In Search of Shared and Nonshared Environmental Factors in Security of Attachment: A Behavior-Genetic Study of the Association Between Sensitivity and Attachment Security

R. M. Pasco Fearon
University College London

Marinus H. Van IJzendoorn
Leiden University

Peter Fonagy
University College London

Marian J. Bakermans-Kranenburg
Leiden University

Carlo Schuengel
Vrije Universiteit Amsterdam

Caroline L. Bokhorst
Leiden University

The current article presents results from a twin study of genetic and environmental components of maternal sensitivity and infant attachment and their association. The sample consisted of 136 twin pairs from 2 sites: Leiden, the Netherlands, and London, UK. Maternal sensitivity was assessed in the home at 9–10 months, and infant attachment security was observed in the laboratory at 12 months. The study yielded little evidence that genetic factors are involved in variations between twins in maternal sensitivity ratings but did find that shared variance in maternal sensitivity was able to account for some of the similarity between twins in attachment security. Weak nonshared associations between sensitivity and attachment appeared to suppress the magnitude of the correlation between attachment and sensitivity in twin children. The results could indicate that the attachment security of one twin may depend on the relationship the parent has with the other twin. The results are brought to bear on the validity of attachment theory as a theory of primarily shared environmental effects in children’s development and the continuing challenge posed to attachment theory by within-family differences in socioemotional processes.

Keywords: attachment, maternal sensitivity, genetics, shared environment, nonshared environment

Behavior-genetic studies have had a substantial impact on the way developmentalists think about the kinds of influences that produce individual differences in cognitive abilities, socioemo-
Attachment research is a prime example of a socioemotional research program that has generally presumed environmental mechanisms without, until recently, testing for possible genetic effects or considering specific environmental experiences that make children in the same family different. Nevertheless, attachment theory makes particularly clear predictions about environmental causal processes and has generated a wealth of data on the associated precursors and consequences of attachment security using nongenetic research designs.

On balance, the evidence from the small set of attachment studies that have used a univariate behavior-genetic design (Bakermans-Kranenburg, Van IJzendoorn, Bokhorst, & Schuengel, 2004; Bokhorst et al., 2003; Finkel, Wille, & Matheny, 1998; O’Connor & Croft, 2001; Ricciuti, 1992) has shown little difference between genetically identical twins (monozygotic [MZ] twins) and twins who share on average 50% of their genes (dizygotic [DZ] twins) in the degree of similarity of their attachment classifications, which suggests that genetic effects on attachment security are limited. It is interesting that organized and disorganized attachment classifications appear to behave differently when subjected to behavior-genetic analysis (Bokhorst et al., 2003). In an earlier analysis of the attachment data presented in this article, no similarity was detected in twins (MZ or DZ) with respect to attachment disorganization, which was estimated to be affected only by the nonshared environment and measurement error. In contrast, the overall security of twins in a family was quite similar when pairs with disorganized classifications were excluded. Among the organized cases, 50% of the variance was attributable to shared environmental effects, and 50% was attributable to the nonshared environment (Bokhorst et al., 2003). With the organized attachment patterns, twins thus appeared relatively similar to each other in terms of attachment security, regardless of genetic differences, which points to the importance of shared environmental factors, such as parental sensitivity.

Parental sensitivity is generally regarded as the single most important determinant of infant attachment security, particularly for the main organized attachment strategies (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969; Pederson & Moran, 1995). However, researchers do not know whether parental sensitivity is shared or nonshared across twins and siblings or, critically, whether sensitivity is associated with the shared or nonshared aspects of attachment security. Studies that have examined parental sensitivity in relation to one child’s attachment security are incapable of illuminating the extent to which any association is specific to a single parent–infant dyad or generalizes to other children in the family. In the current article, we examine the degree of environmental and genetic contribution to variations in maternal sensitivity as it impinges on the child and examine the extent to which genetic and environmental aspects of maternal sensitivity can account for the pattern of similarity and dissimilarity in twins’ attachments to their mother.

Since Ainsworth et al. (1978) original work on naturalistic patterns of mother–infant interaction and attachment, the role of maternal sensitivity has been central to contemporary attachment theory (Bretherton, 1990). The results of observational and experimental studies of attachment have broadly confirmed the idea that sensitive responsiveness to a child’s attachment signals is significantly and causally related to security, although the mean effect size is relatively modest (in De Wolff & Van IJzendoorn’s, 1997, meta-analysis, the mean effect was $r = .24$; see also Bakermans-Kranenburg, Van IJzendoorn, & Juffer, 2003). It is important to note that the association between maternal sensitivity and attachment appears to be restricted to the organized attachment patterns, in a way that is reminiscent of the aforementioned restriction of shared environmental effects to organized cases. Disorganized attachment and sensitivity have been found to be very weakly associated. In Van IJzendoorn, Schuengel, and Bakermans-Kranenburg’s (1999) meta-analysis of 13 studies, the mean effect size for the association between sensitivity and disorganized attachment was $r = .10$.

The evidence base to date is mute as far as the relative role of genetic and environmental factors (shared or nonshared) in shaping parental sensitivity as it impinges on the child or the extent to which identified associations between sensitivity and attachment are shared or nonshared in nature. On the basis of existing evidence, there are three plausible ways of conceptualizing the determinants of sensitivity. First, shared environmental effects on sensitivity are suggested by studies that have shown an association between parental state of mind in the Adult Attachment Interview (Main & Goldwyn, 1998) and maternal sensitivity (Fonagy, Steele, & Steele, 1991; Van IJzendoorn, 1995). Theoretically, the parent’s state of mind in relation to attachment is held to be the consequence of his or her own attachment history and should exert a fairly constant effect on caregiving behavior (Steele & Steele, 1994). Such an account (which we refer to as Hypothesis 1, shared environmental effects) leads to the prediction that twins should experience similar levels of sensitivity or insensitivity to each other regardless of their genetic similarity (Bokhorst et al., 2003).

A second, competing account invokes genetic explanations of consistency of parenting across siblings. By this account, genetically based characteristics of the child determine the parent’s capacity to react sensitively to his or her child (O’Connor, Croft, & Steele, 2000). Consistent with this, studies of children who are twins or adoptees have indicated significant genetic effects on measures of the parenting the children receive (Feinberg, Neiderhiser, Howe, & Hetherington, 2001; Kendler, 1996; Plomin, 1995; Rowe, 1981), including retrospective studies of parental negativity and acceptance–rejection (Braungart-Rieker, Rende, Plomin, & DeFries, 1995; Feinberg et al., 2001) and observational studies of parenting in infancy and childhood (Braungart, 1994; Braungart, Fulker, & Plomin, 1992; DiLalla & Bishop, 1996; Rende, Slomkowski, Stocker, Fulker, & Plomin, 1992). It is critical to note that what these studies refer to is not whether parents’ genes influence the way they parent but whether parenting as it impinges on the child is affected in any way by that child’s genes (i.e., genetically based child effects). This account predicts that identical twins should experience more similar levels of maternal sensitivity than nonidentical twins (Hypothesis 2, genetic effects).

Finally, one could also envisage an account in which the level of maternal sensitivity emerges as a unique characteristic of a specific parent–child relationship (Hypothesis 3, nonshared effects). Twin studies of parenting behavior, although they point to genetic factors, also highlight the extent to which parenting behavior may be a feature of the nonshared environment (Moore, Cohn, & Campbell, 1997). For example, typically, at least 50% of the variance in observed parenting behavior is estimated to be nonshared (although this also includes measurement error). In Deater-Deckard’s (2000) study of preschoolers (mean age 43 months), for example,
between 50% and 80% of the variance in observed parenting (including measures of positive and negative affect, responsiveness, and control) was estimated to be attributable to nonshared environmental effects and measurement error. Somewhat lower (32%) estimates were obtained by Leve, Winebarger, Fagot, Reid, and Goldsmith (1998) in a sample of 159 school-aged twins (median age 8 years). The prediction from a nonshared account of maternal sensitivity is that twins should be no more similar in the levels of sensitivity they experience than children taken from entirely different families, once genetic effects have been accounted for. These differing possibilities regarding the determinants of variation in maternal sensitivity as it impinges on the child raise important issues not only for theory but also for practice. For example, if maternal sensitivity were substantially influenced by shared environmental factors, interventions might be appropriately directed toward a parent’s general capacity to be sensitive (perhaps because of limited skills or state of mind with respect to attachment). In contrast, both genetic and nonshared accounts point toward the importance of intervening at the level of the individual parent–infant dyad. These three competing hypotheses are tested in the current study with standard behavior-genetic models for MZ and DZ twins, which decompose the variance in maternal sensitivity ratings into that attributable to genetic, shared environmental, and nonshared environmental factors.

Standard methods for estimating genetic and environmental effects on behavior involve one dependent variable and are referred to as univariate behavior-genetic designs. Essentially, they rely on the level of correlation between twins in a single trait or domain of measurement and differences in the magnitude of this correlation between MZ and DZ twins. In recent years, there has been increasing recognition of the limitations of univariate methods and the benefits that accrue when multivariate designs are used (e.g., Rutter, 2003). One important weakness of univariate designs is that, rather than testing theoretical accounts of specific developmental mechanisms, univariate genetic analyses quantify only the cumulative effects of a potentially large number of unspecified processes (genetic or environmental). This measurement-driven approach (as opposed to a theory-driven one) brings with it several problems. First of all, estimates of genetic and environmental effects are very sensitive to the exact method by which the dependent variable is measured, which is especially problematic when the goal is simply to quantify genetic and environmental sources of variance. Consumers of behavior-genetic data thus routinely have to bear in mind that estimates are often inflated or deflated in one direction or another because of various sources of error (e.g., rater bias in parent reports; see Rutter, 2003). Second, not taking measurements of proposed mechanisms or established correlates makes it difficult for researchers to evaluate the extent to which behavior-genetic findings fit with what is already known about the subject under study or whether causal interpretations of one kind or another are plausible. Finally, the identification of components of variance that can be broadly attributed to genetics or environment tells us little, if anything, about what specific factors are actually responsible (Deater-Deckard, 2000).

Multivariate behavior-genetic designs, in which more than one dependent variable is measured, avoid many of these problems by examining the links between independent constructs (and hence testing relatively error-free associations). With these methods, behavior-genetic studies also have the potential to address more theory-oriented questions by examining the extent to which genetic and environmental factors underlie associations between putative causes and effects. In the present context, a common interpretation of attachment theory would lead one to expect that the association between sensitivity (the putative cause) and attachment (the putative effect) would be accounted for by shared environmental factors (Hypothesis 4, shared correlation). For example, if it is true, as many attachment researchers have argued, that maternal sensitivity is influenced by a parent’s (singular) state of mind with respect to attachment (and the evidence suggests that, to a significant degree, it is; see Van IJzendoorn, 1995) and this, in turn, causes variations in attachment security, then consistencies in parenting behavior between twins ought to lead to consistencies in the twins’ attachment classifications (regardless of their zygosity). Statistically, this can be tested in terms of the correlation between the shared environmental effects on sensitivity, on the one hand, and attachment, on the other, in a bivariate behavior-genetic model (Plomin et al., 2001). In effect, evidence for this shared environmental model would be found if the attachment classification of each twin could be equally well predicted from the sensitivity shown toward either twin, regardless of genetic similarity. In other words, the correlation between sensitivity and attachment for one twin would be of a similar magnitude to the cross-correlation between one twin’s sensitivity rating and the other twin’s attachment security. Bivariate behavior-genetic models use these differences in within-twin and cross-twin correlations to estimate genetic, shared environmental, and nonshared environmental correlations between two measures (see Purcell, 2001). Given that disorganized attachment has been found to only very weakly correlate with maternal sensitivity (Van IJzendoorn et al., 1999), we also hypothesized that this shared pathway model would be most evident among infants with organized attachment patterns (i.e., most evident when disorganized cases were excluded).

In contrast, an account could be considered in which common genetic factors underlie the association between maternal sensitivity and attachment. In other words, sensitivity and attachment might be associated because they are both influenced by the same genes. For example, genetically influenced temperamental proneness to distress or difficulty could directly influence attachment behavior and evoke less sensitivity from parents. In such scenarios, genetic modeling would estimate a significant correlation between the genetic effects on sensitivity and attachment. This genetic correlation would be evident if the sensitivity expressed to one twin was a better predictor of the other twin’s attachment classification in identical twins than in nonidentical twins (Hypothesis 5, genetic correlation). Finally, a nonshared account would lead to the prediction that, despite a positive association between a parent’s sensitivity to one twin and that same twin’s attachment security, the sensitivity rating would provide no prediction in relation to the other twin’s attachment security. In other words, the association between sensitivity and attachment represents a dyad-specific process that does not generalize to the other twin (by genetic or environmental means). This nonshared account is tested by the correlation between the nonshared environmental effects on sensitivity and attachment and would be indicated by a strong within-twin correlation between sensitivity and attachment but a low cross-twin correlation (Hypothesis 6, nonshared correlation).

Several recent studies have used bivariate behavior-genetic designs to test theoretically derived predictions regarding associa-
tions between two or more developmentally salient variables. For example, Deater-Deckard (2000) has presented a twin study that investigated the link between parenting and behavior problems in young children. Deater-Deckard argued that socialization models of behavior problems predict that the association between parenting and behavior problems should be characterized by shared environmental pathways, in the sense that parenting is assumed to be a shared environmental variable and should lead to similarities between twins in their behavior problems. This is precisely what was found. Not only was evidence found for shared environmental effects on behavior problems, but also a specific measure of the proposed environmental mechanism (parenting behavior) was shown to account for it. It is notable that such a shared environmental pathway was only apparent when objective observational measures of parenting or child behavior were used. Parent reports yielded substantially larger genetic estimates and predominantly genetic mediation between parenting and child behavior problems. A variety of studies have also found meaningful correlates of genetic effects on measures of cognitive abilities and psychopathology (e.g., Rijndijk & Boomsma, 1997; Thapar, Harold, & McGuffin, 1998).

The current study thus set out to

1. examine the extent of genetic and environmental contribution to variation in maternal sensitivity as it impinges on the child by contrasting three hypotheses: a shared environmental effects account (Hypothesis 1), a genetic effects account (Hypothesis 2), and a nonshared effects account (Hypothesis 3); and

2. examine the genetic and environmental contributions to the correlation between sensitivity and attachment by contrasting three parallel hypotheses: a shared environmental correlation account (Hypothesis 4), a genetic correlation account (Hypothesis 5), and a nonshared correlation account (Hypothesis 6).

It should be noted that, to the extent that the predictions outlined above are quantitative, finding a significant effect for one hypothesis does not preclude that other hypotheses will also be supported by the data. The present article is a follow-up of an earlier report on this sample that examined genetic and environmental effects on attachment security in isolation (Bokhorst et al., 2003).

Method

Participants

Participants came from two twin studies, conducted in London, UK, and Leiden, the Netherlands. The two studies used similar designs and measures.

The Leiden twin study. Seventy-six twin pairs participated in this study, with 27 MZ and 49 DZ same-sex pairs. The families were recruited through the Netherlands Twin Register (Boomsma, Orlebeke, & Van Baal, 1992), which contains 40%–50% of all multiple births after 1986. DZ pairs are somewhat overrepresented among same-sex twins of this age, because of the dizygosity of multiple births that are the result of fertility treatment. Eighteen families in our sample reported fertility treatment. The sample nevertheless contains comparatively more DZ twins than the London sample (in which rates of fertility treatment were also relatively high). It is likely that the relatively large proportion of same-sex DZ twins in this sample, compared with the London sample, is a result of minor differences in recruitment (community recruitment in Leiden, hospital recruitment in London) and chance sampling variation. Most families in the sample were middle class. On a scale for educational level, ranging from 1 (elementary school) to 7 (university degree), the mean level was 4.5 (SD = 1.7). Half the sample (50%) had 4 years of secondary education, and only 13% held university degrees. The mean age of the mothers was 32 years (SD = 3.6). Fifty-one percent of children in the Leiden sample were boys, and 49% were girls. In this sample, 61% of infants were first born (i.e., no siblings), 27% were second born, and 12% were third or fourth born.

The London twin study. The original sample consisted of 81 twin pairs, with 30 MZ twin pairs, 32 same-sex DZ twin pairs, and 19 DZ opposite-sex twin pairs, living in and around London. The families were recruited through the Multiple Births Foundation of Queen Charlotte’s and Chelsea Hospital in London. In 32% of cases, the twins were born after fertility treatment (primarily in vitro fertilization or intracytoplasmic sperm injection). Most of the families were middle class and well educated. Nearly half the sample (49%) held university degrees, whereas 20% had high-school-aged qualifications only. The mean age of the mothers was 35 years (SD = 5.4). The twins were between 12 and 13 months when observed in the Ainsworth Strange Situation (Ainsworth et al., 1978). Opposite-sex twins were excluded from the current article because the small different-sex subset would preclude statistical tests of gender and reduce the comparability of DZ twins and MZ twins. In this sample, 59% were boys and 41% were girls (not a significant departure from 50%). χ²(1, N = 62) = 2.32, p = .13. Sixty-one percent of the infants were first born, 29% were second born, and 10% were third or fourth born.

Design and Procedure

All infants were observed in the Ainsworth Strange Situation for assessment of mother–child attachment (Ainsworth et al., 1978) at 12 months of age, after correction for prematurity. In London, both twins were tested during the same visit to the laboratory, with the twins’ father looking after the child who was not currently being assessed. In Leiden, the Strange Situations for each twin were conducted on separate days. Home observations of maternal sensitivity were conducted by two trained researchers when the infants were between 9 and 10 months, and in both locations (Leiden and London) the twins were observed together with their mother. The visits were scheduled at a time when the mother expected the babies to be lively and when a feeding could be observed. During the observation, mothers and babies were filmed during normal, unstructured activities around the home and during two more structured activities: feeding and free play with the mother and each infant separately. Moreover, mothers were asked to complete a questionnaire as a demand that competed with attention to the children (see Pederson & Moran, 1995, 1996). Each session lasted approximately 2 hr, of which 1.5 hr were videotaped. In both studies, the mothers were asked to assess the temperament of their children by completing a questionnaire for each child separately. Mothers filled out the Infant Behavior Questionnaire (Rothbart, 1981) in Leiden and the Infant Characteristics Questionnaire (Bates, Freeland, & Lounsbery, 1979) in London. In designing a sensitivity assessment for twin infants, we faced a dilemma about whether to observe parents with both children present or to conduct a separate assessment of each child. We reasoned that the former strategy would be preferable, as assessments of sensitivity are designed to measure aspects of parenting that represent routine parenting behavior as it typically occurs at home (Pederson & Moran, 1995). In that sense, they are designed to be naturalistic and ecologically valid as far as possible. In twins, the ecology is such that observations of parental sensitivity in the absence of the other twin might be unrepresentative of typical interactions in the home and hence might be less likely to detect processes that are directly implicated in the development of that relationship. In choosing this option, we also followed the only previous study of this kind that looked at sensitivity and attachment in twin pairs (Goldberg, Perrotta, Minde, &
Corter, 1986). Whether and to what extent the results of this study hold up under different observational conditions need to be evaluated in future research.

In two cases from the London sample, scheduling problems meant that sensitivity assessments could not be conducted. The total sample with complete data on sensitivity and attachment was thus 136 twin pairs.

Measures

Strange Situation procedure. The well-known and standard Strange Situation procedure was used to assess infant–mother attachment security in both samples. The procedure consists of three stressful components: the infant and the mother enter an unknown laboratory playroom, a stranger comes in and tries to play with the infant, and the mother leaves the room twice for a brief period. In particular, infants’ behavior at reunion with the mother is essential for coding the quality of the attachment relationship. On reunion, secure infants (B category) seek proximity but, after being cuddled or otherwise reassured, explore the environment again. Nonscure–avoidant infants (A category) avoid the mother and seem to remain focused on the environment, whereas nonscure–resistant infants (C category) seek proximity but resist contact with the mother (Ainsworth et al., 1978). Some secure or nonscure attachment relationships appear to be characterized by the (sometimes momentary) absence or breakdown of an otherwise organized strategy, hence defined as disorganized attachment (D classification; Main & Solomon, 1990). Indexes of disorganized attachment behavior expressed in the Strange Situation are as follows: sequential or simultaneous display of contradictory behaviors, such as distress and avoidance; undirected or misdirected movements and expressions; stereotypies and anomalous movements or postures; freezing or stilling behaviors; expressions of fear or apprehension regarding the parent; and clear indications of confusion and disorganization in the presence of the parent (Main & Solomon, 1990). At each site, Strange Situations were coded by experienced coders who reached satisfactory intercoder reliability on both three-way classifications (\(<\chi^2 > .73\)) and four-way classifications (\(<\chi^2 > .70\)). In Leiden two coders rated the attachment assessments, whereas in London ratings were conducted by three coders. Overall, 43 infants were classified as avoidant, 135 were classified as secure, 46 were classified as resistant, and 50 were classified as disorganized. Compared with the meta-analytic distribution in Van IJzendoorn et al. (1999) of 15% avoidant, 62% secure, 9% resistant, and 15% disorganized classifications (N = 2,104), the current distribution showed an overrepresentation of resistant attachments and an underrepresentation of secure infants, \(\chi^2(3) = 26.66, p < .01\); standardized residual was 4.30 for the resistant classification and −2.49 for the secure infants. Of the 50 disorganized cases, 13 were given subclassifications of avoidant, 15 were subclassified as secure, and 22 were subclassified as resistant. In the present study, the insecure categories (A, C, D) were analyzed as a group in comparison with the secure group because of the relatively small numbers in each insecure category and because of the significance of the basic distinction between security and insecurity in attachment theory (e.g., Waters & Beauchaine, 2003). This binary dummy variable (secure vs. insecure) was used in all attachment analyses in this report.

Maternal sensitivity. Ratings of maternal sensitivity were conducted for each twin by an independent coder from videotaped observations. In London two raters coded the data (one coded Twin 1 and the other coded Twin 2), whereas in Leiden four raters coded the data. Ainsworth et al.’s (1978) coding system for rating maternal sensitivity was used at both sites. The Sensitivity Scale is rated from 1 to 9, with each point anchored by detailed descriptions of typical mother–infant dyads for that score. In Ainsworth et al.’s scheme, maternal sensitivity comprises four aspects: (a) the mother’s awareness of her baby’s signals, (b) an accurate interpretation of them, (c) an appropriate response, and (d) a prompt response. The final score represents the extent to which all four of these components of sensitivity were evident during the observations for a specific mother–infant dyad. An example of an anchoring statement for Point 7 on the Sensitivity Scale (Ainsworth, Bell, & Stayton, 1974) follows:

This mother also interprets the baby’s communications accurately, and responds to them promptly and appropriately—but with less sensitivity than mothers with higher ratings. She may be less attuned to the baby’s more subtle behaviors than the highly sensitive mother. Or, perhaps because she is less skillful in dividing her attention between baby and competing demands, she may sometimes “miss her cues.” (p. 132)

Interrater reliability was .85 and .81 in Leiden and London, respectively.

Zygosity determination. In the Leiden sample, zygosity was determined with Goldsmith, Rieser Danner, and Briggs’s (1991) Zygosity Questionnaire for Young Twins. This questionnaire was completed three times by the mother: when the twins were 10 months of age, 12 months of age, and 3 years of age. Questions concern similarities of physical features of the twins and experiences of mistaking one twin for another. To validate our decision about the twins’ zygosity, we used Bieda et al.’s (2000) discriminant equation. Although this equation was developed for 6-year-old children, it seemed reasonable to use it for validation of our decision in 3-year-olds. According to this equation, all but 1 pair were correctly classified. This pair was reclassified as DZ.

For a substantial number of the London twins, information about placentation was available. From this information, 47% of these twins were identified as MZ. For the other twins, zygosity was determined via a genetic test (Freeman et al., 1997). Parents were given a test kit and instructions when they were at the laboratory for the Strange Situation procedure. They were asked to take the DNA samples of their twins (a sample of cheek cells) at home and to send them to Freeman’s laboratory (Freeman et al., 1997) for zygosity determination.

Data Preparation

Because in the Leiden data set twins were assigned to the Twin 1 and Twin 2 groups on the basis of birth order, whereas in the London data set assignment to Twin 1 and Twin 2 was done at random, the combined data set was rerandomized.

Statistical Analysis

To test the hypotheses outlined in the introduction, we used standard univariate and multivariate behavior-genetic models (Neale & Cardon, 1992). To test the extent of genetic and environmental effects on maternal sensitivity (Hypotheses 1–3), we used the program Mx (Neale, Boker, Xie, & Maes, 1999) to decompose the covariance between twins in sensitivity into that due to genetic factors, shared environment and nonshared environment, and measurement error using maximum likelihood estimation. The method is based on the expectation that (a) genetic effects will lead to greater similarity (covariance) in MZ than in DZ twins, (b) shared environmental effects will lead to similarities between twins irrespective of zygosity (i.e., with genetic effects controlled), and (c) nonshared effects will lead to differences between twins that cannot be accounted for by zygosity. Using statistical models based on these assumptions, one can derive terms from covariance matrices that provide estimates of the proportion of variance in a measure that is attributable to genes (labeled A), shared environments (labeled C), and nonshared environments (labeled E). The fit of the models is tested with the likelihood ratio chi-square statistic and Akaike’s information criterion (AIC). The significance of model parameters (A, C, or E) can be tested by the increase in chi-square when a parameter is deleted from the model. An increase of the chi-square value exceeding 3.84 for a single degree of freedom indicates significant deterioration of model fit. The AIC statistic can also serve as a guide to the best fitting model and is large and negative when model fit is good. It favors parsimony by giving greater weight to models with fewer parameters. The
significances of these parameters (C, A, and E) represent direct tests of Hypotheses 1–3 (respectively) outlined in the introduction.

To test the extent to which genetic and environmental effects underlie the association between sensitivity and attachment, bivariate genetic models were used (Neale & Cardon, 1992). Multivariate genetic analysis (of which the bivariate model is an instance) is an extension of the genes–shared environments–nonshared environments (ACE) model for analyzing genetic and environmental contributions to relationships between two or more measured variables (Plomin et al., 2001). The method works by decomposing the correlation between the measures into that due to genetics, that due to the shared environment, and that due to the nonshared environment (for more details, see Neale & Cardon, 1992).

The basic bivariate model is shown in Figure 1. Estimates of genetic and environmental effects (a, c, and e) on each measured variable are derived from cross-twin correlations or covariances (on the same measure) in a manner parallel to that for univariate analysis. However, in addition to that, the genetic and environmental latent variables are allowed to correlate across measures, and these correlations represent the extent to which common genetic or environmental factors underlie the observed association between the two measures. How much of the association between them is due to common genetic or environmental factors is estimated from the pattern of within-twin and cross-twin correlations between one measure and the other. Thus, if the cross-twin correlation does not vary between MZ and DZ twins, this suggests that environmental factors are responsible for the association between the two measures. If the within-twin correlation is relatively high and the between-twins correlation is also high (in both MZ and DZ twins), this indicates that the environmental processes responsible for the association are shared across twins (and a shared environmental correlation would be detected by the model, $r_{c}$). The significance of this shared environmental correlation represents a test of Hypothesis 4. However, if the within-twin correlation is high but the cross-twin correlation is low, this suggests that the association derives from a process that is twin specific (nonshared), and bivariate models would reveal a nonshared environmental correlation ($r_{e}$). The significance of this correlation thus represents a test of Hypothesis 6.

The magnitude of the contribution that common genes or environments make to the phenotypic correlation is estimated as the product of the paths from one measure to the other via the relevant genetic or environmental latent variable (e.g., the common environmental component of the phenotypic correlation between sensitivity and attachment is $c_{sens} \times r_{c} \times c_{att}$). Thus, when any term in this path is zero or near to zero (e.g., $c_{sens}$, $r_{A}$, $r_{C}$, or $r_{E}$), this path necessarily becomes zero or near to zero.

Standard analyses of this kind assume multivariate normality, and, hence, researchers have to use special methods when analyzing categorical data. Following standard practice (see Neale et al., 1999), we conducted analyses involving the binary attachment security variable by estimating tetrachoric and biserial correlation matrices in the LISREL program PRELIS 2 (Jöreskog & Sörbom, 1996) and weighting these by the asymptotic covariance matrix in Mx. We used the asymptotically weighted least squares estimation procedure for these analyses (Neale et al., 1999).

We also conducted some additional analyses to test for possible moderation prior to the essentially mediational analyses described above. To test for background factors (e.g., gender, maternal age) that might moderate the genetic, shared environmental, and nonshared environmental effects on maternal sensitivity, we tested a set of nested structural models following methods developed by Purcell (2002) for continuous dependent variables. This method allows the examination of the impact of both continuous and categorical moderator variables on genetic and environmental effects on maternal sensitivity. The model introduces linear interaction terms that represent the varying extent of genetic and environmental effects on sensitivity with changes in the moderator. In each analysis, chi-square tests were conducted that tested the significance of the change in fit when a

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**Figure 1.** Path diagram of the bivariate genetic model. A, C and E refer to genetic, shared environmental and nonshared environmental latent variables, respectively. Lowercase a, c and e refer to the effects (path coefficients) of these respective latent variables on the measured variables. Genetic and environmental latent variables and path coefficients for sensitivity and attachment are distinguished by the subscripts sens and att, respectively. MZ = monozygotic twins; DZ = dizygotic twins.
moderator term was dropped from a model in which all moderators were initially present. They thus represent single degree of freedom tests of the moderator effect in question.

**Results**

The results of this study are divided into four sections. In the first, we examine associations between observations of maternal sensitivity in MZ and DZ twins from a behavior-genetic perspective and estimate genetic and environmental effects on sensitivity. In this section, we thus test Hypotheses 1–3 outlined in the introduction. In the second section, we present analyses that test for possible gene–environment interaction so that we can rule out (or in) moderation prior to testing the mediational hypotheses that are at the center of this inquiry (see Baron & Kenny, 1986). In the third section, we conduct a basic univariate genetic analysis of attachment security to provide the relevant context and background for the mediational analyses in the final section. Thus, in the fourth section we present analyses aimed at testing the extent to which genetic, shared environmental, and nonshared environmental effects underlie the association between maternal sensitivity and attachment (Hypotheses 4–6).

**Behavior-Genetic Analyses of Maternal Sensitivity**

As a means of examining similarity of maternal sensitivity to both twins as a function of their genetic similarity, we began by computing intraclass correlation coefficients for MZ and DZ twins for descriptive purposes. In each case, we separated the London and Leiden samples for comparison. The intraclass correlation coefficients are shown in Table 1.

As can been seen from Table 1, the intraclass correlation coefficients were high in both MZ and DZ twins and relatively consistent across samples. The 95% confidence intervals (CIs) for the intraclass correlation coefficients overlapped considerably for all four estimates. To further explore basic genetic modeling assumptions, we carried out a multivariate analysis of variance to check for systematic differences in means among the maternal sensitivity ratings, with the two sets of sensitivity ratings (Twin 1 and Twin 2) as dependent variables. The mean sensitivity ratings by zygosity and sample (London and Leiden) are shown in Table 2. There were no significant effects of sample (Wilks’s λ = .97), F(1, 131) = 2.31, p = .11, or zygosity (Wilks’s λ = .972), F(1, 131) = 1.89, p = .16, and no Sample × Zygosity interaction (Wilks’s λ = .997), F(2, 131) = 0.21, p = .81.

We then conducted standard genetic modeling of genetic and environmental components of maternal sensitivity using the program Mx (Neale et al., 1999). The results of the univariate genetic analysis are summarized in Table 3. The ACE model with all three parameters included proved to be an acceptable fit to the data, $\chi^2(3) = 2.54, p = .47$ (root-mean-square error of approximation [RMSEA] = .042). The genetic term was estimated to be very close to zero and negative ($-2.17 \times 10^{-7}$), because the DZ correlation was somewhat higher than the MZ correlation. Genetic and environmental parameters are not permitted to be negative in the ACE model; thus, this term was subsequently constrained to zero. As a result, the CE model inevitably fitted as well as the ACE model, and the significance of dropping the genetic term could not be directly tested. However, the AE model was not a good fit to the data, $\chi^2(4) = 19.37, p < .01$ (AIC = 11.37; RMSEA = .16), indicating that the model including a shared environmental component and a nonshared environmental component (plus measurement error) was the best fitting model. The parameter estimates associated with this reduced model indicated a large shared environmental component ($e^2 = .66$ standardized estimate, 95% CI = .56, .74) and a somewhat smaller nonshared environmental component ($e^2 = .34$ standardized estimate, 95% CI = .26, .45). There was thus little evidence for Hypothesis 2 (genetic effects on sensitivity) but clear evidence in favor of Hypotheses 1 and 3.

**Moderated Effects**

As outlined in the introduction to this section, prior to testing mediating hypotheses, it is important to rule out (or in) possible moderation (Baron & Kenny, 1986). In twin analyses, moderators can be divided into those that are necessarily shared by twins (e.g., maternal age) and those that might differ between twins (e.g., attachment security, birth weight). The shared moderators selected for analysis were maternal age, number of children in the family, and child gender (gender was necessarily shared because all twins in this sample were same sex). Twin-specific moderators were attachment security and birth weight. By selecting attachment security (secure vs. insecure) as a moderator, we thus chose to ask the following question: Are genetic and environmental effects on maternal sensitivity similar or different in secure compared with insecure infants? For example, this could reveal that child effects (genetic effects on sensitivity) are more prominent for insecure infants than for secure ones. We acknowledge that the question

### Table 1

**Maternal Sensitivity: Twin Intraclass Correlations (ICCs) for the Leiden and London Samples (N = 136 pairs)**

<table>
<thead>
<tr>
<th>Sample</th>
<th>ICC</th>
<th>CI</th>
<th>p</th>
<th>ICC</th>
<th>CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leiden</td>
<td>.69</td>
<td>.43, .85</td>
<td>&lt;.001</td>
<td>.66</td>
<td>.47, .79</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>London</td>
<td>.64</td>
<td>.37, .82</td>
<td>&lt;.001</td>
<td>.68</td>
<td>.43, .83</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note. CI = confidence interval.*

<table>
<thead>
<tr>
<th>Twin</th>
<th>M</th>
<th>SD</th>
<th><strong>Confidence interval</strong></th>
<th>M</th>
<th>SD</th>
<th><strong>Confidence interval</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Twin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>6.34</td>
<td>.30</td>
<td>5.76, 6.93</td>
<td>5.59</td>
<td>.31</td>
<td>4.98, 6.23</td>
</tr>
<tr>
<td>2</td>
<td>5.66</td>
<td>.29</td>
<td>5.08, 6.23</td>
<td>5.35</td>
<td>.30</td>
<td>4.75, 5.95</td>
</tr>
<tr>
<td>DZ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>5.94</td>
<td>.29</td>
<td>5.37, 6.51</td>
<td>5.52</td>
<td>.23</td>
<td>5.07, 5.97</td>
</tr>
<tr>
<td>2</td>
<td>5.81</td>
<td>.28</td>
<td>5.25, 6.37</td>
<td>5.61</td>
<td>.22</td>
<td>5.17, 6.06</td>
</tr>
</tbody>
</table>

*Note. MZ = monozygotic; DZ = dizygotic.*
could also be asked the other way around, but genetic moderation analyses with continuous moderators and binary dependent variables are not currently available.

For descriptive purposes, the correlations between the moderator variables and the difference between twins in maternal sensitivity are shown in Table 4. As can be seen in Table 4, no significant correlations emerged between the moderator variables and twin differences in maternal sensitivity. The results of formal model fitting for moderator effects are shown in Table 5. The table shows the magnitude of the moderator term for A, C, and E (i.e., the linear amount by which the effects of A, C, and E changed as a function of the moderator) and the maximum likelihood chi-square test statistics for each moderator (a chi-square of 3.84 or greater was significant at $p < .05$). As can be seen in Table 5, no significant moderating effects were detected.

**Genetic and Environmental Effects on Attachment**

Although analyses of the genetic and environmental effects on attachment security have been presented for this sample in an earlier article (Bokhorst et al., 2003), this analysis is repeated here because (a) the current article reports on a slightly smaller subsample than the Bokhorst et al. (2003) article and (b) the parameter estimates for this model are needed for a full appreciation of the meaning of the bivariate analyses that follow. The correspondences between twins in attachment classifications are presented for MZ and DZ twins in Table 6. We estimated genetic and environmental effects on attachment security (secure vs. insecure) using standard behavior-genetic models with the program MX, estimated directly from the MZ and DZ contingency tables (Neale et al., 1999). As in the previous analysis of sensitivity, when the ACE model was estimated with the full sample, the genetic term was extremely small and negative ($-5.4 \times 10^{-9}$) and hence had to be constrained to zero. However, the CE model was a good fit to the data, $\chi^2(1) = 0.015, p > .90$ (AIC = $-5.99$; RMSEA = .023), with an estimated shared environmental effect of .50 (95% CI $=.00, .72$), accounting for 25% of the variance in attachment security. The nonshared environmental effect was estimated to be .87 (95% CI $=.66, 1.00$) and hence accounted for 75% of the variance. Deletion of the shared environmental effect led to reduction in model fit that was, for all intents and purposes, significant, $\chi^2(1) = 3.58, p = .06$.

In an earlier article (Bokhorst et al., 2003), we found minimal shared environmental effects on disorganized attachment and strong shared environmental effects in the organized patterns (secure vs. insecure). Consistent with this, the degree of correspondence between the three-way attachment classifications for pairs in which one or both cases was disorganized was substantially lower than that for pairs in which neither twin was disorganized (see Table 7). For the disorganized pairs (in which one or both cases was classified D), there was no significant association between the three-way classification of one twin and the other, $\chi^2(4) = 4.68, p = .32$. For the organized cases, the degree of association was highly significant, $\chi^2(4) = 27.9, p < .01$. To test the significance of the difference in association between these two groups of cases, we carried out a hierarchical log-linear analysis, with Twin 1 and Twin 2 three-way attachment classification as two factors and pair attachment organization (disorganized pairs vs. organized pairs) as a third. The three-way interaction, representing the difference in association between the two groups, was significant, likelihood ratio $\chi^2(4) = 15.14, p < .01$. In addition, the correlation between twins in terms of attachment security (secure vs. insecure) was also significantly different between organized pairs and those in which one or both twins was disorganized ($p < .01$). A second univariate genetic analysis was thus performed with the disorganized pairs removed (this analysis corresponds to the ACE analysis performed on the organized cases in Bokhorst et al., 2003). This second analysis was also important in this context because in later analyses we aimed to test the association between attachment and maternal sensitivity both with and without the disorganized cases (in the light of evidence that disorganization is very weakly associated with sensitivity; see Van IJzendoorn et al., 1999).

The full ACE model with disorganized cases removed ($n = 94$) proved to have an acceptable fit to the data, $\chi^2(2) = 3.30, p = .19$

**Table 3**

<table>
<thead>
<tr>
<th>Sensitivity</th>
<th>$a^2$</th>
<th>$c^2$</th>
<th>$e^2$</th>
<th>$\chi^2$</th>
<th>$df$</th>
<th>$p$</th>
<th>AIC</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE</td>
<td>$&lt;-.001$</td>
<td>.66</td>
<td>.34</td>
<td>2.54</td>
<td>3</td>
<td>.47</td>
<td>$-3.45$</td>
<td>.042</td>
</tr>
<tr>
<td>AE</td>
<td>.83</td>
<td>.17</td>
<td>19.37</td>
<td>4</td>
<td>$&lt;.01$</td>
<td>11.37</td>
<td>.160</td>
<td></td>
</tr>
<tr>
<td>CE</td>
<td>.66</td>
<td>.34</td>
<td>2.54</td>
<td>4</td>
<td>.64</td>
<td>$-5.46$</td>
<td>.023</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>1.00</td>
<td>69.06</td>
<td>5</td>
<td>$&lt;.01$</td>
<td>61.06</td>
<td>.464</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. $n = 56$ pairs monozygotic, 80 pairs dizygotic. CE is the preferred model. A, C, and E indicate which latent variables (genetic [A], shared environmental [C], and nonshared environmental [E]) are included in the four models. Lowercase a, c, and e refer to the standardized effects of these latent variables on sensitivity. AIC = Akaike information criterion; RMSEA = root-mean-square error of approximation.*

**Table 4**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Twin differences in sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>$-0.076$</td>
</tr>
<tr>
<td>Age</td>
<td>$.066$</td>
</tr>
<tr>
<td>Birth order</td>
<td>$-0.007$</td>
</tr>
<tr>
<td>Birth weight Twin 1</td>
<td>$-0.137$</td>
</tr>
<tr>
<td>Birth weight Twin 2</td>
<td>$-0.158$</td>
</tr>
<tr>
<td>Attachment security Twin 1</td>
<td>$-0.037$</td>
</tr>
<tr>
<td>Attachment security Twin 2</td>
<td>$-0.087$</td>
</tr>
</tbody>
</table>

*Birth weight data were available only for 127 out of 136 twin pairs from the current sample.*
However, removal of the genetic term did not lead to a significant reduction in model fit \((\Delta \chi^2 < .10, p = .99)\), whereas removal of the shared environmental term led to a significant reduction in model fit \((\Delta \chi^2 = 4.11, p = .04)\). The model containing only nonshared variance and measurement error \((E)\) was not a good fit to the data, \(\chi^2(4) = 13.68, p < .01\) (AIC = 5.69; RMSEA = .13). The model containing shared environment and nonshared environment plus measurement error was thus the best fitting model (as in our earlier report). In this best fitting model, 50% of the variance was attributable to shared environmental effects (95% CI = 2.5%, 72.9%), and 50% was attributable to nonshared environmental effects (95% CI = 26.1%, 79.1%).

### Pathways of Association Between Sensitivity and Attachment

Having found (a) shared and nonshared environmental effects on both sensitivity and attachment security and (b) no evidence that demographic factors or attachment security amplified or attenuated genetic and environmental effects on maternal sensitivity (i.e., no moderation), we tested the mediational hypotheses concerning the association between attachment security and maternal sensitivity. We did this by testing the significance of correlations between shared environmental and nonshared environmental effects on maternal sensitivity, on the one hand, and attachment security, on the other (Hypotheses 4 and 6). We were not able to test such mediational hypotheses in relation to genetic effects, as the genetic contribution to maternal sensitivity was constrained to zero in earlier analyses (Hypothesis 5). Consequently, we pooled the MZ and DZ groups and conducted bivariate analyses on this single sample.

The models were specified as shown in Figure 1, but with the genetic parameters removed. Thus, the models estimated shared and nonshared environmental components of each measure (sensitivity and attachment) and the correlations \((r_c, r_e)\) between these sources of influence. The significance tests associated with these two correlations represent tests of Hypotheses 4 and 6, namely that shared environmental and nonshared environmental processes (respectively) underlie the link between sensitivity and attachment. As noted earlier, we chose to test these hypotheses with the full sample first and then with the disorganized cases removed. Our reasoning was that the correlation between sensitivity and attachment was likely to be higher for the organized

### Table 5

**Moderator Effects on Genetic and Environmental Influences on Maternal Sensitivity**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Genetic Effect × Moderator</th>
<th>B</th>
<th>(\chi^2)</th>
<th>Shared Environment × Moderator</th>
<th>B</th>
<th>(\chi^2)</th>
<th>Nonshared Environment × Moderator</th>
<th>B</th>
<th>(\chi^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age</td>
<td>(-4.42 \times 10^{-6})</td>
<td>&lt;.01</td>
<td>.024</td>
<td>.80</td>
<td>-.001</td>
<td>&lt;.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>(1.95 \times 10^{-6})</td>
<td>&lt;.01</td>
<td>.178</td>
<td>.77</td>
<td>.124</td>
<td>.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth order</td>
<td>-.159</td>
<td>.20</td>
<td>.186</td>
<td>1.00</td>
<td>.088</td>
<td>.50</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attachment</td>
<td>-.67</td>
<td>.80</td>
<td>.22</td>
<td>1.49</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth weight</td>
<td>(6.6 \times 10^{-4})</td>
<td>&lt;.01</td>
<td>5.46 \times 10^{-5}</td>
<td>&lt;.01</td>
<td>1.65 \times 10^{-4}</td>
<td>&lt;.01</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. The chi-square values refer to the change in fit when the respective moderator term is dropped from a model with all other moderator terms present. All chi-square tests are single df tests; df = 1. Birth weight \(N = 127\); all other \(Ns = 136\).

### Table 6

**Distribution of Attachment Classifications by Zygosity**

<table>
<thead>
<tr>
<th>Twin 2 attachment classification</th>
<th>Twin 1 attachment classification</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>A</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>B</td>
<td>B</td>
<td>5</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>C</td>
<td>C</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>D</td>
<td>D</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>8</td>
<td>9</td>
<td>2</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>Monzygotic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>A</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>B</td>
<td>B</td>
<td>4</td>
<td>5</td>
<td>12</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>C</td>
<td>1</td>
<td>8</td>
<td>0</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>D</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>14</td>
<td>15</td>
<td>18</td>
<td>80</td>
<td></td>
</tr>
</tbody>
</table>

Note. A = nonsecure–avoidant; B = secure; C = nonsecure–resistant; D = disorganized.

### Table 7

**Twin Correspondence for Three-Way Attachment Classifications for Pairs Classified as Disorganized and Organized**

<table>
<thead>
<tr>
<th>Twin 2 three-way attachment classification</th>
<th>Twin 1 three-way attachment classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorganized</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>2 (−0.1)</td>
</tr>
<tr>
<td>B</td>
<td>4 (0.0)</td>
</tr>
<tr>
<td>C</td>
<td>2 (0.1)</td>
</tr>
<tr>
<td>Organized</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>9 (3.3)</td>
</tr>
<tr>
<td>B</td>
<td>9 (−1.0)</td>
</tr>
<tr>
<td>C</td>
<td>2 (−1.2)</td>
</tr>
</tbody>
</table>

Note. Standardized residuals are in parentheses. A = nonsecure–avoidant; B = secure; C = nonsecure–resistant.
Table 8
Twin Correlation Matrices for Sensitivity and Attachment Security

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full sample&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Twin 1 sensitivity</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Twin 1 security</td>
<td>.14</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Twin 2 sensitivity</td>
<td>.65*</td>
<td>.31**</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>4. Twin 2 security</td>
<td>.13</td>
<td>.24*</td>
<td>.15</td>
<td>—</td>
</tr>
</tbody>
</table>

Organized attachment patterns only<sup>b</sup>

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Twin 1 sensitivity</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Twin 1 security</td>
<td>.15</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Twin 2 sensitivity</td>
<td>.63**</td>
<td>.43**</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>4. Twin 2 security</td>
<td>.25*</td>
<td>.56**</td>
<td>.26**</td>
<td>—</td>
</tr>
</tbody>
</table>

<sup>a</sup> n = 136.  <sup>b</sup> no Ds, n = 94.
<sup>*</sup> p ≤ .05.  <sup>**</sup> p ≤ .01.

attachment classifications (secure, avoidant, and resistant), given the meta-analytic findings of Van IJzendoorn et al. (1999).

The correlations between sensitivity and attachment that formed the basis of the structural equation model are shown in Table 8. These correlations were generated in PRELIS 2 (Jöreskog & Sörbom, 1996) and include tetrachoric correlations between twins’ attachment securities, biserial correlations between sensitivity and attachment security, and Pearson correlations between twins’ sensitivity scores, on account of the binary nature of the attachment security variables. It is notable that the cross-twin correlations between sensitivity and attachment varied somewhat between the Twin 1 and Twin 2 groups. For example, in the full sample the Twin 1 attachment–Twin 1 sensitivity correlation was .31, whereas the Twin 2 attachment–Twin 1 sensitivity correlation was .13. Similarly, in the organized cases, the Twin 1 security–Twin 2 sensitivity correlation was .43, whereas the Twin 2 security–Twin 1 sensitivity correlation was .25. Because the assignment of twins was carried out entirely at random, this difference can only be a chance function of sampling error. This difference was the main source of error in the model fit statistics shown below.

The results of the model testing are shown in Table 9. The full model (including shared and nonshared environmental correlations) was found to be a reasonable fit to the data, $\chi^2(3) = 6.21$, $p = .10$ (AIC = .21; RMSEA = .08). Deletion of the shared environmental correlation, $r_C$, between sensitivity and attachment led to a significant reduction in model fit, $\Delta \chi^2(1) = 15.27, p < .01$. Deletion of the nonshared correlation, $r_N$, from the full model led to a reduction in model fit that was essentially significant, $\chi^2(1) = 3.56, p = .06$. The full model was thus selected as the best fitting model. The parameter estimates for this model are summarized in Figure 2.

As can be seen in Figure 2, shared environmental effects on sensitivity correlated with shared environmental effects on attachment ($r_C = .58$, 95% CI = .27, 1.00). This thus confirmed our principal hypothesis that shared environmental effects would underlie the association between maternal sensitivity and attachment security and hence that sensitivity would account for a significant portion of the similarity between twins in attachment security. Using an alternative representation of the model, described by Loehlin (1996), it is possible to calculate the proportion of the correlation between twins’ attachment securities attributable to (or at least associated with) similarities in the maternal sensitivity they receive. These calculations indicated that the shared environmental component of maternal sensitivity could account for approximately 33% of the correlation between twins in attachment security (which was .24, from Table 8). This same result implies that shared components of sensitivity accounted for 33% of the shared variance in attachment security. These results are consistent with Hypothesis 4. However, in addition to this positive association, there was also a negative correlation between the nonshared components of sensitivity and attachment ($r_N = -.14$, 95% CI = −1.00, .01), consistent with Hypothesis 6 (dyad-specific links between sensitivity and attachment). The negative nonshared pathway does not contribute to the phenotypic correlation between twins in attachment security but suggests that nonshared environmental effects created an inverse relationship between sensitivity and attachment (greater sensitivity to one twin that was not expressed toward the other was associated with lower likelihood of security). This negative nonshared effect was small in size, accounting for only 2% of the nonshared variance in attachment.

The phenotypic correlation between attachment and sensitivity consisted of two parts—the positive association via the shared sensitivity latent variable ($0.78 \times 0.58 \times 0.47 = .213$) and a negative part via the nonshared sensitivity latent variable ($0.63 \times -1.4 \times .89 = -.078$). These two paths sum together to .14—the phenotypic correlation between sensitivity and attachment found in the full sample of this study. What this implies is that nonshared environmental effects had the effect of suppressing the magnitude of the phenotypic association resulting from the shared environmental pathway between sensitivity and attachment. The non-

<table>
<thead>
<tr>
<th>Model</th>
<th>Full sample&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Organized&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\chi^2$</td>
<td>df</td>
</tr>
<tr>
<td>Full model</td>
<td>6.21</td>
<td>3</td>
</tr>
<tr>
<td>Shared environment cross-measure path removed</td>
<td>21.48</td>
<td>4</td>
</tr>
<tr>
<td>Nonshared cross-measure path removed</td>
<td>9.76</td>
<td>4</td>
</tr>
</tbody>
</table>

<sup>a</sup> n = 136.  <sup>b</sup> no Ds, n = 94.

Note.  AIC = Akaike information criterion.
Discussion

In this first twin study on the association between maternal sensitivity and infant attachment security, the role of the environment for individual differences in attachment security is supported. First, we found no evidence for a significant contribution of genetic factors (residing in the infants) to differences in maternal sensitivity. Instead, maternal sensitivity, at least as measured in this study, was found to be a strongly shared environmental variable. These findings support and extend the absence of evidence for genetic effects on individual differences in infant attachment security reported in a previous article (Bokhorst et al., 2003). Second and more important, we were also able to document the potential of the shared component of maternal sensitivity to explain some of the similarity of attachment security between twins in the same family. In addition, given the striking degree of variance in attachment that was attributable to nonshared effects, we also explored possible links between sensitivity and twin differences in attachment. We found a weak nonshared correlation between sensitivity and attachment that was negative in sign (contrary to our expectations), which suggests that sensitivity might explain some twin differences in attachment, but not in ways we had anticipated.

Contemporary thinking in attachment research views the primary causes of attachment security and insecurity as being located in dimensions of parental caregiving behavior, and research has focused on maternal sensitivity as the key component of maternal behavior in the development of attachment, particularly in relation to the organized attachment categories. Studies using the Adult Attachment Interview have suggested that these patterns of maternal behavior may be shared by children in the same family because they are guided by a parent’s internal working model of attachment.

1 It should be noted that the negative nonshared pathway could also be modeled as a cross-twin effect, with sensitivity toward the co-twin (that is not shown to the target twin) impacting positively on the target twin’s attachment security (i.e., the parameter changes sign).
(Van IJzendoorn, 1995). As might be anticipated from attachment theory, maternal sensitivity, as observed by two independent raters, was found to be highly correlated between twins, regardless of their zyosity. Ainsworth et al.’s (1978) characterization of sensitivity emphasizes the need for parents to attune their caregiving to the individual contours of the child’s behavior and interactional tempo, which perhaps counterintuitively helps to explain why it is primarily a shared environmental measure. This molding of the parent’s behavior to the unique particularities of the child would be expected to diminish the impact of the child’s behavior on sensitivity because a parent would receive similar ratings as long as his or her caregiving were responsive to the specific needs and characteristics of the child in question. The intraclass correlation between twins was found to be approximately .65, a figure that is somewhat higher than that found in siblings measured several years apart (correlations ranged from .30 to .61; see Van IJzendoorn et al., 2000; Ward, Vaughn, & Robb, 1988). The fact that the observations of sensitivity toward each twin were conducted together during the same home visit could have introduced correlated errors of measurement, which would lead to an inflation of the estimate of this association (although these ratings were conducted by independent raters). However, the fact that the twin correlation was only modestly greater than similar sibling assessments measured several years apart may suggest that this inflation is relatively small. Nevertheless, differences in the way sensitivity was measured in our twin study compared with existing sibling studies could affect the comparability of the estimates in ways that are difficult to ascertain. As such, this result should be treated cautiously. As noted previously, univariate genetic designs are highly sensitive to method of measurement, and we cannot exaggerate the possibility of correlated errors when considering the genetic and environmental estimates for sensitivity in the current study. It may well be that had sensitivity been measured separately for each twin (i.e., only one present at a time), ACE estimates would have been discrepant from those presented. Further studies of maternal sensitivity in genetically informative designs would be highly beneficial in this regard, particularly if multiple methods of measurement could be used. Nevertheless, in the context of available evidence, the current findings do broadly converge on the view that maternal sensitivity is expressed relatively consistently between twins and siblings in ways that may not be especially contingent on their genetic similarity.

It should also be noted that the current design only allowed us to test whether children’s genes have an effect on sensitivity, not parents’ genes. To test for the effects of parental genetic factors, a sample of adult twin parents would be required. Also, little is known about the possibly changing contributions of genetics and environment (shared or nonshared) to parental sensitivity (or, indeed, attachment) over the course of development. Furthermore, the low-risk nature of the current sample may not represent the extent of genetic and environmental contribution to variation in sensitivity or attachment in higher risk groups.

The current study is able to partially confirm a second prediction grounded in attachment theory—that the association between maternal sensitivity and infant attachment would be mediated by shared environmental processes. In other words, inconsistencies in the way sensitivity was experienced by twins could explain some of the similarity in their attachment security. This finding is consonant with formulations of attachment theory that emphasize parental working models of attachment and with the extent evidence concerning the relationship between maternal sensitivity and attachment (De Wolff & Van IJzendoorn, 1997; Van IJzendoorn, 1995). This aspect of the findings converges with similar results presented by Van IJzendoorn et al. (2000) with non-twin siblings; these authors also found that similarities in sibling attachments could be partially explained by similarities in parental sensitivity. The current findings, considered against a background of research showing the predictive power of the Adult Attachment Interview and the effectiveness of sensitivity-based interventions for later attachment security (Bakermans-Kranenburg et al., 2003), provide further evidence that the emphasis in attachment theory on the shared family environment is, at least to a degree, well placed.

Despite these positive results, it is important not to lose sight of the fact that a large proportion of the variance in attachment security (with and without twin pairs in which one or both twins was disorganized) was estimated to be nonshared (see Bokhorst et al., 2003). This striking finding, confirmed in other twin and sibling samples, poses major challenges to attachment theorists. There is clearly an urgent need to understand how differences in attachment security between children in the same family arise. In that regard, we reasoned that differences in maternal sensitivity would be a compelling place to start. We had hypothesized that sensitivity that was specific to one child would relate positively to that child’s attachment security. The effect of this would have been to increase the within-twin correlation between sensitivity and attachment relative to the cross-correlation between one child’s experience of sensitivity and the other child’s attachment security. What we observed was, in fact, the opposite: The cross-twin correlation between sensitivity and attachment was higher than the direct within-twin correlation. The bivariate model characterized this as a negative effect; that is, the model inferred that a nonshared process had lowered the within-twin correlation (relative to the cross-twin correlation). This would happen if sensitivity expressed to one twin but not to the other actually led to insecurity in the former twin. One should remember, of course, that this nonshared process weighs against a stronger shared one, by which generally higher sensitivity (expressed to both twins) increases both twins’ security.

One way of looking at this nonshared effect is to imagine that when attachment outcomes are discordant, mothers try to compensate by increasing their sensitivity to the insecure child. This would tend to lower the correlation between sensitivity and attachment within a dyad. It could also be that insecure attachment more generally evokes greater sensitivity in parents when attachments in the family are discordant. Another way of thinking about the effect is to note that the pattern of correlations could just as easily be generated by a process that raises the cross-twin correlation (rather than lowering the within-twin one). This could suggest that the attachment security of one twin depends on the relationship the parent has with the other twin. Mechanisms that have been described in the literature to account for sibling dependencies such as this include sibling comparison or barricade effects (Reiss et al., 1996). The pattern of correlations could suggest that insensitive parenting expressed to one twin (but not expressed to the other) leads to decreases in the other twin’s security. How such an effect might occur is unclear. One possibility is that observing insensitivity toward one’s sibling reduces one’s own sense of felt security, regardless of its effects on the sibling to whom the parenting is
directed. Although one might argue that such comparative effects require greater cognitive sophistication than has been acquired by 1-year-old infants, some evidence suggests otherwise. Hart, Field, Del Valle, and Letourneau (1998), for example, found that infants were more likely to protest, look at their mother, and seek their mother’s proximity when she attended to a toy baby than when she was attending to a book, which suggests some degree of monitoring by the infant of the parent’s attention toward others.

However one conceptualizes this finding, if it is reliable it raises interesting questions and might suggest that the same parenting behaviors can have opposite effects depending on whether they are shared or nonshared within a sibling pair. The implication of this might be that behaviors that have environmental mediating functions may be heterogeneous rather than homogenous in terms of their associations with outcome. The shared environmental pathway in the current study appeared to behave in a manner consistent with traditional attachment theory, whereas the second independent, nonshared process might be understood to intersect with more complex aspects of the family system. In other words, superimposed on a background of predominantly shared environmental connections between siblings’ attachments and maternal behavior, the nonshared environment may create rather complex interrelationships among different members of the family. Such complexity seems to point to the need for a family systems approach, in which there is direct consideration of the ways relationships affect relationships (Auhagen & Hinde, 1997).

It should be noted that the overall magnitude of the association between sensitivity and attachment in this study was low, averaging at approximately .15 (the cross-twin correlation was somewhat larger, at .22) in the full sample. This increased noticeably to around .24, on average, when only the organized patterns of attachment were included. Both these values are within the range observed in Van IJzendoorn’s (1995) meta-analysis, which found a mean effect size of $r = .24$. With the negative path partialled out, the phenotypic correlation between attachment and sensitivity was estimated to be .30 when disorganized pairs were removed. It should also be noted that the negative nonshared pathway detected in this study might be a factor that is most evident in twin samples, and this, in turn, might account for the somewhat lower correlation between attachment and sensitivity found in this study. Furthermore, there was a lack of consistency in the sample in the cross-twin correlation, which could be taken to suggest that the non-shared correlation was a sampling error.

Overall, with correlations between attachment and sensitivity of the magnitude found in this study, it seems clear, at least within the limitations noted above, that much of the variance in attachment remains to be explained, as originally discussed by Van IJzendoorn (1995). It is notable that the association between twins’ attachment classifications (secure vs. insecure) was relatively substantial, but again only when disorganized infant pairs were excluded. This finding is consistent with previous analyses of this data set, with 50% of the variance attributable to shared environment for organized attachment patterns, whereas all the variance in disorganization (disorganized vs. organized) was estimated as nonshared (Bokhorst et al., 2003). Within the organized groups, a third of the shared environmental variance in attachment security was explainable in terms of shared features of maternal sensitivity, leaving two thirds still to be accounted for. This gap in the accounted-for shared variance in attachment security is reminiscent of the so-called transmission gap, first described by Van IJzendoorn (1995), in which maternal sensitivity is only able to account for a third of the correlation between parental state of mind with respect to attachment and infant attachment security. Whether parental Adult Attachment Interview assessments would account for the majority of the shared environmental component of attachment security remains to be seen.

In addition, it is important to consider that a single assessment of parental state of mind with respect to attachment is unable, on its own, to account for differences between twins or siblings in attachment security. The fact that between 50% and 75% of the variance in security (excluding and including disorganized cases, respectively) was of the nonshared kind indicates that alternative means of bridging that gap are required. Clearly, some proportion of this nonshared variance is measurement error, so studies that use multiple assessments of attachment, ideally at multiple points in time, might well drive this figure down. Nevertheless, it seems unlikely that only measurement error is at play. In this study, a small but significant proportion of the nonshared variance in attachment was found to be associated with nonshared variance in maternal sensitivity (and hence does not appear to be measurement error), but this only accounted for between 2% and 6% of the nonshared variance. As noted in the introduction, the large proportion of variance in attachment that is nonshared continues to pose significant challenges to the way that attachment researchers conceptualize and investigate this important area of socioemotional development. Traditionally, attachment theory has failed to explicitly consider how children may have different attachment relationships to the same parent, and hence the current data, along with others presenting attachment classifications for twins and siblings, present a challenge to attachment researchers. Further investigations into the sources and mechanisms of nonshared variance in attachment are no doubt an important avenue for future research. All manner of family factors and processes might be relevant to this research endeavor, such as differences in infant characteristics (biobehavioral regulation, illness, sleep, temperament), parental behavior (sensitivity; frightened or frightening behavior; attributions of the infant; distal factors, e.g., psychological health; marital processes), and their interplay. It may be that more qualitatively oriented studies would help focus research onto the appropriate domains most effectively at this early stage.

When considering the results of this study, it is important to recognize some of the limitations of twin methodology. A variety of questions have been raised about the extent to which various assumptions of the twin method hold, such as the equal environments assumption. Although some studies have found support for this assumption (see Plomin et al, 2001), we currently lack direct independent evidence regarding the validity of the twin method for assessing attachment and its correlates. In this respect, one should remember that resistant infants were overrepresented in our sample compared with a normative meta-analytic sample (Van IJzendoorn et al., 1999), and twin-specific child rearing factors may be responsible for this divergence—for example, the greater burden that twins place on effective and consistent parenting. It is also possible that the Strange Situation is less valid (and possibly more stressful) for twins than for nontwins, which may also limit the study’s generalizability.

These limitations of the twin method echo recent discussions in this journal by Partridge (2005), Greenberg (2005), and McGue,
Elkins, Walden, and Iacono (2005). Partridge (2005) and Greenberg (2005) rejected the idea that differences in the extent of similarity between genetically related individuals can tell researchers anything about genetics (and, by extension, the environment). Although we agree that the interpretation of twin correlations is not straightforward, the sheer reliability of these differences in correlation, tested in a variety of measurement conditions, with a variety of different genetic relationships (twins, siblings, stepsiblings, cousins, children reared apart and together) attests to the basic validity, within reasonable limits, of the method. However, it is certainly true that behavior-genetic data have been used and interpreted too simplistically at times, particularly with respect to the potential complexity of the mechanisms that could give rise to differences in twin correlations for developmental variables (e.g., see arguments provided by Rutter, 2003). However, we believe that genetic methodologies have an important place in scientific research, when used as natural experiments to test refined developmental theories, with sensitive, high-quality measures, and when interpreted with appropriate regard for the broader evidence base and the limitations of the methodology. In that context, the convergent findings of twin and sibling studies of attachment have been very illuminating, largely because attachment theory makes such clear predictions about the environmental processes that give rise to individual differences in attachment and because of the availability of well-validated and intensive observational instruments, not only of the phenotype but also of the environment.

In summary, the current twin study of maternal sensitivity and attachment provides some support for predictions of attachment theory. Maternal sensitivity was found to show little evidence of genetic effects from the child, and shared environmental effects on sensitivity were found to correlate significantly with shared environmental effects on attachment. Thus, the similarity between twins in attachment security was partially explained by consistencies in their experience of maternal sensitivity. The study provides partial support for the coherence of parent–child relationships across siblings in a family. However, the study had limited success in identifying associations between sensitivity and the substantial nonshared aspects of attachment. Research into the specific causes and effects of nonshared environmental influences on development is still in its infancy, and further work is clearly needed to identify factors that might account for the sizable discrepancies in children’s attachment relationships in the family.

References
Greenberg, G. (2005). The limitations of behavior–genetic analyses: Com-


