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The Etiology of Behavioral Problems and Competencies in Very Young Twins

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Abstract

Although genetic and environmental influences on behavior problems in middle childhood and adolescence have been well-studied, little is known about the etiology of behavior problems in very early childhood. The present study explores genetic and environmental contributions to individual differences in behavior problems and competences in an infant–toddler sample of twins. There were 1,950 twin pairs (mean age = 23.8 months) who were rated by parents on the Infant-Toddler Social and Emotional Assessment. All four domains (Externalizing, Internalizing, Dysregulation, Competence) and 20 subscales–indices on the Infant-Toddler Social and Emotional Assessment displayed significant heritability. There were also substantial shared environmental influences operating on most of the domains and subscales. Compared with behavior problems, behavioral competencies were less heritable and more influenced by shared environments.

Keywords

twins; behavior problems; genetics; model fitting

The toddler period may be a critical time for the early emergence of clinically significant problem behaviors (Campbell, 1985; Keenan & Wakschlag, 2000). Externalizing problems (e.g., aggression, conduct disorders, hyperactivity) and internalizing problems (e.g., anxiety, depression, and emotional problems) can be reliably identified in very early childhood and show considerable stability across age (e.g., Briggs-Gowan et al., 2006; Campbell, Pierce, March, Ewing, & Szumowski, 1994; Fischer, Rolf, Haszi, & Cummings, 1984; Keenan, Shaw, Delliquadri, Giovannelli, & Walsh, 1998; Lavigne et al., 1998; Mathiesen & Sanson, 2000; Olson, Bates, Sandy, & Lanthier, 2000). Toddlers who display problem behaviors are at risk for a variety of nonoptimal developmental outcomes including poor academic performance, conflictual parent–child interactions, peer rejection, delinquency, and later

maladjustment (e.g., Caspi, Moffitt, Newman, & Silva, 1996; Hofstra, van der Ende, & Verhulst, 2002; Patterson, DeBaryshe, & Ramsey, 1989; Stevenson & Goodman, 2001). Given their developmental significance, it is important to understand the factors that influence problem behaviors in very young children.

The etiology of behavior problems in middle childhood and adolescence has been well studied. Twin and adoption studies examining externalizing, internalizing, and related behavior problems consistently reveal substantial genetic influence, with heritabilities in the range of .40–.70 (e.g., Bartels et al., 2003, 2004; Edelbrock, Rende, Plomin, & Thompson, 1995; Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998; Saudino, Ronald, & Plomin, 2005). The influence of shared environments on behavior problems is typically more modest, explaining between 0% and 40% of the variance in behavior problems (e.g., Bartels et al., 2003, 2004; Deater-Deckard & Plomin, 1999; Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; Happonen et al., 2002; Saudino et al., 2005; Scourfield et al., 2003). Little is known, however, about the relative contributions of genetic and environmental factors to individual differences in very early childhood. Studies looking at age effects across middle childhood and adolescence have found that the importance of genetic influences on specific behavior problems (e.g., depression, anxiety, antisocial behavior) increase with age (e.g., Eley & Stevenson, 1999; Rice, Harold, & Thapar, 2002; Rhee & Waldman, 2002; Scourfield et al., 2003; Thapar & McGuffin, 1994), thus reminding researchers that despite the phenotypic stability of behavior problems across age, the etiology of behavior problems in early childhood need not be the same as that for older children.

Most of the research examining genetic and environmental influences on behavior problems in early childhood comes from the Netherlands Twin Registry (NTR; Boomsma, 1998), a large population-based sample of several thousand twin pairs assessed longitudinally beginning at age 3. Analyses of the Child Behavior Checklist (CBCL; Achenbach, 1992) indicated that at age 3, genetic factors explain at least 50% of the variance on the two broadband scales of Externalizing and Internalizing. Shared environmental influences were significant but more modest, explaining no more than a third of the variance for Externalizing and roughly 10% of the variance for Internalizing behaviors (Bartels et al., 2004; van den Oord, Verhulst, & Boomsma, 1996; van der Valk, van den Oord, Verhulst, & Boomsma, 2001). All CBCL problem syndromes were significantly heritable at age 3 years; however, the pattern of genetic and environmental effects was variable. Over-active and anxious-depressed behavior problems were highly heritable (i.e., .70 and .69, respectively) with negligible shared environmental influences (Derks, Hudziak, van Beijsterveldt, Dolan, & Boomsma, 2004). In contrast, individual differences in oppositional behavior were more strongly influenced by environmental (.43 shared environmental variance, .24 nonshared environmental variance) than genetic (.33) factors (Derks et al., 2004). Aggression and withdrawn behavior problems displayed heritabilities and environmentalities similar to those of the broadband scales (Derks et al., 2004; Hudziak et al., 2003). Sex differences have also emerged in the NTR data. Specifically, for aggression and withdrawn behaviors, genetic influences were stronger for girls and shared environmental influences were stronger for boys (Derks et al., 2004), whereas the reverse was true for overall externalizing problems on the CBCL broadband Externalizing scale (Bartels et al., 2004).

Studies outside the NTR suggest stronger evidence of shared environmental influence than found in the Dutch data. For example, neither the Externalizing nor Internalizing scales on the CBCL displayed significant heritability in a sample of 229 twin pairs with a mean age of 2 years (Schmitz, Cherny, Fulker, & Mrazek, 1994). For both scales, shared environmental influences accounted for more than 40% of the variance. Although based on a small sample, this finding hints that shared environmental influences may be important in younger children; however, to our knowledge, there are no other published studies exploring the etiology of behavior problems in children below age 3. Similarly, to date, almost all the behavioral genetic research on behavior problems in early childhood has relied on the CBCL to assess problem behaviors. An exception is the Twins Early Development Study (TEDS; Trouton, Spinath, & Plomin, 2002), a large community sample of young twins in the United Kingdom, that assesses hyperactivity at ages 2, 3, and 4 using the Strengths and Difficulties Questionnaire (Price, Simonoff, Waldman, Asherson, & Plomin, 2001) and anxiety and related behaviors in 4-year-olds using items from a variety of parent-rating measures of temperament and psychopathology (Eley et al., 2003). Consistent with the Dutch CBCL findings, hyperactivity in TEDS was substantially heritable with little evidence of shared environmental influences (Price et al., 2001); however, in contrast to the Dutch CBCL results that found modest shared environmental influence (Bartels et al., 2004; van den Oord et al., 1996; van der Valk et al., 2001), in the TEDS sample, anxiety-related behaviors displayed significant and substantial shared environmental influences (Eley et al., 2003). These findings highlight the need to consider the extent to which empirical results converge across different measures.

The present study extends prior research on the etiology of behavior problems in early childhood in two important ways. First, we examined the relative influences of genetic and environmental effects on behavior problems in a large sample of very young twins (mean age = 23.8 months). Fifty-eight percent of the sample was under 2 years of age, thus allowing us to explore the factors that influence individual differences in very early emerging behavior problems. Given the Schmitz et al. (1994) findings that showed substantial shared environmental influences in their 2-year-old sample and the research from middle childhood that suggests that genetic effects may increase with age (Eley & Stevenson, 1999; Rice et al., 2002; Rhee & Waldman, 2002; Scourfield et al., 2003), we hypothesized that shared environmental factors would play an important role in the variability of behavior problems in our toddler sample.

The second way in which we extend prior research is through our use of a novel measure, the Infant Toddler Social Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 2006; Carter, Briggs-Gowan, Jones, & Little, 2003), to assess behavior problems. The extent to which behavioral genetic findings replicate across measures informs about the robustness of the effects. As indicated above, the CBCL has dominated the research exploring behavior problems in young children. This was due, in part, to the fact that there were few well-established measures to assess problem behaviors in very young children. The ITSEA was designed to address this weakness and is appropriate for children younger than those who can be assessed on the CBCL. Moreover, the ITSEA offers some advantages to the CBCL. In addition to including scales that address problem behaviors falling under the broad domains of externalizing and internalizing, the ITSEA includes behaviors related to

regulatory problems (e.g., sleeping and eating problems, negative emotionality, and sensory sensitivity). Dysregulation, often considered a temperament dimension, has been linked to a range of both externalizing and internalizing behavior problems (Eisenberg, Cumberland, & Spinrad, 2001; Eisenberg, Guthrie, & Fabes, 2000; Rubin, Burgess, & Dwyer, 2003). In fact, dysregulation in early infancy has been shown to predict conduct disorder several years later in middle childhood (Morrell & Murray, 2003). Despite being a developmentally significant behavior, dysregulation has generally been ignored in behavioral genetic studies of problem behaviors. Given its links to temperament, we predicted that in the present study behaviors falling under the domain of dysregulation would be genetically influenced.

The ITSEA also includes measures of socioemotional and behavioral competencies in addition to behavior problems, thus giving a fuller picture of socioemotional development in very young children. Competencies (e.g., compliance, attention regulation, prosocial peer behaviors, empathy, and mastery motivation) reflect the presence of age-appropriate skills (Carter et al., 2003) and may serve to buffer risk for later adjustment problems (Carter, 2002; Cicchetti & Cohen, 1995). As is the case for dysregulation, the study of early socioemotional competencies has received relatively little attention from a behavioral genetic perspective, however, two specific aspects of socioemotional competence, empathy and prosocial behavior, have been examined in a handful of twin studies. Overall, both competencies in childhood appear to be genetically influenced (Knafo & Plomin, 2006b; Scourfield, John, Martin, & McGuffin, 2004; Saudino et al., 2005; Stevenson, 1997; Zahn-Waxler, Robinson, & Emde, 1992; Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001). Similar to findings for problem behaviors, genetic influences on prosocial behavior tend to increase, and shared environmental influences tend to decrease with age (Knafo & Plomin, 2006b; Scourfield et al., 2004). There are also hints of sex differences in genetic effects on prosocial behaviors, but the results are inconsistent across raters, methods, and studies (e.g., Knafo & Plomin, 2006b; Saudino et al., 2005; Scourfield et al., 2004; Zahn-Waxler et al., 2001). In the present study, we look at a broad range of behaviors that fall under the ITSEA Competence domain. On the basis of prior behavioral genetic research with empathy and prosocial behavior, we predicted that behavioral competencies on the ITSEA would be influenced by genetic factors. Moreover, because prosocial behaviors in toddlers have been linked to parenting practices (e.g., Deater-Deckard, Dunn, O'Connor, Davies, & Golding, 2001; Eisenberg & Fabes, 1998; Knafo & Plomin, 2006a; Krevans & Gibbs, 1996; Whiteside-Mansell, Bradley, Tresch Owen, Randolph, & Cauce, 1998), it was also predicted that early behavioral competencies would show substantial shared environmental variance.

Method

Sample and Recruitment

Twins were recruited via *Jumeaux et plus* ("Twins and more"), an organization for parents of multiples in France. Prospective participants were sent questionnaires by regular mail. The recruitment letter included a description of the study, the questionnaire, a consent form, and a prepaid envelop for return of questionnaires. There were 2,793 families who completed questionnaires. Of these, 2,680 were families with twins (triplets and quadruplets

are not included in the present analyses). We further restricted analyses to twins within the age range of 1–4 years ($n = 2,274$). Zygosity was determined through parent reports of twins' blood type, physical similarity, and instances of identity confusion. This method of zygosity classification has been shown to yield accuracy of over 90% when cross-validated with zygosity diagnoses based on blood typing or DNA markers (Price, Freeman, Craig, Ebersole, & Plomin, 2000). Using stringent criteria for zygosity classification, it was possible for us to determine the zygosity of 1,950 twin pairs. Twins who could not be identified as monozygotic (MZ) or dizygotic (DZ) were excluded from all analyses. The final sample included 204 monozygotic male and 189 monozygotic female twin pairs, 412 dizygotic male and 390 dizygotic female twin pairs, and 755 dizygotic opposite (DZO) sex twins. The mean age at time of assessment was 23.8 months ($SD = 9.0$). The sample was predominantly urban and middle to upper-middle class (i.e., factory workers and farmers were relatively rare). Although some questionnaires were completed by the twins' fathers, 96% were completed by their mothers.

Measure

Parents rated the behaviors of their twins on a French adaptation of the ITSEA, the Evaluation Sociale et Emotionnelle de Jeunes Enfants (ESEJE; Bracha et al., 2004). The ITSEA and its French counterpart consist of 169 items comprising 17 subscales grouped into four domains, (a) Externalizing (Activity/Impulsivity, Aggression/Defiance, Peer Aggression), (b) Internalizing (Depression/Withdrawal, General Anxiety, Separation Distress, Inhibition to Novelty), (c) Dysregulation (Sleep, Negative Emotionality, Eating, Tactile Sensitivity), and (d) Competence (Compliance, Attention, Imitation/Play, Mastery Motivation, Empathy, Prosocial Peer Relations), and three behavioral indices (Maladaptive, Social Relatedness, and Atypical). Items are rated on a 3-point scale: 0 = *not true/rarely*, 1 = *somewhat true/sometimes*, and 2 = *very true/often*. A *no opportunity* code allows parents to indicate that they have not had the opportunity to observe certain behaviors (e.g., behavior with peers in daycare). The ITSEA domains have demonstrated good internal consistency ($\alpha = .80-.90$) and test-retest reliability ($r = .82-.90$), as well as validity relative to observational measures and other parent-report checklists (Carter & Briggs-Gowan, 2006). Additionally, the ITSEA has demonstrated validity in distinguishing children with diagnosed psychiatric conditions and Autistic disorder from matched controls (Carter & Briggs-Gowan, 2006). Prior confirmatory factor analyses of the ESEJE (Bracha et al., 2004) revealed a factor structure mirroring that of the ITSEA and similar levels of reliability for domain scales ($\alpha = .73-.87$), thus providing a strong test of measurement invariance across the two cultures. In the present sample, domain scales demonstrated levels of reliability consistent with both the ITSEA and its French counterpart ($\alpha = .73-.81$).

Data Transformation

Scores for Peer Aggression, Depression/Withdrawal, General Anxiety, Eating, and Maladaptive and Atypical Behaviors were positively skewed and were log-transformed to create a more normal distribution. Because twin covariances can be inflated by variance due to age, all scores were residualized for age effects (see McGue & Bouchard, 1984). These residualized scores were used in all behavioral genetic analyses.

Twin Intraclass Correlations

Twin intraclass correlations for all five zygosity groups were calculated using a double entry procedure. Although the major results of a twin analysis can be gleaned from twin intraclass correlations, model-fitting procedures analyze all of the data simultaneously, provide tests of fit of models, yield confidence intervals for parameter estimates, and tests the fit of alternative models (Plomin, DeFries, McClearn, & McGuffin, 2001).

Model-Fitting Analyses

A sex-limitation model (Neale & Cardon, 1992) was fit to observed covariance matrices of each of the four ITSEA domains and the 20 subscales using Mx structural equation modeling software (Neale, Boker, Xie, & Maes, 2003). The model, illustrated as a path diagram in Figure 1, uses data from all five zygosity groups (male and female MZ and DZ pairs and opposite-sex DZ pairs) to estimate genetic and environmental variance components and to evaluate possible sex differences in genetic and environmental contributions to phenotypic variation of the measure under study. The rectangles represent the phenotypic variances of each twin. The circles represent latent genetic and environmental factors. The curved double-headed arrows indicate correlations between the variables they connect, whereas the single-headed arrows represent partial regressions of the measured variable on the latent variable.

Within same-sex twin pairs, the model is a standard univariate twin model sometimes referred to as the *ACE model*. The A factors refer to additive genetic influences—the sum of the average effect of all genes that influence a trait. On the basis of the degree of genetic relatedness, the A factors correlate, as indicated by the r_g paths, 1.0 and .5 for MZ and DZ twins, respectively. The C factors refer to the influence of shared rearing environments on twin resemblance. Because all twins were reared in the same family, the correlation for shared environment (i.e., r_c) is 1.0 for both MZ and DZ twins. Finally, the E factors reflect nonshared environmental variance and measurement error. Nonshared environmental influences are unique to each member of a twin pair and therefore are depicted in the path diagram as residual arrows for each twin representing the remaining variance not explained by genetics or shared environment. The path coefficients a , c , and e are partial regressions that indicate the relative influence of the latent variables on the phenotype. When path coefficients have been standardized, the percentage variance explained by a latent variable is the square of the path coefficient. As indicated by the m and f subscripts for the path coefficients in Figure 1, the model allows a , c , and e , to be estimated separately for each sex.

The phenotypic variation for opposite-sex twins is also a function of additive genetic variance and shared and nonshared environmental influences, but the genetic correlation between opposite-sex twin pairs may be less than .5 if there are qualitative sex-specific genetic effects (i.e., different genes operate on the behavior for each sex). Similarly, although opposite-sex twins are reared in the same family, they may have fewer shared environmental experiences than same-sex twins and, hence, the correlation for shared environment DZO twins may be less than 1.0. In Figure 1, the genetic and shared environmental correlations for DZO twins are depicted as dashed double-headed arrows. The sex-limitation model allows estimation of either a specific DZO genetic correlation

(r_gO) or DZO shared environmental correlation (r_cO); however, both parameters cannot be estimated simultaneously.

The full sex-limitation model estimating phenotypic variances, the parameters a_m , c_m , e_m , a_f , c_f , e_f , and either r_gO or r_cO permits quantitative sex differences in ACE parameters, qualitative sex differences as assessed by r_gO , and phenotypic variance differences between the sexes. To test the significance of sex effects, we compared the fit of this full-sex limitation model against a series of three nested reduced models for each variable: (a) a *common effects* (quantitative sex differences) model in which ACE parameter estimates for males and females were allowed to differ, but r_g for opposite-sex DZ twins was constrained to be the same as r_g for same-sex DZ twins, thereby excluding qualitative sex-specific genetic effects; (b) a *scalar model* (phenotypic variance differences between the sexes) that allows phenotypic variances between males and females to differ but equates ACE parameters for males and females and constrains r_gO to be the same as r_g same sex; (c) a *null model* (no sex differences) in which ACE parameters and phenotypic variances for males and females are constrained to be equal and r_gO is constrained to be the same as r_g for same-sex DZ twins. Because these alternative models are hierarchically related (i.e., one model is nested within the other), the relative fit of each alternative model is determined by the difference in chi-square between the two models with degrees of freedom equal to the difference in degrees of freedom between the two models. The full r_gO and full r_cO models are not hierarchically related; therefore, we computed Akaike's information criterion for both models (see Neale & Cardon, 1992, for formula) and the model with the lowest Akaike's information criterion was judged to be the better fitting model.

When the above model-fitting analyses suggested a sex difference in the etiology of a behavior (i.e., either the full sex-limitation model or the common effects model were the best fitting models), specific tests of sex differences in variance components were conducted. Here, we fit a series of submodels of the best fitting model (base model) where either the genetic (a^2), shared environmental (c^2), or nonshared environmental (e^2) variances were equated across sex. The fits of these models were then compared with the fit of the base model. A significant difference in chi-squares between the base model and the submodel indicates that boys and girls could not be equated for the parameter being tested.

Results

Descriptive Statistics

Table 1 lists the ITSEA domain and subscale means and standard deviations by zygosity and sex. We evaluated the effects of sex, zygosity, and age using generalized estimating equations implemented in the SAS GENMOD procedure to account for dependence in the data due to the fact that our sample comprised pairs of twins. Generalized estimating equations are an extension of the standard generalized linear models that allow modeling of correlated data (Liang & Zeger, 1986; Zeger & Liang, 1986). As would be expected given our large sample size, many significant mean group differences were found. Consistent with previous research with the ITSEA, boys displayed higher levels of externalizing behavior problems involving activity/impulsivity, aggression/defiance, peer aggression, and negative emotionality, and girls displayed higher levels of competence, compliance, attention,

imitation/play, empathy, prosocial behaviors, and social relatedness (Bracha et al., 2004; Carter et al., 2003). Also consistent with previous research was the finding of significant age effects. In general, behavior problems tend to increase across early childhood, perhaps illustrating the shift into the “terrible twos.” Despite this, behavioral competencies also increase across age. Of interest to our twin design, MZ and DZ twin groups did not significantly differ on any ITSEA variable.

To evaluate the proportion of the sample at risk for clinically relevant behavior problems, we used cut points from U.S. normative data (Carter & Briggs-Gowan, 2006). For each domain, cut points identify the mean scores for each sex within 6-month age groups that reflect the extreme 10th percentile in the normative sample. Using these criteria, we found that approximately 20% of the sample had externalizing problems, 13% had internalizing problems, 10% had problems of dysregulation, and 31% had an extreme score on any of the ITSEA behavior problem domains (i.e., Externalizing, Internalizing, or Dysregulation). Seventeen percent of the sample had low scores on the Competence domain. These rates of behavior problems are largely comparable with those reported elsewhere for the ITSEA (Briggs-Gowan et al., 2006; Carter & Briggs-Gowan, 2006).

Intraclass Correlations

Twin intraclass correlations are presented in Table 2. For all ITSEA domains and subscales, twin correlations were high indicating substantial cotwin resemblances. Nonetheless, MZ twins are consistently more similar than DZ twins, suggesting genetic influences. With the exception of Activity/Impulsivity, Negative Emotionality, and Eating Problems, DZ correlations were considerably higher than one half the MZ correlation, indicating that shared environmental influences also contribute to variability in ITSEA domains and subscales. Although not high enough to be suggestive of substantial shared environmental influence, the large and significant DZ correlations for Activity/Impulsivity are particularly noteworthy. Previous research with parent ratings of hyperactivity and related behaviors typically find DZ correlations that are much less than one half the magnitude of the MZ correlations, suggesting rater contrast biases for parent-report measures (Eaves et al., 1997; Nadder, Silberg, Eaves, Maes, & Meyer, 1998; Price et al., 2001; Saudino et al., 2005; Thapar, Hervas, & McGuffin, 1995). In the present study, the pattern of twin correlations, combined with a pattern of no significant variance differences between zygosity groups, suggests that contrast biases were not operating.

There are also hints of possible sex differences in the etiology of ITSEA behaviors. The DZO correlations for Externalizing, Competence, Peer Aggression, and Prosocial Peer Relations were lower than those of same-sex DZ twins, suggesting potential qualitative genetic differences between boys and girls (i.e., genetic influences on behavior problems that are specific to one sex will reduce within-pair similarity). Similarly, for Peer Aggression, Depression/Withdrawal, and Inhibition to Novelty, the magnitude of MZ–DZ differences differed markedly across boys and girls, indicating possible quantitative genetic differences (i.e., differences in the magnitude of heritability across sex). Although suggestive, this examination of patterns of twin correlations does not take into consideration sample sizes and possible sex differences in variances, nor do they simultaneously analyze

all available information—for this reason, model-fitting analyses are needed to provide tests of sex differences.

Model-Fitting Analyses

Tables 3 and 4 present the fit indices and parameter estimates from the best fitting models for ITSEA domains and subscales, respectively. For some variables the best fitting models yielded significant chi-squares; however, with large samples such as ours significant chi-squares can result from trivial failures of the model (Neale et al., 2003). Under these circumstances, the root-mean-square error of approximation (RMSEA) statistic, which is relatively independent of sample size, provides a better indication of fit. RMSEA values below .10 indicate a good fit and those below .05 a very good fit (Neale et al., 2003). Thus, according to the RMSEA criterion all models fit the data well.

Variance components—Model-fitting analyses confirmed our impressions garnered from the intraclass correlations regarding genetic and environmental influences. All four domains and 20 subscales—indices displayed significant heritability. There were also substantial shared environmental influences operating on most of the domains and subscales. Although most ITSEA variables are moderately heritable, the magnitude of genetic effects varied considerably, explaining from 9% to 75% of the total variance, depending on the behavior assessed. Activity/Impulsivity, Negative Emotionality, and Eating Problems were the subscales that were most strongly influenced by genetic factors. For these three subscales, genetic influences accounted for approximately two thirds to three quarters of the total variance, with the remaining variance being largely due to nonshared environmental influences (i.e., shared environmental influences were trivial and nonsignificant). The least heritable variables were Prosocial Peer Relations and Maladaptive Behaviors, with genetic factors explaining no more than 15% of the total variance. These variables did, however, display prodigious shared environmental influences (80% to 86% of variance). Thus, for these two behaviors, familial resemblance is largely due to shared family environments rather than shared genes. Other ITSEA subscales for which shared environmental influences were consistently larger than genetic influences included Competence, Peer Aggression, Separation Distress, Compliance, Imitation/Play, Empathy, Social Relatedness, and the Atypical Index. For these variables, shared environmental variance explained approximately 50% to 75% of the phenotypic variance. Across all domains and subscales, nonshared environmental influences, which include error, were generally modest (4% to 34% of variance).

Sex differences—As indicated in Table 3, sex-limitation models suggested few differences in the etiology of the four ITSEA domains. The null model, which equates boys and girls for all parameters, was the best fitting model for both Internalizing and Dysregulation. For Externalizing, the scalar model provided the best fit to the data, indicating that there was a sex difference in phenotypic variance (boys higher than girls) but not in the proportion of variance that was accounted for by genetic and environmental factors. Competence was the only ITSEA domain to display a significant sex difference at the level of etiology. For this domain, the best fitting model was the full sex-limitation model including r_gO , which allows for quantitative and qualitative genetic differences

between boys and girls. The genetic correlation between DZO twins ($r_g = .35$) was significantly lower than that for DZ same-sex twins ($r_g = .50$). Boys and girls, however, did not significantly differ in the magnitude of genetic and environmental variance (see Table 5). Thus, the sex difference for Competence appears to be solely qualitative in nature and suggests that, to some extent, different genes may be operating across the sexes.

Qualitative sex differences were also indicated for two ITSEA subscales. The full r_gO sex-limitation model was the best fitting model for Peer Aggression. Similarly, the full sex-limitation model provided the best fit to the data for Prosocial Peer Relations; however, it was not possible to distinguish between the r_gO model and the r_cO model. Both models fit the data equally well: r_gO model, $\chi^2(8) = 2.10$, $p = .98$; r_cO model, $\chi^2(8) = 2.10$, $p = .98$. Thus, there may be qualitatively different genetic or shared environmental differences between the sexes for Prosocial Peer Relations. Follow-up tests on both Peer Aggression and Prosocial Peer Relations (see Table 5) indicated that in addition to qualitative sex differences, there were significant quantitative sex differences in variance components. Peer Aggression was more heritable in boys and more environmentally influenced in girls, whereas the reverse was true for Prosocial Peer Relations.

Other ITSEA subscales displayed only quantitative sex differences (i.e., the same genes and/or environments operate across sex, but the magnitude of genetic and/or environmental effects differed for boys and girls). For example, the Compliance and Empathy subscales displayed substantial environmental influences for both boys and girls; however, individual differences on both subscales were more heritable in boys and more strongly influenced by environmental (both shared and nonshared) factors in girls. In contrast, girls displayed a higher heritability and boys displayed higher shared and nonshared environmental influences for Attention Problems. Finally, there were significant sex differences in nonshared environmental influences in Depression/Withdrawal (boys lower than girls), Eating Problems (boys higher), and Sensory Sensitivity (boys higher). The remaining ITSEA scales displayed no significant sex differences in genetic or environmental etiology. There were, however, some differences in phenotypic variance for several scales, as indicated by the scalar model being the model that best described the data. In all cases, the variances for boys exceeded those for girls.

Discussion

Despite the fact that early appearing behavior problems have been shown to be stable and clinically significant (e.g., Briggs-Gowan et al., 2006; Campbell et al., 1994; Caspi et al., 1996), most research exploring genetic and environmental influences on behavior problems has focused on middle childhood and adolescence. Knowledge about the etiology of behavior problems in young children is largely based on data from a single research project (the NTR) using a single measure (the CBCL). While this research has provided valuable information regarding the factors that influence individual differences in behavior problems, it is critical to know whether the NTR findings replicate in a different population, using a different measure. The present study provides novel evidence of genetic and environmental influences on behavior problems and competencies in very early childhood. We discuss these results by behavior problem domain.

Externalizing and Internalizing

Consistent with prior research with older children, behaviors related to the broad domains of Externalizing and Internalizing were moderately heritable. On average, genetic factors explained approximately 50% of the variance in the ITSEA Externalizing and Internalizing domains and subscales. As predicted, shared environmental factors were also significant, accounting for roughly 30% of the variance in these domains and subscales. The remaining variance in externalizing and internalizing behavior problems in very young children can be attributed to nonshared environmental effects. The similarity of our findings with other studies of externalizing and internalizing behaviors in early childhood is remarkable. Our estimates of genetic and environmental variances components for the ITSEA Externalizing domain were comparable with those found for CBCL Externalizing for 3-year-old twins participating in the NTR (Bartels et al., 2004). Similarly, the heritability of the ITSEA Internalizing domain was close to that reported in the NTR; however, as with Schmitz et al. (1994), we found substantial shared environmental influences in our younger sample. Prior research exploring internalizing behaviors in older twins has found that heritability increases and shared environmental factors decrease with age (Eley & Stevenson, 1999; Rhee & Waldman, 2002; Rice et al., 2002; Scourfield et al., 2003). Our findings for Internalizing on the ITSEA with younger twins would be consistent with these prior conclusions.

ITSEA Externalizing and Internalizing subscales also yielded results that were consistent with previous findings on comparable measures. For example, our estimates of heritability and environmental variances for ITSEA Aggression/Defiance, Activity/Impulsivity, and Depression/Withdrawal subscales are similar to those reported for the CBCL Oppositional, Overactivity, and Withdrawn scales, respectively, in the NTR (Derks et al., 2004). As in previous studies with older children (e.g., Eaves et al., 1997; Kuntsi & Stevenson, 2001; Price et al., 2001; Saudino et al., 2005; Thapar et al., 1995), Overactivity was one of the subscales with the highest heritability, and comparable with previous studies of conduct disorder (Deater-Deckard & Plomin, 1999; Eaves et al., 1997; Eley, Lichtenstein & Stevenson, 1999), the heritability for Aggression/Defiance was one of the lowest, with a high proportion of variance accounted for by shared environmental influences. In addition, our findings for General Anxiety and Separation Distress on the ITSEA were similar to those for general distress and separation anxiety at 4 years of age in the TEDS sample (Eley et al., 2003). This consistency of results across measures and samples provides strong evidence for the role of genetic and environmental influences on externalizing and internalizing behaviors in early childhood.

There were, however, some differences between our findings and those reported in the literature. We found no sex difference in the etiology of individual differences on the Externalizing domain, whereas in the NTR, boys and girls differed in the magnitude of genetic and environmental influences on the CBCL Externalizing scale (Bartels et al., 2004). This difference could be a result of greater statistical power afforded by the NTR sample, which was approximately four times the size of our sample. It should be noted, though, that the NTR analyses provided an omnibus test of sex differences across four ages (3, 7, 10, and 12 years) simultaneously and did not look at sex effects within each age. Thus, it is possible that sex effects on externalizing behaviors emerge after early childhood. Indeed, earlier

analyses of the NTR data that were restricted to early childhood found no sex differences for Externalizing on the CBCL at age 3 (van den Oord et al., 1996). Moreover, in the more recent NTR analyses, the magnitude of genetic and environmental effects were very similar for boys and girls at age 3 (boys: $a^2 = .54$, $c^2 = .27$, $e^2 = .16$; girls: $a^2 = .50$, $c^2 = .32$, $e^2 = .18$). Thus, the sex effect, if present, appears to be small.

Similarly, although both our study and the NTR study find a sex effect in the etiology of depressive/withdrawn behaviors in early childhood, the source of this sex difference differs across samples. In the present study, nonshared environmental influences on the ITSEA Depression/Withdrawn subscale were higher for girls than boys (i.e., genetic and shared environmental variances did not differ). In contrast, in the NTR, genetic effects on the CBCL Withdrawn scale at age 3 were stronger for boys and shared environmental effects were stronger for girls (Derks et al., 2004). The NTR findings were based on ratings from both mothers and fathers, and it is possible that the additional source of information, along with the benefits of a larger sample (i.e., almost 10,000 twin pairs), allowed for the detection of sex differences. Nonetheless, research with older samples produces a confused picture regarding sex effects for the etiology of depression in middle childhood and adolescence, with several studies using parent ratings showing no effects (Eaves et al., 1997; Happonen et al., 2002; Rice et al., 2002), one study using teacher ratings showing greater heritability for girls (Happonen et al., 2002), and two studies using self-reports suggesting greater heritability for boys (Eley & Stevenson, 1999; Rice et al., 2002). It may be that sex effects in the etiology of depressive behaviors are dependent on sample characteristics, the rater, or the measure used to assess behaviors (Saudino et al., 2005).

Our findings for peer aggression also differ from NTR results for CBCL aggression at age 3. In the Dutch sample, aggressive behaviors were substantially heritable, explaining between 50% and 67% of the variance (Bartels et al., 2003; Hudziak et al., 2003; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). Father reports, but not mother reports, suggested that aggressive behaviors were more heritable for girls than for boys (Hudziak et al., 2003). In the present study, genetic factors on the ITSEA Peer Aggression subscale were modest and shared environmental variance substantial. Moreover, in comparing the relative importance of genetic and environmental influences, we found that genetic influences were stronger for boys, and environmental influences (shared and nonshared) were stronger for girls. There was also evidence of qualitative sex-specific effects suggesting that the genes that influence peer aggression differ for boys and girls. This difference in outcomes may result from differences in the types of behaviors that are assessed on the two aggression scales. Aggression on the CBCL is broadly defined to include disobedience, hostility, and overt aggression within social and nonsocial contexts. In contrast, the ITSEA Peer Aggression subscale is more focused on relational and overt aggression within a peer social context. Aggression that is directed against adults and defiance toward adults is captured on the Aggression/Defiance scale of the ITSEA (discussed above). It has been suggested that measures that are more specific may be more sensitive to detecting sex differences (Vierikko, Pulkkinen, Kaprio, Viken, & Rose, 2003). Indeed, Vierikko et al. (2003) also found sex-specific effects for teacher ratings of behavior problems related to social aggression in early adolescence, but they found that girls had higher heritabilities than boys

on both teacher and parent measures. A cautious interpretation of the data is that the etiology of peer aggression differs across sex and, possibly, across age.

Dysregulation

The ITSEA provides a unique opportunity to explore the factors that influence individual differences in dysregulation in early childhood. Because of its association with child temperament, which is assumed to have a constitutional foundation (Goldsmith et al., 1987), we expected that dysregulation would be genetically influenced. This was true for the overall domain score as well as for each of the subscales. With two exceptions, Negative Emotionality and Eating Problems, there were also substantial shared environmental influences on dysregulation behaviors. The lack of shared environmental influences on the ITSEA Negative Emotionality subscale is not surprising given that this is a finding that frequently emerges in the temperament literature (e.g., Saudino & Cherny, 2001; Saudino, McGuire, Reiss, Hetherington, & Plomin, 1995; Spinath & Angleitner, 1998) and the ITSEA Negative Emotionality subscale is conceptualized as a measure of temperament rather than a problem behavior scale (Carter & Briggs-Gowan, 2006).

To our knowledge, this is the first study to examine genetic influences on eating problems in young children; consequently, it is difficult to put these findings into context. A small study of twin temperament found that shared family environments, not genes, influenced children's reactivity to food (Plomin & Rowe, 1977). Reactivity to food is clearly a component of the ITSEA Eating Problem scale; it also has a moderate association with Sensory Sensitivities, another scale in the Dysregulation domain that displayed moderate genetic and shared environmental effects. Two other studies suggest that genetic, shared, and nonshared environmental factors are important to children's eating behaviors, but these studies did not address eating problems (Falciglia, & Norton, 1994; Keski-Rahkonen, Viken, Kaprio, Rissanen, & Rose, 2004). Nonetheless, our finding of substantial genetic influence and little shared environmental influence on young children's eating problems is consistent with research exploring the etiology of eating disorders in adolescents and adults (see Bulik, Sullivan, Wade, & Kendler, 2000; Gorwood, Kipman, & Foulon, 2003).

Behavioral Competencies

The etiology of behavioral competencies in very early childhood differs considerably from that of behavior problems. Variability in the overall ITSEA Competence domain score and most of the competence subscales was largely due to shared environments. Genetic influences contributed only modestly to individual differences in behavioral competencies. Similar results have emerged from studies of early prosocial behavior (Knafo & Plomin, 2006b; Zahn-Waxler et al., 2001); however, the present study extends these findings by demonstrating that shared environmental influences are important across a range of early socioemotional competencies.

There is an abundance of phenotypic research that shows that parenting practices are associated with compliance, empathy, and prosocial behaviors (e.g., Braungart-Rieker, Murphy Garwood, & Stifter, 1997; Deater-Deckard et al., 2001; Eisenberg & Fabes, 1998; Krevans & Gibbs, 1996; Whiteside-Mansell et al., 2003). Our findings of prodigious shared

environmental influences on behavioral competencies would be consistent with parenting research in early childhood and suggest that there are family-wide effects that influence the development of behavioral competencies and thus make young siblings similar within this behavioral domain. Recent research by Knafo and Plomin (2006a) sheds some light on the types of family effects that influence early prosocial behavior. Multivariate genetic analyses looking at genetic and environmental links between parent discipline/affection and child prosocial behavior found that the association between parent positivity (positive discipline and affection) and prosocial behavior at age 3 was almost entirely due to common shared environmental influences. Interestingly, the association between parent negativity and prosocial behavior was genetically mediated. Therefore, parental positivity, but not negativity, is a family-wide environment that makes siblings similar in prosocial behavior (Knafo & Plomin, 2006a). However, this effect appears to be salient only in early childhood as Knafo and others (Scourfield et al., 2004) have found that shared environmental influences on prosocial behavior decrease with age, perhaps as a result of the child's increasing interactions with others outside the family. Nonetheless, the present results highlight the need for more multivariate behavior genetic research to explore genetic and environmental contributions to the association between parenting and other aspects of behavioral competencies in early childhood.

Compared with other ITSEA domains and subscales, behavior competencies also tend to show more sex differences at the level of etiology. The finding of qualitative sex effects for the Competence domain and for the Prosocial Peer Relations subscale suggests that to some extent, the specific genes or types of environments that influence these behaviors may differ for boys and girls. This is consistent with evidence that child sex moderates the association between parenting styles and the emergence of self-regulatory skills (Carter, Mayes, & Pajer, 1990; Weinberg, Tronick, Cohn, & Olson, 1999). Although intriguing, these results should be viewed cautiously as qualitative sex differences did not emerge in other large-scale twin studies that have tested for such effects (Knafo & Plomin, 2006b; Scourfield et al., 2004).

Sex differences in the magnitude of genetic and environmental effects (i.e., quantitative sex differences) were evident for Compliance, Attention, Empathy, and Prosocial Peer Relations. For Compliance and Empathy, heritabilities were higher for boys, and environmental influences were higher for girls. A possible explanation for this finding may be that girls experience greater socialization pressures to be "nice young ladies" who are compliant, empathic, and nurturing (Maccoby, 1998). This could also explain why a similar pattern of sex differences appeared on the Peer Aggression subscale. Interestingly, in contrast to prior twin studies of prosocial behavior that have found higher heritabilities for boys and higher shared environmental influences for girls (Knafo & Plomin, 2006b; Saudino et al., 2005), the Prosocial Peer Relations subscale did not display this pattern of sex effects. In fact, although the effects were very small, the heritability for this competence subscale was slightly higher for girls; however, as indicated above, the sex-limitation model did indicate that different environments may be operating across sex.

Girls also displayed a higher heritability than boys for the Attention subscale. This is somewhat interesting as the inattentive subtype of attention-deficit/hyperactivity disorder is

the subtype that is most frequently observed in girls (Weiler, Bellinger, Marmor, Rancier, & Waber, 1999). It should be noted, however, that the finding of sex differences in the magnitude of genetic effects for a given behavior problem or competency does not necessarily mean that genetic factors explain sex differences in prevalence rates (Rutter, Caspi, & Moffitt, 2003).

Limitations

In the present study, behavior problems in early childhood were primarily influenced by genetic factors, although shared environments, and to a lesser extent, nonshared environments, also contributed significantly to variability in behavior problems. Behavioral competencies, on the other hand, displayed considerably smaller genetic effects. Individual differences in competencies were largely due to shared environmental influences. The finding that behavior problems and competencies in early childhood are influenced by genetic factors likely comes as no surprise given the abundance of research that finds significant heritabilities for individual differences in a wide range of normal and abnormal behaviors across the lifespan. However, the presence of substantial shared environmental influences is noteworthy. In general, behavioral genetics research suggests that the importance of shared environmental influences on many behaviors and traits is typically modest. The types of environmental influences that are often most relevant in behavioral genetics studies are of the nonshared variety (Plomin et al., 2001). This was not the case for most ITSEA variables in the present study.

Although our finding of substantial shared environmental influences is intriguing, it should be noted that twin studies indirectly assess genetic and environmental influences on the basis of the resemblances of twin siblings. Such designs do not identify specific genes that influence behaviors, nor do they inform about specific environmental influences (i.e., beyond identifying “shared” and “nonshared” environments). Moreover, there are possible methodological explanations for our finding of substantial shared environmental influences. As with most large-scale twin research, we used a parent-report questionnaire to determine whether twins were MZ or DZ. Although previous research has found such questionnaire methods to be valid and highly accurate (Price et al., 2000), errors in identifying the zygosity of twins would inflate estimates of shared environmental variance. That is, misclassification twins would artificially lower MZ twin correlations and increase DZ twin correlations, thereby reducing the differences between MZ and DZ resemblances and inflating shared environmental variance. However, in the present study, the twin correlations for same-sex twins identified as DZ by questionnaire are very similar in magnitude to those for opposite-sex twins (who have to be DZ), suggesting that zygosity was correctly identified.

Rater bias may also explain shared environmental variance. When both members of a twin pair are assessed by the same rater, rater biases act to inflate estimates of shared environmental variance. That is, there is covariance between the biases that affect the ratings of each twin such that the rater tends to consistently overestimate or underestimate the behavior of both cotwins (Saudino, 2005b). This consistency across cotwins would act to inflate the similarity of both MZ and DZ twins. Our reliance on a single rater (usually the

mother) leaves open the possibility of undetected rater bias. However, in the NTR, multivariate analyses of mother and father ratings of behavior problems on the CBCL have found that rater bias models do not provide the best fit to the data (Derks et al., 2004; van der Valk et al., 2001). Moreover, as discussed above, our findings of significant shared environmental variance are not discrepant from previous research using multiple raters (e.g., Derks et al., 2004; van den Oord, Boomsma, & Verhulst, 2000). In addition, the impact of shared environmental effects varied in predictable ways across domains (i.e., behavioral competencies > behavioral problems) and subscales. For example, the two ITSEA subscales that are most related to specific temperament dimensions, Negative Emotionality and Activity/Impulsivity, showed no significant shared environmental influence—a finding that is ubiquitous in the temperament literature (Saudino, 2005a). The variable importance of shared environmental influences across behaviors within the study coupled with the consistency of our findings with other research suggests that rater bias may not be a problem and that shared environments are an important source of variability in young children's behavioral problems and competencies. Nonetheless, replication with multiple raters would strengthen conclusions about shared environmental influences. If, as we suspect, the finding of shared environmental influences is not merely methodological, then the inclusion of measured environments (e.g., parenting, socioeconomic status) would be an important next step in the study of genetic and environmental influences on early behavior problems.

A final limitation has to do with the representativeness of our sample, which was recruited via volunteer membership in a parents-of-multiples organization. As is usually the case with developmental research, the sample was predominantly middle class. This limitation raises the question of the generalizability of results. Replication with a more diverse sample is needed. However, as indicated earlier, our ITSEA findings showed considerable consistency with prior research based on samples recruited using birth registries—both in terms of phenotypic analyses of the ITSEA (e.g., Carter et al., 2003) and in terms of behavioral genetic analyses of early behavior problems assessed with different measures (e.g., Bartels et al., 2004; Derks et al., 2004; Knafo & Plomin, 2006b). Thus, the effects studied appear to be robust to the limitations posed by having a convenience sample.

Clinical Implications

Although it may not be fully appropriate in clinical use to impose cut points derived from the U.S. standardization sample in this French sample of twins, it is striking that for the most part, the percentages of children in the extremes were comparable with those observed in the U.S. standardization sample (Carter & Briggs-Gowan, 2006). Consistent with U.S. findings, approximately one third of our sample had an extreme score indicating they were at risk for clinically relevant behavior problems on at least one of the ITSEA problem domains. Our French sample displayed a slightly higher incidence of externalizing problems (20% vs. 10%) and lower competencies (17% vs. 10%). The higher percentage of low competence scores may be attributed to the higher rate of language delay expected in twin samples (Dale et al., 1998) as language delay is associated with lower competence on the ITSEA (Carter & Briggs-Gowan, 2006; Irwin, Carter, & Briggs-Gowan, 2002). The lower competence and higher externalizing scores may also reflect cultural differences in rating children's behaviors. It is especially noteworthy, however, that despite these modest variations in the

incidences of extreme scores for these two behavioral domains, the etiology of individual differences in these domains is consistent with prior research conducted in the United States, the United Kingdom, and the Netherlands.

The varied patterns of genetic and environmental influence across domains and scales of the ITSEA lend further support to the argument that parents can provide differentiated views of their very young children's socioemotional and problem behaviors. Although for some problem scales genetic influence is quite high, environmental influence is always present. Given previous work documenting that early emerging problems are stable (e.g., Briggs-Gowan et al., 2006), early detection and intervention efforts are warranted. Indeed, the fact that early behavior problems show significant and substantial shared environmental influences, whereas such environmental influences are much more modest (and sometimes negligible) in later childhood, suggests that family-wide interventions may be most effective at earlier ages.

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Figure 1.

The sex-limitation model. The latent variables A, C, and E refer to additive genetic variance, shared environmental variance, and nonshared environmental variance, respectively. The path coefficients a , c , and e indicate the relative influence of the latent variables on the phenotype. The m and f subscripts for the path coefficients refer to effects for males and females, respectively. r_g and r_c are the genetic and shared environmental correlations between same-sex twins, respectively. r_gO and r_cO are the genetic and shared environmental correlations between opposite-sex twins, respectively.

Table 1

Infant-Toddler Social and Emotional Assessment Means (With Standard Deviations in Parentheses) by Sex and Zygosity

Domain or subscale	Boys		Girls		Main effects		
	MZ	DZ	MZ	DZ	Sex	Zygosity	Age
Domain							
Externalizing	13.5 (7.2)	12.9 (6.7)	11.6(6.4)	11.5 (6.0)	***		***
Internalizing	16.1 (7.0)	16.5 (6.8)	17.1 (7.2)	16.2 (6.5)			***
Dysregulation	16.3 (8.0)	16.5 (8.0)	16.2 (8.0)	15.7 (7.7)	*		***
Competence	44.0 (12.3)	43.4 (11.9)	44.8 (12.2)	46.1 (12.0)	***		***
Subscale							
Externalizing							
Activity Impulsivity	4.7 (2.8)	4.3 (2.8)	3.9 (2.6)	3.7 (2.6)	***		*
Aggression/Defiance	7.3 (4.3)	6.9 (3.8)	6.2 (3.5)	6.4 (3.4)	***		***
Peer Aggression ^a	1.6 (2.2)	1.6(2.2)	1.5 (2.1)	1.4(2.0)	*		***
Internalizing							
Depression Withdrawal ^a	0.6(1.1)	0.8 (1.5)	0.7 (1.3)	0.6 (1.1)	*		**
General Anxiety ^a	4.4 (3.6)	4.6 (3.5)	4.8 (4.1)	4.4 (3.3)			***
Separation Distress	6.6 (2.9)	6.7 (2.9)	6.9 (2.8)	6.6 (2.8)			
Inhibition to Novelty	4.5 (2.6)	4.3 (2.3)	4.7 (2.4)	4.5 (2.4)	**		***
Dysregulation							
Sleep Problems	2.1 (2.0)	2.2 (2.1)	2.1 (2.0)	2.1 (2.0)	*		
Negative Emotionality	8.7 (4.8)	9.0 (5.0)	8.1 (4.6)	8.0 (4.6)	***		***
Eating ^a	2.3 (2.4)	2.2 (2.4)	2.5 (2.5)	2.5 (2.5)	**		
Sensory Sensitivity	3.1 (2.2)	3.1 (2.2)	3.5 (2.5)	3.2 (2.2)			***
Competence							
Compliance	9.6 (3.1)	9.7 (3.1)	10.0(3.1)	10.3 (3.0)	***		***
Attention	6.9 (2.3)	6.7 (2.3)	7.1 (2.1)	7.3 (2.2)	***		***
Imitation/Play	8.8 (2.5)	8.6 (2.4)	9.1 (2.3)	9.1 (2.2)			***
Mastery Motivation	8.3 (2.5)	8.3 (2.3)	8.4 (2.4)	8.4 (2.3)			***
Empathy	6.9 (3.7)	6.5 (3.7)	6.9 (3.9)	7.0 (3.8)	***		***
Prosocial Peer Relations	3.4 (2.8)	3.4 (2.8)	3.4 (2.8)	3.9 (3.0)	***		***
Problem Indices							
Maladaptive Behaviors ^a	2.0 (2.4)	2.0 (2.6)	2.1 (3.1)	1.8 (2.2)			
Social Relatedness	16.2 (2.7)	16.2 (2.6)	16.8 (2.4)	16.5 (2.5)	**		**
Atypical Index ^a	1.0(1.5)	1.0 (1.5)	0.9 (1.7)	0.9 (1.4)			
No. of individuals	408	1,579	378	1,535			

Note. Means are for untransformed data. MZ = monozygotic; DZ = dizygotic.

^a Generalized estimating equations analyses were performed on log-transformed data.

* $p < .05$.

**
 $p < .01$.

 $p < .001$.

Table 2

Twin Intraclass Correlations (With 95% Confidence Intervals in Parentheses)

	Same-sex boys		Same-sex girls		
Domain or subscale	MZ	DZ	MZ	DZ	Opposite-sex DZ
Domain					
Externalizing	.81 (.76–.85)	.56 (.49–.62)	.83 (.78–.87)	.64 (.59–.70)	.50 (.44–.55)
Internalizing	.79 (.73–.83)	.48 (.41–.55)	.81 (.76–.86)	.51 (.43–.58)	.56 (.51–.61)
Dysregulation	.79 (.74–.84)	.50 (.42–.57)	.82 (.77–.86)	.55 (.47–.61)	.52 (.47–.57)
Competence	.94 (.92–.95)	.84 (.81–.87)	.93 (.91–.95)	.85 (.82–.88)	.79 (.76–.81)
Subscale					
Externalizing					
Activity Impulsivity	.78 (.72–.83)	.41 (.32–.48)	.79 (.72–.83)	.48 (.40–.55)	.37 (.30–.43)
Aggression/Defiance	.82 (.77–.86)	.56 (.49–.62)	.81 (.76–.86)	.60 (.53–.66)	.53 (.48–.58)
Peer Aggression	.90 (.88–.93)	.72 (.67–.76)	.84 (.79–.88)	.77 (.72–.81)	.64 (.60–.68)
Internalizing					
Depression Withdrawal	.70 (.63–.77)	.60 (.53–.65)	.76 (.69–.81)	.43 (.35–.51)	.57 (.51–.61)
General Anxiety	.75 (.69–.81)	.52 (.45–.59)	.78 (.71–.83)	.45 (.37–.53)	.53 (.48–.58)
Separation Distress	.84 (.80–.88)	.64 (.58–.69)	.86 (.81–.89)	.68 (.63–.73)	.67 (.63–.71)
Inhibition to Novelty	.78 (.72–.8)	.37 (.28–.45)	.76 (.69–.81)	.51 (.43–.58)	.45 (.39–.51)
Dysregulation					
Sleep Problems	.84 (.79–.87)	.52 (.45–.59)	.80 (.75–.85)	.57 (.50–.64)	.59 (.54–.63)
Negative Emotionality	.68 (.60–.75)	.37 (.29–.45)	.75 (.68–.81)	.45 (.37–.53)	.39 (.33–.45)
Eating	.69 (.61–.75)	.27 (.18–.36)	.76 (.69–.81)	.34 (.25–.43)	.28 (.21–.34)
Sensory Sensitivity	.77 (.71–.82)	.54 (.47–.60)	.87 (.83–.90)	.65 (.59–.70)	.61 (.57–.66)
Competence					
Compliance	.93 (.91–.94)	.77 (.72–.80)	.90 (.87–.92)	.80 (.76–.84)	.75 (.72–.78)
Attention	.77 (.71–.82)	.60 (.53–.66)	.82 (.77–.86)	.52 (.44–.59)	.52 (.47–.57)
Imitation/Play	.93 (.91–.95)	.82 (.79–.85)	.94 (.92–.95)	.82 (.79–.85)	.78 (.75–.81)
Mastery Motivation	.84 (.79–.87)	.59 (.52–.65)	.78 (.72–.83)	.54 (.46–.61)	.59 (.54–.64)
Empathy	.94 (.93–.96)	.82 (.79–.85)	.91 (.89–.94)	.85 (.82–.88)	.80 (.78–.83)
Prosocial Peer Relations	.95 (.93–.96)	.90 (.88–.92)	.95 (.94–.96)	.88 (.86–.90)	.86 (.84–.87)
Problem Indices					
Maladaptive Behaviors	.89 (.86–.92)	.83 (.80–.86)	.89 (.86–.92)	.79 (.75–.82)	.78 (.75–.81)
Social Relatedness	.82 (.77–.86)	.66 (.60–.71)	.82 (.76–.86)	.70 (.64–.74)	.62 (.57–.66)
Atypical Index	.90 (.87–.92)	.74 (.70–.78)	.91 (.89–.93)	.71 (.66–.76)	.73 (.70–.77)
No. of pairs of twins	204	412	189	390	755

Note. All correlations are significant at $p < .05$. MZ = monozygotic; DZ = dizygotic.

Table 3

ITSEA Domains: Estimates of Genetic and Environmental Variance (With 95% Confidence Intervals in Parentheses) and Fit Indices From Best Fitting Model

Domain	Best fitting model	χ^2	df	p	AIC	RMSEA	a^2	c^2	e^2	r_gO	Var _{boys}	Var _{girls}
Externalizing	Scalar	22.62	11	.02	0.62	.026	.46 (.38-.54)	.34 (.27-.41)	.20 (.17-.23)		44.67	33.90
Internalizing	Null	16.99	12	.15	-7.01	.009	.50 (.41-.58)	.29 (.21-.36)	.22 (.09-.25)		42.64	42.64
Dysregulation	Null	14.93	12	.25	-9.07	.011	.56 (.48-.65)	.25 (.17-.32)	.19 (.17-.22)		60.40	60.40
Competence	Full r_gO	11.26	8	.19	-4.74	.004						
Boys							.21 (.16-.27)	.73 (.67-.77)	.06 (.05-.08)	.35 (.17-.49)	98.12	92.07
Girls							.15 (.10-.20)	.78 (.73-.82)	.07 (.06-.09)	.35 (.17-.49)	98.12	92.07

Note. ITSEA = Infant-Toddler Social and Emotional Assessment; a^2 = genetic variance; c^2 = shared environmental variance; e^2 = nonshared environmental variance; r_gO = genetic correlation between opposite-sex twins; AIC = Akaike's information criterion; RMSEA = root-mean-square error of approximation; Var = variance.

Table 4

ITSEA Subscales and Indices: Estimates of Genetic and Environmental Variance (With 95% Confidence Intervals in Parentheses) and Fit Indices from Best Fitting Model

Subscale	Best fitting model	χ^2	df	p	AIC	RMSEA	a^2	c^2	e^2	$r_g O$	Var _{boys}	Var _{girls}
Activity/Impulsivity	Scalar	10.93	11	.50	-11.71	.005	.75 (.65-.82)	.04 (.00-.13)	.21 (.18-.24)		8.11	6.74
Aggression/Defiance	Scalar	14.59	11	.20	-7.41	.014	.45 (.37-.53)	.34 (.27-.41)	.21 (.18-.24)		14.18	11.68
Peer Aggression	Full $r_g O$	6.75	8	.56	-9.25	.000						
Boys							.35 ^b (.26-.44)	.56 ^b (.50-.63)	.10 ^b (.08-.12)	-.18 (-.39-.12)	.007	.006
Girls							.16 (.06-.25)	.68 (.60-.75)	.16 (.13-.20)	-.18 (-.39-.12)	.007	.006
Depression/Withdrawal	Common	66.12	9	.00	48.12	.087						
Boys						.39 (.27-.50)	.40 (.30-.49)	.21 ^b (.17-.26)			.003	.002
Girls							.41 (.29-.53)	.29 (.21-.38)	.29 (.24-.36)		.003	.002
General Anxiety	Scalar	19.88	11	.05	-2.12	.032	.45 (.35-.55)	.29 (.21-.37)	.28 (.22-.30)		.019	.017
Separation Distress	Scalar	4.95	11	.93	-17.05	.000	.36 (.30-.43)	.48 (.42-.54)	.15 (.13-.18)		8.39	7.77
Inhibition to Novelty	Null	14.17	12	.29	-9.83	.018	.63 (.53-.73)	.13 (.05-.22)	.24 (.21-.27)		5.32	5.32
Sleep Problems	Null	12.51	12	.41	-11.49	.015	.54 (.46-.61)	.30 (.23-.36)	.17 (.14-.19)		4.13	4.13
Negative Emotionality	Scalar	13.95	11	.24	-8.05	.012	.65 (.54-.76)	.08 (.00-.17)	.26 (.23-.31)		24.82	20.17
Eating Problems	Common	18.10	9	.03	0.10	.019						
Boys							.64 (.52-.71)	.02 (.00-.11)	.34 ^b (.28-.42)		.009	.010
Girls							.71 (.57-.78)	.05 (.00-.16)	.25 (.20-.31)		.009	.010
Sensory Sensitivity	Common	9.98	9	.35	-8.02	.012						
Boys							.42 (.30-.53)	.35 (.26-.45)	.22 ^b (.18-.27)		4.79	4.88

Subscale	Best fitting model	χ^2	df	p	AIC	RMSEA	a^2	c^2	e^2	r_gO	Var _{boys}	Var _{girls}
Girls							.38 (.27-.49)	.47 (.37-.56)	.15 (.12-.19)		4.79	4.88
Compliance	Common	4.74	9	.86	-13.26	.000						
Boys							.32 ^b (.27-.38)	.61 ^b (.55-.66)	.07 ^b (.06-.09)		8.20	7.98
Girls							.15 (.08-.23)	.73 (.66-.79)	.11 (.09-.14)		8.20	7.98
Attention	Common	3.19	9	.96	-14.82	.000						
Boys							.37 ^b (.23-.51)	.41 ^b (.28-.52)	.23 ^b (.19-.28)		4.37	3.90
Girls							.63 (.53-.72)	.20 (.13-.29)	.16 (.13-.20)		4.37	3.90
Imitation/Play	Scalar	15.29	11	.17	-6.71	.016	.24 (.20-.27)	.69 (.66-.72)	.07 (.06-.08)		5.06	4.40
Mastery Motivation	Null	8.73	12	.73	15.26	.000	.44 (.36-.52)	.37 (.30-.43)	.20 (.17-.23)		5.05	5.05
Empathy	Common	8.09	9	.53	-9.91	.000						
Boys							.25 ^b (.21-.30)	.69 ^b (.64-.73)	.06 ^b (.05-.07)		10.39	9.65
Girls							.11 (.05-.17)	.80 (.74-.84)	.09 (.08-.12)		10.39	9.65
Prosocial Peer Relations	Full r_gOa	2.10	8	.98	13.90	.000					6.04	6.18
Boys							.09 ^b (.06-.13)	.86 ^b (.83-.89)	.05 (.04-.06)	.26 ^a (.05-.43)	6.04	6.18
Girls							.15 (.12-.20)	.80 (.76-.84)	.04 (.04-.06)	.26 ^a (.05-.43)	6.04	6.18
Maladaptive Behaviors	Null	31.23	12	.002	7.23	.046	.15 (.10-.19)	.73 (.70-.77)	.12 (.11-.14)		.008	.008
Social Relatedness	Scalar	26.46	11	.006	2.32	.036	.33 (.26-.40)	.49 (.43-.54)	.18 (.16-.21)		6.93	6.10
Atypical Index	Scalar	16.91	11	.11	-5.09	.019	.31 (.26-.36)	.59 (.54-.63)	.11 (.09-.12)		.0032	.0029

Note. ITSEA = Infant-Toddler Social and Emotional Assessment; a^2 = genetic variance; c^2 = shared environmental variance; e^2 = nonshared environmental variance; r_gO = genetic correlation between opposite-sex twins; AIC = Akaike's information criterion; RMSEA = root-mean-square error of approximation; Var = variance.

^aThe full r_cO model fit equally as well. Fit statistics and parameter estimates for a^2 , c^2 , and e^2 are the same as for the full r_gO model; r_cO , the shared environment correlation between opposite-sex twins, was estimated as .97 (confidence interval = .94-.99).

b_j Parameter estimate significantly differs across sex ($p < .05$).

Table 5

Chi-Square Tests of Specific Sex Differences in Variance Components

Domain or subscale	Base model	Model 1 equate a^2	Model 2 equate c^2	Model 3 equate e^2
Competence				
χ^2 (df)	11.26 (8) ^a	14.22 (9)	13.70 (9)	12.54 (9)
χ^2 (df = 1)		2.96	2.44	1.28
Peer Aggression				
χ^2 (df)	6.75 (8) ^a	15.62 (9)	11.60 (9)	18.85 (9)
χ^2 (df = 1)		8.88 ^{**}	4.86 [*]	12.11 ^{**}
Depression/Withdrawal				
χ^2 (df)	66.12 (9) ^b	66.25 (10)	69.27 (10)	71.60 (10)
χ^2 (df = 1)		0.12	3.15	5.48 [*]
Eating Problems				
χ^2 (df)	18.10 (9) ^b	18.74 (10)	21.13 (10)	22.35 (10)
χ^2 (df = 1)		0.63	3.02	4.25 [*]
Sensory Sensitivity				
χ^2 (df)	9.98 (9) ^b	10.24 (10)	12.15 (10)	16.99 (10)
χ^2 (df = 1)		0.26	2.17	7.01 ^{**}
Compliance				
χ^2 (df)	4.74 (9) ^b	14.36 (10)	11.53 (10)	14.01 (10)
χ^2 (df = 1)		9.61 ^{**}	6.78 ^{**}	9.27 ^{**}
Attention				
χ^2 (df)	3.19 (9) ^b	10.94 (10)	9.06 (10)	8.03 (10)
χ^2 (df = 1)		7.76 ^{**}	5.88 [*]	4.84 [*]
Empathy				
χ^2 (df)	8.09 (9) ^b	19.33 (10)	16.23 (10)	17.93 (10)
χ^2 (df = 1)		11.24 ^{**}	8.14 ^{**}	9.84 ^{**}
Prosocial Peer Relations				
χ^2 (df)	2.10 (8) ^a	7.93 (9)	7.99 (9)	2.82 (9)
$\Delta\chi^2$ (df = 1)		5.83 [*]	5.89 [*]	.72

Note. Base model refers to the best fitting model from Tables 3 and 4. Models 1, 2, and 3 refer to submodels of the base model where either the genetic (a^2), shared environmental (c^2), or nonshared environmental (e^2) variances were equated across sex. A significant chi-square difference between the base model and the submodel indicates that boys and girls could not be equated for the parameter being tested.

^a Base model was full r_{gO} (genetic correlation between opposite-sex twins) model.

^b Base model was common effects model.

* $p < .05$.

** $p < .01$.