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Characteristics Associated with Presence of Depressive Symptoms in Adults with Autism Spectrum Disorder

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Abstract Evidence suggests that individuals with autism spectrum disorders (ASD) often exhibit associated psychiatric symptoms, particularly related to depression. The current study investigated whether individual characteristics, specifically, severity of ASD symptoms, level of cognitive ability, and/or presence of other psychiatric disorders, are associated with occurrence of depressive symptoms in adults with ASD. Forty-six adults with ASD were administered a standardized psychiatric history interview. Twenty participants (43%) endorsed depressive symptoms. It was found that individuals with less social impairment, higher cognitive ability, and higher rates of other psychiatric symptoms, were more likely to report depressive symptoms. These characteristics may be vulnerability factors for the development of depression, and should be considered when screening and treating adults with ASD.

Keywords Autism · Depression · Anxiety · Comorbidity · Associated psychopathology · Cognitive ability

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Individuals with autism spectrum disorder (ASD) vary in terms of their symptom expression and level of functioning (DSM-IV; American Psychiatric Association (APA) 1994). Many individuals with ASD also suffer from associated medical and psychiatric conditions, which contribute to this variability (Gillberg & Billstedt 2000; Lainhart 1999). Because the core symptoms comprising ASD can cause substantial impairment for an individual, coexisting psychiatric symptoms are oftentimes not the primary focus of screening, diagnosis, or treatment. Research suggests, however, that such conditions can exacerbate the core symptoms of ASD, further compromising functioning, quality of life, and long-term outcome (Matson & Nebel-Schwalm 2006; Perry et al. 2001).

A variety of coexisting psychiatric symptoms have been commonly reported in individuals with ASD, including depression, mania, hyperactivity, inattention, aggression, obsessive–compulsive disorder, Tourettes disorder, specific phobias, and generalized anxiety (Gadow et al. 2004; Ghaziuddin et al. 1998; Green et al. 2000; Lecavalier 2006; Leyfer et al. 2006; Sverd 2003). A coexisting psychiatric disorder reportedly occurs in 65%–80% of individuals diagnosed with an ASD (de Bruin et al. 2006; Ghazziuddin et al. 1998; Leyfer et al. 2006), with rates tending to be higher than in groups of individuals with intellectual disability without autism (Brereton et al. 2006).

Evidence suggests that depressive symptoms are the most common psychiatric concern among individuals with ASD, and are more likely to occur in adolescence and adulthood (Ghaziuddin et al. 2002). In fact, the presence of depressive symptoms in adolescents and adults with ASD has been recognized since some of the earliest studies of autism and Asperger's Disorder. In a review of studies that followed children with ASD to adulthood, Rutter (1970) reported increased general behavioral difficulties in

adolescence, noting one patient who developed manic-depressive episodes in adolescence. Similarly, in a sample of 34 individuals with Asperger's Disorder, Wing (1981) reported "clinically diagnosable anxiety and varying degrees of depression… especially in late adolescence and adult life, which seem to be related to the painful awareness of handicap and difference from other people" (p. 118). Several studies and case reports have confirmed the high risk for depression in this population (Clarke et al. 1989; Ghaziuddin & Tsai 1991; Komoto et al. 1984; Lainhart 1999; Lainhart & Folstein 1994; Matson & Nebel-Schwalm 2006; Stewart et al. 2006; Skinner et al. 2005). Such symptoms may exacerbate the core ASD characteristics, resulting in reduced communication, social withdrawal, and an increase in psychomotor agitation, self-injurious behavior, stereotypic behavior, obsessive behavior, aggression, and sleep disturbance (Lainhart 1999; Perry et al. 2001).

Better understanding of coexisting psychiatric conditions such as depression is needed to improve accurate diagnosis and conceptualization of symptoms in individuals with ASD. A clear understanding of how these symptoms are expressed in individuals with ASD, in addition to the factors (individual, family, genetic, environmental) that are associated with vulnerability to developing these symptoms, would facilitate the development of a standardized and systematic method of assessment. If depression is found to be present, effective behavioral and pharmaceutical treatments exist that may provide benefit both to individuals with ASD and to those who care for them. For example, antidepressant medications have been used to successfully treat depressive symptoms (including observed depressed mood, sleep disturbance, self-injurious behavior, psychomotor agitation, weight loss, reduced communication, tearfulness, and loss of interest) in individuals with ASD (Perry et al. 2001).

While there is clearly a growing body of literature that has documented psychiatric disorders in ASD, particularly depression, there have been fewer studies that have investigated potential risk and protective factors related to the development of depressive symptoms in this population. Ghaziuddin and Greden (1998) reported that children with ASD presenting with depression are more likely to have a family history of depression. Others (Bolten et al. 1998; Piven & Palmer 1999) have reported high rates of major depression in non-autistic relatives of individuals with ASD, which do not appear to be accounted for by the stress of raising a child (or children) with autism. Similar to findings with the general population (e.g., Kendler et al. 1999; Patton et al. 2003), significant negative life events, such as family sickness and bereavement, may contribute to the occurrence of depression in children with ASD (Ghaziuddin et al. 1995).

With regard to individual factors, Wing (1981) suggested that those with more social awareness are more likely to experience depressive symptoms. More recently, investigators (Meyer et al. 2006) have examined whether certain patterns of social information processing are related to the development of coexisting psychiatric conditions, including anxiety and depression in ASD. For example, Meyer and colleagues suggested that a negative attributional style may be linked to poor psychosocial adjustment and hence higher levels of internalizing and externalizing psychopathology in a sample of 7–13 year-old children with Asperger's Disorder. Importantly, children in the study showed the ability to interpret and make attributions about social situations, and had enough insight to self-report about social problems. Such findings imply that many children with Asperger's Disorder have an awareness of their social difficulties and lack of success in the social arena. Awareness of one's own role in failed social situations and assumption of responsibility for negative events contributes to lower self-esteem and discouragement, which may increase the risk for depression in an individual with ASD (Barhill & Smith Myles 2001). Similarly, in a sample of adolescents with Asperger's Disorder, it was reported that participants attributing school-related social failure to their own abilities (rather than effort, chance, or task difficulty) were more likely to experience depressive symptoms (Barnhill 2001). Other research (Capps et al. 1995; Tantam 2000) has corroborated the suggestion that higher functioning individuals with ASD are aware of their social difficulties and that this awareness may lead to increased distress and the development of associated psychiatric conditions.

Reports of elevated levels of psychiatric conditions particularly among individuals with Asperger's Disorder (Frith 2004; Khouzam et al. 2004; Meyer et al. 2006; Szatmari et al. 1989; Tonge et al. 1999) suggest that level of intelligence may also be related to the presence of depression and anxiety in this population. In an intervention study conducted with boys with ASD, Solomon et al. (2004) reported that children with higher cognitive ability were less likely to demonstrate improvement on a measure of depression than boys of lower cognitive ability. In another study examining children with depressive symptoms, depressed patients were found to have higher intellectual ability than non-depressed patients (Ghazziudin & Greden 1998). To date, there have been few studies that have directly investigated whether higher intellectual ability or increased social awareness are associated with higher rates of depressive symptoms in adults with ASD.

In summary, depressive symptoms are common among adolescents and adults with ASD. Furthermore, based on the available studies conducted primarily with children and adolescents with ASD, both higher cognitive ability and

higher levels of social functioning appear to be vulnerability factors for the development of depressive symptoms. In the present study, we sought to determine whether these same factors are associated with depression in adults with ASD. Following from previous studies, we hypothesized that adults with ASD who have higher cognitive ability and social functioning would be more likely to endorse experiencing depressive symptoms than adults with ASD who have lower cognitive ability and social functioning.

Methods

Participants were assessed at the University of Washington Autism Center as part of the NIMH STAART Center of Excellence Study or the NICHD CPEA Family Study of Autism (U54MH066399N or U19HD34565, Geraldine Dawson, PI). Potential participants were excluded for having identifiable genetic disorders associated with autism (fragile X syndrome, Norrie syndrome, neurofibromatosis, tuberous sclerosis), sensory or motor impairment, seizures, cerebral palsy, major physical abnormalities, current psychoactive medications, or inability to undergo an MRI (as part of another study). Forty-six adults with ASD, ranging in age from 18 to 44 years (M = 23.70, SD = 7.21), participated. Participant self-report of race and ethnicity yielded the following information: 44 Non-Hispanic or White, 1 African American, 1 more than one race. After complete description of the study, written informed consent was obtained.

Clinicians administering the measures were blind with respect to the hypothesis being tested in this study. All participants were diagnosed with an autism spectrum disorder using a parent interview, the Autism Diagnostic Interview-Revised (ADI-R; Lord et al. 1994), which uses criteria for autism according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; APA 1994) and the Autism Diagnostic Observation Schedule (ADOS), a semi-structured observation and interview administered directly to the participant. Participants interviewed as part of the NIMH STAART Center of Excellence Study (n = 37) were given the WPS version of the ADOS (ADOS–WPS; Lord et al. 2000), and the 9 participants interviewed as part of the NICHD CPEA Family Study of Autism were administered the Generic version of the ADOS (ADOS-G). The subset of items being compared on the ADOS in the current study did not differ between the two versions. The ADOS yields a total score as well as separate communication and social domain scores. The communication domain measures symptoms related to verbal and nonverbal communication (e.g., speech abnormalities, stereotypic speech, gestures), while the social domain measures symptoms related to empathy, insight, social reciprocity, use of social overtures, and social response to others. All participants in the current study were administered Module 4 of the ADOS, given their ability to use fluent speech. Inter-rater reliability was assessed by double coding a subset of the sample using procedures outlined by Shrout and Fleiss (1979); intraclass correlations were .88 or higher on all ADOS and ADI subscales. IQ was estimated using four subtests of the Wechsler Adult Intelligence Scale- Third Edition (WAIS-III; Wechsler 1997): Vocabulary, Comprehension, Block Design, and Object Assembly.

Psychiatric symptoms were assessed using a standardized psychiatric history interview. The University of Washington research team adapted a Family History Interview developed by Rutter and Folstein (1995), by adding research diagnostic criteria (FH RDC; Andreasen et al. 1977) for psychiatric conditions not originally included in the interview, including depression, manic-depressive disorder, anxiety disorders, obsessive–compulsive disorder (OCD), drug and alcohol abuse, conduct disorders, and psychosis. Specific probes for each interview item, as well as criteria used for coding each item, were based on DSM-IV (APA 1994) definitions. A trained clinician administered the 15–30 min interview with each participant. Clinicians evaluated the participant's depressive symptoms, including feelings of misery, loss of interest, sleep and appetite disturbance, and feelings of self-blame and hopelessness, as well as associated impairment and/or history of professional treatment for depression. Based on self-report of symptoms, clinicians assigned the participant to one of the following categories of depression: (a) None (no depressive symptoms; n = 26); (b) Probable but insufficient information to meet criteria for more severe levels of depression (n = 4); (c) Definite disorder, including at least one month of unhappiness, anhedonia and social impairment or treatment (social impairment includes missing school or having decreased performance at school or other activities, or decrease in socialization; n = 14); (d) Hospitalized for at least 2 days or electro-convulsive therapy (ECT) before age 16 (n = 1); or (e) Definite episode of at least 2 weeks, but less than 1 month (n = 1). Participants in categories 2 through 5, endorsing symptoms of depression, were collapsed into the 'depressed' group. This group consisted of 20 individuals (43% of the participants). The 'nondepressed' group consisted of the 26 individuals who did not endorse any symptoms related to depression. Table 1 presents the gender and ages of both groups. The two groups differed significantly on age, $t(44) = -2.76$, $p < .01$, such that on average, the depressed group contained older individuals.

Symptoms related to general anxiety and OCD were assessed according to a similar system, described in more detail below. The number of participants in each group (depressed and nondepressed) endorsing symptoms related

Table 1 Gender and age for depressed and nondepressed groups

	Depressed (n = 20)	Nondepressed (n = 26)
Gender		
Male	19	23
Female	1	3
Age (years)		
Mean(SD)	26.82(8.76)	21.30(4.62)
Range	18.33–44.75	18.00–35.08

to anxiety and OCD are presented in Table 2. To evaluate general anxiety, participants were asked about their overall level of fear, anxiety, and worry, in addition to experiences of agoraphobia and panic attacks. Categories included (a) None; (b) Probable, but insufficient information to meet criteria for more severe anxiety; (c) Chronic or recurrent severe anxiety, agoraphobia or panic, with at least one clear example of observable behaviors (e.g., staying at home, reassurance seeking, trembling, pacing, treatment); (d) Hospitalized for at least 2 days or ECT before age 16; or (e) Subthreshold level of symptoms (significant but not definite symptoms). In order to evaluate symptoms related to OCD, participants were asked whether they have specific rituals or routines he or she needs to carry out, or thoughts that he or she cannot get out of mind that are inappropriate or disturbing. Categories for self-report of OCD related symptoms included (a) None; (b) Probable, but insufficient information to meet criteria for more severe OCD; (c) Definite symptoms; or (d) Subthreshold level of symptoms.

Table 2 Number of participants in each group endorsing symptoms related to general anxiety and obsessive–compulsive disorder

Symptoms	Depressed (n = 20)	Nondepressed (n = 26)
Anxiety disorder total	9	4
None	11	22
Probable	3	0
Chronic or recurrent severe anxiety	5	2
Hospitalized at least 2 days or ECT	1	0
Subthreshold level of symptoms	0	2
Obsessive–compulsive disorder total	11	5
None	9	21
Probable	5	3
Definite	4	0
Subthreshold, significant but not definite	2	2
Endorsing both anxiety and OCD symptoms	4	1

Results

Cognitive Ability

To assess whether the depressed and nondepressed groups differed in terms of cognitive ability, independent samples t-tests were conducted, comparing Full Scale, Performance, and Verbal WAIS scores by group. Table 3 presents the IQ scores for each group and levels of statistical significance for each analysis. Independent samples t-tests revealed significant cognitive differences between groups, such that on average, individuals who endorsed symptoms of depression had significantly higher overall Full Scale cognitive scores and significantly higher Verbal abilities as estimated by the WAIS. Although the depressed group also showed a trend toward higher Performance IQ scores, this difference was not significant. The results suggest that in this sample, individuals endorsing symptoms of depression had higher levels of cognitive ability, with IQs in the average to above average range.

Symptom Severity

Independent samples t-tests were also conducted in order to assess for group differences in terms of symptom severity; groups were compared on the ADOS total score, ADOS communication domain score, and ADOS social domain score. Table 4 presents ADOS scores and significance values for each group. Results indicate that, on average, individuals who endorsed depressive symptoms had significantly fewer symptoms on the ADOS social domain (i.e., lower ADOS scores). Although the depressed group also tended to have fewer symptoms on the ADOS communication domain, the difference between groups was not significant. Combining the social and communication domains yielded a significant difference, such that the depressed group had fewer symptoms overall, across the

Table 3 WAIS scores for depressed and nondepressed groups

	Depressed	Nondepressed	t-value	Sig. (2-tailed) (df = 44)
Full Scale IQ				
Mean(SD)	112.10(18.01)	98.38(22.17)	−2.252	.029
Range	85–139	57–134		
Performance IQ				
Mean(SD)	109.80(18.09)	99.81(21.06)	−1.694	.097
Range	50–133	50–133		
Verbal IQ				
Mean(SD)	111.45(18.13)	97.35(21.98)	−2.324	.025
Range	76–140	52–140		

Table 4 ADOS scores for depressed and nondepressed groups

	Depressed	Nondepressed	*t*-value	Sig. (2-tailed) (df = 44)
Social domain				
Mean(SD)	7.45(1.96)	9.31(2.94)	2.439	.019
Range	4–12	4–14		
Comm. domain				
Mean(SD)	3.45(1.50)	4.12(1.53)	1.472	.148
Range	0–6	2–7		
Social + Comm.				
Mean(SD)	10.85(2.58)	13.42(3.96)	2.519	.015
Range	6–16	7–20		

two domains. On average, both groups exhibited more impaired social scores than communication scores.

Symptom Severity Among Individuals with Higher Cognitive Ability

It is possible that higher cognitive and verbal ability facilitates better capability to self-report symptoms. To address the question of whether the association between improved social abilities and depressive symptoms would be found even among individuals with comparable levels of verbal ability, we re-ran the analyses, excluding all individuals with full scale IQs in the below average range. Thus, data only from those participants with cognitive abilities in the average to high range ($\leq$1 SD below the mean on the WAIS) were included. This average-high IQ group consisted of 39 participants (20 depressed, 19 nondepressed), with IQs ranging from 85–139. Independent samples *t*-tests revealed that individuals in the depressed group still demonstrated significantly fewer social symptoms on the ADOS social domain, $t(37) = 2.30$, $p = .027$. Evaluation of symptoms across communication and social domains suggested that the depressed group continued to have fewer symptoms overall, although this difference only approached significance, $t(37) = 2.00$, $p = .053$. Thus, even within a group that scored at or above the average range on cognitive testing, those who endorsed depressive symptoms tended to have fewer communicative and social impairments based on the ADOS.

Additional Psychopathology

Finally, to determine whether the presence of other psychiatric symptoms was associated with higher levels of depressive symptoms, we examined the percentage of anxiety and obsessive–compulsive related symptoms for the depressed and nondepressed groups. Almost half (45%) of the individuals endorsing depressive symptoms also endorsed symptoms related to general anxiety, whereas only 15% of the nondepressed group endorsed such symptoms. Non-parametric (Pearson chi-square) tests revealed that these proportions significantly differed by group, $\chi^2(1, N = 46) = 4.89$, $p = .027$. Similarly, a significantly greater proportion of the depressed group (55% versus only 19% of the nondepressed group) endorsed symptoms related to OCD, $\chi^2(1, N = 46) = 6.38$, $p = .012$. There was also a significantly greater proportion of individuals in the depressed group (20%) who reported symptoms of *both* anxiety and obsessive–compulsive disorder, as compared to the nondepressed group (4%), $\chi^2(2, N = 46) = 7.28$, $p = .026$.

Discussion

The present study investigated individual characteristics associated with depressive symptoms in adults with autism spectrum disorder. Our analyses indicated that among a group of adults with ASD, 43% endorsed significant levels of depressive symptoms. These individuals were found to have higher cognitive abilities as estimated by the Verbal and Full Scale scores on the WAIS, and showed less impaired social functioning as indicated by fewer symptoms on the social domain of the ADOS. Even within a group of individuals who scored at or above average on cognitive testing, follow-up analyses confirmed that individuals endorsing depressive symptoms exhibited fewer social impairments. These findings demonstrate that having better social skills was a specific vulnerability factor associated with the presence of depressive symptoms in adults with ASD.

The finding that higher functioning individuals, particularly those with more intact social skills, are more likely to endorse symptoms of depression has important clinical implications. Individuals with higher cognitive abilities and better social skills may have more awareness of their difficulties in social settings and of their differences from peers. A study by Hedley and Young (2006) supported this notion. They found that perception of dissimilarity between self and others was related to depressive symptoms in a sample of high functioning individuals with ASD. Such individuals may be expected to cope with more complex and challenging social situations, which they might find stressful. Social expectations, imposed on themselves or by others, may be higher and more difficult to achieve for these individuals. Previous research has suggested that high functioning individuals with ASD, particularly those with Asperger's Disorder, have more interest in social interaction and relationships, but may not have the skills

necessary to execute successful social relations (Ozonoff et al. 2002). Having the motivation and desire to form social relationships, combined with impairments in the skills necessary to do so, can lead to failed attempts to interact and the inability to form successful relationships. Repeated rejection coupled with lack of understanding, insight, or skills needed to modify strategies, could certainly lead to the development of depressive symptoms.

Interestingly, it was found that the group of individuals who endorsed symptoms of depression was significantly older than those who did not. This is consistent with previous findings. In their review of depression in individuals with autism, Ghaziuddin et al. (2002) noted that based on clinical experience, the rate of depression rises with age. Perhaps as individuals with ASD reach early and even middle adulthood, societal expectations to establish significant relationships, complete their education, become independent, and have a job begin to have more impact. Especially for those who are higher functioning and who have performed relatively well in school and in other childhood activities, the early 20s and beyond can mark a period of time in which the developmental gap between themselves and same-aged peers becomes more pronounced. Stress incurred in the transition to adulthood may add to difficulties relating to peers. Adults with ASD may be aware that they are behind their peers developmentally, express desire to achieve adult developmental milestones, but do not have the adaptive skills or the executive planning abilities to take the necessary steps toward independent living. Feelings of depression may also impact the motivation needed to make these life changes. In addition, by the time an individual with ASD reaches adulthood, he or she may have incurred a long history of negative social experiences and peer rejection. For example, in a sample of children ages 8–14, Bauminger and Kasari (2000) reported high levels of loneliness and poor quality of friendships. A history of experiencing unsuccessful peer interactions and social isolation may contribute to poor self-esteem, frustration, and other feelings related to depression (Tantam 2000).

This study found that individuals with ASD who endorse depressive symptoms are also significantly more likely to have symptoms related to general anxiety and obsessive–compulsive disorder (OCD). Depression, anxiety, and OCD are highly co-morbid in the general population as well (McLaughlin et al. 2006; Overbeek et al. 2002; Pollack 2005; Tukel et al. 2002). The presence of anxiety and rituals or compulsions may exacerbate difficulties in adults with ASD and further impair functioning. In their review of case reports in the literature, Stewart et al. (2006) suggested that for individuals with ASD, symptoms of OCD, aggression, and even self-injury can increase during an episode of depression. Clinicians involved with screening, diagnosis, and treatment of adults with ASD should be aware of the possibility that these symptoms can cluster together and have the potential to cause substantial impairment.

In addition, professionals should recognize that individuals with ASD suffering from depression and related difficulties may have thoughts related to suicide and intentionally harm themselves. In a review of psychiatric disorders in ASD, Lainhart (1999) noted that individuals with ASD who are higher functioning may attempt or commit suicide, especially when experiencing frustration or depressive symptoms. In fact, suicidal thoughts and attempts have previously been reported in individuals with ASD (Lainhart & Folstein 1994; Wing 1981). Given that individuals with ASD do not consistently self-report or confide in others when experiencing depressed mood (Stewart et al. 2006), it is imperative that such behaviors are thoroughly assessed during clinical evaluation or treatment. The high rate of depression among the current sample underscores the need for screening for suicidal thoughts when appropriate. A total of 20 out of 46 participants in our sample endorsed symptoms related to depression, whereas only 13 (9 in the depressed group and 4 in the nondepressed group) endorsed symptoms related to anxiety, which are often thought to be more commonly associated with ASD. In this sample of adults, it is the depressive symptoms that are more prevalent and deserve attention.

It is worth noting that in the current sample, potential participants were excluded for taking psychoactive medications. This may have resulted in screening out potential participants already being treated for depression. The high proportion (43%) of participants endorsing depressive symptoms in this sample may actually be an underestimate of the rate of depression in adults with ASD. Future studies are needed to determine prevalence and better understand depressive symptom expression by conducting psychiatric diagnosis in individuals with ASD. The development of promising measures to assess psychiatric symptoms in children with ASD has recently been a focus of research (e.g., Leyfer et al. 2006), which has contributed to our knowledge regarding prevalence of coexisting psychiatric disorders in children. Future research endeavors should continue to focus on reliable assessment measures and further characterization of depressive symptoms in adults with ASD.

In summary, the findings from this study suggest that higher cognitive ability and social functioning might constitute vulnerability factors for the development of depression in individuals with ASD. As such, clinicians should be aware that as children with ASD improve their cognitive and social functioning as a result of early intervention, increased vigilance for the emergence of

depressive symptoms is warranted. These results underscore the need for professionals to attend to signs of depression in higher functioning individuals, and to consider these symptoms when diagnosing, referring, and developing treatment plans.

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References

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington DC.

Andreasen, N. C., Endicott, J., Spitzer, R. L., & Winokur, G. (1977). The family history method using diagnostic criteria. Reliability and validity. *Archives of General Psychiatry, 34*, 1229–1235.

Barnhill, G. P. (2001). Social attributions and depression in adolescents with Asperger syndrome. *Focus on autism and other developmental disabilities, 16*, 46–53.

Barhill, G. P., & Smith Myles, B. (2001). Attributional style and depression in adolescents with Asperger syndrome. *Journal of Positive Behavior Interventions, 3*, 175–182.

Bauminger, N., & Kasari, C. (2000). Loneliness and friendship in high-functioning children with autism. *Child Development, 71*, 447–456.

Bolten, P. F., Pickles A., Murphy M., & Rutter, M. (1998). Autism, affective and other psychiatric disorders: Patterns of familial aggregation. *Psychological Medicine, 28*, 385–395.

Brereton, A., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders, 36*, 863–870.

Capps, L., Sigman M., & Yirmya, N. (1995). Self-competence and emotional understanding in high functioning children with autism. *Development and Psychopathology, 7*, 137–149.

Clarke, D. J., Littlejohns, C. S., Corbett, J. A., & Joseph, S. (1989). Pervasive developmental disorders and psychoses in adult life. *British Journal of Psychiatry, 155*, 692–699.

de Bruin, E. I., Ferdinand, R. F., Meester, S., de Nijs, P. F. A., & Verheij, F. (2006). High rates of psychiatric co-morbidity in PDD-NOS. *Journal of Autism and Developmental Disorders, 37*, 877–886.

Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*, 672–686.

Gadow, K., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders, 34*, 379–393.

Green, J., Gilchrist, A., Di Burton, & Cox, A. (2000). Social and psychiatric functioning in adolescents with Asperger Syndrome compared with conduct disorder. *Journal of Autism and Developmental Disorders, 30*, 279–293.

Ghaziuddin, M., Alessi, N., & Greden, J. F. (1995). Life events and depression in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 25*, 495–502.

Ghaziuddin, M., Ghazziuddin, N., & Greden, J. (2002). Depression in adults with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, 32, 299–306.

Ghaziuddin, M., & Greden, J. (1998). Depression in children with autism/pervasive developmental disorders: A case-control family history study. *Journal of Autism and Developmental Disorders*, 28, 1998.

Ghaziuddin, M., & Tsai, L. (1991). Depression in autistic disorder. *British Journal of Psychiatry, 159*, 721–723.

Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disabilities Research*, *4*, 279–283.

Gillberg, C., & Billstedt, E. (2000). Autism and Asperger syndrome: coexistence with other medical disorders. *Acta Psychiatrica Scandinavica, 102*, 321–330.

Hedley, D., & Young, R. (2006). Social comparison processes and depressive symptoms in children and adolescents with Asperger syndrome. *Autism, 10*, 139–153.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *The American Journal of Psychiatry, 156*, 837–841.

Khouzam, H. R., El-Gabalawi, F., Pirwani, N., & Priest, F. (2004). Asperger's disorder: A review of its diagnosis and treatment. *Comprehensive Psychiatry, 45*, 184–191.

Komoto, J., Usui, S., & Hirata, J. (1984). Infantile autism and affective disorder. *Journal of Autism and Developmental Disorders, 14*, 81–84.

Lainhart, J. E. (1999). Psychiatric problems in individuals with autism, their parents, and siblings. *International Journal of Psychiatry, 11*, 278–298.

Lainhart, J. E., & Folstein, S. E. (1994). Affective disorders in people with autism: A review of published cases. *Journal of Autism and Developmental Disorders, 24*, 587–601.

Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders*, *36*, 1101–1114.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders, 36*, 849–861.

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2000). *Autism diagnostic observation schedule manual*. Los Angeles: Western Psychological Services.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview–revised: A revised version of the autism diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*, 659–685.

Matson, J. L., & Nebel-Schwalm, M. S. (2006). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities*, *28*, 341–352.

McLaughlin, T. P., Khandker, R. K., Kruzikas, D. T., & Tummala, R. (2006). Overlap of anxiety and depression in managed care population: prevalence and association with resource utilization. *The Journal of Clinical Psychiatry, 67*, 1187–1193.

Meyer, J. A., Mundy, P. C., Van Hecke, A. V., & Durocher, J. S. (2006). Social attribution processes and comorbid psychiatric symptoms in children with Asperger syndrome. *Autism, 10*, 383–402.

Overbeek, T., Schruers, K., Vermetten, E., & Griez, E. (2002). Comorbidity of obsessive-compulsive disorder and depression: prevalence, symptom severity, and treatment effect. *The Journal of Clinical Psychiatry, 63*, 1106–1112.

Ozonoff, S., Dawson, G., & McPartland, J. (2002). *A parent's guide to Asperger syndrome and high-functioning autism*. New York: Guilford Press.

Patton, G. C., Coffey, C., Posterino, M., Carlin, J. B., & Bowes, G. (2003). Life events and early onset depression: cause or consequence? *Psychological Medicine, 33*, 1203–1210.

Perry, D. W., Marston, G. M., Hinder, S. A., Munden, A. C., & Roy, A. (2001). The phenomenology of depressive illness in people with a learning disability and autism. *Autism, 5*, 265–275.

Piven, J., & Palmer, P. (1999). Psychiatric disorder and the broad autism phenotype: Evidence from a family study of multiple-incidence autism families. *The American Journal of Psychiatry, 156*, 557–563.

Pollack, M. H. (2005). Comorbid anxiety and depression. *The Journal of Clinical Psychiatry, Supp. 8*, 22–29.

Rutter, M. (1970). Autistic children: infancy to adulthood. *Seminars in Psychology, 2*, 435–450.

Rutter, M., & Folstein, S. (1995). Modified autism family history interview for developmental disorders of cognition and social functioning. Baltimore: The Johns Hopkins University School of Medicine.

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: uses in assessing rater reliability. *Psychological Bulletin, 86*, 420–428.

Skinner, S. R., Ng, C., McDonald, A., & Walters, T. (2005). A patient with autism and severe depression: medical and ethical challenges for an adolescent medicine unit. *The Medical Journal of Australia, 183*, 422–424.

Solomon, M., Goodlin-Jones, B. L., & Anders, T. F. (2004). A social adjustment enhancement intervention for high functioning autism, Asperger's syndrome, and pervasive developmental disorder NOS. *Journal of Autism and Developmental Disorders, 34*, 649–668.

Sverd, J. (2003). Psychiatric disorders in individuals with pervasive developmental disorder. *Journal of Psychiatric Practice, 9*, 111–127.

Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: a review. *Autism, 10*, 103–116.

Szatmari, P., Bartolucci, G., & Bremmer, R. (1989). Asperger's syndrome and autism: comparison of early history and outcome. *Developmental Medicine and Child Neurology, 31*, 709–720.

Tantam, D. (2000). Psychological disorder in adolescents and adults with Asperger syndrome. *Autism, 4*, 47–62.

Tonge, B. J., Brereton, A. V., Gray, K. M., & Einfeld, S. L. (1999). Behavioural and emotional disturbance in high-functioning autism and Asperger syndrome. *Autism, 3*, 117–130.

Tukel, B., Polat, A., Ozdemir, O., Aksut, D., Turksoy, N. (2002). Comorbid conditions in obsessive-compulsive disorder. *Comprehensive Psychiatry, 43*, 204–209.

Wechsler, D. (1997). WAIS-III: Administration and Scoring Manual: Wechsler Adult Intelligence Scale. Psychological Corporation.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine, 11*, 115–129.