisposition for coldness in
13 close relationships will tend to treat their children coldly, and their children will tend to have
14 lower quality intimate relationships when they grow up. This relationship will emerge even if
15 there is absolutely no causal influence of the parent's cold behaviour, as the parents will
16 have given their children the genetic predisposition for coldness. Attachment traits might be
17 affected by parenting behaviour (as in the case of dismissiveness), but that cannot be
18 established without controlling for genetic influences, usually with a family-based design.

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

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

1 Handren, & Crano). The paper does not mention the possibility that the association could be
2 driven entirely by the offspring's inheritance of their parents' genetic predisposition to drink
3 alcohol, despite the extensive evidence that alcohol consumption is heritable (Verhulst,
4 Neale, & Kendler, 2015). Another study, in *Developmental Psychology* (Prenoveau et al.,
5 2017), interpreted an association between maternal postnatal depression and infant
6 emotional negativity at 24-months of age as causal. Again, the possibility that a heritable
7 disposition might underlie this association was not mentioned, even though substantial
8 heritability of child negative affect (Saudino, 2005) and perinatal (including pre- and post-
9 natal) depression (Viktorin et al., 2016) are well-established.

10 Even molecular genetic studies are susceptible to this confound. For instance, a
11 paper published this year in *Social Psychological and Personality Science* (Stanton et al.,
12 2017) found associations between maternal attachment anxiety and avoidance and offspring
13 expression of a gene thought to be involved in stress regulation (NR3C1). The authors
14 interpreted these associations as causal: "our results suggest that mothers' adult attachment
15 orientations influence children's expression of a gene relevant to both adaptive stress
16 regulation and the inflammatory response". However, attachment orientation (see above)
17 and NR3C1 expression (Wright et al., 2014) are both substantially heritable, creating the
18 familiar potential for genetic confounding. The researchers acknowledge the possibility of
19 these confounds, but interpret them as "additional mechanism[s] by which children respond
20 to parental behavior", which misses the point that the results might not reflect children
21 responding to parental behaviour at all.

22 A danger of reports such as these, beyond misleading other researchers, is that
23 unjustified interpretations can be picked up in the broader culture, leading to misplaced
24 blame on parents for negative outcomes in their children. W&S's study was reported by high-
25 profile media outlets such as *Scientific American* (Caruso, 2016), where the findings were
26 said to show that "how we take care of children is just so vitally important". This type of
27 reporting continues a long history of unjustified blame on parents for children's outcomes.
28 For example, the term "refrigerator mothers" was coined as a label for mothers whose lack of

1 maternal warmth was said to cause autism (Kanner, 1949). This idea persisted for decades,
2 even though it is now well-established that autism is highly genetic and is not caused by lack
3 of maternal warmth (Tick, Bolton, Happé, Rutter, & Rijdsdijk, 2016). Parents have also been
4 blamed for schizophrenia (Hooley, 1985) and obesity (Kokkonen, 2009), whereas both
5 disorders are primarily genetically transmitted and do not appear substantially influenced by
6 parental behavior (Gejman, Sanders, & Duan, 2010; Wardle, Carnell, Haworth, & Plomin,
7 2008). Such reporting compounds the effects of negative childhood outcomes by adding
8 parental guilt to the grief they are already feeling for their children's suffering.

9 To avoid such harmful ideas arising from data that do not justify them, researchers
10 and journal editors should consider genes, just as any other possible confound, when
11 considering associations between variables. Whenever genetic factors might influence the
12 variables of interest (e.g., a parental trait or behavior and their child's trait or behavior), even
13 a small overlap in the genetic influences on the two variables can cause significant
14 confounding (Barbaro, Boutwell, Barnes, & Shackelford, 2017). Where potential for genetic
15 confounding exists, researchers must demonstrate that genetic effects are not responsible
16 for the association if they wish to posit a causal interpretation. McAdams et al. (2014)
17 provide a review of genetically controlled methodologies, and give examples of studies that
18 reveal previously identified behavioral correlations as spurious. When appropriate study
19 designs are not possible, consideration should be given to all plausible explanations, with a
20 balanced accounting of evidence relating to each. Given that almost every studied trait is
21 heritable to some degree (Polderman et al., 2015), genetic contributions should almost
22 always be considered when dealing with associations between parent and child behavior.
23 Researchers, reviewers, and journal editors are all responsible for insisting on appropriate
24 standards of evidence for causal interpretations of such associations, even when the
25 longitudinal relationship rules out reverse causality.

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