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Exploring the Relationship Between Autistic-Like Traits and ADHD Behaviors in Early Childhood: Findings from a Community Twin Study of 2-Year-Olds

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Abstract

Behaviors characteristic of autism and ADHD emerge in early childhood, yet research investigating their comorbidity has focused on older children. This study aimed to explore the nature of the relationship between autistic-like traits and ADHD behaviors in a community sample of 2-year-olds. Twins from the Boston University Twin Project (*N*=312 pairs) were assessed by their parents on autistic-like traits and ADHD behaviors using the Childhood Behavior Checklist. Phenotypic analyses showed that after controlling for general cognitive ability and socioeconomic status, autistic-like traits (total scale as well as social and nonsocial subscales) correlated positively with ADHD behaviors (*r*=0.23–0.26). Structural equation model-fitting analyses revealed that there were modest shared genetic influences between ADHD- and autistic traits (genetic correlation = 0.27) as well as some common environmental influences explaining their covariation. Implications for identifying shared biological pathways underlying autistic-like traits and ADHD behaviors are discussed.

Keywords

Autistic-like traits; ADHD; Twins; Genetics; Autism

Autistic spectrum disorders (ASD) and attention deficit hyperactivity disorder (ADHD) are both neurodevelopmental disorders that begin in early childhood. Although their core diagnostic symptoms do not explicitly overlap, high levels of comorbidity between ASD and ADHD have been reported. Rates of ADHD in samples of individuals with autism and ASD

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have been reported as between 28% and 78% (de Bruin et al. 2007; Gadow et al. 2005; Ghaziuddin et al. 1998; Goldstein and Schwebach 2004; Hattori et al. 2006; Lee and Ousley 2006; Leyfer et al. 2006; Simonoff et al. 2008; Sinzig et al. 2009; Yoshida and Uchiyama 2004). For example, the Special Needs and Autism Project, the only population-derived sample used to address this question with structured assessments, found that 28% of their sample with ASD also had ADHD (Simonoff et al. 2008). With one exception (a study of 3–5-year-olds; Gadow et al. 2004) all of these studies have employed samples of children in middle to late childhood.

Elevated rates of autistic symptoms have been observed in samples of children with ADHD. Overall, many studies of children in middle or late childhood have shown that a substantial proportion of individuals with ADHD show severe autistic symptoms (Clark et al. 1999; Hattori et al. 2006; Mulligan et al. 2009a, b; Nijmeijer et al. 2009; Reiersen et al. 2007; Rommelse et al. 2009; Santosh and Mijovic 2004). For example, in one recent large study of combined type ADHD, that excluded cases with comorbid ASD, 7% of children with ADHD had a high level of sub-clinical autistic symptoms while another 59% show modest autistic-like difficulties (Mulligan et al. 2009a).

Behaviors characteristic of ASD and ADHD both show quantitative variation in the general population (e.g., Constantino and Todd 2000; Mulligan et al. 2009b). Across three studies with large samples (>400 individuals), strong correlations between autistic-like traits and ADHD-behaviors in middle childhood and early adulthood have been reported, ranging from 0.48 to 0.57, and this finding has been reported using several different measures and using parent, teacher and self report (Constantino et al. 2003; Reiersen et al. 2008; Ronald et al. 2008). In sum, evidence shows that ASD and ADHD show high rates of comorbidity in clinical samples and that their trait behaviors covary strongly in general population samples.

Both autism and ADHD are known to be highly heritable conditions (e.g., Bailey et al. 1995; Levy et al. 1997). Furthermore, individual differences in autistic-like traits in middle childhood or adolescence are highly heritable in the general population and show modest nonshared environmental influence (Constantino and Todd 2000, 2003, 2005; Ronald et al. 2005; Ronald et al. 2006; Hoekstra et al. 2007; Skuse et al. 2005). Similarly, ADHD behaviors in the general population are also highly heritable and show none or modest shared environmental influences, both in early childhood (e.g. Price et al. 2001) and later childhood (e.g. McLoughlin et al. 2007; Saudino et al. 2005). The first twin study of autistic-like traits in early childhood studied 2-year-old twins and reported modest heritability (39%) and modest shared and nonshared environmental influences (21% and 40%, respectively) (Edelson and Saudino 2009).

Evidence concerning the causes of the association between autistic-like traits and ADHD behaviors comes from family, twin and molecular genetic studies. In a recent large family study of 821 ADHD probands and 1,050 siblings aged 5–17 from the International Multi-Centre ADHD Genetics (IMAGE) study, it was found that autism symptoms showed shared familial influences with ADHD, with 56% of the phenotypic correlation between ADHD and autistic symptoms explained by shared familial influences (Mulligan et al. 2009a). A second somewhat smaller family study, again from the IMAGE project, of 256 sibling pairs

aged 5–19 years, found low and nonsignificant cross-correlations between proband PDD symptoms and sibling ADHD symptoms (r=−0.01–0.10) (Nijmeijer et al. 2009). The authors concluded that familiality of PDD symptoms in the context of ADHD was largely independent from familiality of ADHD symptoms. The contrasting findings of these two family studies could be due to the smaller sample size employed in the latter study or the different measure of PDD symptoms, but these differences need to be resolved with further research on other samples.

Findings from twin studies have concurred with the findings from the first family study by Mulligan and colleagues in suggesting that there are overlapping causal influences on PDD and ADHD symptoms. First, the Missouri twin study has reported, using an epidemiological sample of 219 male twin pairs aged 7–15 years, that variation in attention problems explained a significant proportion of variation in autistic traits (Constantino et al. 2003). A second twin study in a UK general population sample of >6,000 male and female 8-year-old twins reported substantial genetic overlap between autistic-like traits and ADHD symptoms and this was found both for the whole sample and in extreme-scoring groups, and using both parent and teacher reports (Ronald et al. 2008). Finally, a recent twin study of 674 adult Australian twins who provided self reports of their autistic traits and inattentive and impulsive symptoms reported substantial genetic overlap between these two measures (Reiersen et al. 2008). These two latter, more recent twin studies reported genetic correlations (which estimate the degree of genetic overlap between two variables) in middle childhood and young adults as 0.54–0.57 (depending on sex and rater) and 0.72, respectively (Reiersen et al. 2008; Ronald et al. 2008). Therefore, with the exception of one family study on an ADHD sample (Nijmeijer et al. 2009), across family and twin studies, and across different types of raters (parent, teacher and self report), findings suggest that significant genetic overlap exists across autistic-like traits and ADHD behaviors in middle childhood and early adulthood.

Initial molecular genetic findings suggest that similar areas of the genome might be involved in ASD and ADHD (Smalley et al. 2002). Candidate gene studies are beginning to explore genetic associations that may explain the comorbidity of ASD and ADHD (e.g. Gadow et al. 2008, 2009) and the serotonin transporter gene (SLC6A4) has shown associations in both ASD and ADHD samples (e.g., Kim et al. 2002, 2005).

According to several twin studies, different autistic-like traits (social-communication problems and nonsocial behaviors) are highly heritable in the general population but appear to be caused by largely distinct genetic influences (Ronald et al. 2006; Ronald et al. 2005). Low genetic overlap between social and nonsocial autistic-like traits has also been reported in a community sample of 2-year-old twins (Edelson et al. 2009). Interestingly, one of the family studies found significant positive correlations between a total ADHD symptoms scale and all types of PDD symptoms: social, communication and nonsocial (Nijmeijer et al. 2009).

The present study aimed to explore for the first time the association between autistic-like traits and ADHD behaviors in early childhood, in a community sample of 2-year-olds. We hypothesized that PDD and ADHD behaviors would be positively correlated at this early

age. Our second aim was to explore the extent to which genetic and environmental influences overlapped for autistic-like traits and ADHD behaviors in 2-year-olds. Our hypothesis was that was there would be some shared genetic influences across these two sets of behaviors. We did not have specific expectations about how the degree of genetic overlap between autistic-like traits and ADHD behaviors in this sample would compare with the findings in older samples. However, the general finding in behavior genetics that shared environment plays a greater role in early childhood (Plomin et al. 2008), and the possibility that some genetic influences on ADHD behaviors might emerge later in development, would both lead to the prediction that genetic overlap between autistic-like traits and ADHD behaviors would be lower in early childhood than at later ages. Finally, analyses explored to what extent ADHD behaviors were associated with social and nonsocial autistic-like traits separately. Based on the finding by Nijmeijer et al. (2009), that all the types of autism symptoms correlated with ADHD behaviors, we predicted that both social and nonsocial autistic-like traits would show an association with ADHD behaviors in our sample.

Methods

Sample

The Boston University Twin Project sample was recruited from birth records supplied by the Massachusetts Registry of Vital Records. Twins were selected preferentially for higher birth weight and gestational age. No twins with birth weights <1,750 g or with gestational ages <34 weeks were included in the study. One twin was excluded due to chromosomal abnormalities. Parents completed questionnaire measures within two weeks of their twins' second birthday. The present analyses include 312 same-sex pairs of twins (144 MZ, 168 DZ), mean age 2.07 years (SD= 0.05). Although the sample was predominately Caucasian (85.4%), ethnicity was generally representative of the Massachusetts population (3.2% Black, 2% Asian, 7.3% Mixed, 2.2% Other). Socioeconomic status (SES) according to the Hollingshead Four Factor Index of Social Status (Hollingshead 1975) provided a raw score from 8 to 66 (higher scores reflect higher SES) and in this sample ranged from low to upper middle class (range = 20.5–66.0; *M*=50.9, SD=14.1).

Zygosity was determined via DNA analyses using DNA obtained from cheek swab samples. In the cases where DNA was not available (*n*=3), zygosity was determined using parents' responses on physical similarity questionnaires which have been shown to be more than 95% accurate when compared to DNA markers (Price et al. 2000).

Measures

Autistic-Like Traits and ADHD Behaviors—The Pervasive Developmental Problems (PDP) and ADHD subscales from the *Child Behavior Checklist for Ages 1 1/2–5* (*CBCL/1 1/2–5*; Achenbach and Rescorla 2000) provided parent-rating measures of autistic-like traits and ADHD behaviors. No changes were made from the published instrument. The PDP scale consisted of 13 statements regarding the child's social-communicative and repetitive behaviors (e.g., "Avoids looking others in the eye"; "Repeatedly rocks head or body"). The ADHD scale consisted of 6 statements that assess a child's inattentive and hyperactive behavior (e. g., "Can't concentrate, can't pay attention for long", "Can't sit still, restless, or

hyperactive"). Parents were asked to indicate how well each statement described their child's behavior as observed within the past two months on a 3-point Likert scale. The CBCL is widely used in behavioral genetic research (e.g., Constantino et al. 2003) and has demonstrated adequate reliability and validity (Achenbach et al. 1987).

The ADHD PDP preschool scale has been shown to have test–retest reliability of 0.86 and an odds ratio of 11, that is, children who score in the clinical range have 11 times greater odds of being from a referred sample than those who do not score in the clinical range (Achenbach and Rescorla 2000). In addition, the PDP CBCL subscale has been shown to have high sensitivity for detecting children with ADOS-G classification of autism (80%) but modest specificity (42%) (Sikora et al. 2008). The PDP CBCL shows significant positive correlations with other autism screening measures such as the Gillian Autism Rating Scale (r=0.66) (Sikora et al. 2008). In the present sample, internal consistency was modest, estimated at 0.64 by Cronbach's alpha. Scores on the CBCL PDP scale ranged from 0 to 13.

For the additional analyses on social and nonsocial autistic-like traits, the CBCL PDP scale was divided into two subscales. The subscales were created based on the social/communicative or nonsocial nature of the items, using symptom categories for autistic disorder from the Diagnostic and Statistical Manual for Psychiatric Disorders (DSM-IV: American Psychiatric Association 1994) as guidance for categorizing each item. Six items were considered to relate to social impairments; these included avoiding eye contact and not answering to name. Five items were determined to be nonsocial in nature; these included preference for sameness, and inflexible and repetitive behaviors. Two items from the full CBCL PDP subscale, "speech problem" and "strange behavior", were excluded from the subscales because they did not explicitly refer to behaviors from either social or nonsocial domain. The alphas for these social or nonsocial subscales were 0.56 and 0.50, respectively.

The ADHD CBCL preschool scale has been shown to have test–retest reliability of 0.74 and an odds ratio of 4, that is, children who score in the clinical range have 4 times greater odds of being from a referred sample than those who do not score in the clinical range (Achenbach and Rescorla 2000). In the present sample, internal consistency for the ADHD scale was 0.78 as estimated by Cronbach's alpha. Scores on the CBCL ADHD scale ranged from 0 to 12.

Bayley Mental Development Index (MDI)—The Mental Scale of the Bayley Scales of Infant Development (BSID-II) was used to yield a Mental Development Index (MDI) score. The BSID-II is a commonly used assessment of mental development in early childhood and is reliable and valid (Bayley 1993). The MDI data were collected as part of a visit to the laboratory, conducted within approximately two weeks of the twins' second birthdays within a standardized laboratory situation.

Analyses

Data Transformation and Confounding Variables—The PDP scale from the CBCL showed a moderate positive skew (see Table 1); consequently, this variable was log transformed to create a more normal distribution. The ADHD subscale had only modest skew (see Table 1) and therefore did not require transformation.

In terms of confounding variables, both autistic and ADHD quantitative traits show a male bias and both are negatively correlated with IQ (e.g., Kuntsi et al. 2004; Hoekstra et al. 2009). Factors relating to socioeconomic status have also been associated with psychopathology (e.g., Langley et al. 2007). Therefore these possible confounding variables were controlled for in the analyses. Using standard quantitative genetic procedures, all scores were residualized for sex effects. The PDP subscale was further residualized to remove the effects of cognitive abilities and SES. These residualized scores were used in all the behavioral genetic analyses.

Phenotypic Analyses—Pearson correlations were employed to explore the strength of the relationship between ADHD and PDP scales. Mean differences were evaluated using generalized estimating equations (GEE) implemented in the SAS GENMOD procedure to account for dependence in the data due to the fact that our sample comprised pairs of twins. GEE are an extension of the standard generalized linear models that allow modeling of correlated data (Liang and Zeger 1986).

The Twin Design—The twin design is based on comparing the within-pair similarity of monozygotic (MZ) and dizygotic (DZ) twins on a measure or trait of interest (Plomin et al. 2008). The design is based on the assumption that MZ twins share all of their DNA and DZ twins share on average half of their DNA.

Heritability refers to the proportion of variation of a trait in a population explained by genetic influences. ‘Environmental influences’ in the twin design refer to all variance that is not explained by genetic influences, and is split into two types, shared and nonshared. Shared environment refers to experiences that make children growing up in the same family similar; nonshared environment refers to environmental influences that make children growing up in the same family different (Plomin et al. 2008).

Twin Correlations—Twin similarity coefficients (intraclass correlations; Shrout and Fleiss 1979) were used for an initial examination of the twin data to compare MZ and DZ twin similarity. Evidence that a trait shows additive genetic influences (A) is indicated when twin similarity is greater for MZ twins than DZ twins. Shared environmental influence (C) is indicated if DZ twin correlations are greater than half the MZ twin correlations. Nonshared environmental influences (E) are indicated by the extent to which MZ correlations are less than unity.

When MZ twins are more than twice as similar than DZ twins, this suggests nonadditive genetic influences (D) such as dominance (interaction of two alleles at the same loci); however since this pattern of twin correlations was not observed in the present data, nonadditive genetic influences were not modeled. Hence, causative influences were divided into additive genetic (A), shared environment (C), and nonshared environment (E). To evaluate genetic and environmental sources of covariance across variables, cross-trait cross-twin (CTCT) correlations were calculated.

Model-Fitting Analyses—Multivariate genetic models decompose the variance of each phenotype and the covariances *between* phenotypes into additive genetic effects (A), shared

environmental effects (C), and nonshared environmental effects (E). Model-fitting procedures are used to analyze all of the data simultaneously, provide tests of the fit of models, yield confidence intervals for parameter estimates, and test the fit of alternative models. The A latent variables are fixed to correlate 1.0 and 0.5 for MZ and DZ twins, respectively. The C latent variables refer to the influence of shared environment on twin resemblance. Because all twins in the sample were reared in the same family, shared environments are assumed to correlate 1.0 for both MZ and DZ twins. Finally, the E latent variables reflect nonshared environmental influences that are unique to each member of a twin pair, including measurement error and therefore are fixed to correlate 0 in the model.

In the present study, a Cholesky decomposition model was used to investigate the sources of covariance between autistic-like and ADHD behaviors. The Cholesky model was presented here in the form of a correlated factors model, which is mathematically equivalent to the Cholesky (Loehlin 1996). Importantly, the correlated factors model provides estimates of the genetic correlation (r_g) and the shared (r_c) and nonshared (r_e) environmental correlations.

These are particularly useful statistics because they give a direct indication of the proportion of overlap in causal influences across two measures. They can vary between 0, indicating complete independence in causal influences, to 1, indicating that all influences are shared across two variables.

The following bivariate models were tested: ACE, CE, AE, E, AEs. "*s*" refers to another parameter that can be added to the model, representing a form of phenotypic interaction between twins (Neale et al. 2003). When modeling parent report data, a negative phenotypic interaction might occur because it exists in the children's behavior towards each other, or because there is a contrast effect in the parental ratings of their behavior (i.e., parents contrast twins and thereby exaggerate behavioral differences between the children). Contrast effects are implicated in parent ratings of twins when DZ variance is significantly greater than MZ variance.

Models were fit to raw data using a maximum likelihood pedigree approach implemented in Mx structural equation modeling software (Neale et al. 2003). This approach does not yield a χ^2 for assessing the fit of the model, however, the fit of a model can be assessed by calculating the difference between the negative log-likelihood (−2LL) of the model and that of a saturated model (i.e., a model in which the variance/covariance structure is not estimated and all variances and covariances for MZ and DZ twins are estimated). The difference in −2LL is asymptotically distributed as χ^2 with degrees of freedom (df) equal to the difference in the number of parameters in the full model and that in the saturated model. Fit was also assessed using Akaike's Information Criterion (AIC) which was calculated as follows: $\Delta\chi^2$ −2*Δdf. Negative AIC values indicate a good fit of the data to the model.

Results

To ascertain the representativeness of the sample on the PDP and ADHD measures, the percentage of the sample scoring at the impaired extreme in the sample was compared to the CBCL standard percentile scores. 98.1% of the sample scored ≤10 on the ADHD CBCL

scale, mirroring the CBCL norms which indicate that a score of greater than 10 indicates children falling above 98% of the population. 97.7% of our sample scored ≤9 on the PDP CBCL scale, which is in line with the CBCL standard percentile scores which indicate that scoring >9 falls within the clinical range (above 98%).

The PDP and ADHD scales were mainly completed by mothers in the sample, with a small proportion completed by fathers (94% and 6%, respectively). There were no significant mean differences in mother and father ratings for the PDP scale (z=0.75, p=0.45) or the ADHD scale (z= 1.92, p=0.06). More important to our behavior genetic analyses, MZ and DZ twin correlations were not significantly different when including only the maternal ratings compared to including both maternal and paternal ratings (values did not differ by more than 0.02) and the phenotypic correlation between ADHD and PDP scales was not significantly different. Therefore maternal and paternal ratings were both included in the analyses in order to maximize the sample size in the analyses.

Confounding Variables

General cognitive ability, as assessed by the Bayley MDI, correlated −0.09 (n.s.) with the PDP subscale and −0.12 (p<0.05) with the ADHD subscale. Similarly, SES correlated negatively with the PDP subscale (−0.12, p<0.05) and with the ADHD subscale (−0.07, n.s.). As outlined in the Methods section, in order to control for their possible confounding effects, we regressed out these variables from the PDP total score and subscales prior to the analyses.

Descriptive Statistics

Table 2 lists the means and standard deviations by sex and zygosity. The ADHD scale showed significant mean sex differences, with males scoring significantly higher than females (effect size = −0.81). The PDP scale and the social and nonsocial PDP subscales showed no significant mean sex or zygosity effects. The ADHD scale correlated positively with the PDP scale (r=0.27, p<0.001) and with the social and nonsocial subscales (r=0.23 for both, p< 0.001).

Twin Correlations

The top half of Table 3 displays the univariate twin correlations. MZ twin correlations were higher than DZ twin correlations, suggesting modest genetic influences on autistic-like and ADHD behaviors. DZ twin correlations were greater than half the MZ correlations for the PDP scales, suggesting shared environmental influence. Finally, for all scales, MZ twin correlations were significantly less than unity, showing that nonshared environment also explained some of the variation in these behaviors. The cross-trait cross-twin (CTCT) correlations are displayed in the lower part of Table 3. MZ similarity was not greater than DZ similarity and confidence intervals for MZ and DZ CTCT correlations overlapped.

Model-Fitting Results Between PDP and ADHD Scales

Saturated models were run to derive a saturated fit with which to compare the more constrained variance components models and to test for mean and variance differences between the MZ and DZ groups. MZ and DZ means could be equated without a significant

deterioration in fit for both the ADHD and PDP scales. For all scales, variances could not be equated across MZ and DZ groups without a significant deterioration in fit of the model. DZ twins showed greater variance than MZ twins on all the scales, suggesting the presence of contrast effects in the data.

Table 4 presents the fit statistics for the bivariate models run for the PDP total and ADHD scales. The fit of the ACE model was not significantly worse than the saturated model fit, indicating that the model fit the data well. This was also indicated by the negative AIC fit value for the ACE model. The only change to the ACE model that did not cause a significant ($p<0.05$) deterioration in fit (as indicated by the 'Relative fit of model' *p*-values on the right hand side of the table) was dropping the covariance path for A ($\Delta\chi^2=1.28$ (1*df*), p = n.s.). The 's' parameters in the AEs models could be dropped without a significant deterioration in fit ($\Delta\chi^2= 4.15$ ($\Delta 2df$), p = n.s.).

Parameter estimates are presented for the full ACE model in the lower part of Table 4. Both the PDP and ADHD scales showed additive genetic, shared and nonshared environmental influences. The heritability estimate was higher for the ADHD than the PDP scale (54% versus 19%) although confidence intervals overlapped. There was a trend towards the PDP scale showing higher estimates of both shared and nonshared environmental influences (35% and 46%, respectively) than the ADHD scales (17% and 30%, respectively), although again confidence intervals overlapped for these estimates.

The genetic correlation between the PDP and ADHD scales was estimated at 0.27, indicating a moderate degree of genetic overlap, and 33% of the phenotypic correlation was explained by additive genetic influences. However, as noted by the model fits above, this estimate was not statistically significant in the model. The shared environmental covariance showed complete overlap between measures ($r_c=1$) and was in the positive direction, indicating that the same shared environmental factors influenced both autistic-like traits and ADHD behaviors. Finally the nonshared environmental correlation was small and negative ($r_e=-0.17$), suggesting that most nonshared environmental influences did not overlap across the two scales, and those that did operated to increase behaviors in one domain and decrease them in the other.

Model-Fitting Results Between Social and Nonsocial PDP Subscales and ADHD Scale

Unlike for the total PDP-ADHD models the ACE bivariate models between the PDP subscales and ADHD fit significantly worse than the saturated models ($p<0.05$) indicating that these models overall did not fit the data so well. Fit statistics for these bivariate models are available from the first author on request. For both bivariate models, the covariance paths for both additive genetic influences (A) and nonshared environmental influences (E) could be dropped without a significant deterioration in fit. In addition, for the social-ADHD bivariate model, shared environment (C) could be completely dropped from the model without a significant deterioration in fit ($\Delta\chi^2=7.00$ (3*df*), p = n.s.). For both of these bivariate models the 's' parameters in the AEs models could be dropped without a significant deterioration in fit.

Table 5 presents the parameter estimates for the full ACE bivariate models between social autistic-like traits and ADHD behaviors and between nonsocial autistic-like traits and ADHD behaviors. The full ACE models path estimates are presented to demonstrate the results of the full model, but it is explained in the text where path estimates could be dropped. The heritability estimates were higher for ADHD (55–58%) than for either social or nonsocial PDP subscales (21% and 12%, respectively). There was a trend towards the PDP subscales showing higher estimates of shared environmental influences (both 29%) than the ADHD scales (13–16%).

The genetic correlation between social autistic-like traits and ADHD behaviors was estimated at 0.14, indicating a small degree of genetic overlap between social PDP traits and ADHD behaviors, and this estimate was not statistically significant. The proportion of the phenotypic correlation explained by genetic influences was 21%. However, it is notable that in the more parsimonious AE model, which did not fit significantly worse than the ACE model, the genetic correlation increased to 0.41 (0.25–0.57).

The genetic correlation between nonsocial autistic-like traits and ADHD behaviors in the full ACE model was also modest (r_g=0.32) and 38% of the phenotypic correlation was explained by genetic influences. Shared environment correlations were estimated at unity in both full ACE models (r_c=1), indicating that the same shared environmental factors influenced both social/nonsocial autistic-like traits and ADHD behaviors. Finally the nonshared environmental correlation was small and negative in both models (r_e=−0.04 for the social-ADHD bivariate model and −0.17 for the nonsocial-ADHD bivariate model).

Discussion

Data are presented from this first twin study to assess the degree to which behaviors characteristic of ASD and ADHD covary in 2-year-olds and the extent to which this covariation is explained by genetic and environmental influences. These findings from a community sample of 2-year-olds are compared to previous studies that have addressed this question using samples from later childhood and early adulthood.

Autistic-Like Traits and ADHD Behaviors

Autistic-like traits and ADHD behaviors were found to show a positive significant correlation in this community sample of 2-year-olds. Compared to studies of older children and young adults (Constantino et al. 2003; Reiersen et al. 2008; Ronald et al. 2008), the relationship between autistic-like traits and ADHD behaviors was weaker (*r*=0.23–0.26 compared to *r*=0.48–0.57), suggesting that their covariance increases with age. One alternative methodological explanation for this lower phenotypic correlation is that regressing out the confounding variables (cognitive ability and SES) diminished the phenotypic correlation between PDP and ADHD scales. However this explanation was not supported by the data because the phenotypic correlation between PDP and ADHD scales was only slightly higher (*r*=0.29) before the confounding variables were regressed out. A second possibility is that not all types of behaviors characteristic of ADHD and ASD have emerged at this young age, resulting in less reliable measurement of these behaviors compared to older ages. Supporting this possibility, the alpha values for the CBCL ADHD

and PDP scales were somewhat lower (0.64 and 0.78) in this 2-year-old sample than those reported in the twin studies on older samples (0.75–0.92; Reiersen et al. 2008; Ronald et al. 2008), although the use of different measures in these previous studies should be considered.

As described in the Introduction, findings from the sample used here have reported previously that autistic-like traits at age 2 show a modest heritability (39%) as well as significant shared and nonshared environmental influences (Edelson and Saudino 2009). The heritability estimate of autistic-like traits in the bivariate model presented here was somewhat lower (19%) than the first estimate, which is likely to be explained by the fact that it was modeled in a bivariate model and because the scale had been corrected for confounding variables. Nevertheless this modest heritability estimate concords with the previous report in suggesting that heritability of autistic-like traits is lower in early childhood than at later ages. Regarding the finding of shared environment on autistic-like traits in 2-year-olds, it is common in twin studies of other behavior problems and cognitive ability to report significant shared environmental estimates in early childhood that tend to reduce in older age groups (e.g., Saudino et al. 2008).

The heritability of ADHD behaviors in 2-year-olds was estimated at 54%, which is slightly lower than what has been reported previously (e.g. Price et al. 2001), and is possibly due to the different measures used across these studies (the CBCL versus the Rutter scale).

The findings suggested that a modest proportion of genetic influences on autistic-like traits also influence ADHD behaviors. The genetic correlation estimate of 0.27 between the PDP and ADHD scales found here was lower than that reported in previous studies of middle childhood (r_g=0.54–0.57; Ronald et al. 2008) and still lower than that estimated in early adulthood (r_g=0.72) (Reiersen et al. 2008). When considering this pattern of genetic correlations across studies it is worth noting differences with respect to confounding variables. In the present study, with the lowest genetic correlation, both cognitive ability and SES were regressed out of the scales prior to the analysis. The Ronald et al. (2008) study regressed out cognitive ability from the analysis in middle childhood; the Reiersen et al. (2008) study on adults did not regress out any confounding variables. To test the impact of confounding variables on the model-fitting result, we re-ran the best-fitting ACE bivariate model without regressing out any confounding variables. We found that the genetic correlation was still modest (0.34) and not significantly different from the genetic correlation when the effects of the confounding variables were removed, and still lower than the results in the studies on older children. In sum, these results from twin studies of early childhood, middle childhood, and early adulthood suggest that some genetic influences overlap between autistic-like traits and ADHD behaviors and that this degree of overlap increases across development. Genes influencing both autistic-like and ADHD behaviors may increase in their effect across development, or new genes influencing both autistic-like and ADHD behaviors may come online after age 2.

The shared environmental correlation was estimated at unity, suggesting that there are some shared environmental factors affecting both phenotypes. Another possibility is that assortative mating artificially inflated the shared environment component. Although the present data could not test for the presence of assortative mating (ratings of parent traits are

needed) a previous twin study has reported the presence of assortative mating on autistic traits (Constantino and Todd 2005), suggesting this could be a possible explanation. Another possibility is that there was correlated rater variance across the two measures because the same rater was used for both measures; this would also act to artificially inflate the shared environmental estimates. This latter scenario could be tested by collecting data from multiple raters.

Social and Nonsocial Autistic-Like Traits and ADHD Behaviors

Children who were rated as having more ADHD behaviors showed both more social autistic-like traits and more nonsocial autistic-like traits. This concurs with a recent family study that found that both social and nonsocial autism symptoms were elevated in children with ADHD and their siblings (Nijmeijer et al. 2009). The results suggested that in 2-year-olds the association between autistic-like traits and ADHD behaviors in the general population is driven equally by both social and nonsocial autistic-like traits, and that both social and nonsocial autistic behaviors contribute equally to the genetic and environmental influences that are shared with ADHD behaviors.

Limitations

Like many studies, this study should be considered in light of its general and specific limitations. The more general limitations of the twin design can be resolved by using other research designs to address the same research question. To our knowledge, no family studies investigating the overlap between autistic-like traits and ADHD behaviors in young children have yet been carried out, but the present findings concur to some extent with recent family studies of older children (Mulligan et al. 2009a; Nijmeijer et al. 2009). This is a cross-sectional study: in the future a longitudinal study will be more informative for examining the relationship between autistic-like traits and ADHD behaviors across development.

Some characteristics of the sample warrant discussion. The sample size was modest which led to wide confidence intervals for some estimates and the sample was underpowered to detect etiological sex differences. The sample did not include children at the very severe end of the autism spectrum. Nevertheless, eight individuals out of the sample of 624 individuals were formally diagnosed with an ASD at age 3. After taking into account relatedness between twin pairs, this prevalence is still within current estimates of the prevalence of ASD (Baird et al. 2006), suggesting that the sample was reasonably representative. Furthermore, the sample was selected not to include children who were preterm or that had a very low birth weight. Therefore the current findings should be considered in light of the sample's characteristics.

In terms of the measures, it was advantageous to employ scales from the CBCL, which have been widely used and assessed for reliability and validity (Achenbach and Rescorla 2000). Nevertheless, the reliability, as assessed by Cronbach's alpha, for the PDP scale and subscales was modest in the present sample, and may have led to inflated estimates of the nonshared environment term (which includes measurement error). As described in the Methods section, the PDP CBCL scale has been shown to have modest specificity for detecting children with ADOS-G classification of autism, and therefore this limitation

regarding the measure's validity should be taken in account when assessing the results. The social and nonsocial subscales were devised by the authors, based on DSM-IV symptom categories for autistic disorder, but they have not been externally validated. Parent report contains some bias and shows only modest correlation with other raters (Najman et al. 2001). However, parental assessment of problem behavior is a practical option for large preschool samples, and parents are familiar with behavior across time and a range of situations.

Clinical Implications

In terms of clinical diagnoses, current criteria exclude the diagnosis of ADHD when ASD is present. It is important to exercise caution when interpreting findings from community samples in relation to clinical issues but it is notable that these findings concur with clinical studies in supporting the notion that autistic-like traits and ADHD behaviors show substantially greater overlap than chance alone in preschool children (Gadow et al. 2004). The combined impact of having developmental data showing that this comorbidity is present in preschool children, as well as the evidence from twin studies that there are shared genetic and environmental influences on autistic and ADHD behaviors, suggests that the overlap between ASD and ADHD may be partly explained by shared biological pathways that come into play early in development. The present results also suggest that there may be some shared and nonshared environmental influences that explain the overlap between autistic and ADHD behaviors in early childhood. An alternative 'phenotypic causality' hypothesis, that developing autistic-like traits can lead individuals to manifest symptoms of ADHD at a later time-point (or vice-versa, that ADHD symptoms lead to autistic-like traits), is not well supported by these data. A significant relationship between the degree of autistic-like traits and degree of ADHD behaviors is already present in 2-year-olds.

Several implications stem from the existing results on the overlap between autistic and ADHD behaviors in both clinical and community samples. Molecular genetic research exploring the genomic regions and gene pathways associated with ASD may benefit from exploring candidate regions and genes associated with ADHD, and vice-versa. Furthermore, the overlap in etiological factors suggests that there will also be overlap in common neurobiological pathways and that this overlap appears to become greater in magnitude across development. Second, this evidence for overlap between ASD and ADHD behaviors both clinically and when assessed as quantitative traits should be used as a spring board for re-evaluating the diagnostic rules concerning dual diagnoses of ASD and ADHD.

Summary

The present findings suggest that autistic- and ADHD-like traits begin to covary as early as 2-years of age in the general population after controlling for the confounding effects of cognitive ability, SES and gender. This covariation is caused by a modest proportion of common genetic influences across autistic traits and ADHD behaviors as well as by common shared environmental influences. Within autistic traits, both social and nonsocial types of autistic traits are equally associated with ADHD behaviors in 2-year-olds and both contribute to the modest genetic overlap between these two categories of behavior. A future research goal should be to explore how specific social impairments (e.g., aloofness, social

reciprocity, etc) and specific nonsocial behaviors (e.g., resistance to change, repetitive motor behaviors, etc) overlap with specific ADHD symptoms. A second goal is to explore how genetic and environmental influences interact in the processes that influence the covariation of these behaviors.

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Table 1

Descriptive Statistics for the Raw Scales

	CBCL ADHD Scale	CBCL PDP Scale	Bayley MDI	SES
M	4.26	2.56	90.70	51.04
SD	2.70	2.34	13.89	10.68
Skew	0.47	1.30	−0.35	−0.81
Kurtosis	−0.08	2.27	−0.50	0.10

N=312 individuals (one twin per pair selected randomly for birth order) *CBCL PDP* child behavior checklist pervasive developmental problems subscale; *CBCL ADHD* CBCL attention deficit hyperactivity disorder subscale; *MDI* mental development index

Table 2

Descriptive Statistics for the CBCL Pervasive Developmental Problems and Attention Deficit Hyperactivity Disorder Scales Used in the Model-Fitting, Split by Zygosity and Gender

	MZM; *M* (SD)	DZM; *M* (SD)	MZF; *M* (SD)	DZF; *M* (SD)	ANOVA		Effect size	
					Sex	Zyg	Sex	Zyg
CBCL ADHD Scale	4.77 (2.68)	4.58 (2.80)	3.68 (2.40)	4.02 (2.55)	−3.16*	0.23	−0.81	0.06
CBCL PDP Scale[a]	0.45 (0.31)	0.47 (0.30)	0.42 (0.28)	0.48 (0.30)	−0.28	1.19	−0.01	0.03
CBCL PDP Social subscale[a]	0.48 (0.54)	0.49 (0.51)	0.46 (0.49)	0.52 (0.54)	0.28	1.07	<0.01	0.02
CBCL PDP Nonsocial subscale[a]	0.63 (0.57)	0.72 (0.61)	0.62 (0.56)	0.81 (0.57)	0.83	2.10*	0.02	0.06

Effect size (ES) estimated as Cohen's *d*, which expresses group differences in standard deviation units

CBCL PDP child behavior checklist pervasive developmental problems subscale; *CBCL ADHD* CBCL attention deficit hyperactivity disorder subscale; *MZM* monozygotic males, *DZM* dizygotic males, *MZF* MZ females, *DZF* DZ females; Zyg zygosity

[a]Transformed scale

*$p<0.05$

Table 3

Twin Intraclass Correlations, and Cross-Trait Cross-Twin Correlations (with 95% Confidence Intervals)

Measure	Univariate Intraclass Correlations	
	MZ	**DZ**
PDP Total	0.57 (0.49–0.65)	0.40 (0.30–0.49)
	N=136	*N*=155
ADHD	0.72 (0.63–0.79)	0.34 (0.19–0.46)
	N=144	*N*=167
Social PDP subscale	0.52 (0.42–0.60)	0.35 (0.25–0.44)
	N=133	*N*=154
Nonsocial PDP subscale	0.46 (0.36–0.55)	0.29 (0.19–0.39)
	N=129	*N*=154
	Cross-trait cross-twin correlations	
	MZ	DZ
PDP Total—ADHD	0.30 (0.19–0.40)	0.37 (0.27–0.46)
Social PDP—ADHD	0.15 (0.03–0.26)	0.34 (0.24–0.44)
Nonsocial PDP—ADHD	0.28 (0.16–0.38)	0.34 (0.23–0.43)

PDP child behavior checklist (CBCL) pervasive developmental problems subscale; *ADHD* CBCL attention deficit hyperactivity disorder subscale. *MZ/DZ* monozygotic/dizygotic twins

Table 4

Fit Statistics and Parameter Estimates for Bivariate Models Between Autistic-Like Traits and ADHD Behaviors

Model	Overall Fit of Model[a]						Relative Fit of Model[b]		
	−2LL	*df*	LRT	Δ *df*	*p*	AIC	Δ χ^2	*df*	*p*
Saturated	3,159.97	1,187							
Full ACE	3,184.47	1,202	24.50	15	0.057	−5.50			
AEs	3,191.90	1,203	31.92	16	0.010	−0.08			
AE	3,196.05	1,205	36.07	18	0.007	0.07	11.57[b]	3	0.009
CE	3,214.96	1,205	54.99	18	0.000	18.99	30.49[b]	3	0.000
E	3,396.37	1,208	236.39	21	0.000	194.39	211.89[b]	6	0.000
Drop A-cov	3,185.75	1,203	25.78	16	0.057	−6.22	1.28[b]	1	0.259
Drop C-cov	3,195.55	1,203	35.57	16	0.003	3.57	11.07[b]	1	0.001
Drop E-cov	3,188.80	1,203	28.83	16	0.025	−3.17	4.33[b]	1	0.038
	a^2	c^2	e^2				r_g	r_c	r_e
ADHD	0.54 (0.36–0.68)	0.17 (0.04–0.31)	0.30 (0.23–0.39)				0.27 (−1.00–1.00)	1.00 (0.73–1.00)	−0.17 (−0.32–−0.01)
PDP	0.19 (0.00–0.44)	0.35 (0.14–0.54)	0.46 (0.36–0.58)						

−2LL = log likelihood fit statistic; *df* = degrees of freedom; LRT(*df*) = likelihood ratio χ^2 test with Δ *df* comparing model to the saturated model; *AIC* = Akaike's Information Criterion. A = Additive genetic influences, C = Shared environmental influences, E = Nonshared environmental influences, s = sibling interaction path; a^2 = Additive genetic parameter estimate, c^2 = Shared environmental parameter estimate, e^2 = Nonshared environmental parameter estimate; r_g = Genetic correlation, r_c and r_e = Shared and nonshared environmental correlations, respectively

[a] Overall fit of the model is determined by the difference in −2LL of each model and that of the saturated model

[b] Relative fit of the model determined by the χ^2 difference ($\Delta\chi^2$) between full bivariate ACE model and each reduced model

Table 5

Parameter Estimates for Bivariate ACE Models Between Social (Top Panel) and Nonsocial (Bottom Panel) Autistic-Like Traits and ADHD Behaviors

	a^2	c^2	e^2	r_g	r_c	r_e
Social PDP-ADHD						
ADHD	0.58 (0.40–0.72)	0.13 (0.02–0.28)	0.28 (0.22–0.37)	0.14 (−1.00–1.00)	1.00 (0.63–1.00)	−0.04 (−0.20–0.12)
Social	0.21 (0.00–0.47)	0.29 (0.06–0.50)	0.51 (0.40–0.63)			
Nonsocial PDP-ADHD						
ADHD	0.55 (0.36–0.70)	0.16 (0.03–0.31)	0.30 (0.23–0.39)	0.32 (−1.00–1.00)	1.00 (0.67–1.00)	−0.17 (−0.32–0.00)
Nonsocial	0.12 (0.00–0.39)	0.29 (0.08–0.46)	0.60 (0.46–0.72)			

A = Additive genetic influences, C = Shared environmental influences, E = Nonshared environmental influences, s = sibling interaction path; a^2 = Additive genetic parameter estimate, c^2 = Shared environmental parameter estimate, e^2 = Nonshared environmental parameter estimate; r_g = Genetic correlation, r_c and r_e = Shared and nonshared environmental correlations, respectively