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Association Between Depression and Anxiety in High-Functioning Children with Autism Spectrum Disorders and Maternal Mood Symptoms

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Abstract

Research suggests that children with autism spectrum disorders (ASDs) and their relatives have high rates of depression and anxiety. However, relatively few studies have looked at both factors concurrently. This study examined the potential relationship between maternal mood symptoms and depression and anxiety in their children with ASD. Participants were 31 10- to 17-year-old children with an ASD diagnosis that was supported by gold-standard measures and their biological mothers. Mothers completed the *Autism Comorbidity Interview* to determine whether the child with ASD met criteria for any depressive or anxiety diagnoses and a questionnaire of their own current mood symptoms. As expected, many children with ASD met criteria for lifetime diagnoses of depressive (32%) and anxiety disorders (39%). Mothers' report of their own current mood symptoms revealed averages within the normal range, though there was significant variability. Approximately 75% of children with ASD could be correctly classified as having a depressive or anxiety disorder history or not based on maternal symptoms of interpersonal sensitivity, hostility, phobic anxiety, depression, and anxiety. The results provide preliminary evidence that maternal mood symptoms may be related to depression and anxiety in their children with ASD. Although the design did not allow for testing of heritability per se, the familial transmission patterns were generally consistent with research in typical populations. While larger follow-up studies are needed, this research has implications for prevention and intervention efforts.

Keywords

Autism; Asperger's disorder; psychiatric comorbidity; anxiety; depression; mood disorders; familial aggregation; maternal symptoms

Both depression (e.g. Ghaziuddin, Ghaziuddin, & Greden, 2002) and anxiety (White, Oswald, Ollendick, & Scahill, 2009) have each been described as the most common psychiatric concern for children with ASD. Review papers report prevalence rates in the range of 4% to 57% (Ghaziuddin et al., 2002: Lainhart, 1999) and 7% to 84% (e.g. Lainhart, 1999; White et al., 2009) for depression and anxiety disorders in ASD respectively. This variability in ranges is not unexpected due to differences across studies in age ranges, level of intellectual ability, recruitment methods, focus on current versus lifetime symptoms, and method of determining comorbidity status. Nonetheless, it is clear that comorbid depressive and anxiety disorders are quite problematic in ASD, increase impairment in the child, and add family strain (see Ghaziuddin et al., 2002; Stewart, Barnard, Pearson, Hasan, &

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O'Brien, 2006; White et al., 2009 for review). Understanding factors associated with a higher likelihood of comorbid depression and anxiety could aid in swifter and more specific treatments.

A better understanding of comorbidity in ASD may also help inform etiological research. Specifically, questions regarding the genetic meaning of the high rates of such comorbidities have been raised due to similar observations of high rates of depressive and anxiety disorders in parents of children with ASD. Given that previous research has found that most parents with an affective disorder report an onset prior to the birth of their child with an ASD (e.g., Bolton, Pickles, Murphy, & Rutter, 1998; Mazefsky, Folstein, & Lainhart, 2008; Piven et al., 1991; Piven & Palmer, 1999), it seems unlikely that the sole explanation for this occurrence is the stress related to raising a challenging child.

Reported rates of depressive and anxiety disorders in the family members of children with ASD have varied, due to similar reasons as those mentioned above, as well as different family history research methods with varying degrees of sensitivity. A literature review reported rates of major depression ranging from 20-37% in the first degree relatives of individuals with ASD (Lainhart, 1999). Other studies have found substantially higher prevalence rates for mood disorders among first-, second-, and third-degree relatives, ranging from 60% to 74% (Bolton et al., 1998; Delong, 2004; Ghaziuddin, 2005). Despite differences in the specific rates, most studies do indicate that there are more mood disorders in families with an individual that has an ASD compared to families without any children with disabilities and families with a member that has Downs Syndrome (Bolton et al., 1998; DeLong, 2004; Ghaziuddin, 2005; Mazefsky, Williams, & Minshew, 2008b). Female relatives of children with ASD, in particular, are significantly more likely to have major depression compared to families of individuals with Downs Syndrome (Bolton et al., 1998). Conversely, bipolar disorder has not been found to be consistently elevated among the first-degree relatives of individuals with ASD (Bolton et al., 1998; Piven & Palmer, 1999). Some studies have found highly elevated levels of anxiety in parents of probands with autism compared to parents of an individual with Downs Syndrome (Murphy et al., 2000; Piven et al., 1991). Yet, another study found that rates of anxiety and obsessive-compulsive disorder were higher, but only when including multiple generations in the analysis (Bolton et al., 1998).

The transmission rate of depression and anxiety among typical families is well established (Nes, Roysamb, Reichborn-Kjennerud, Harris, & Tambs, 2007; Singer, 2006; Tambs, 1991). A meta-analysis of depression in family, twin, and adoption studies suggests that depression has a strong familial (and mostly genetic) aspect in typically developing families (Sullivan, Neale, & Kendler, 2000). For example, results from multiple twin studies for major depression are converging to indicate an estimated heritability of approximately 0.33, which refers to the proportion of individual difference in risk for depression accounted for by genetics (Kendler, 2001). A study that utilized the *Symptom Checklist 90* (SCL-90; Derogatis, 1994) to capture symptom presentation in a typical population found a slightly higher heritability estimate of 0.43 for both depression and anxiety (Tambs, 1991). These previous findings suggest that depression and anxiety are highly transmittable through nuclear families, although it still is not clear whether genetics, shared environment, or an interaction of these factors is the cause.

Few studies have yet to investigate the potential relationships between psychiatric symptoms in family members and comorbidity in individuals with ASD. Ghaziuddin and Greden (1998) compared the family history of inpatient children with autism or ASD that had comorbid depression to children with ASD but without comorbid depression. Results indicated that 77% of the comorbid depression group had a family history of depression

compared to 30% of the children without comorbid depression (Ghaziuddin & Greden, 1998). Interpretation of these findings is complicated by the use of an inpatient sample, and the fact that diagnoses were based solely on clinical impressions rather than structured psychiatric interview. However, these findings were partially supported by a study by Mazefsky et al. (2008a) comparing the presence of psychiatric disorders in adults with ASD and their first-degree relatives using a structured psychiatric interview. A considerable number (88%) of probands with ASD were found to have a mood or anxiety disorder, and 60% of families had at least one parent with depression. One particularly pertinent finding was that 80% of probands had depression when their mothers were also diagnosed with depression, compared to only 16% of those whose mothers did not have depression meeting criteria for comorbid depression. However, some of Mazefsky et al.'s (2008a) findings did not suggest an increased risk based on parental history of mood and anxiety disorders. Specifically, the presence of anxiety in probands did not differ based on family history of mood disorder, and mood disorder in fathers did not seem to have any significant bearing on patterns of comorbidity in their adult children with autism either. This study had a very small sample size, however, so results must be considered preliminary.

Further research needs to be conducted on the relationship between comorbidity and patterns of psychiatric symptoms and disorders in family members in order to determine what services should be made available to individuals with ASD and their families to prevent comorbid psychiatric conditions and facilitate faster and more targeted treatment. Additionally, information on whether there is an association may aid in understanding how affective disorders may bear any common genetic risk with ASD. This study aimed to investigate the relation between psychiatric comorbidity for children and adolescents with ASD and their mothers' mood symptoms on a psychiatric survey. In particular, analyses focused on comorbid depression and anxiety in ASD because of the literature suggesting an especially high prevalence of comorbidity and ASD family history for these disorders. Patterns of maternal mood symptoms were compared between mothers of children with an ASD and a comorbid mood and/or anxiety disorder and those without a comorbid mood or anxiety disorder. It was expected that mothers of children with comorbid depression and/or anxiety would report more mood-related symptoms themselves than the mothers of children without a comorbid depressive or anxiety disorder.

Methods

Participants

The participants were 31 children and adolescents (10-17 years old) with ASD who were part of a study on the assessment of psychiatric comorbidity in ASD. All participants from the primary study who had data on maternal mood symptoms were included in this study. Participants were consecutive referrals recruited through word of mouth and fliers posted within a children's hospital (including a developmental clinic specialized in diagnosing ASDs). The advertisement did not mention comorbidity nor were recruitment efforts focused on a psychiatric hospital; rather the flyers targeted any children with ASD within our age range. This method of recruitment was chosen in hopes of attaining a representative sample of children with ASD both with and without comorbid psychiatric disorders in order to allow us to study patterns of associated factors and assessment instruments designed to differentiate comorbid disorders.

DSM-IV/ICD-10 diagnostic criteria based on *Autism Diagnostic Observation Schedule* (ADOS)/*Autism Diagnostic Interview* (ADI) results agreed upon by all National Institute of Health Collaborative Program of Excellence in Autism sites in June of 2004 was followed to assign specific ASD study diagnoses, and these were confirmed by the expert clinical opinion of a licensed clinical psychologist specialized in ASD [DSMIV-TR; American

Psychiatric Association, 2000]. The majority of participants had Asperger's disorder (64%; $n = 20$), 26% had autism ($n = 8$), and 10% had pervasive developmental disorder not otherwise specified (PDD-NOS; $n = 3$). Participants were excluded if they had comorbid mental retardation (e.g. an intellectual quotient of 70 or below). The restriction of subjects to those without mental retardation reflected a desire to define patterns of comorbidity and maternal symptoms that are associated with ASD and not the nonspecific consequence of mental retardation. Additionally, it has been suggested that higher cognitive functioning is associated with more depressive and anxiety symptoms in individuals with ASD (Sterling, Dawson, Estes, & Greenson, 2008; Tsankanikos et al., 2006). The focus was on pre-adolescents and adolescents due to the high risk for the development of psychiatric disorders during this time period; approximately 2/3 of the sample was in the 10-12 age range and 1/3 was in the 13-17 age range.

The biological mothers of each child with ASD also participated. The demographic information for the probands with ASD and their mothers can be seen in Table 1. Adaptive behavior and intellectual quotient scores in this sample were consistent with other studies of high-functioning individuals with ASD (e.g. Mazefsky et al., 2008b).

Measures

Probands' diagnoses of high-functioning autism, Asperger's disorder, or PDD-NOS were supplemented by Module 3 or 4 of the (ADOS) and the *Autism Diagnostic Interview-Revised* (ADI-R) (Lord et al., 2000; Lord, Rutter, & LeCouteur, 1994). Intellectual ability was determined via administration of the *Wechsler Abbreviated Scale of Intelligence* (Weschsler, 1999).

To assess comorbidity, the *Autism Comorbidity Interview-Present and Lifetime Version* (ACI-PL; Leyfer et al., 2006) was administered to the mothers by a licensed clinical psychologist. The ACI-PL was modified from the *Kiddie Schedule for Affective Disorders and Schizophrenia* to be used for the diagnosis of psychiatric comorbidities in children and adolescents with ASD (Leyfer et al., 2006). Specific modifications included allowing a modified diagnosis for disorders for which the DSM-IV-TR considers ASD an exclusion and symptom specifications such as social-related (and not lack of interest) for social phobia. Subsyndromal categories were also created in order to capture symptoms with impairment that did not reach full DSM-IV-TR criteria. The ACI-PL has at least adequate inter-rater reliability (kappas between 0.7 and 0.8) and test-retest reliability (kappas 0.61-0.7) across the various disorders. Criterion and concurrent validities were found to be good for various disorders, including depression. Sensitivity for the Boston and Salt Lake City labs that originally tested the ACI-PL was 100% for depression and specificity ranged from 83 to 93.7% (Leyfer et al., 2006).

Mothers completed the *Symptom Checklist-90-Revised* (SCL-90-R; Derogatis, 1994), a self report inventory of current psychiatric symptoms. Each response is a number rating from 0 to 4 of severity ("not at all" to "extremely"). The nine indices cover somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism, as well as 3 global indices. Each of the indices is then converted to a *t-score*, normed for males and females separately. Reliability is good, with internal consistency ranging from alpha levels of 0.77 –0.90 for the nine indices, and test-retest reliability ranging from 0.68 to 0.83. Convergent-discriminant validity was also found in relation to the MMPI and other measures (Derogatis, 1994). Studies have also found that the ability of the SCL-90-R to screen between patients and typical community members is good (Rauter, Leonard, & Swett, 1996; Schmitz et al., 2000).

Analyses

Means and standard deviations were calculated for SCL-90-R t-scores. The frequency of specific disorders was determined for the ACI-PL. Point-biserial correlations were calculated between the ACI-PL diagnoses and Full Scale, Verbal, and Performance IQ scores to ensure that that was no relationship to intellectual ability before proceeding with analyses using ACI-PL diagnoses. Five categories were created that measured both past and current syndromal proband diagnoses (e.g. whether a proband "ever" had a disorder): major depression, generalized anxiety disorder, transition/change-related anxiety, social phobia, and specific phobia. In addition, two broader variables were created: (1) Any depression (if a participant met at least subsyndromal criteria for either major depression, dysthymia, or depression not otherwise specified), and (2) any anxiety (if a participant met at least subsyndromal criteria for generalized anxiety disorder, social phobia, or specific phobia). Transition/change-related anxiety was not utilized in the any anxiety category because of its provisional nature and lack of inclusion in the DSM-IV-TR.

To investigate the relationship between comorbidity and maternal mood symptoms, logistic regressions were chosen. These analyses were conducted to determine whether mothers' patterns of mood symptoms could explain a significant amount of variance regarding the presence or absence of comorbidity in the probands. The any depression and any anxiety measures were used as the dependent variables, because they represented a general predisposition to depression or anxiety symptoms that could correspond to the nature of SCL-90-R scores.

In addition, due to the high correlation among SCL-90 variables (results available upon request) and the high number of variables raising risks of Type I error, only a subset of SCL-90 variables was utilized in the regression analyses. Excluded variables included: Paranoid ideation and psychoticism; Obsessive-compulsive symptoms (due to concerns regarding overlap with broader autism phenotype symptoms; see Piven and Palmer, 1999); and Somatization (due to its strong cross-correlation with both depression and anxiety causing problems with multicollinearity; see Carpenter and Hittner, 1995; Rauter et al., 1996). Variables included focused on mood-related symptoms that were expected to have a strong relationship to the dependent variables (comorbid anxiety or depression in the child), including Interpersonal Sensitivity, Hostility, Phobic Anxiety, Depression, and Anxiety. Depression and anxiety are often comorbid in individuals and frequently co-present in families (Middledorp, Cath, Van Dyck, & Boosma, 2005). Middledorp et al. (2005) also argue that neuroticism is a heritable personality trait that is present in many people with depression or anxiety disorders, and thus provides the basis for heritability. Interpersonal Sensitivity may be considered an aspect of neuroticism in that it reflects a tendency to demonstrate a negative reaction to perceived stimuli and a tendency to be highly emotional. Previous research has also found a significant relationship between hostility and neuroticism (Carmody, Crossen, & Wiens, 1989).

Results

Table II displays the mean and ranges of SCL-90-R t-scores for mothers. The mean t-scores were within a normal deviation for each index, and ranges suggest a normal distribution. There was sufficient variability in SCL-90 scores, ranging from ~1 standard deviation (SD) below the mean to 3 SDs above the mean. Most mothers scored in the abnormal range (1.5 SDs above the mean) for depression and hostility.

Point-biserial correlations did not reveal any significant associations between Full Scale, Verbal, or Performance IQ scores and any of the ACI-PL variables of interest, $p > 0.20$ in all cases (specific results available upon request). Table III displays the probands' comorbidity

results based on the ACI-PL. Nineteen percent (6) of the probands met full criteria for a diagnosis of syndromal major depression either currently or in the past, and 32% (10) had a syndromal or subsyndromal diagnosis under the any depression category. Thirty-nine percent (12) of probands had a diagnosis that qualified for the any DSM-IV-TR anxiety category.

Two logistic regressions were performed to explore the potential relationship between mothers' SCL-90 interpersonal sensitivity, hostility, phobic anxiety, depression, and anxiety scores and the dependent variable of comorbid depression and anxiety in their children with ASD. For the any depression regression, the model was significant overall χ^2 (5) = 11.27, p<0.05. The SCL-90-R anxiety subscale was a significant predictor (P = 0.03). The model accounted for approximately 43% of the variance in the presence or absence of depression in probands. Additionally, the model correctly identified the proband's comorbid depression diagnosis (present or not present) for 77.4% of participants.

For the any anxiety regression, the model was also significant overall χ^2 (5) = 12.09, p<0.05. Significant predictors included hostility (P = 0.03) and phobic anxiety (P = 0.05). Approximately 44% of the variance in the presence or absence of anxiety was accounted for by the model. The model correctly identified 74.2% of probands as having or not having comorbid anxiety.

Discussion

This study examined the potential relationship between co-morbid depression or anxiety in adolescents with high-functioning ASDs and the mood symptoms of their mothers. Results of a self-report questionnaire of current mood symptoms in mothers revealed averages within the normal range, though there was significant variability. With regard to proband comorbidity, 32% of adolescents with ASD met criteria for lifetime history of a co-morbid depressive disorder and 39% met criteria for lifetime history of an anxiety disorder. Participants with ASD could be correctly classified as having one of these comorbid conditions or not based on mothers' current mood symptoms for the majority of cases.

Findings of comorbid depressive and anxiety disorders in this study support previous research indicating that these disorders are common among individuals with ASD (Lefyer et al., 2006; Lainhart, 1999). Previously reported prevalence rates for both comorbid depression and anxiety have varied widely (Lainhart, 1999), and the results are within the reported range for both. Leyfer et al. (2006) also utilized the ACI-PL and found that nearly a fourth of participants had syndromal or subsyndromal major depression, but only about 10% met syndromal criteria. The current study found that 19% of probands received a syndromal diagnosis, and nearly a third of probands met criteria for the any depression category, which also included dysthymia and depression not otherwise specified. The somewhat higher rates in this study could be due to the fact that the mean age for our sample was 2.5 years older than Leyfer et al.'s (2006) study which had a lower age limit of 5 years compared to our lower age limit of 10. If similar factors account for depression in typically developing individuals and individuals with ASD, the higher age range may have increased the prevalence of depression in the sample due to increases in depression with age (Kessler et al., 2005). In addition, this study included only higher-functioning individuals whereas Leyfer et al.'s (2006) study included some children with ASD and comorbid mental retardation. Therefore, our slightly higher rate of syndromal depression is also consistent with research that has suggested a higher prevalence of comorbidity in ASD among individuals with higher cognitive ability (Sterling et al., 2008; Tsankanikos et al., 2006).

For anxiety disorders, Leyfer et al. (2006) found that almost half of their sample (44%) met criteria for a syndromal specific phobia diagnosis. The current study found a lower rate of specific phobia (13%), and this was comparable to the rates found for social phobia and generalized anxiety disorder (13 and 10%, respectively). Differences between the studies in rates of specific anxiety disorders may reflect difficulty reliably differentiating anxiety subtypes, as well as the influence of age and level of functioning. Overall, 40% of this sample met criteria for the any anxiety category, further contributing to the argument that comorbid anxiety is a significant issue in ASD.

Although our research design does not allow us to factor out environmental versus genetic influences, the pattern of results generally supports previous research on the relationship between depression and anxiety heritability in the typical population (Middledorp et al., 2005; Nes et al., 2007; Singer, 2006; Tambs, 1991). Regression results suggest that maternal symptoms of depression, anxiety, phobic anxiety, interpersonal sensitivity, and hostility can explain a large amount of variance of whether individuals with ASD have comorbid depression or anxiety, which implies similarities with typical populations. Specifically, the maternal measures of Phobic Anxiety and Hostility were significant predictors of proband anxiety, and the maternal measure of Anxiety was a significant predictor of proband depression. The significance of the Hostility scale in the anxiety regression may reflect the relationship between hostility and neuroticism seen in previous research (Carmody et al., 1989) given that studies suggest that neuroticism plays a major role in the familial transmission of depression and anxiety (Middledorp et al., 2005).

This study also partially supports results from two previous studies that suggest a strong relationship between psychiatric symptoms in family members and ASD comorbidity (Ghaziuddin & Greden, 1998; Mazefsky et al., 2008a). The logistic regression results indicated that approximately 75% of the probands could be correctly classified as having comorbid depression or anxiety or not based on current maternal symptoms of interpersonal sensitivity, hostility, phobic anxiety, depression, and anxiety. Ghaziuddin and Greden (1998) found that more than twice as many children with autism (77%) had comorbid depression with a parent's positive history than those that did not (30%). Additionally, Mazefsky et al. (2008a) found a similarly strong relationship among individuals whose mothers had a diagnosis of depression; 80% of children had comorbid depression compared to only 16% of children whose mothers did not have depression. These results all suggest that parent's psychiatric symptoms have a relationship with comorbidity in ASD that requires further study. However, there were some differences in the specific parental symptoms that were related to comorbidity. Most notably, this study did not find maternal depressive symptoms to account for a significant amount of variance in either comorbid depression or anxiety in their children with ASD.

All three of these studies employed different methodologies which need to be considered when interpreting the patterns of relationships between parental and child symptoms and any differences in findings. First, age groups included children (Ghaziuddin & Greden, 1998), pre-adolescents and adolescents (the current study), and adults (Mazefsky et al., 2008a). Second, Ghaziuddin and Greden (1998) employed an inpatient sample, which may have skewed their prevalence rates for both families and children with ASD. Third, there are many differences in how the comorbid and parental diagnoses were established. Specifically, Ghaziuddin and Greden (1998) based diagnoses on clinician judgment only, and Mazefsky et al. (2008a) used diagnostic assessments designed for typically-developing populations (SADS-L; Spitzer & Endicott, 1978) for both the probands and their relatives. Assessments used for typical populations may not reflect a distinct difference in psychiatric comorbidity among individuals with ASD (Leyfer et al., 2006). The present study used a measure specifically developed for assessing comorbidity in ASD to establish proband

diagnoses, which could be perceived as a strength over these earlier studies, but used a self-report measure for mood symptoms for the mothers. An additional important related difference is that maternal information used in the regressions in current study only included *current symptoms*, whereas the other two studies investigated parental *lifetime history of psychiatric disorders*. These differences represent the challenges that plague comparing previous research in this area, as many factors may have potentially influenced results.

Limitations of this study should be considered when interpreting results. The sample size was small and it was not an epidemiological sample, which may not provide accurate prevalence rates for probands. This study also only examined the current mood symptoms of mothers of individuals with ASD, which may not provide results as comparable or valid as those of psychiatric diagnoses from structured interview assessments (it was not possible to cross-check or confirm maternal ratings of their own psychiatric symptoms). There may also be a self-selection bias in this type of research; even though children with comorbid disorders were not specifically recruited, families of children with many emotional and behavioral concerns could have been more likely to find a study of this nature intriguing.

The fact that the mothers provided all information for sources of data (both for the SCL-90-R and for the ACI-PL) may also be a limitation, though this was felt to be the best approach when weighing the strengths and weaknesses of using self-report. Participants in this study were also part of a study aimed at better understanding self-report limitations in children with ASD (Mazefsky, Kao, & Oswald, 2010). Therefore, they each completed the ACI-PL themselves. However, participants were unable to provide the time course and severity information necessary to make diagnostic decisions, and had markedly limited insight into their own emotions and behaviors even when evidence existed to support maternal report (e.g. even children with hospitalizations for past suicide attempts did not endorse any depressive symptoms). This problem is common for comorbidity research (e.g., Mazefsky et al., 2008a) and reflects the difficulties in administering structured interviews to individuals with ASD. Incorporating teacher data or some other source of information would improve the design, but was not feasible due to the extensive time required to administer the ACI-PL and other measures.

Despite these limitations, this study builds on earlier research on family history and comorbidity in ASD, and adds to the very limited body of research on the *relationship* between maternal psychiatric diagnoses/symptoms and child comorbidity in ASD. The results provide preliminary evidence that maternal anxiety in particular may be related to comorbidity in their children with ASD. An increased knowledge of comorbidity in ASD and potential risk factors for it (such as certain familial psychiatric symptom patterns) could lead to the development of prevention measures, and earlier and more effective treatment for those affected. In addition, although most current ASD treatments are focused on the proband, it is possible that shedding light on this important issue could lead to family-based treatments that could improve overall quality of life and outcome.

Future research should utilize a larger sample in order to determine the validity of these preliminary findings. Future research on this topic would be most effective if both comorbid diagnoses and parental psychiatric history were established via standardized diagnostic interviews. Additionally, the contribution of subscales beyond depression and anxiety in the regressions implies that personality traits may contribute to family history and deserve further study. Finally, it would be helpful for future studies to include siblings and fathers as well in order to create a fuller understanding of potential genetic and shared environmental links.

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Table I

Proband and Mother Demographics (N=31)

		Mean (SD)	Range
Proband Age in Years		11.90 (1.94)	10-17
Race	*Caucasian*	87.1% (27)	
	African-American	3.2% (1)	
	Hispanic	3.2% (1)	
	Biracial	6.5% (2)	
Parental Marital Status	*Married*	77.4% (24)	
	Remarried	6.5% (2)	
	Single	9.7% (3)	
	Other	3.2% (1)	
	Missing	3.2% (1)	
Percent of Probands who were Male		80.6%	
Proband ABAS Adaptive Behavior Composite SS[a]		65.90 (14.88)	43-97
Proband WASI Verbal IQ		106.35(19.53)	71-147
Proband WASI Performance IQ		102.55 (16.40)	77-132
Proband WASI Full Scale IQ		104.84 (17.76)	71-144
Education Level[b] – Mother		2.19 (.87)	1 - 4

Note.

[a] SS = Standard Score (mean = 100; SD = 15);

[b] 1 = high school; 2 = college; 3 = graduate school; 4=other

Table II

SCL90-R Scores for Mothers (n = 31)

SCL90-R Indices	Mean (SD)	Range	% (#) with t ≥ 65[A]
Interpersonal Sensitivity	52.74 (8.59)	39-69	9.68% (3)
Depression	55.68 (9.50)	42-72	22.58% (7)
Anxiety	49.68 (10.68)	37-72	12.90% (4)
Hostility	54.55 (10.84)	40-74	25.81% (8)
Phobic Anxiety	47.87 (6.51)	44-65	3.22% (1)

Table III

Prevalence of Lifetime Comorbid Conditions in the Participants with ASD based on Mothers' Report on the Autism Comorbidity Interview

Comorbid condition (%)--only syndromal diagnosis unless otherwise indicated	Percent of current or past diagnosis (N)
Major depression	19.4 (6)
Any depression (syndromal and subsyndromal major depression, dysthymia, or depression NOS)	32.3 (10)
Generalized anxiety disorder	9.7 (3)
Transition/change-related anxiety	32.3 (10)
Social phobia	12.9 (4)
Specific phobia	12.9 (4)
Any DSM Anxiety (syndromal and subsyndromal generalized anxiety disorder, Social phobia, or specific phobia)	38.7 (12)

Note. All results represent a combination of current and past diagnoses.