Varied presentations of emotion dysregulation in autism complicate diagnostic decision making and may lead to inaccurate psychiatric diagnoses or delayed autism diagnosis for high-functioning children. This pilot study aimed to determine the concordance between prior psychiatric diagnoses and the results of an autism-specific psychiatric interview in adolescents with high-functioning autism. Participants included 35 predominantly Caucasian and male verbal 10- to 17-year-olds with a confirmed autism spectrum disorder and without intellectual disability. The average age of autism spectrum diagnosis was 11 years old. Lifetime psychiatric diagnoses were established via the Autism Comorbidity Interview, developed to identify comorbid conditions within the context of autism. Autism Comorbidity Interview results were compared to parent report of prior community psychiatric diagnoses. Approximately 60% of prior psychiatric diagnoses were not supported on the Autism Comorbidity Interview; the lowest diagnostic concordance was for prior bipolar disorder and obsessive-compulsive disorder diagnoses. Although 51% of children met Autism Comorbidity Interview criteria for at least one psychiatric disorder, rates of prior diagnoses were much higher, with 77% having at least one prior psychiatric diagnosis and 60% having two or more. Although many participants met criteria for comorbid psychiatric disorders, the majority of previous...
Studies are converging on the finding that the majority of children with autism spectrum disorders (ASD) meet criteria for at least one concurrent psychiatric disorder. In a population-based sample of 10- to 14-year-old children, 71% of children with ASD met criteria for at least one current psychiatric disorder, 41% had two or more, and 24% had three or more diagnoses (Simonoff et al., 2008). These numbers are quite similar to studies with wider age ranges that found 72% (Gjevik, Eldevik, Fjeran-Granum, & Sponheim, 2011) and 71% (Leyfer et al., 2006) of children with ASD had at least one comorbid disorder. Comorbidity rates in samples of referred youth are even more striking, and substantially higher than children in psychiatric clinics without ASD. For example, Joshi et al. (2010) found that children with ASD referred to a pediatric psychopharmacology program met criteria for an average of 6.4 comorbid diagnoses, with 95% of these children meeting criteria for three or more diagnoses.

There are several mechanisms that could lead to the co-occurrence of two separate disorders, such as shared environmental or biological risk factors (Caron & Rutter, 1991). For example, family history studies have highlighted the high rates of affective disorders in relatives of children with ASD, raising the possibility of a genetic link (e.g., Mazefsky, Folstein, & Lainhart, 2008). Having one disorder can also increase risk of a second disorder. For example, more negative feedback from peers, awareness of social difficulties, and so on, may contribute to the co-occurrence of ASD and social anxiety disorder (White, Bray, & Ollendick, 2011).

Alternatively, it is possible that what is considered comorbidity actually reflects poor diagnostic boundaries and overlapping symptoms (Caron & Rutter, 1991). In this case, the “comorbid disorder” may actually be a manifestation of impairment related to the “primary” disorder. Many disorders, and ASD in particular, are quite heterogeneous, which further contributes to nosological complexities. In addition, variable progression of symptom presentation in the same individual over time may contribute to the diagnosis of separate disorders that are in fact reflective of the same underlying pathology.

Studies of the manifestations of ASD in infancy suggest that disturbances in the capacity for self-regulation are apparent early in the course of ASD. Specifically, infants later diagnosed with ASD exhibit more intense and frequent distress reactions by 12 months, greater levels of irritability and negative affect by 36 months, and higher activity levels compared to non-ASD siblings and typically developing controls (Garon et al., 2009; Zwaigenbaum et al., 2005). In addition, sleep problems, which often begin in the 1st year of life, are significantly more common and severe in ASD than in typically developing children (Sounders et al., 2009).

Because emotional and behavioral dysregulation is so interfering and difficult to address, it may overshadow diagnostic symptoms of ASD, contributing to inappropriate treatments and delayed diagnosis of ASD. A study of Medicaid claims found that only 43% of children with ASD were diagnosed as such on their first mental health visit (Mandell, Ittenbach, Levy, & Pinto-Martin, 2007). In addition, 63% of children enrolled in three studies of childhood bipolar disorder, severe mood dysregulation, major depression, and/or anxiety disorders scored in the “ASD-likely range” on at least one ASD screening measure, despite these studies excluding children with ASD diagnoses (Towbin, Pradella, Gorrindo, Pine, & Leinbenlifu, 2005). A study of behavioral psychiatry clinics found that nearly 30% of children presenting as and diagnosed with conduct disorder had undiagnosed ASD (Gilmour, Hill, Place, & Skuse, 2004). Taken together, these studies suggest that children with ASD often first receive mood or behavioral disorder diagnoses. However, it is unclear whether these diagnoses reflect accurate comorbidity or if they were misattributions of ASD-related impairment.

There is a new opportunity to address such questions with the development of an interview designed to more carefully tease apart ASD-related impairment from psychiatric comorbidity, called the Autism Comorbidity Interview (ACI; Lainhart, Leyfer, & Folstein, 2003; Leyfer et al., 2006). The ACI is a modification of the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997) for developmentally disabled and/or ASD populations. Utilizing a measure developed for ASD is important for accurate differential diagnosis. Otherwise, some symptoms that are taken as evidence of a secondary disorder may in fact be better explained by associated features of the ASD itself, just as some symptoms mistakenly attributed to the ASD may be manifestations of new comorbid disorders.

The ACI provides a more accurate measure of the behavioral manifestation of psychiatric comorbidity by gathering information on symptoms that cluster together in time, are significantly impairing, and, when acute,
represent a change from the child’s baseline emotional state, behavior, and functioning (Lainhart et al., 2003; Leyfer et al., 2006). The ACI accomplishes this by including an introductory section that assesses the child’s emotions and behaviors at baseline and explanations of differential diagnosis considerations in ASD. Screening questions were added to capture the unique ways that disorders may manifest in ASD (e.g., added inquires about increased agitation and self-injury when probing possible depression). Applicability of symptoms is determined before something is coded as “not present” (e.g., a child must be considered capable of expressing a symptom). Additional probing is included to make sure that ASD-related impairments are not considered evidence of a new disorder (e.g., questions to make sure separation anxiety is due to attachment to the caregiver and not avoidance of social or nonsocial aspects of the situation). Caregivers are also asked how difficult they find the child’s symptoms to control and how distressing the symptoms are, given that in many cases children with ASD may be unable or not inclined to report such concepts themselves.

No previous studies, to our knowledge, have administered structured, ASD-modified psychiatric diagnostic interviews to children with ASD and compared the findings to their history of prior diagnoses to help clarify the level of concordance. Thus, this was a pilot study to determine the concordance between prior psychiatric diagnoses in children with high-functioning ASD (HFASD) and the results of an ASD-specific psychiatric interview. Determining the concordance or lack thereof between this type of assessment and psychiatric diagnosis (HFASD) and the results of an ASD-specific psychiatric interview. The focus on “higher functioning” individuals with ASD in this initial study was due to this population presenting a particular challenge related to diagnosis (in comparison to when children have significant early delays or very frank autism symptoms). Most (80%) participants were newly diagnosed with ASD prior to entering the study, with an average age of first ASD diagnosis of almost 11.

Participants were thirty-five 10- to 17-year-old children with HFASD (see Table 1). Clinical diagnoses included autistic disorder; Asperger’s disorder; or pervasive developmental disorder, not otherwise specified. Participants were excluded if they had less than fluent nonechoed speech or intellectual disability. Participants’ mean Full-Scale IQ was in the average range and their mean adaptive behavior composite was in the impaired range.

Participants were recruited through word of mouth and fliers at a developmental disorders diagnostic clinic at a Midwest children’s hospital, and four participants were recruited at a children’s psychiatric hospital with an ASD clinic. Both hospitals are in small cities surrounded by rural communities. Recruitment materials advertised the study as aimed to better understand the types of difficulties that adolescents with HFASD face; psychiatric comorbidity was not incorporated into recruitment efforts. All participants’ parents/guardians provided informed consent to participate, and participants provided assent. Participants were provided with a $35 incentive and assessment results. No families turned down participation once they were informed of the study procedures. All eligible participants completed the entire study, with the exception of one 14-year-old male whose psychiatric comorbidity interview was never completed due to repeated failed appointments. Family income was not gathered, but most mothers had college or higher degrees, and there was a fairly even distribution among fathers of high school, college, and graduate degrees. Most (69%) of the participants were living with biological, married parents.

ASD diagnoses were supported by Module 3 or 4 of the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000), a structured interactive assessment for ASD, and the Autism Diagnostic Interview–Revised (ADI–R; Lord, Rutter, & LeCouteur, 1994), a caregiver interview. These instruments were administered by individuals with research-level reliability, and all diagnoses were confirmed by the expert opinion of a licensed clinical psychologist who specializes in ASD. Participants satisfied Diagnostic and Statistical Manual of Mental Disorders (4th ed., text. rev. [DSM–IV–TR]; American Psychiatric Association, 2000) criteria for their respective ASD diagnoses. The mean social and communication

### TABLE 1

<table>
<thead>
<tr>
<th>Demographic Characteristics of Participants (n = 35)</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Age</td>
<td>12.11</td>
<td>2</td>
<td>10–17</td>
</tr>
<tr>
<td>Age When First Diagnosed With ASD</td>
<td>10.89</td>
<td>3</td>
<td>3–16</td>
</tr>
<tr>
<td>WASI Full-Scale IQ</td>
<td>105</td>
<td>17</td>
<td>71–144</td>
</tr>
<tr>
<td>ABAS–II Adaptive Behavior Composite</td>
<td>67</td>
<td>15</td>
<td>43–97</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>%</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>80.0%</td>
</tr>
<tr>
<td>Caucasian Race</td>
<td>88.6%</td>
</tr>
</tbody>
</table>

Note: ASD = autism spectrum disorders; WASI = Wechsler Abbreviated Scale of Intelligence (Psychological Corporation, 1999); ABAS–II = Adaptive Behavior Assessment System–II (Harrison & Oakland, 2003).
total ADOS score was 12 (e.g., above the autism cutoff), with a standard deviation of 3. Only two participants were below the ADOS cutoff for ASD. Careful clinical reviews were conducted to ensure these participants had ASD, and both had elevations on the ADI and exceeded at least one area on the ADOS. Seven additional children were screened but did not meet the research criteria for ASD.

Measures

Prior diagnoses and treatment. Prior psychiatric diagnoses were determined by parent/guardian report on a nonstandardized history questionnaire that is used in a child development clinic of a children’s hospital. This questionnaire included questions related to prior psychiatric diagnoses that the child received in the community, previous psychiatric hospital stays, outpatient mental health treatment, and any psychotropic medication use.

Psychiatric comorbidity. At least one parent/guardian was interviewed by a licensed psychologist with the ACI (Lainhart et al., 2003). The ACI is a semistructured psychiatric interview that determines the presence of both lifetime and current (past 3 months) diagnoses for most major psychiatric disorders based on the DSM-IV-TR (American Psychiatric Association, 2000) criteria (suspending exclusions pertaining to ASD). It was designed to allow the investigator to elicit enough specific information to make a clinical judgment about coding whether symptoms are present. Validity and reliability of the ACI was established by parent report on 109 children (age = 5–17) with a diagnosis of autistic disorder (Leyfer et al., 2006). Criterion validity was determined by comparison of treatment histories to ACI diagnoses made blind to treatment history. Sensitivity was 100%, and specificity ranged from 83% to 93%. Interrater reliability was determined by sharing and rescoring ACI tapes across sites; agreement was 93%. Interrater reliability was determined by sharing and rescoring ACI tapes across sites; agreement was 93%. Interrater reliability was determined by sharing and rescoring ACI tapes across sites; agreement was 93%.

Lifetime ACI diagnoses were utilized. Summary codes were created for depressive disorders (major depression, dysthymic disorder, and mood disorder not otherwise specified) and anxiety disorders (generalized anxiety disorder, separation anxiety disorder, panic disorder, specific phobia, and social phobia; obsessive-compulsive disorder [OCD] was kept separate). This was done to improve comparability to parent report of prior community diagnoses. Thus, ACI diagnoses included in comparisons were any depressive disorder, bipolar disorder, any anxiety disorder, OCD, attention deficit hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD). Composite “internalizing disorder” and “externalizing disorder” variables were also created.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Frequencies of Number of Psychiatric Diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ACI Prior Diagnoses</td>
</tr>
<tr>
<td>M (SD) Number of Diagnoses</td>
<td>0.86 (1.06)</td>
</tr>
<tr>
<td>Range in Number of Diagnoses</td>
<td>0–4</td>
</tr>
<tr>
<td>% (n) of Sample With ≥1 Diagnosis</td>
<td>51.4 (18)</td>
</tr>
<tr>
<td>% (n) of Sample With ≥2 Diagnoses</td>
<td>22.9 (8)</td>
</tr>
<tr>
<td>% (n) of Sample ≥3 Diagnoses</td>
<td>8.6 (3)</td>
</tr>
</tbody>
</table>

Note: Recall that anxiety disorders and depressive disorders were combined, and counted as only one disorder each (e.g., anxiety would count as one disorder, even if a child had been diagnosed with both generalized anxiety and specific phobia). ACI = Autism Comorbidity Interview.

RESULTS

As shown in Table 2, the majority of children had at least one psychiatric disorder diagnosis, both based on the ACI (51%) and based on prior diagnoses (77%). The frequency of multiple comorbid diagnoses differed between ACI results and prior diagnoses. Specifically, of the participants who had at least one ACI diagnosis, 43% had more than one diagnosis. Of the participants who had at least one prior diagnosis, 88% had more than one prior diagnosis. Overall, 23% of participants received two or more ACI diagnoses compared to 60% with two or more prior diagnoses. These findings may be an underestimate for both ACI and prior diagnoses, given that anxiety disorders and depressive disorders were combined into broader diagnostic categories (any anxiety and any depression) rather than counted as separate disorders.

There was very poor agreement between the ACI and prior diagnoses, with kappas ranging from 0 to .5 (see Table 3). The diagonal in Table 3 represents the percent of prior diagnoses that were supported by the ACI. Overall, 59% (41 of 69) of prior diagnoses were not supported by the ACI, with none of the prior bipolar disorder or OCD diagnoses supported. Nearly one third of the entire sample had prior ADHD diagnoses that were not supported, and around 20% of the sample had received depressive, anxiety, and ODD diagnoses that were not supported on the ACI. The highest rate of agreement was for whether or not any internalizing disorder was present (the ACI confirmed 65% of prior diagnoses of any internalizing disorder), but the highest kappa was for depressive disorders, with 57% of prior diagnoses supported by the ACI and only two previously undetected depressive disorders. Previously undetected psychiatric diagnoses were rarer (e.g., only 11 previously undetected psychiatric diagnoses in the entire sample), but were most common for anxiety disorder diagnoses (14.3%, n = 5).

The number of diagnoses and medications were not significantly related to age of initial ASD diagnosis
However, as shown in Table 4, the number of prior diagnoses and medications increased as the age of initial ASD diagnosis increased, whereas there did not appear to be a pattern to the number of ACI diagnoses by ASD diagnosis age. Current age and IQ were not related to the number of prior or ACI diagnoses or treatment history variables (p > .05).

The majority of participants had received some form of outpatient mental health treatment (88%, n = 30). The mean number of psychotropic medications attempted was 2.85 (SD = 3.09), with substantial variability (range = 0–14). Medication use was especially pronounced among participants who had been previously diagnosed with bipolar disorder (M number attempted = 7.0) or ODD (M = 5.1). The number of prior psychiatric diagnoses was significantly correlated with the number of psychotropic medications attempted, r(35) = .781, p < .001.

Eight participants had prior psychiatric hospitalizations (see Table 5). Hospitalizations were prior to their ASD being identified for all but one child (Child 3).

Hospitaized children had an average of 3.38 prior diagnoses, with low (25%) diagnostic agreement with the ACI. All but one had a prior ODD diagnosis, none of which were confirmed on the ACI. Depression was the most common ACI diagnosis for these children, as well as the diagnosis with the highest rate of agreement.

### DISCUSSION

Children and adolescents with ASD commonly receive additional psychiatric diagnoses. However, the majority (nearly 60%) of prior psychiatric diagnoses were not supported by a structured psychiatric interview that carefully took ASD-related impairment into account. In particular, none of the bipolar disorder or OCD diagnoses were supported.

There are many possible explanations for the lack of concordance between community diagnoses and ACI diagnoses. The first is that the lack of concordance reflects diminished sensitivity of the ACI. We feel this is unlikely, given evidence for the validity and reliability of the ACI (Leyfer et al., 2006). The ACIs for this study were administered by licensed psychologists who specialize in ASD and have significant experience in general child psychopathology. In addition, there was a striking concordance between ACI rates found in this study and prior and independent published work with the ACI that reported on samples in two different states (Leyfer et al., 2006). Further, the ACI stems from the K-SADS, which has been studied extensively and is thought to be very useful in promoting the valid and reliable identification of disorders (Ambrosini, 2000; Kaufman et al., 1997).

Lack of concordance may stem from high rates of false positive diagnoses—diagnosing comorbid disorders

### TABLE 4

<table>
<thead>
<tr>
<th>Age of ASD Dx</th>
<th>No. of Prior Dx</th>
<th>No. of ACI Dx</th>
<th>No. Meds Attempted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before 10</td>
<td>6</td>
<td>1.33 (0.81)</td>
<td>0.83 (1.60)</td>
</tr>
<tr>
<td>10 or 11</td>
<td>14</td>
<td>1.86 (1.41)</td>
<td>1.00 (0.88)</td>
</tr>
<tr>
<td>12 or 13</td>
<td>8</td>
<td>2.00 (1.31)</td>
<td>0.25 (0.46)</td>
</tr>
<tr>
<td>14 or Older</td>
<td>7</td>
<td>2.71 (2.63)</td>
<td>1.29 (1.25)</td>
</tr>
</tbody>
</table>

*Note: ASD = autism spectrum disorder; Dx = diagnosis; ACI = Autism Comorbidity Interview.*
when they were not actually present—in combination with some false negative diagnoses—not diagnosing comorbid disorders that actually were present. Although difficult to ascertain in a pilot study, this could imply that the symptoms and dysregulation related to the diagnostic and cognitive features of ASD are sometimes attributed to the presence of another disorder. Notably, 80% of participants were referred to the study immediately following their initial ASD diagnoses as this sample was predominantly diagnosed with ASD during late childhood = adolescence. Given that their ASD diagnoses had been missed for so long, it seems plausible that some of the psychiatric diagnoses they received were mislabeling of ASD-related concerns. In other words, the multiple psychiatric diagnoses may reflect clinicians’ attempts to make sense of complex presentations. Numerous parents, after listing the child’s prior diagnoses, commented that they were “since taken away” or “later determined not to be the case,” providing further support for mislabeling of concerns.

Avoiding inaccurate psychiatric diagnoses in children with ASD is critical given the potential for inappropriate treatments and diverting attention away from an underlying ASD. There is sound evidence demonstrating that early ASD diagnosis and subsequent treatment leads to significantly better outcomes (e.g., Reichow & Wolery, 2008). Although age of ASD diagnosis was not significantly correlated with the number of prior psychiatric diagnoses in this study (possibly due to low power), there was a trend of more prior psychiatric diagnoses and a higher number of medications attempted with a later age of ASD diagnosis. The number of prior community diagnoses was positively correlated with the number of medications, so it is possible that these diagnoses steered clinicians toward medication treatments. This possibility is consistent with a large national registry study which found that 13% of children with ASD without any comorbid psychiatric diagnoses were on psychotropic medications, compared to 94.2% of those with bipolar disorder diagnoses, 82.6% of those with depressive diagnoses, and so on (Rosenberg et al., 2010; comorbid diagnoses were not verified by the authors).

Further research indicates that later ASD diagnosis is related to a higher likelihood of psychiatric hospitalization (Mandell, 2008). Of the eight children with hospitalizations histories in this study, only one had been identified as having ASD at the time of hospitalization. Further, there was a very low rate of agreement between the ACI and their prior diagnoses (with almost all receiving unsupported ODD diagnoses). Although this is certainly a challenging population with a high rate of aggressive and disruptive behaviors, it’s possible that outpatient interventions could have been more successful if their concerns had been conceptualized within the framework of their ASD.

In addition to treatment implications, conceptualization of emotional and behavioral presentation in children with ASD can inform neurobiological research and vice versa. Growing evidence implicates ASD as a “distributed neural systems disorder” with “broad involvement of cortical systems and higher order abilities” (Minshew & Keller, 2010, p. 124). Therefore, one would expect the

<table>
<thead>
<tr>
<th>Participant</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>12</td>
<td>12</td>
<td>17</td>
<td>13</td>
<td>16</td>
<td>10</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Age of ASD Dx</td>
<td>12</td>
<td>12</td>
<td>15</td>
<td>13</td>
<td>16</td>
<td>10</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Gender</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>F</td>
<td>M</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Full-Scale IQ</td>
<td>144</td>
<td>105</td>
<td>83</td>
<td>71</td>
<td>76</td>
<td>101</td>
<td>109</td>
<td>100</td>
</tr>
<tr>
<td>ADOS Scorea</td>
<td>13</td>
<td>12</td>
<td>11</td>
<td>11</td>
<td>10</td>
<td>17</td>
<td>7</td>
<td>13</td>
</tr>
</tbody>
</table>

Note: Higher scores on the Autism Diagnostic Observation Schedule (ADOS) generally suggest more autism spectrum disorder (ASD) symptoms. Dx = diagnosis; ACI = Autism Comorbidity Interview; PD = prior diagnoses; OCD = obsessive-compulsive disorder; ODD = oppositional defiant disorder; ADHD = attention deficit hyperactivity disorder.

aADOS diagnostic cutoffs are 7 for ASD in general and 10 for autistic disorder.

bExcluding OCD.
behavioral manifestation of ASD to involve more than just the three diagnostic domains (e.g., differences in emotion regulation would be expected; Mazefsky & Minshew, 2010). For example, Souders et al. (2009) hypothesized, “The synaptic pathway and gene anomalies associated with ASD alter levels of monoaminergic neurotransmitters” (p. 1576) leading to arousal dysregulation that may underlie some cases of sleep problems, agitation, anxiety, and fears in ASD.

The policies of many third-party payers contradict this notion, and may in some cases lead to diagnoses in children with ASD that are otherwise not necessary (and may be responsible for some of the poor concordance found in this study). Unfortunately, claims for insurance reimbursement by psychologists and psychiatrists are often denied if the primary condition is listed as ASD, thereby forcing clinicians to add an additional diagnosis (Leslie & Martin, 2007). If the presenting symptom(s) are better explained by the ASD, this may drive a misconceived understanding of the disorder in general, as well as lead to diagnoses that follow the child when records are reviewed in later settings.

Limitations and Future Directions

This pilot study highlights the complexity of psychiatric diagnosis and conceptualization of emotion and behavioral dysregulation in ASD. With the many implications of diagnostic practices for treatment, research, and public policy, this topic is deserving of further attention in larger scale studies. Future studies on this topic should also include both typically developing and other psychiatric disorder control groups, particularly given research demonstrating that children with other non-ASD diagnoses demonstrate similar social and communication impairments (e.g., Towbin et al., 2005; White et al., 2011).

Characteristics of this study’s participants should be considered when interpreting findings and addressed in follow-up studies. Participants were a clinical (as opposed to epidemiological) sample of preadolescents and adolescents with HFASD, with the majority not receiving their ASD diagnosis until late childhood or adolescence. Children with HFASD typically receive their diagnoses later than lower functioning children, with diagnosis delayed until adolescence or even adulthood in some cases (White et al., 2011). Thus, studies of diagnostic patterns for this group of individuals with HFASD may be particularly important in order to develop a better understanding of what contributes to the delay. However, the results of this study may not generalize to lower functioning children with ASD, or those who are identified earlier as having ASD. Further, this sample was predominantly Caucasian. Extensions of this work with other ethnicities should be completed, particularly given evidence of ethnic disparities in the diagnosis of ASD (Mandell et al., 2007).

Assessment of ASD through the ADOS and ADI–R and of comorbid psychiatric diagnoses (i.e., ACI) is quite time- and training intensive, presenting a challenge to research. However, the type of careful clinical characterization employed in this study is necessary to begin to distinguish psychiatric comorbidity from what may be more accurately conceptualized as ASD-related impairment. This study relied on parent report; it would be helpful for future studies to utilize record review or other means to confirm the accuracy of parental report of prior diagnoses. We also would like to emphasize that our use of “comorbid psychiatric disorders” reflects empirically defined, clinically distinct syndromes, rather than neurobiologically and neuropathologically defined disorders. Questions that should be addressed in future research include whether the psychiatric symptoms in question are more accurately considered part of the ASD but defined separately based on the DSM, whether they reflect a shared underlying genetic and neural pathway, or whether they are separately co-occurring phenom-enon. Thus, understanding of the causal mechanisms linking psychiatric symptoms and ASD would be advanced by longitudinal studies combining the type of in-depth clinical evaluation reported in this manuscript with examination of neuropsychological profiles and neuroscience and genetic approaches.

REFERENCES


