Child Development, November/December 2006, Volume 77, Number 6, Pages 1656–1667

The Limits of Genetic Influence: A Behavior-Genetic Analysis of Infant–Caregiver Relationship Quality and Temperament

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This report presents data on 9-month-old twin pairs ($n_{\mathrm{MZ}} = 172$; $n_{\mathrm{DZ}} = 333$) from the Early Childhood Longitudinal Study, demonstrating that the role of genetic variation among infants is trivial and the shared and nonshared environment is substantial in accounting for the observed quality of infant–caregiver relationships. In contrast, maternal reports of temperament were best accounted for by genetic variation and nonshared environmental influences. Interestingly, however, shared environmental effects were documented for *observations* of temperament. Consistent with a growing database, the current study calls into question the ubiquity of heritability effects in all domains of psychological inquiry. It also bolsters consensus in the field of developmental psychology that shared environmental effects are not as elusive as had once been thought.

Critics of socialization research (e.g., Harris, 1995; Rowe, 1994; Scarr, 1992) have argued that the ubiquitous correlations that have been documented between parenting behaviors and child outcomes may not be due to parenting per se, but instead to the effects of genes that are shared within families. There are at least two foundations for this conclusion. First, for a wide variety of outcomes, identical twins are more similar to one another than are fraternal twins (Bouchard, 2004), raising the possibility that genetic variation plays a key role in individual differences. Second, the results of biometric modeling, in which the relative effects of genetic, shared environmental, and nonshared environmental variance are estimated, have suggested that the effects of shared environments are often relatively small (Plomin & Daniels, 1987), leading some writers to speculate that parental influences are minimal and have essentially no long-term effects on development (Harris, 1995).

Developmental scholars have attempted to address the criticisms raised by Rowe, Harris, and others in several ways. Some of these rebuttals have focused on defending existing theoretical perspectives on parenting and highlighting the limitations of the ways in which behavior-genetic data have been interpreted (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Vandell, 2000). For example, Rutter and colleagues (Rutter 2002; Rutter, Pickles, Murray, & Eaves, 2001) have emphasized that nonshared environmental effects in behavior-genetic research need not reference extrafamilial experiences, as has been suggested by Harris (1995). It could be the case that nonshared environmental effects reflect shared experiences that have different consequences for individuals in the same family (but see Turkheimer & Waldron, 2000).

More important perhaps, developmental scholars have noted that the effects of shared environments are not as trivial as was once thought. For example, a growing body of genetically informed developmental research has documented consistent behavior-genetic effects of the shared environment in at least two important domains: parenting (Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998; Neiderhiser et al., 2004; O'Connor, Hetherington, Reiss, & Plomin, 1995) and delinquency (for a selective review, see Leve et al., 1998), as well as their covariation over time (Burt, McGue, Krueger, & Iacono, 2005; Spotts, Neiderhiser, Hetherington, & Reiss, 2001). As anticipated by an early review by Hoffman (1991), shared environmental effects have been especially clear in *observational* studies of parenting and delinquency (e.g., Spotts et al., 2001). Such genetically informed research is important not only because it tempers the long-held assertion that most environmental experiences make individuals in the same family different (i.e., are nonshared within families), but also because it reflects an explicit acknowledgement that the vast majority of data on parental socialization in developmental psychology, which are almost exclusively drawn from between-family research designs,

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confound shared experiences and shared genes within families.

Although a clear consensus has been built in the field that shared environmental effects are readily documented in domains central to developmental theory, it is still an open question as to whether, as some commentators have claimed, ''everything is heritable'' (Turkheimer, 2000, p. 160). Recently, scholars within both behavior-genetic (Bouchard, 2004; Turkheimer, 2000) and developmental circles (Rutter, 2002) have highlighted the fact that a wide variety of psychological phenotypes are heritable, leading one prominent developmental scientist to note that ''any dispassionate reading of the literature leads to the inescapable conclusion that genetic factors play a substantial role in the origins . . . of all psychological traits'' (Rutter, 2002, p. 2). Although some scholars have used the term ''trait'' to reference a subset of psychological phenotypes that are heritable (i.e., personality, mental ability, psychological interests, psychiatric illnesses, and social attitudes; Bouchard, 2004), Rutter (2002) clearly had a broader set of outcomes in mind in his presidential address to the Society for Research in Child Development, reviewing compelling behavior-genetic evidence for the heritability of attitudes, social behavior, divorce, religiosity, as well as parenting styles. From these provocative reviews emerges a critical question: Are there any important, developmentally significant exceptions to Turkheimer's (2000) ''first law of behavior genetics'' which states that ''all human behavioral traits are heritable'' (p. 160)?

To be sure, if heritability effects are truly ubiquitous in psychology, there would be major consequences for classic theoretical perspectives on parental socialization. Some attachment theorists, for example, have suggested that the quality of infant–caregiver relationships is largely the product of shared as well as nonshared experiences with primary caregivers (O'Connor, Croft, & Steele, 2000; Weinfield, Sroufe, Egeland, & Carlson, 1999). This rare ''point prediction'' (i.e., $h^2 = 0$) is notable because so few remaining developmental theorists so boldy (or perhaps naively) reject genetic influence. Importantly, there is reason to expect that the quality of infant–caregiver relationships may in fact be one of the rare developmental phenotypes for which small or nonexistent heritability estimates might obtain (see also Waller & Shaver, 1994). Such results would be consistent with relatively small-sample twin data on attachment security in the early life course (Bakermans-Kranenburg, van IJzendoorn, Bokhorst, & Schuengel, 2004; Bokhorst et al., 2003; O'Connor & Croft, 2001; Ricciuti, 1993). In each of these twin studies the estimated additive genetic effects on security were very modest and often close to zero. Moreover, there was evidence in each of these studies of substantial shared and nonshared environmental influences on the development of attachment security (but see Finkel, Wille, & Matheny, 1998). A recent study of Canadian infant twins similarly revealed that adults' *perceptions* of their parenting of their 5-month-olds in general did not differ by zygosity (i.e., was not heritable). Instead, shared environmental effects were strongly implicated in explaining variation in perceived quality of parenting (Boivin et al., 2005).

Given the relatively small-scale nature of prior observational studies focused on the heritability of infant–caregiver relationships, in this paper we use one of the largest datasets available to determine whether the observed variation in infant–caregiver relationship quality is in fact attributable in large part to shared and nonshared environmental factors and to determine whether genetic variation among infants is largely irrelevant. Using the same sample we also examine whether, in contrast, individual differences in commonly studied dispositional factors, such as infant temperament, have sizable heritability coefficients and negligible shared environmental effects. However, we will introduce an important qualification to this issue by examining whether parental reports of temperament—the kind of reports typically used in the literature—lead to negligible estimates of the effects of shared environment, whereas observer reports of temperament lead to estimates of the effects of the shared environment that are quite considerable. Before presenting these analyses, we provide an overview of the biometric modeling used in this report as well as a description of the unique sample under investigation.

The Additive Behavioral Genetic Model Applied to Developmental Phenomena

Behavior-genetic research is designed to partition the variation in an outcome into several distinct effects: (a) additive genetic influences, (b) shared environmental effects, and (c) nonshared environmental influences. Because shared environmental factors and shared genes are difficult to unconfound, behavior-genetic researchers often take advantage of twin designs in which genetic similarity is known and varies across twin groups. In univariate behavior-genetic analyses of twin data, the heritability (h^2) of an outcome is estimated by examining the degree to which identical (monozygotic or MZ) and fraternal (dizygotic or DZ) pairs differ in terms of similarity on a given variable. As DZ twins share on average 50% and MZ twins 100% of their genetic variation, evidence that MZ twin pairs are more alike on average than DZ twin pairs suggests a role for genetic variation in the development of a phenotype. Controlling for genetic covariation, twin designs also allow for the estimate of shared and nonshared *environmental* effects. By definition, the shared environmental effect provides an aggregate estimate of the degree to which individuals living in the same family are similar to one another on a particular outcome. In contrast, nonshared environmental effects reference intra- and extrafamilial (nongenetic) processes that make siblings *different*. The nonshared environmental effect also includes measurement error.

Genetically informed research designs, such as the one described above, have been in use for some time in developmental psychology. Such approaches, however, are hardly widespread in their implementation in research on socialization. To be sure, two major longitudinal research projects—the Nonshared Environment Adolescent Development Study (NEAD; Reiss, Neiderhiser, Hetherington, & Plomin, 2000) and the Environmental-Risk Longitudinal Twin Study (E-Risk; Moffitt & the E-Risk Study Team, 2002)—include genetically informed designs. In addition, other programs of research, including the National Longitudinal Study of Adolescent Health, have over-sampled twin participants, therefore allowing researchers to begin to disambiguate the differential effects of shared genes and shared family experiences (e.g., Cleveland & Crosnoe, 2004). However, relatively few longitudinal, genetically informed studies exist that have tracked the role of *parenting* experiences in the developmental adaptation of children from infancy forward (but see Finkel et al., 1998). Given this state of affairs, one of the more exciting recent developments in the field of child psychology was the initiation of the Early Childhood Longitudinal Study–Birth Cohort (ECLS–B), the first nationally representative, longitudinal, methodologically rigorous study of an American birth cohort recruited in infancy that is explicitly designed to examine the effects of contexts on human development.

More specifically, the ECLS–B identified the parents of 10,688 children born in 2001, who have thus far been extensively assessed with their primary caregivers at 9 months. Most critical for the current analysis is the fact that (a) a large group of twin participants of known zygosity were over-sampled in the ECLS–B and (b) this study included observational data on the quality of the infant–caregiver relationship—a construct that has been of great interest to socialization researchers. The ECLS–B also collected maternal reports and observations of other aspects of infant behavior, such as temperament, that many researchers have argued are strongly influenced by genetic variation. We note also that this is the largest sample of co-twin infants observed interacting with their caregivers in the psychological literature and one of the few samples of infant twins examined in naturalistic observations in their homes.

The fact that the ECLS–B includes naturalistic observational assessments of the quality of the parent–child relationship in infancy is significant because this interpersonal relationship has been the focus of many theories of socialization and figures centrally in attachment theory. In contrast, much theory and data suggest that other aspects of infant development, including variation in temperament, may be strongly influenced by genetic factors. As a point of fact, most definitions of temperament presuppose that such variations are largely or even exclusively the product of endogenous factors that predate the birth of the child (e.g., Rothbart & Bates, 1998). In addition, data recently presented in Bokhorst et al. (2003) replicate a consistent finding that temperamental variation is largely heritable and that, to the extent to which environmental influences on its development can be identified, they appear to be nonshared within families. Nonetheless, given recent evidence that observations tend to yield larger effects of the shared environment than do self-reports and maternal reports (Emde et al., 1992; Plomin, 1981; Rhee & Waldman, 2002), in this study we conducted behavior-genetic decompositions of both maternal report and observer ratings of temperament. There is some evidence that parental reports of temperament in particular tend to underestimate the effects of the shared environment in large part because parents tend to exaggerate differences between their DZ infants, a phenomenon known as the *rater contrast effect* (Loehlin, 1992).

Informed debate about whether and how parents and genetic variation "matter" in development requires that developmental scientists disambiguate the effects of shared genes and shared environments in their research (Rutter, 2002). In this analysis, we therefore present data from the ECLS–B to examine whether the genetic and environmental contributions to infant temperament and infant–caregiver relationship quality differ as a function of outcome and method. More specifically, we expected that the role of genetic variation among infants would be minimal and the shared environment substantial in accounting for the observed quality of infant–caregiver relationships. We also expected that genetic factors and the nonshared environment would best explain variability in maternal reports (but not observations) of infant temperament.

Method

Participants

Participants for this study were drawn from the twin subsample of the ECLS–B 9-month assessment, which consists of approximately 800 twin pairs. More specifically, twin pairs were included in the current analysis if they met four criteria: (a) both twins participated in the ECLS–B, (b) they resided in the same home (too few twins were reared apart to conduct separate analyses), (c) they were the same sex, and (d) they were of known zygosity. Note that twin zygosity was ascertained by parental report in the ECLS–B—parents in this sample reported learning the zygosity of their children via a host of means, including cheek swab, blood, placental membrane, and blood typing DNA tests; doctors' observations of the placenta; doctor/midwife report; questionnaire; etc. (More detailed data on zygosity determination can be found in a restricted dataset available from the Department of Education.)

The criteria described above yielded a total of 172 MZ and 333 DZ co-twin pairs for this study (note, first, that MZ and DZ twins pairs were roughly half male and half female and, second, sampling was not based on whether the twins were identical or fraternal; therefore, the MZ versus same-sex DZ distribution above is simply a feature of prevalence in the 2001 birth cohort, despite the fact that, typically, a third of all twin births are MZ, same-sex DZ, and opposite-sex DZ; G. Mulligan, personal communications, April 17, 2006). The diverse subsample that is the focus of this study was 57.8% Caucasian, 16.4% African American, 18.2% Hispanic, 3.4% Asian or Hawaiian or Pacific Islander, 0.6% American Indian or Native Alaskan, and 3.6% multiracial. In addition, this subsample represents the entire range of the socioeconomic spectrum, from the first through the fifth quintile ($M = 3.2$, $SD = 1.4$; range = 1 [*lowest quintile*] to 5 [*highest quintile*]), as assessed by a socioeconomic status composite variable consisting of measures of paternal and maternal education and occupation, as well as household income (for detailed information see Nord et al., 2005). Note also that the final subsample for this study was comparable to (i.e., did not differ significantly from) the full, representative twin sample drawn from the ECLS–B on measures of socioeconomic status (full twin sample: $M = 3.3$; $SD = 1.4$) or ethnicity (full twin sample: 61.1% Caucasian, 15.8% African American, 15.7% Hispanic, 3.0% Asian or Hawaiian or Pacific Islander, 0.5% American Indian or Native Alaskan, and 3.8% multiracial).

As this is a secondary analysis, we provide below summary information regarding the measures highlighted in this report. One of the major strengths of the ECLS–B is the care and attention given to measure selection and development, including issues pertinent to ensuring validity and reliability. Complete information regarding the measures described below, as well as similarly detailed information regarding sampling, are available in two documentation manuals (Andreassen, Fletcher, & West, 2005; Bethel, Green, Nord, Kalton, & West, 2005) that can be freely downloaded from the U.S. Department of Education, National Center for Education Statistics Web site maintained at http://nces.ed.gov/ecls/Birth.asp.

Measures

Observed infant–caregiver relationship quality. A teaching task involving primary caregivers and target infants was rated by trained and reliable coders using the Nursing Child Assessment Teaching Scale (NCATS; NCAST, 1995), a well-validated assessment used in clinical practice and research to screen mothers and infants for early intervention programs. In the NCATS procedure, the infant's primary caregiver is asked to teach the target child a task, such as block building, just beyond the capacity of the infant. The focus of the coding was not on task success or failure, however, but the quality of the interaction between infant and caregiver. This observational assessment was selected by the ECLS–B as it has been used in several major national studies of child development (e.g., Early Head Start Research and Evaluation Project; Early Intervention Collaboration Study, Shonkoff, Hansen-Cram, Krauss, & Upshur, 1992, etc.) where it has demonstrated validity as an assessment of infant–caregiver relationship quality. For twins, interactions between primary caregivers and each target infant were conducted separately, recorded on separate videotapes, and used slightly different teaching activities.

In the ECLS–B, teaching interactions were videotaped in the home and subsequently coded using 73 binary (yes/no) items relevant to both the mother's and child's behavior during these interactions. A total score was derived by summing all items that characterize the degree to which parents and their children successfully employed a "teaching loop," whereby the primary caregiver (a) is observed to properly alert the child, thereby setting up expectations, (b) effectively instructs the child by making suggestions, asking questions, and so forth, (c) provides time for the child to respond to the instruction and (d) offers adequate and sensitive feedback to the child, and the target infant (e) sends clear cues to the caregiver and (f) is appropriately responsive to the cues of the caregiver.

In addition to extensive quality control efforts regarding properly administering the NCATS procedure, intercoder reliability on the NCATS was ensured in several ways. First, primary coding was completed by Westat staff who received extensive training by four individuals certified for this purpose by NCAST at the University of Washington. All of these trainers passed NCAST reliability training and in turn identified a set of Westat coders who themselves met the criteria established by NCAST ($>90\%$ reliability). Throughout coding, all raters at Westat and NCAST reliability coders at the University of Washington maintained at least 85% reliability across labs to prevent drift (based on a randomly selected set of 20 cases per month). Ultimately, the intercoder reliability correlation coefficient for the total score of the NCATS was .72 (see Andreassen et al., 2005, for additional details).

Mother-reported temperament. Selected items from the Infant/Toddler Symptom Checklist (ITSC; DeGangi, Poisson, Sickel, & Wiener, 1995) were administered to mothers that describe target participants' temperament. Of the total of 19 items represented on the ITSC, a subset of 8 items covering the domains of self-regulation, sleep–wake regulation, and attending were selected by the ECLS–B team, including (a) "is fussy or irritable," (b) "goes from whimper to crying," (c) "demands attention and company," (d) "wakes up 3 or more times," (e) "needs help to fall asleep," (f) "startled by loud sounds," (g) "cries for food or toys," and (h) "difficulty to raise on average." A principal components analysis of these ratings (standardized) indicated that three components were necessary to reproduce the associations among the ratings. The first component captured fussiness and demanding behavior ($\lambda = 2.56$; items a, b, c, and h), the second captured sleeping difficulties ($\lambda = 1.60$; items d and e), and the third reflected a tendency to be easily startled or upset ($\lambda = 1.02$; items f and g). In the analyses we report below, we focus on a composite based on the 4 irritability and attention-seeking items that loaded on the first component ($\alpha = .69$ for both twins). Note that the 8 items used in the ECLS–B were selected for the shortened form of the ITSC due to their ability to "identify children with sensorimotor and self regulatory difficulties" (Andreassen et al., 2005, p. 159; see also DeGangi et al., 1995).

Observed temperament. A subset of items drawn from the Behavior Rating Scale (BRS; Bayley, 1993), a supplementary component of the Bayley Short Form–Research Edition (BSID–II; Bayley, 1993), was used to characterize infants' behavior while participants completed the Bayley, a measure of infant cognitive development. Seven ratings were completed by trained observers: (a) "child displays positive affect," (b) "child displays negative affect," (c) "child is adaptable to change in materials," (d) "child shows interest in materials," (e) "child pays attention to tasks," (f) "child displays social engagement," and (g) "child shows control of movements." A principal components analysis of these ratings indicated that two or three components were necessary to reproduce the associations among the ratings. The first component captured positive affect ($\lambda = 3.21$; items a, d, e, and f), the second negative affect ($\lambda = 1.01$; items b and c), and the third motor control ($\lambda = 0.88$; item g). For simplicity in presentation, we report below analyses based on a single composite that combines the positive and negative affect items (i.e., items a–f), keyed in the direction of positive affect ($\alpha = .79$ for both twins). When we analyzed the data separately for positive and negative affect ratings, we reached the same conclusions we did based on the full composite. Note that observed temperament ratings were coded by the in-home interviewer after the home visit was completed as per instructions established in the BRS (Bayley, 1993). Interviewers underwent training in coding the BRS scales and achieved reliability with a standard developed by the ECLS–B (Andreassen et al., 2005).

Biometric Modeling

To examine the sources of individual differences in each variable of interest, we estimated the parameters of the ACE model—a commonly used biometric model in the behavioral genetics literature (see Neale & Cardon, 1992). According to this model, variation in an outcome arises from three latent sources: additive genetic variation (A), variation in environments that are shared between siblings (C), and variation in environments that are not shared between siblings (E). The additive genetic component represents the degree to which genetic variability contributes to variation in the outcome of interest. The shared environment component represents the degree to which variation common to siblings contributes to the outcome of interest. Finally, the nonshared effect represents the degree to which variation not shared by siblings contributes to the outcome of interest. The nonshared environment component also reflects random measurement errors.

We estimated the parameters of the ACE model using the computer program Mx (Neale, Boker, Zie, & Maes, 1999) and evaluated the fit of the model using several statistics, including chi-square, Akaike's information criterion (AIC), and root mean square error of approximation (RMSEA). Chi-square is a commonly used statistic for quantifying model fit; higher values of chi-square indicate larger deviations between the model's implied values and the empirical ones. AIC (Akaike, 1983) is proportional to the chi-square statistic (specifically, it is equal to chi-square minus two times the degrees of freedom), but it is designed to benefit more parsimonious models. Smaller AIC values indicate a better fit. RMSEA represents the degree of error involved in reproducing the observed covariance matrix from the estimated model. Smaller values indicate better model performance. A well-fitting model should have a nonsignificant chi-square, a low AIC, and a low RMSEA.

In addition to evaluating the full ACE model for each outcome of interest, it is also possible to constrain certain paths to zero as a way of determining whether the data can be explained just as well by assuming that one or more variance components are irrelevant in explaining variation in a given outcome. In the analyses we report below, we evaluated all possible submodels of the full ACE model: AE (i.e., a model that does not assume a role of shared environmental factors), CE (i.e., a model that assumes no additive genetic effects), and E (i.e., a model that assumes that all phenotypic variation is due to nonshared factors). In cases in which removing one or more components does not significantly diminish the fit of the model, we focused on the more parsimonious model.

Results

Phenotypic Correlations

Phenotypic correlations among the three constructs explored in this report (observed infant–caregiver quality, observed temperament, and maternal reports of temperament) were trivial to small in magnitude by Cohen's (1992) criteria. As expected, observed infant–caregiver relationship quality was trivially associated with observed ($r = .03$, *ns*) and maternal-reported temperament ($r = -.09$, $p < .05$). In contrast, we identified a small association between observed and maternal reports of temperament ($r = -.12$, $p < .01$). (Note that this association is negative because observed temperament was keyed such that higher scores reflect more positive relative to negative affect.)

Because phenotypic correlations were small in magnitude, we did not follow up univariate behavior-genetic analyses described below with bivariate analyses to decompose heritable and environmental sources of variation to the associations among these variables. We do note, however, that the results of supplementary multilevel analyses in HLM 6.02 revealed that in only one instance there was evidence that zygosity (treated as a Level II covariate) explained variation in the associations described above. More specifically, a marginally significant Level II HLM effect suggested that the association between maternal-reported and observed temperament varied by zygosity (as might be expected, the association was marginally larger for MZ than for DZ twins).

Intraclass Correlations

The intraclass correlations among MZ and DZ twins for each of the variables of interest are reported in Table 1. With the exception of observer ratings of infant–caregiver relationship quality, the MZ correlations tend to be higher than the DZ correlations. More specifically, results of *r*-to-*z* tests of the difference between two independent correlations revealed statistically significant differences between MZ and DZ correlations for observed temperament ($t = 3.2$; $p < .01$) and maternal reports of temperament ($t = 3.5$; $p < .01$), but not infant–caregiver relationship quality ($t = -0.1$; $p = .91$). Note that MZ and DZ *n*s vary somewhat in analyses based on participation in each part of the 9-month assessment (see Table 1). Subsamples were comparable, however, to the full set of twins selected for this study on all key demographic data described in the Methods section.

Biometric Modeling

To evaluate the relative contribution of genetic, shared environmental, and nonshared environmental sources of variance more formally, we estimated the parameters of the full ACE model as well as the various derivatives of it. Each set of analyses was based on the variance–covariance matrix for MZ and DZ twins.

Table 1
Intraclass Correlations Among MZ and DZ Co-Twins

Variable	Correlation	
	MZ	DZ
Observer-rated parent–child quality	.42 (122)	.43 (234)
Maternal-report temperament	.48 (172)	.19 (332)
Observer-rated temperament	.64 (165)	.42 (312)

Note. MZ = monozygotic; DZ = dizygotic.
Sample sizes are reported in parentheses. The *n*s vary from analysis to analysis due to missing data.

Observer ratings of parent–child interaction quality. Table 2 reports the results of the model fitting analyses for the full ACE model and its submodels. As can be seen, the contribution of additive genetic variance to relationship quality was virtually zero. In fact, when the additive genetic component of the model was constrained to equal zero, the constrained model was able to explain the data just as well as the full ACE model. It is important to note that placing other constraints on the model (e.g., setting C to zero) led to significant decrements in performance. In short, it appears that the most parsimonious account for the relationship quality data involves shared and nonshared environmental variance.

Maternal reports of child temperament. Table 3 reports the results of the model fitting analyses for the full ACE model and its submodels for maternal reports of child temperament. These results were consistent with what has been reported before in the literature (e.g., Bokhorst et al., 2003). Specifically, there was evidence of a sizable genetic and nonshared environmental component to ratings of temperament, but negligible evidence of a shared environmental component. Indeed, constraining the shared environmental component to equal zero (i.e., an AE model) led to the most parsimonious account of these data.

As can be seen in Table 1, the MZ correlation was substantially higher than twice the DZ correlation, raising the possibility that some of the variation in ratings of temperament was due to nonadditive genetic variance. As such, we also examined an ADE model, a model that assumes that the variation in the outcome of interest is due to an additive genetic component (A), a dominance component (D), and nonshared environmental factors plus measurement noise (E). (Note that, in a twin design, the more inclusive ACDE model cannot be tested because of the confounding of the A, C, and D components in twin pairs raised together.)

The results of this analysis are presented in Table 4. These analyses suggest that the genetic contribution to maternal ratings was comprised of both additive and nonadditive effects. Because some writers have noted that a DE model is not biologically plausible (see Becker-Blease et al., 2004), we do not present this submodel in Table 4. Nonetheless, it is important to note that setting either the A or the D component to zero did not lead to noteworthy decrements in model fit. However, it was not clear statistically whether one submodel was necessarily better than the other. Given that the ADE model performed as well or better than these submodels, we conclude that the full ADE model—one that includes additive and nonadditive genetic influences—provides the best account for variation in maternal reports of temperament.

Observer ratings of child temperament. Table 5 reports the results of the model fitting analyses for the full ACE model and its submodels for observer ratings of child temperament. There was evidence of a sizable genetic and nonshared environmental component to ratings of temperament, as well as a comparable shared environmental component. In-

Table 2
Estimates of the Contribution of Additive Genetic, Shared Environmental, and Nonshared Environmental Sources of Variance to Variation in Observer-Rated Infant–Caregiver Relationship Quality

Model	Model fit					Nested model comparisons			Variance components		
	χ^2	*df*	*p*	AIC	RMSEA	χ^2_{diff}	*df*	*p*	A	C	E
ACE	6.078	3	.108	.078	.071	—	—	—	.01	.40	.59
AE	16.171	4	.003	8.171	.132	10.09	1	.001	.51	—	.49
CE	**6.082**	**4**	**.193**	**− 1.918**	**.051**	**.004**	**1**	**.950**	—	**.41**	**.59**
E	71.999	5	<.001	61.990	.272	65.92	2	<.001	—	—	1

Note. AIC = Akaike's information criterion; RMSEA = root mean square error of approximation.
The statistics for the nested model comparisons test the difference in fit obtained when specific sources of variation in the full ACE model are constrained to zero. The most parsimonious model is highlighted in bold.

Table 3

Estimates of the Contribution of Additive Genetic, Shared Environmental, and Nonshared Environmental Sources of Variance to Variation in Maternal-Report Temperament

	Model fit					Nested model comparisons			Variance components		
Model	χ^2	*df*	*p*	AIC	RMSEA	χ^2_{diff}	*df*	*p*	A	C	E
ACE	2.976	3	.395	− 3.024	.012	—	—	—	.46	0	.54
AE	**2.976**	**4**	**.562**	**− 5.024**	**.000**	**<.001**	**1**	**.999**	**.46**	—	**.54**
CE	11.087	4	.026	3.087	.089	8.11	1	.004	—	.50	.50
E	149.482	5	<.001	139.782	.365	146.51	2	<.001	—	—	1

Note. AIC = Akaike's information criterion; RMSEA = root mean square error of approximation.
The statistics for the nested model comparisons test the difference in fit obtained when specific sources of variation in the full ACE model are constrained to zero. The most parsimonious model is highlighted in bold.

deed, the various submodels, which imposed constraints on A and C, were not able to account for the data as well as one that included both additive genetic and shared environmental components. These findings suggest that variation in child temperament, as rated by disinterested observers, reflects both genetic, shared environmental, and nonshared environmental components of variance.

Discussion

The observation that individual differences in all psychological phenotypes are heritable and that shared environmental factors are relatively inconsequential for that variation have been described as "laws of behavior genetics" (Turkheimer, 2000). Rutter (2002), for example, recently wrote that "it is necessary to note the pervasiveness of genetic influences across all psychological traits, even those involving attitudes or social behavior" (p. 2). One of our objectives in this article was to offer an important caveat to this claim. Data from the ECLS–B suggest that, when it comes to understanding variability in the quality of the relationship shared by infants and their caregivers, genetic variability among infants is largely inconsequential. In contrast, environmental factors, both those that are shared and not shared within families, are paramount. These data, taken together with the findings from other genetically informed observational studies of parent–child relationships in the early life course (Bakermans-Kranenburg et al., 2004; Bokhorst et al., 2003; Dozier, Stovall, Albus, & Bates, 2001; O'Connor & Croft, 2001) and love styles in adulthood (Waller & Shaver, 1994), call into question the ubiquity of heritability effects in all domains of psychological inquiry. They also bolster consensus in the field of developmental psychology that shared environmental effects are not as elusive as had once been believed (Rose, 1995; Rutter, 2002).

Although we are skeptical of claims regarding the far-reaching role of genetic variation in understanding all individual differences, we are equally surprised by the tendency of some developmental researchers to dismiss outright research in behavioral genetics. As such, a secondary objective in this article was to demonstrate how behavior-genetic research designs have the potential to provide

Table 4

Estimates of the Contribution of Additive Genetic, Nonadditive Genetic, and Nonshared Environmental Sources of Variance to Variation in Maternal-Report Temperament

	Model fit					Nested model comparisons			Variance components		
Model	χ^2	*df*	*p*	AIC	RMSEA	χ^2_{diff}	*df*	*p*	A	D	E
ADE	**1.908**	**3**	**.59**	**− 4.092**	**.000**	—	—	—	**.25**	**.24**	**.51**
AE	2.976	4	.562	− 5.024	.000	1.07	1	.301	.46	—	.54
E	148.893	5	<.001	138.893	.365	146.99	2	<.001	—	—	1

Note. AIC = Akaike's information criterion; RMSEA = root mean square error of approximation.
The statistics for the nested model comparisons test the difference in fit obtained when specific sources of variation in the full ADE model are constrained to zero. The retained model is highlighted in bold (see text for explanation). Note that the DE submodel is not presented because it is biologically implausible (see Becker-Blease et al., 2004).

Table 5

Estimates of the Contribution of Additive Genetic, Shared Environmental, and Nonshared Environmental Sources of Variance to Variation in Observer-Rated Temperament

	Model fit					Nested model comparisons			Variance components		
Model	χ^2	*df*	*p*	AIC	RMSEA	χ^2_{diff}	*df*	*p*	A	C	E
ACE	**1.062**	**3**	**.786**	**− 4.938**	**.000**	—	—	—	**.39**	**.23**	**.38**
AE	6.365	4	.173	− 1.635	.033	4.75	1	.029	.65	—	.35
CE	11.054	4	.026	3.054	.089	9.43	1	.002	—	.50	.50
E	148.893	5	< .001	138.893	.365	147.27	2	< .001	—	—	1

Note. AIC = Akaike's information criterion; RMSEA = root mean square error of approximation.
The statistics for the nested model comparisons test the difference in fit obtained when specific sources of variation in the full ACE model are constrained to zero. The most parsimonious model is highlighted in bold.

among the strongest support for socialization theories, even those that posit that genetic variation plays little or no role in the development of a given phenotype. Because behavior-genetic designs allow researchers to separate the effects of shared environmental from shared genetic influences, they offer a compelling, if conservative, test of strong socialization hypotheses. Behavior-genetic research is not without its limitations (see Gottlieb, 1995). Nonetheless, the only way to rule out the potential confounding effects of genetic variability is by taking that variability into account and behavior-genetic designs offer one way to do so.

In recent years researchers have made some headway toward studying developmental processes in a genetically sensitive fashion (e.g., Moffitt & the E-Risk Study Team, 2002; Reiss et al., 2000). We should note, however, that twin designs are only one of several approaches available for doing so. Recent research that involves the direct assessment of molecular genetic functional polymorphisms has the potential to further our understanding of the interplay among genes, parenting, and peer experiences in development (see, e.g., Bakermans-Kranenburg & van IJzendoorn, 2004; Caspi et al., 2002, 2003; Lakatos et al., 2002). What is interesting about the work by Caspi et al. (2002, 2003) in particular is that it suggests main effects of environmental but not genetic variation, as well as the much heralded evidence for interactions whereby risk alleles increase the negative effects of environmental inputs such as maltreatment and life stress.

In addition to illustrating the limits of genetic influence, this study underscores the importance of *method* in the estimate of effects in behavior-genetic analyses (Hoffman, 1991; Rose, 1995). For example, in examining *mothers' reports* of infant temperament, we were unable to identify shared environmental effects in accounting for similarities among twins. In contrast, when examining *observer ratings* of temperament, each component of the ACE model—including the shared environmental component—was necessary to account for the data. We believe the growing literature demonstrating that behavior-genetic findings are moderated by the method used to assess psychological constructs (e.g., Emde et al., 1992; Plomin, 1981; Rhee & Waldman, 2002) is consistent with Hoffman's (1991) assertion that early behavior-genetic research underestimated the effects of shared environments.

Limitations and Caveats

One of the limitations of the present study is that, because data are currently available for only one assessment point in the ECLS–B, this dataset is not yet able to speak to the question of whether the correlates of infant–caregiver relationships are due to shared environments, shared genes, or both (Harris, 1995). Second, as is the case with all classic behavior-genetic twin studies, our findings suggest only an anonymous role for shared environmental effects without identifying the precise mechanisms giving rise to those effects. An implication of this is that the shared environmental effects we have identified may in fact have nothing to do with parents whatsoever. We have framed our discussion as if shared environmental factors are mostly about parental influence, but this may not be the case. Third, we should note that, although we believe these data on parent–child relationship quality violate the ''laws of behavior genetics'' (Turkheimer, 2000), this variable is only one of many that have been studied in the behavior-genetic literature. We believe, however, that it is an important one and that these findings should not be brushed aside as a unique exception to a more inclusive rule. To be sure, the quality of infant–caregiver relationships is much more relevant to theories of socialization than those variables often studied in the behavior-genetic literature (e.g., IQ, self-reported personality, social attitudes; Bouchard, 2004).

Fourth, this study focused on the heritability of *infant*–caregiver relationships due to their central role in attachment theory. There is now ample evidence that social contexts, including parent–child relationships, are associated with genetic variation by adolescence, although several caveats apply. First, behavior-genetic estimates vary across domains of parenting. Neiderhiser et al. (2004, p. 36) summarized the state of affairs in this way: "parental warmth and support and parental negativity have shown substantial genetic and nonshared environmental influences and modest to negligible shared influences ... In contrast, primarily shared and nonshared environmental influences have been found for measures of parental monitoring and control." Even more critically, perhaps, heritability effects (even within the same investigations) are much more readily identified using questionnaire methods but are more modest when direct observations of parenting are conducted (Neiderhiser et al., 2004; O'Connor et al., 1995). Furthermore, no investigation has examined attachment-specific behavior beyond preschool; therefore, it is unclear whether the finding of minimal genetic influence in this and other studies is age specific, domain specific, or both.

Finally, although these data suggest that genetic variability among infants is not associated with variation in the quality of infant–caregiver relationships, it could be that a study of MZ and DZ *parents* would yield contrasting results using a parent-based behavior-genetic design, such as the Children of Twins approach (CoT; D'Onofrio et al., 2005; see also Neiderhiser et al., 2004). If it were determined that the quality of infant–caregiver relationships reliably covaries with genetic variability among parents, for example, a claim could be still made for the role of *passive* genetic processes in the development of these relationships (Neiderhiser et al., 2004). That said, the current analysis, together with that reported by Boivin et al. (2005), is important because it begins to close the book on the long-held notion that genetic variation among infants normatively evokes parental behaviors that account for the quality of the infant–caregiver relationship. This conclusion is consistent with research demonstrating a weak association between the quality of attachment security in infancy and variation in temperament (Vaughn & Bost, 1999). Questions remain, however, about what causal factors lead secure parents to have secure children given that parental sensitivity accounts only for a modest proportion of the variance in the quality of infant–caregiver relationships (De Wolff & van IJzendoorn, 1997). Considering parental genetic variation as a possible closer of this "transmission gap" (van IJzendoorn, 1995) should be a priority for attachment researchers and will necessitate additional genetically informed research (for an excellent example, see the adoption study by Dozier et al., 2001).

More generally, we believe that these data provide important qualifications to what has been claimed about the role, or lack thereof, of genetic variation and shared environments in developmental outcomes. Some behavior-genetic researchers and commentators have made overly broad claims about the limited role of parents and the ubiquitous role of genetic variation in human development. On the other hand, it is equally true that many developmental psychologists have failed to disambiguate shared family environments and shared genes in their studies of human development, making claims about shared environmental influences that are suspect. The current analysis thus speaks to the utility of fully unconfounding ideology and methodology in the study of socialization processes.

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