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# Rethinking the Transmission Gap: What Behavioral Genetics and Evolutionary Psychology Mean for Attachment Theory. A Comment on Verhage et al. (2016)

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**Abstract**

Traditional attachment theory posits that attachment in infancy and early childhood is the result of intergenerational transmission of attachment from parents to offspring. Verhage et al. (2016) present meta-analytic evidence addressing the intergenerational transmission of attachment between caregivers and young children. In this commentary, we argue that their appraisal of the behavioral genetics literature is incomplete. The suggested research focus on shared environmental effects may dissuade the pursuit of profitable avenues of research and may hinder progress in attachment theory. Specifically, further research on the "transmission gap" will continue to limit our understanding of attachment etiology. We discuss recent theoretical developments from an evolutionary psychological perspective that can provide a valuable framework to account for the existing behavioral genetic data.

*Keywords: Evolutionary Psychology; Behavioral Genetics; Attachment.*

# Rethinking the Transmission Gap: What Behavioral Genetics and Evolutionary Psychology Mean for Attachment Theory. A Comment on Verhage et al. (2016)

Verhage et al. (2016) present meta-analytic evidence addressing the intergenerational transmission of attachment between caregivers and young children. The authors arguments and appraisal of the literature are incomplete, however. Their assertion that research on the etiology of attachment should focus on transmission of attachment via parent traits—aspects of the shared environment—may be misguided in light of behavioral genetic evidence. The research emphasis advocated by Verhage et al. (2015) does not account for or interpret the behavioral genetic evidence regarding the sources of variance in attachment throughout the lifespan. Further, the arguments presented by Verhage et al. (2016) do not fully appreciate theoretical developments in the broader attachment literature, specifically as theory and empirical evidence relate to attachment theory as a developmental phenomenon. We argue in this commentary that these limitations may dissuade the pursuit of profitable avenues of research on attachment theory. Specifically, further research on the shared environment, in general, and on intergenerational transmission, in particular, may limit our understanding of the etiology, development, and function of attachment.

## Where is the Environment?

Traditional attachment theory posits that attachment, primarily in infancy and early childhood, is the result of intergenerational transmission of attachment from parents to offspring. Cross-generational continuity of attachment is assumed to be a product of environmental influences and, specifically, shared environmental influences such as parent-based traits. A central tenet of attachment theory is that the caregiver's attachment representations—an aspect of the shared environment—are responsible for cross-generation continuity of attachment and, consequently, are central to the etiology of attachment. According to attachment theory, "working models" of attachment are transmitted early in life from a primary caregiver, and then carried and modified throughout the lifespan (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969, 1982).

But where, exactly, is the environment? Towards which specific aspects of the environment should researchers direct their efforts when searching for environmental influences on attachment development? Recent methodological advances in behavioral genetics provide insight into the multiple sources of environmental variance, including shared environmental influences ($c^2$; aspects of the environment that work to make individuals more similar to one another) and nonshared environmental influences ($e^2$; aspects of the environment that work to make individuals less similar to one another[1]) (Plomin, DeFries, Knopik, Neiderhiser, 2013). Discussions of attachment theory, however, typically focus on shared environmental effects (particularly in infancy), ignoring nonshared influences (or, at best, conflating them with shared environmental influences). Despite the fact that they are aware of the results of behavioral genetic research concerning attachment across the lifespan (the authors cite several behavioral genetic studies in their article), Verhage et al. (2016) argue that transmission (invoking aspects of the shared environment), specifically, should remain the focus of future attachment research.

Although methodological advances in behavioral genetics afford the ability to differentiate shared environmental effects from nonshared environmental effects, traditional attachment researchers assume that parent traits exert a meaningful—and lasting—effect on attachment. One reason for this continued investigation of shared environmental effects, such as transmission, is research indicating negligible heritability of attachment in infancy. In the absence of genetic influence, the environment must explain the observed intergenerational continuity of attachment, thus aligning with a core premise of attachment theory that attachment develops as a result of transmission processes from parent to infant. However, researchers may not appreciate (or acknowledge) the "other" environment—the nonshared environment—that explains a significant proportion of variance in attachment, in addition to potential transmission

[1] It is important to note that $e^2$ also captures measurement error, which may explain why $e^2$ explains a large portion of attachment variance in infancy. As displayed in Table 1, however, $e^2$ maintains a relatively large and stable influence on attachment throughout the lifespan. This suggests that $e^2$ (above and beyond measurement error) explains a large portion of the variance in attachment throughout the lifespan.

effects. Indeed, in terms of variance explained, the nonshared environment parameter in twin studies is generally of equal or greater magnitude than the shared environment parameter.

Verhage et al. (2016) also suggest that genetic influences on attachment transmission can be ignored (at least in infancy and early childhood), given the behavioral genetic evidence produced by several studies documenting negligible heritability of caregiver-infant attachment (e.g., Bokhorst et al., 2003; Fearon et al., 2006; Roisman & Fraley, 2008). Although correct in asserting that heritability has negligible effects on attachment in infancy and early childhood, Verhage et al. (2016) inaccurately conclude that transmission—aspects of the shared environment—is primarily responsible for the development of infant attachment, and therefore its etiology, ignoring the significant role of the nonshared environment.

There are two problems with attributing infant attachment only to transmission processes and, thus, exclusively to the shared environment (i.e., the caregiver's attachment representations) on the basis of negligible heritability estimates during infancy and early childhood. First, this approach ignores effects attributable to the nonshared environment that account for about half of the variance in an infant's attachment to a caregiver. Behavioral genetic research investigating environmental influences on attachment consistently documents substantial effects of the nonshared environment on attachment in infancy and throughout the lifespan. Second, these conclusions are based on exclusive attention to, and interpretation of, research on attachment in infancy and early childhood. If attachment theory is to explain how attachment develops and manifests throughout the lifespan, *all* of the available empirical evidence, including research on attachment beyond early childhood, must be taken into account.

The conclusion that the shared environment and transmission explain attachment etiology is supported only by selective attention to a particular (early) developmental period, and is consistent with the powerful—but unfounded—assertion that parent-based traits have substantial and long-lasting effects on a child's development (Harris, 1995). It is important—and necessary—for researchers to acknowledge that attachment theory is a developmental theory that attempts to explain the etiology of attachment and the manifestation of interpersonal attachment dynamics *throughout the lifespan*. If researchers seek to

understand the etiology and the developmental trajectories of attachment from infancy through adulthood, it is important to focus research on the investigation of the nonshared environment, which consistently explains a significant proportion of variance in attachment throughout the lifespan.

**The Behavioral Genetics of Attachment**

The continued interest in the "transmission gap" has proliferated in the literature because traditional attachment theory assumes that an infant's attachment to a primary caregiver (e.g., the mother) is transmitted by the primary caregiver's attachment representations of their own primary caregiver. As in Verhage et al's. (2016) meta-analysis, caregiver attachment representations are measured with the Adult Attachment Interview (AAI), which are then used to predict infants' attachment behaviors during procedures such as the Strange Situation. By definition, however, AAI scores are aspects of the shared environment, such that a caregiver's attachment representations to their own caregiver cannot vary between offspring. If attachment transmission is central to attachment development and, consequently, is the basis for "working models" throughout the lifespan, we should expect: (1) that the shared environment contributes significantly to attachment throughout the lifespan, and (2) stability of attachment throughout the lifespan.

Ignoring the behavioral genetic evidence on attachment throughout the lifespan, however, can lead to continued propagation of the importance of transmission and the shared environment. To elucidate this point, we present in Table 1 a list of studies that have produced heritability and environmentality estimates of attachment across the lifespan (see the note to the table for inclusion criteria), several of which Verhage et al. (2016) cite to support their arguments. Taking into account the breadth of the behavioral genetic evidence on attachment, several patterns can be identified that have substantial implications for future research: (1) Shared environmental effects on attachment disappear by adolescence, (2) heritability effects emerge by 24 months of age, although they do not reach significant values until adolescence, and (3) nonshared environmental effects are significant and substantial throughout the lifespan (see *Figure 1*). These patterns are in conflict with the core premises of transmission and stability suggested by traditional attachment theory.

Consideration of behavioral genetic evidence throughout the lifespan has important implications for the refinement of attachment theory and future empirical research because this evidence calls into question the assumption that the shared environment—most often, parent-based traits, such as scores on the AAI—can account for the instantiation of attachment in infancy and throughout development. It is important to note that the assumption of stable environmental influences might hold up to future empirical investigation—but, importantly, not for the shared environment. Instead, the empirical evidence renders untenable the assumption that the shared environment and, specifically, transmission of attachment representations, exert lasting and substantive effects on attachment throughout the lifespan.

Evidence of attachment stability from infancy to adulthood, however, is relatively weak (e.g., Groh et al., 2014; but see Fraley, 2002 for meta-analysis of published studies only). Longitudinal research studies assessing stability of attachment in infancy to adulthood often document that significant life events—aspects of the nonshared environment (e.g., abuse)—result in changes in attachment representations (e.g., Waters, Merrick, Treboux, Crowell, & Albersheim, 2000). Research also demonstrates that there is only a small correlation between an individual's attachment to a parent (such as their mother) and their attachment to a romantic partner when assessed in adulthood (Fraley, Hudson, Heffernan, & Segal, 2015). Together, these findings conflict with the assumption that the transmission of attachment from caregiver to child (to the extent that this occurs) has substantial implications for attachment relationships throughout the lifespan (Harris, 1995).

Because heritability of attachment is negligible in infancy, attachment researchers conclude that the environment should be the continued focus of research. By adopting this perspective, however, research has inappropriately concluded that investigations of nonshared environmental effects are not necessary *precisely because* heritability effects are negligible in infancy. In short, researchers cite behavioral genetic studies of infants' attachment to caregivers as evidence of the importance of the shared environment—and, specifically, transmission—but neglect discussion and empirical investigation of the nonshared environment, perhaps because nonshared environmental effects are not consistent with fundamental assumptions of attachment theory as originally proposed. Verhage et al. (2016) seem to

express this confusion when discussing the implications of and future directions for research indicated by their meta-analytic findings. Specifically, Verhage et al. propose a theoretical model of shared environmental mechanisms (e.g., family functioning, couple relationship) that facilitate intergenerational transmission of attachment, despite the fact that shared environmental variance does not meaningfully contribute to attachment after early childhood. Similarly, although the overall effect of attachment transmission identified by Verhage et al. is substantially smaller than originally documented 20 years ago, the authors nevertheless emphasize the importance of future research dedicated to identifying mechanisms of attachment transmission (e.g., how caregiver attachment representations are transmitted to infants).

We contend that research on cross-generational (dis)continuity should also focus on nonshared environmental effects on attachment throughout the lifespan, rather than exclusively on shared environmental effects, such as transmission, only during infancy and early childhood. A research focus on nonshared environmental effects on attachment can better account for the behavioral genetic data. In contrast, that shared environmental effects are apparent only in infancy and early childhood is not consistent with traditional attachment theory from a developmental perspective for two reasons. First, shared environmental effects disappear by adolescence—and likely by middle childhood (behavioral genetic evidence for middle childhood is absent from the literature, see below)—when children are primarily socialized outside the home (Harris, 1995). Second, nonshared environmental effects are the only consistent environmental effects documented throughout the lifespan—precisely what attachment theory proposes and attempts to explain as a developmental theory. Even if transmission occurs—albeit not to the degree demanded by a core pillar of attachment theory—it cannot fully explain the etiology of attachment and, consequently, lacks utility to understand the development of attachment throughout the lifespan.

**Implications of the Evidence and Directions for Future Research**

Behavioral genetic evidence throughout the lifespan suggests that research on nonshared environmental effects on attachment will advance our understanding of attachment as a developmental phenomenon. On average across the lifespan, the nonshared environment accounts for nearly 60% of the

variance in attachment ($M_e^2$ = .58 across the studies listed in Table 1), whereas the shared environment and genes each account for approximately 20% of the variance in attachment ($M_c^2$ = .20, $M_h^2$ = .21 across the studies listed in Table 1)[2]. The nonshared environment is a substantial factor that must be considered, theoretically and empirically, by attachment researchers. The only designs that allow the nonshared environment to be distinguished from the shared environment are behavioral genetic designs (e.g., twin studies). Standard social science methods (such as examining one child and one parent) are not sufficient because they conflate the effects of genes ($h^2$) with those of the shared environment ($c^2$) and with effects of the nonshared environment ($e^2$) (Harris, 1995). Attachment researchers, more generally, appear to be fixated on the importance of transmission—the shared environment (especially in infancy and early childhood)—and may not appreciate the theoretical and methodological refinements that have emerged from behavioral genetics research.

Because a substantial proportion of variance in attachment throughout the lifespan is attributable to the nonshared environment, and because the function of the attachment system changes from infancy to adolescence and adulthood (Hazan & Diamond, 2000), we suggest that greater empirical effort is invested in identifying the nonshared environmental influences on the etiology and development of attachment throughout the lifespan. Shifting the research focus of attachment from shared environmental effects to nonshared environmental effects will more profitably contribute to our understanding of attachment as a lifespan phenomenon.

Specifically, we recommend that research investigates the (nonshared) environmental inputs that activate and regulate the attachment system, instead of further research investigating transmission and other parent-based variables known to be negligible in the development of attachment[3]. Advances in

[2] Average estimates of $c^2$, $e^2$, and $h^2$ were calculated as simple means across the studies listed in Table 1. Variance of the estimates and study sample sizes were not accounted for in the mean calculations.

[3] Although it is likely that parenting traits are captured by $c^2$, it is important to note that, in the case of adoptive twin studies, parenting variables are captured by $e^2$. See Daw et al. (2015) for arguments concerning the assumption that parenting variables are captured by $c^2$ and alternative arguments that $c^2$ could function to make siblings different (e.g., via sibling niching processes). However, the results produced by the non-genetically sensitive analyses conducted by Verhage et al. (2015) cannot support claims of intergenerational transmission via parental sensitivity because they cannot differentiate the various types of environmental influences.

attachment theory from an evolutionary psychological perspective (see also, Simpson & Belsky, 2016) provide a framework of which the behavioral genetic data can be accounted for and, importantly, can be used to generate hypotheses regarding the documented nonshared environmental factors that facilitate the activation of attachment mechanisms throughout the lifespan. Recognizing and appreciating the original evolutionary roots of Bowlby's attachment theory, in combination with behavioral genetic evidence and recent evolutionary psychological perspectives, we next highlight three promising avenues of attachment research.

First, recent theoretical advances integrating life history theory and attachment theory (Del Giudice, 2009; Ellis, Figueredo, Brumbach, & Scholmer, 2009; see also Kaplan & Gangestad, 2005; Simpson & Belsky, 2016) provide a foundation for generating hypotheses about the ecological factors that may affect the etiology and development of attachment (e.g., Andrews & Rosenblum, 1994; Barbaro & Shackelford, 2016; Simpson & Belsky, 2008). For instance, Ellis et al. (2009) and Simpson and Belsky (2008, 2016) provide detailed accounts of the ways in which ecological variation in morbidity-mortality rates in the local environment may predict variation in attachment (see also Barbaro & Shackelford, 2016). Appreciating the evolutionary foundations of the attachment system, more generally, Del Giudice (2009) conceptualizes attachment dimensions as facilitators of evolved sexual strategies, taking into account exposure to local ecological variants and asymmetries between men and women in reproductive biology.

Recognizing nonshared environmental factors as important in the development of attachment throughout the lifespan may account for the observation of cross-generational continuity of attachment—argued by traditional attachment researchers to be a result of shared environmental factors, such as a parent's attachment representations. In ancestral environments, local ecology was likely to be similar between generations, although individual exposure (e.g., within generational fluctuations and the perceptions of and exposure to the local ecology) may be unique to each individual within that ecology. More generally, when relevant ecological variables substantially differ for parents and offspring, we might expect greater cross-generational discontinuity in attachment. When relevant ecological variables

do not differ substantively for parents and offspring, in contrast, we might expect greater cross-generational continuity in attachment.

Vehage et al. (2015) propose "caregiver sensitivity" as a mediating mechanism between caregiver attachment representations and infant attachment. By hypothesizing that differences in parenting between offspring are a result of caregiver attachment representations (theoretically and statistically) constrains "caregiver sensitivity" to shared environmental variance—further neglecting contributions of nonshared factors to attachment etiology. To propose that caregiver sensitivity can vary between offspring is defensible (and likely influences attachment development), but to propose that caregiver sensitivity is determined by caregiver attachment representations (a trait that cannot vary between offspring) theoretically constrains caregiving to be equal across offspring—which does not align with empirical observation that caregiving does vary between offspring. However, insights from evolutionary psychology suggest that the local ecology (e.g., morbidity-mortality rates, resource availability) in which a parent is raising offspring can impact parenting (Simpson & Belsky, 2016)—allowing "caregiver sensitivity" to be captured correctly by nonshared environmental variance.

Findings reported in Verhage et al. (2016) offer peripheral support for these suggestions. In particular, Verhage et al. note the absence of attachment transmission in genetically unrelated dyads in comparison to genetically related dyads. Verhage et al. argue that the absence of transmission in the subsample of genetically unrelated dyads is attributable to small sample size and the late placement of the children into their families (and because genes do not explain attachment similarity in early childhood, the fact that the dyads are genetically unrelated cannot explain the absence of the effect). We suggest, however, that the absence of attachment transmission may be attributable to differences in ecology (aspects of the nonshared environment) between parents and offspring facilitated by the late placement of the children into their families. Similarly, Verhage et al. document a lack of transmission in "at risk" samples, which may be attributable to between-generation differences in rearing environments—in which case, the current ecology in which a parent is raising offspring is more influential to parenting practices than is the ecology in which they themselves were raised.

Alternatively, the "robust and universal" cross-generational continuity of attachment may be a spurious relationship attributable to the fact that parents and children typically do reside in similar ecologies in early childhood before children are socialized by peers. And, especially in infancy and early childhood, young children's exposure to the local ecology is under the control of the caregiver and, thus, caregiver attachment and young children's attachment is expected to correlate. Most informative would be longitudinal investigation of how between- and within-generational differences in ecology influence cross-generational (dis)continuity of attachment.

Second, functional differences between the caregiver attachment system in infancy and the romantic attachment system in adolescence and adulthood (Hazan & Diamond, 2000) may explain the increase in genetic effects on attachment that emerge in adolescence, and highlights the changing functions of the attachment system throughout the lifespan. In infancy, infant-caregiver attachment promotes infant survival—which is strongly influenced by the local ecology (e.g., resource availability, pathogen load). Thus, the caregiver attachment system evolved because it solved adaptive problems of offspring survival during a period of extreme dependency. The adult attachment system, in contrast, is posited to have been "co-opted" or evolved from the caregiver attachment system (Hazan & Diamond, 2000; Hazan & Zeifman, 1999) to solve adaptive problems of pair-bonding.

Differences in the function of the attachment system over the course of development highlights broader questions concerning core premises of traditional attachment theory: attachment is transmitted, and then influences attachment relationships throughout the life span. We have already noted how transmission as traditionally conceptualized cannot explain attachment in adulthood given the lack of shared environmental variance responsible for adult attachment. Over evolutionary history, once offspring have reached adolescence and adulthood, parents are no longer primary attachment figures but, instead, a romantic partner becomes the primary object of attachment. Thus, one must also consider the practical importance of the "transmission gap" given that: (1) Parents are no longer the primary attachment figure in adolescence and adulthood (Hazan & Diamond, 2000; Simpson & Belsky, 2016), (2) the stability of parental attachment throughout the lifespan is relatively weak (Groh et al., 2014), (3) the covariance

between attachment to parents and attachment to romantic partners is small (Fraley et al., 2015; Roisman et al., 2007), and (4) assessment of specific attachment bonds have more predictive utility over general attachment "styles" (Barry, Lakey, Orehek, 2007).

Moreover, because the adult attachment system interacts with other psychological mating mechanisms during and after puberty, the significant heritability estimates for attachment that emerge in adolescence (and perhaps as early as middle childhood, see *Figure 1*) may be attributable to the variance that romantic attachment shares with other heritable reproductively-relevant traits (e.g., pubertal development, fertility). Research examining variation in attachment security from infancy to adulthood would benefit from investigating individual exposure to ecological (nonshared environmental) factors known to affect infant survival, intrasexual and intersexual competition, and mating strategies. Numerous evolutionarily-relevant ecological factors—and, importantly, an individual's perception of and exposure to the local ecology—such as morbidity-mortality rates, fluctuations of morbidity-mortality rates (e.g., Simpson, Griskevicius, I-Chun Kou, Sung, & Collins, 2012; Szepsenwol, Simpson, Griskevicius, & Raby, 2016), pathogen load (e.g., Hill, Prokosch, & DelPriore, 2015), and resource availability (e.g., Andrews & Rosenblum, 1994), are associated with survival, competition, and mating strategies. Thus, these factors may be particularly valuable to assess in future research investigating the effects of the nonshared environment on the development of the attachment system from an evolutionary psychological perspective.

And, lastly, there is an empirical gap in behavioral genetic evidence on attachment in middle childhood. Heritability and environmentality estimates of attachment in middle childhood are important for a comprehensive understanding of the sources of variance responsible for the development of attachment because of the emergence of genetic effects explaining significant variance in attachment documented in the literature (see Table 1 and *Figure 1*). The conclusion that genes do not affect the development of attachment is not warranted, especially given that genetic effects become significant in adolescence (and continue throughout adulthood), when mating becomes a prominent adaptive problem. Recently, theoretical frameworks have been advanced that highlight the importance of middle childhood

as a "developmental switch point" for attachment processes (Del Giudice, 2015). Specifically, Del Giudice (2015) proposes that attachment in middle childhood guides successful navigation of peer relationships concerning social status and competition, which provides information about an individual's relative position in the mating market and may facilitate mating opportunities in adolescence. Support for this notion is gleaned from sex differences in attachment that emerge in middle childhood (Del Giudice & Angeleri, 2016) that parallel sex differences documented in adult attachment (Del Giudice, 2011).

These recent theoretical and empirical advances addressing attachment in middle childhood suggest that genetic effects on attachment may become significant in middle childhood as a transitional state between caregiver attachment and romantic attachment. An important next step in the refinement of attachment theory is the use of behavioral genetic methodology to determine heritability and environmentality estimates of attachment in middle childhood. Knowledge of the developmental progression of heritability effects *vis-à-vis* environmental factors during the first decade of life would afford greater refinement of recent evolutionary theories and tests of hypotheses concerning attachment in middle childhood (Del Giudice, 2015), specifically, and give credence to differing functions of attachment throughout development more generally.

**Conclusions and the Way Forward**

In conclusion, Verhage et al.'s (2016) claims regarding the apparent "robust and universal" support for intergenerational transmission of attachment and, more importantly, their suggestions regarding further investigation of shared environmental effects to explain the etiology and development of attachment are misguided. Attachment theory is one of the most well-researched and generative contributions to psychological science. However, as with any theory, new data—in this case, the full scope of behavioral genetic evidence—requires theoretical refinement. Specifically, nonshared environmental effects remain consistent and important throughout the lifespan and, therefore, should be a substantive focus of research addressing the etiology and development of attachment.

A new focus of attachment research on nonshared environmental effects requires behavioral genetic designs that afford differentiating heritability from shared and nonshared environmental

influences. Recent theoretical developments from an evolutionary psychological perspective provide a valuable framework by which to account for the existing behavioral genetic data, and from which new hypotheses about attachment development can be generated. Overall, continued research on the effects of the shared environment—specifically, the transmission of attachment—belies a broader reluctance to acknowledge and appreciate the theoretical progression of attachment theory over the preceding decades. An evolutionary psychological perspective can better account for the existing data and afford a more comprehensive understanding of attachment throughout the lifespan.

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**Table 1.** *Heritability and Environmentality Estimates from Behavioral Genetic Studies of Attachment throughout the Lifespan.*

| Study | Age | *n* (Twin Pairs) | Attachment Measure | Dependent measure for genetic analyses | $h^2$ | $c^2$ | $e^2$ |
|---|---|---|---|---|---|---|---|
| Fearon et al. (2006)[a] | 12 months | 136 | Strange Situation Procedure | Secure vs. Insecure | .00 | .25 | .75 |
| Bokhorst et al. (2003) | 12-14 months | 157 | Strange Situation Procedure | Secure vs. Insecure | .00 | .52 | .48 |
| Bakermans-Kranenburg et al. (2004) | 14-15.5 months | 56 | Attachment Q-Sort | Security | .00 | .59 | .41 |
| Roisman & Fraley (2008) | 24 months | 485 | Attachment Q-Sort | Security | .17 | .53 | .30 |
| O'Connor & Croft (2001) | 42-45 months | 220 | Strange Situation Procedure | Security | .14 | .32 | .53 |
| Fearon et al. (2014) | 15 years | 551 | Child Attachment Interview | Secure vs. Insecure | .35 | .00 | .65 |
| Donnellan et al. (2008) | 18-28 years (*M* = 20.9) | 273 | Adult Attachment Scale | Anxiety<br>Avoidance | .45<br>.39 | .00<br>.00 | .55<br>.61 |
| Crawford et al. (2007) | 16-79 years (*M* = 30.9) | 239 | Relationship Scales Questionnaire | Anxiety<br>Avoidance | .40<br>.00 | .01<br>.30 | .59<br>.70 |
| Brussoni et al. (2000) | 17-79 years (*M* = 31.3) | 220 | Relationship Scales Questionnaire | Secure<br>Preoccupied<br>Dismissing<br>Fearful | .37<br>.25<br>.00<br>.43 | .00<br>.00<br>.29<br>.00 | .63<br>.75<br>.71<br>.57 |

*Notes*. $h^2$ = heritability estimates; $c^2$ = shared environmental estimates; $e^2$ = nonshared environmental estimates. Estimates obtained from full ACE models when reported. [a]Subsample of Bokhorst et al. (2003). Sample of studies drawn from the PsychINFO database at the university of the corresponding

author using the search terms “behavior genetic” + “attachment”. Studies were excluded if information reported in Table 1 was not provided by the authors.

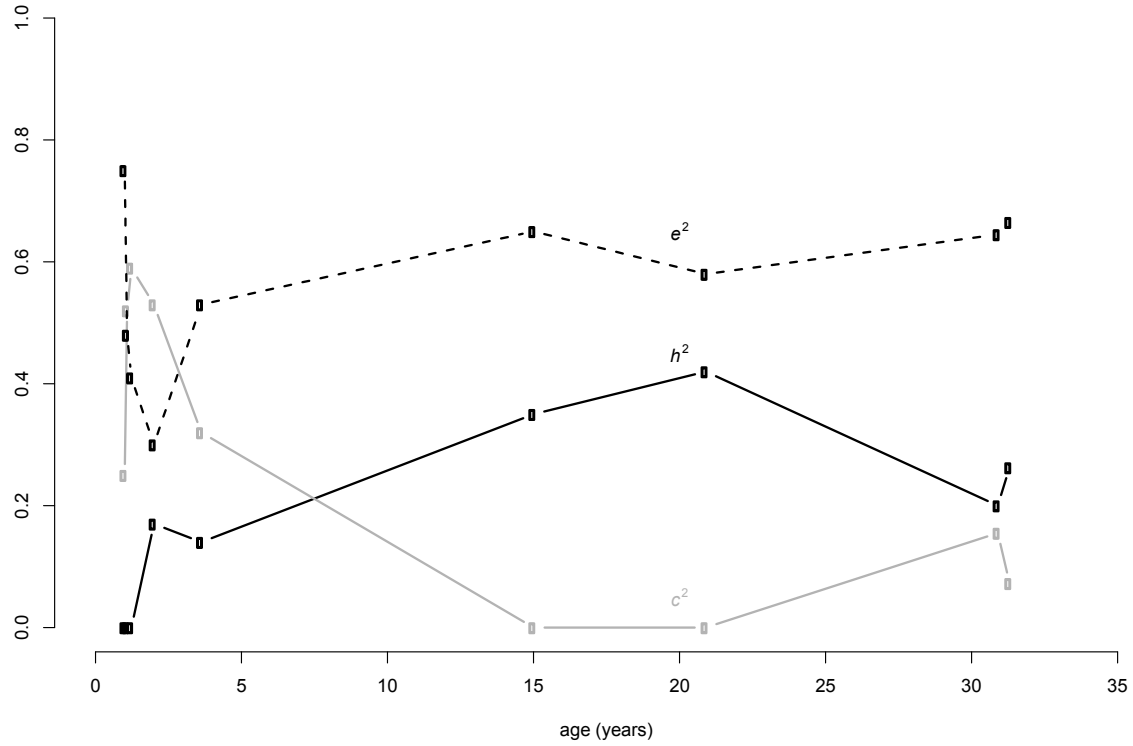


*Figure 1.* Graphical representation of the heritability estimates ($h^2$), shared environmental estimates ($c^2$), and nonshared environmental estimates ($e^2$) reported in Table 1. *Notes*. When multiple estimates are reported within a study (see Table 1), the average is computed and shown here; the *x*-axis (age) reflects the age (in years) of the sample or the mean age of the sample as indicated in Table 1.