2009

Comorbid psychopathology in individuals with autism spectrum disorders and intellectual disabilities

Santino Vincent LoVullo
Louisiana State University and Agricultural and Mechanical College, slovullo@gmail.com

Follow this and additional works at: https://digitalcommons.lsu.edu/gradschool_theses

Part of the Psychology Commons

COMORBID PSYCHOPATHOLOGY IN INDIVIDUALS WITH AUTISM SPECTRUM
DISORDERS AND INTELLECTUAL DISABILITIES

A Thesis

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Master of Arts

in

The Department of Psychology

by
Santino Vincent LoVullo
B.A., California State Polytechnic University Pomona, 1997
May, 2009

TABLE OF CONTENTS

ABSTRACT

While there has been an abundance of research investigating Autism Spectrum Disorders (ASD) in children, very little emphasis has been placed on ASD in adults, especially in regards to comorbid psychopathology. This is of great concern considering that ASD often co-occurs with intellectual disability (ID), and that both may serve as risk factors for additional psychopathology. While instruments exist that measure comorbid psychopathology in adults with ID, these scales are not targeted to the unique expression of comorbidity in adults with ID and ASD. The Autism Spectrum Disorders-Comorbidity for Adults (ASD-CA) was devised for this reason. This paper begins with an overview of ASD, including history, diagnostic features, prevalence, and a discussion of comorbidity. The overview is followed by two studies. Study 1 was conducted to examine the frequency of symptom endorsements among adults classified as having ID; ID and ASD; and ID, ASD, and additional psychopathology. Study 2 was conducted to further increase the utility of the ASD-CA by creating cutoff scores for its subscales. The results of Study 1 showed a general pattern among diagnostic groups in regards to scores on the subscales of the ASD-CA, with the ASD groups scoring the highest. For Study 2, cutoff scores were calculated for each subscale of the ASD-CA, and were defined as values one standard deviation greater than the respective ID and ASD group means. This paper concludes with a discussion of the implications of the results as well as directions for future research.

INTRODUCTION

Until recently, efforts at investigating autism spectrum disorders (ASD) were almost exclusively devoted to children with little emphasis placed on adults (Volkmar, Klin, & Cohen, 1997). However, for children this resulted in the creation of improved diagnostic instruments and the development of effective behavioral interventions. An area that has especially been neglected is research in comorbid psychopathology, particularly as it applies to adults who are lower functioning and lack verbal skills (Bradley, Summers, Wood, & Bryson, 2004). This is alarming when one considers that comorbidity is common in people with intellectual disability (ID; Borthwick-Duffy & Eyman, 1990), with evidence that it also frequently occurs in people with ASD (Gillberg & Billstedt, 2000; Matson & Nebel-Schwalm, 2007). Unfortunately, instruments that accurately measure comorbidity in adults with ID and ASD are not available. The Autism Spectrum Disorders-Comorbidity for Adults (ASD-CA) is a new instrument that was developed to fill this niche. The measure was designed to provide accurate diagnoses, therefore, leading to more tailored and effective treatments. The purpose of this study is to investigate the differences among three groups using the subscales of the ASD-CA, followed by the development of cutoff scores for this measure. The study will be preceded by an overview of autism and a discussion of comorbidity in ID and ASD.

History of Autism

In the first clinical account of autism, Kanner (1943) provided a detailed description of 11 children who exhibited common behavioral characteristics. Although the presentation and severity of their symptoms varied, the children shared abnormalities in social relationships, language acquisition and use, and displayed stereotypical behaviors. While modifications to the definition of autism have occurred over time, Kanner's account has endured, with the abnormalities he noted in the three areas resembling our current notion of the disorder.

1

Kanner suggested that, in children with autism, the primary characteristic was an inability to relate to people or things in a normal way. The children he described were disconnected from the outside world and showed an inability to develop typical social relationships. Kanner contrasted this inability to the withdrawal displayed in people with schizophrenia, who were able to engage in social relationships prior to the onset of their disorder. Conversely, Kanner believed that children with autism never acquired relationships, and this occurred as a result of, what he considered, an ingrained desire to be alone that began during infancy. This desire to be alone could be seen in the absence of an anticipatory posture when being picked up by a caregiver, and by the lack of acknowledgement of others. Kanner described the children's relationships with people as analogous to their relationships with inanimate objects; they would walk into a room and not recognize the presence of others.

In addition to a lack of social desire and awareness, Kanner also noted abnormalities in communication. Of the 11 children he described, three were non-verbal and the remaining eight demonstrated severe deficits in functional speech. Kanner believed that, for the children with limited speech, problems with communication were further compounded by the presence of normal articulation and strong rote memories. This combination resulted in their tendency to repeat nonfunctional phrases. Even for the language that was useful, much of it was situation specific, with generalization taking an extended amount of time. Another characteristic Kanner observed was their difficulty in correctly using first and third person pronouns, such as "I" and "you."

Kanner elaborated on the children's' stereotypic behaviors, need for sameness, and abnormal reactions to sensory stimuli. They were horrified by loud noises, moving objects, and, from what Kanner suggested, any intrusion into their preferred state of aloneness. All aspects of the children's' behavior were characterized by a need for sameness that, when violated, resulted

2

in behavior outbursts. From what Kanner described, evidence for this desire for sameness was supported by their repetitive movements and speech, and in a limitation of spontaneous activities. The children showed an obsessive need for objects in their environment to be arranged in particular configurations and the elements of their routines to be performed in predictable sequences. Kanner made reference to their preoccupation with parts of objects and an inability to experience things as a whole. Furthermore, he noted that the children had an extreme pleasure for sensory experiences and especially enjoyed spinning, jumping, and other rhythmic movements.

Kanner noted other characteristics that he felt were associated features of the disorder. He viewed the children as essentially physically normal, although he considered five to have larger than average heads, while several others demonstrated gross motor impairments. Although he believed that they all came from intelligent and well-educated families, he thought that their parents failed to display affection and instead were preoccupied with academic endeavors, showing little interest in people. Kanner believed, at least to some extent, that this lack of affection contributed to what he described as an "autistic disturbance of affective contact."

Definition of Autism. While Kanner was the first to use the word "autism" to reflect its present meaning, the term was coined by the Swiss psychiatrist Eugene Bleuler in 1908. He used the word to describe the active withdrawal from reality that was characteristic of patients with schizophrenia. Bleuler described the clinical manifestations of "autism" as a tendency towards delusional thinking, a reduced ability to interact with others, and a withdrawal into fantasy life. In contrast, the disorders described by Kanner and Hans Asperger, who will be discussed next, were characterized by an innate inability to form relationships.

A year after Kanner classified autism as a separate disorder, Hans Asperger (1944), who was unaware of Kanner's work, described four children with difficulties in socialization, but who

had relatively normal cognitive and language abilities. While Kanner's work was internationally known, Asperger's account was originally written in German and, as a consequence, received far less attention. In fact, the translation of his work did not occur until 1991 when a German psychologist, Uta Frith, incorporated the disorder into a chapter of her book on the very topic (Asperger & Frith, 1991).

In Asperger's characterization, the primary impairments were in socialization and in understanding the unwritten rules of interaction. The children observed by Asperger demonstrated difficulties in the proper use of eye contact, gesturing, maintaining appropriate proximity to others during conversation, and in exhibiting social behaviors that come naturally to most people. They also had the tendency to develop unusual preoccupations with specific subject matter, resulting in long, one-sided conversations. While the children Kanner described had severe limitations in the development of speech, Asperger's children followed a typical developmental course. Asperger also noted deficits in gross motor coordination, a resistance to change, and an attachment to special objects. Whereas Kanner's autism became associated with low functioning, nonverbal individuals, Asperger's disorder was associated with quite the opposite (Volkmar et al., 1997). Another difference was that, in comparison, Asperger's children had better social skills and displayed less pronounced stereotypic behaviors.

Although Asperger's Syndrome was described in 1944, the development of the definition of autism, which is currently part of the broader category of Pervasive Developmental Disorders (PDD) in the DSM-IV-TR (APA, 2000), evolved from Kanner's original (1943) depiction. Despite Kanner's recognition that autism was distinct from schizophrenia, the early history of autism is rampant with a confusion of the disorders. This confusion was most likely a result of Bleuler's use of the word "autism" to describe a feature of schizophrenia, and the inconsistent use of "childhood schizophrenia, child psychosis, and autism as labels for the same disorder"

(Rutter, 1978). Along these lines, Creak (1964) provided a lengthy definition of "Childhood Schizophrenia," which, despite the label, was in fact a reference to autism. Unfortunately, her criteria were unclear and difficult to apply consistently in clinical practice. The boundaries between autism and schizophrenia remained undifferentiated until the 1970's when research began to emerge showing that they were indeed distinct disorders. For instance, Kolvin (1971) showed that the two disorders could be differentiated by age of onset and the development of language and cognitive skills.

One of the most influential definitions of autism was developed by Rutter (1978). He attempted to further refine Kanner's concept of autism by providing four primary criteria: (1) onset before 2 ½ years of age, (2) impairments in social skills, (3) abnormal language development, and (4) insistence on sameness. In addition, Rutter felt it was important to view the symptoms in light of the person's intellectual development, and medical and neurological status. He believed that these variables could substantially expand the range of symptom expression in autism, and that a multiaxial approach would be the best account for this variability.

During the same year, Edward Ritvo (1978) developed a competing definition in conjunction with the National Society for Autistic Children (NSAC). The NSAC was a powerful lobbying group composed of parents seeking to find improved services for their children. Not only did he suggest a biological basis for autism, but in comparison to Rutter's (1978) definition, Ritvo's was more specific. His definition included the following criteria: (1) impaired development of language, physical, and social skills; (2) the absence of or delay in speech; (3) unusual reactions to sensory stimuli; and, (4) abnormal relations with people, events, and objects. Although influential, the NSAC definition was partially intended to influence social policy, thus compromising its scientific rigor (Rutter & Schopler, 1988). As a consequence, the key

components of Rutter's definition were included in the Diagnostic and Statistical Manual, Third Edition (DSM-III; APA, 1980).

The DSM, which in its current form is the DSM-IV-TR (APA, 2000), is a handbook used by mental health professionals to diagnose mental disorders according to specified criteria. The first version of the DSM was published in 1952 by the American Psychiatric Association (APA, 1952), with a second version, DSM-II, appearing in 1968 (APA). However, the DSM-III (APA, 1980) is of particular importance because it was the first to employ a multiaxial approach to diagnosis, as well as provide specific criteria for each disorder (Matson & Minshawi, 2006). The DSM-III was also the first to incorporate the category of PDD. From its inception into the DSM-III in 1980, PDD have gone through several modifications that have occurred in synchrony with revisions to the DSM. Over this period of time, the trend has been a broadening of the diagnostic category of PDD in order to include variations of the disorders at various ages and developmental levels, while simultaneously controlling for issues of specificity and sensitivity (Matson & Minshawi, 2006).

Diagnostic Criteria

At present, there are two systems that clinicians use to diagnose mental health problems. One is the DSM-IV-TR (APA, 2000), and the other is the International Statistical Classification of Diseases and Related Health Problems, 10th Edition (ICD-10; World Health Organization, 1992). The ICD-10 is used to classify a variety of diseases and conditions, a portion of which is mental health related. The ICD-10 provides a universally accepted method of comparing mortality and morbidity rates. Due to the similarity between the two systems, specifically in the diagnosis of PDD, and the fact that the DSM-IV-TR is the more widely used instrument, the present discussion will be limited to DSM-IV-TR criteria.

As previously mentioned, the DSM-IV-TR employs a multiaxial approach, with psychiatric disorders classified into five dimensions. Axis I consists of the clinical disorders and the overarching category of Disorders usually First Diagnosed in Infancy, Childhood, or Adolescence. A portion of this category is the Pervasive Developmental Disorders, including: Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder, Asperger's Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). Axis II consists of the personality disorders and intellectual disability. Axis III is designated for general medical conditions that may be contributing factors to the development of psychopathology, or that may provide useful information in the understanding and treatment of mental disorders. Axis IV is designated for environmental and social factors that may have an impact on the diagnosis, treatment, and prognosis of a mental disorder. Finally, Axis V is used for reporting an individual's overall level of functioning as assessed by a clinician (APA, 2000).

Recently, PDD have been more commonly referred to in the literature as autism spectrum disorders (ASD; Matson & Minshawi, 2006). Because of this trend, the term ASD will be used in reference to these disorders for the remainder of this paper. ASD are characterized by impairments in three clusters of behavior which are abnormal in comparison to one's developmental level (APA, 2000). ASD consist of severe impairments in reciprocal social interaction, communication, and are also characterized by the presence of stereotypical behavior. Although there are five disorders under the category of ASD, for the purposes of this study, only the criteria for Autistic Disorder and PDD-NOS will be described. It is important to note that the DSM-IV-TR employs a "Chinese Menu" approach to operationalizing diagnoses. In essence, in order for an individual to be diagnosed with a particular disorder, he/she must exhibit a minimum number of symptoms per cluster of behavior that defines the disorder.

Autistic Disorder. A diagnosis of Autistic Disorder requires that an individual has two endorsements from the socialization domain, one from communication, and one from stereotypies. In addition, there must be delays or abnormal functioning prior to three years of age in at least one of the following areas: (1) social interaction, (2) social communication, or (3) symbolic or imaginative play. Furthermore, the behaviors should not be better explained by Rett's Disorder or Childhood Disintegrative Disorder. Items within the socialization domain include: (a) impairment in the use of nonverbal behaviors; (b) developmentally inappropriate peer relationships; (c) a lack of seeking to share enjoyment, interests, or achievements with other people; and, (d) a lack of social or emotional reciprocity. Impairments in communication consist of: (a) an absence of, or a delay in, the development of speech; (b) an inability to maintain a conversation with others; (c) stereotyped use of language; and, (d) a lack of varied and spontaneous social play. Items in the stereotypy domain include: (a) a preoccupation with a narrow and stereotyped pattern of interest; (b) need for sameness in routines or rituals; (c) repetitive and stereotyped movements; and, (d) an enduring preoccupation with parts of objects (APA, 2000).

A diagnosis of Autistic Disorder relies on its differentiation from other ASD. While autism is more common in males, Rett's Disorder is almost exclusively exhibited in females. Furthermore, Rett's Disorder is distinguished by a loss of purposeful hand skills, poorly coordinated gait or trunk movements, and acquired microcephaly. Often the social deficits that develop in Rett's Disorder are transient and become less obvious over time. Childhood Disintegrative Disorder, unlike autism, is characterized by severe regression in several areas of functioning after two years of normal development. Finally, in Asperger's Disorder, there are no significant delays in language, and ID is usually not observed (APA, 2000).

Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). PDD-NOS is a diagnosis within ASD that encompasses a combination of symptoms that are not better accounted for by the other, more defined ASD. Consequently, there is a wide variability in the expression of symptoms in people with PDD-NOS. The designation of PDD-NOS may be used as a default diagnosis when there is a lack of information for diagnosis, or a sparse developmental history prevents the diagnosis of another ASD. For instance, a diagnosis of Autistic Disorder or Childhood Disintegrative Disorder requires an onset of symptoms prior to age three. If the age of onset cannot be established, then a diagnosis of PDD-NOS may be more appropriate. PDD-NOS may also refer to conditions that are within the boundaries of ASD, but have symptoms that are less severe. Furthermore, PDD-NOS can also be used for those who have a late onset of symptoms (APA, 2000). According to the DSM-IV-TR, PDD-NOS is diagnosed when "there is a severe and pervasive impairment in the development of reciprocal social interaction associated with impairment in either verbal or nonverbal communication skills or with the presence of stereotyped behavior, interests, and activities, but the criteria are not met for a specific Pervasive Developmental Disorder, Schizophrenia, Schizotypal Personality Disorder, or Avoidant Personality Disorder" (APA, 2000).

Of particular difficulty is drawing the line between PDD-NOS and the autistic-like symptoms that are displayed in individuals with ID. There's a general consensus that individuals with ID are more likely to exhibit ASD symptomotology (Bregman, 1991; Towbin, 1997). However, it is important to note that, for a diagnosis of an ASD, deviations in these areas must extend beyond what is expected for a person's cognitive development (APA, 2000). The importance of cognitive development in the expression of the core symptoms of ASD was demonstrated in a study by Matson, Dempsey, LoVullo, and Wilkins (2008). The study examined the effects of IQ on the expression of ASD symptoms in individuals with Autistic

9

Disorder and ID, PDD-NOS and ID, and ID only. Overall, the individuals with autism exhibited more symptoms than those with PDD-NOS, who exhibited more symptoms than controls. While level of IQ was a strong moderator of ASD symptomotology for individuals with ID, this was less the case for the group with PDD-NOS, and not the case for the group with autism. In summary, it appears that as the level of cognitive impairment increases, the more difficult it becomes to differentiate ASD from ID.

Diagnostic Instruments

Despite the availability of classification systems such as the DSM-IV-TR and the ICD-10, the diagnosis of an ASD can present a clinician with "unique challenges" (Lord, 1997). As mentioned, the expression of an ASD can be compounded by varying degrees of intellectual and verbal abilities (Volkmar et al., 1997). Furthermore, the initial approach to the assessment of these disorders was unsystematic and unreliable, and as a consequence, structured assessment instruments were developed (Matson, Nebel-Schwalm, & Matson, 2007). The following is a discussion of the most commonly used instruments in the identification of ASD in children and adults.

Aberrant Behavior Checklist (ABC). The ABC is a part of the Autism Screening Instrument for Educational Planning (ASIEP; Krug, Arick, & Almond, 1980) developed for use with individuals with severe ID. The ABC, which helps identify the severity of behaviors related to autism, consists of 57 questions, assessing five areas: sensory, relating, body and object use, language, and social and self-help. The questions are presented in a yes/no format, with higher scores indicating greater impairment. This scale was designed to use teachers as primary raters; however, parents may provide additional information. The output of this assessment is an overall score that falls into one of three categories indicating the probability of autism: high, questionable, and unlikely probability.

The initial estimates of inter-rater reliability for the ABC were high (Krug et al., 1980); however, the methods used to obtain these figures have been criticized. In an additional analysis, Volkmar (1988) found the ABC's inter-rater reliability to be significantly lower than previously reported. Furthermore, Volkmar reported a sensitivity of 75% and a specificity of 81%.

According to Volkmar (1988), advantages of the ABC include ease of administration, the use of teachers as primary raters, and the availability of scoring norms for different ages and disabilities (e.g., normal, deaf, blind). Disadvantages include a large percentage of false-negatives, particularly when used with higher functioning individuals. Furthermore, Volkmar (1988) suggests that the ABC appears to be more appropriate as a screening tool rather than substitute for thorough clinical assessment.

Childhood Autism Rating Scale (CARS). The CARS (Schopler, Reichler, DeVellis, & Daly, 1980) was created in order to diagnose children who were referred to the Treatment and Education of Autistic and related Communication-handicapped CHildren (TEACCH) program in North Carolina, and was developed in response to limitations of earlier classification systems that were based on Kanner's (1943) and Creak's (1964) criteria. According to Schopler et al. (1980), these systems lacked sensitivity and were not suitable for very young children. The CARS consists of 15 subscales: Impairment in Human Relationships, Imitation, Inappropriate Affect, Bizarre Use of Body Movement and Persistence of Stereotypes, Peculiarities in Relating to Nonhuman Objects, Resistance to Environmental Change, Peculiarities of Visual Responsiveness, Peculiarities of Auditory Responsiveness, Near Receptor Responsiveness, Anxiety Reaction, Verbal Communication, Nonverbal Communication, Activity Level, Intellectual Functioning, and General Impressions. Each item is rated on a 7-point scale with higher numbers indicating greater abnormality (Schopler et al., 1980). A total score is calculated

that indicates the severity of autism according to the following labels: nonautistic, mild to moderately autistic, and severely autistic.

The CARS is reported to have good reliability, which is reflected in its high internal consistency (alpha = .94), inter-rater reliability (.55-.93), and test-retest reliability (.88; Schopler et al., 1980). The CARS also has excellent concurrent validity in terms of its agreement with the DSM-III (Sevin, Matson, Coe, & Fee, 1991) and DSM-IV-TR (Perry, Condillac, Freeman, Dunn-Geier, & Belair, 2005).

Overall the CARS is a psychometrically sound instrument that can be used to assist in the identification of children with autism over 2 years of age by assessing the severity of symptoms. The CARS is a widely used instrument that is written in several languages; it has very good validity and reliability, and requires minimal training for use by clinicians familiar with autism. A limitation of this instrument, however, is that it was created before the concept of a spectrum of autistic disorders, and therefore is not useful for differential diagnosis in this regard (Klinger & Renner, 2000; Matson & Minshawi, 2006).

Autism Diagnostic Interview-Revised (ADI-R). The ADI-R (Lord, Rutter, & Le Couteur, 1994) is a structured interview used to aid in the diagnosis of ASD. It was developed in response to some of the shortcomings of the original version of the instrument, the ADI (Le Couteur, Rutter, Lord, & Rios, 1989). The ADI was created for research purposes, as a tool to provide reliable diagnosis of ASD in genetic studies for individuals older than 5 years of age. However, criticisms of the ADI were its excessive length, overlapping sections for areas measuring social and communication skills, and an extensive section on general behavior problems (Lord et al., 1994). In response to this, along with the desire of the test's creators to increase the measure's clinical utility, with a trend towards earlier diagnosis of ASD, the ADI-R was created.

The ADI-R consists of five sections: (1) opening questions, (2) early and current communication, (3) early and current social development and play, (4) repetitive and restricted behaviors (currently or ever), and (5) questions regarding general behavior problems. In addition, in order to better discriminate between children with autism and ID, and children with severe ID, modifications were made to the items in the communication, socialization, and stereotypy domains (Lord et al., 1994). For the ADI-R, new items were added to help identify autism prior to 5 years of age, while other items were eliminated that were judged to be redundant or unreliable. The interview is conducted by a clinician, with scoring based on the behaviors described by the parent or caretaker. The items are then coded according to a four-point scale: (0) no definite behavior of the type specified; (1) behavior of the type specified is probably present but defining criteria are not fully met; (2) definite abnormal behavior of the type described in the definition and coding; and, (3) extreme severity (Lord et al., 1994). The scores are then calculated using an algorithm that exists in three different versions: lifetime, current behavior, and a version for children less than 4 years of age. For this measure, an indication of autism is based on the individual meeting criteria for each of the three domains characteristic of autism, with at least one abnormality in a domain by 36 months of age (Lord et al., 1994).

The psychometric data for the ADI-R as reported by Lord et al. (1994) were good in terms of both reliability and validity. In terms of inter-rater reliability, kappa scores ranged from .62 to .89. Test-retest reliability was good; however, it was based on a small sample size and should be interpreted with caution. Validity was also reported as good with the social and nonverbal communication algorithm items able to discriminate preschoolers with autism from those with language impairments, and ID (Lord et al., 1994).

Overall, the ADI-R is a psychometrically sound instrument that is able to differentiate individuals on the autism spectrum from those who are not. The scale includes a scoring

13

algorithm that is based on DSM-IV-TR and ICD-10 diagnostic criteria. Furthermore, it provides an interview that is structured and thorough, including sections for the assessment of behavior problems and early development. However, the ADI-R is limited in that it does not discriminate among the different ASD. In addition, it takes a considerable amount of time to administer, and clinicians are recommended to receive extensive training in order to be deemed proficient.

Autism Diagnostic Observation Schedule -Generic (ADOS-G). The ADOS-G is a semi-structured observation tool used to assess the social interactions, communication skills, and play skills of children and adults who are suspected of having ASD. This instrument is a revised and combined version of two previously used instruments, the ADOS (Lord, Rutter, Goode, & Heemsbergen, 1989) and the Pre-Linguistic ADOS (PL-ADOS; DiLavore, Lord, & Rutter, 1995). Like the ADI, the ADOS was originally intended for research purposes, and as an observational supplement to the ADI in diagnosing children with autism between the ages of 5 and 12. However, due to the desire to use these instruments in clinical settings with children under the age of five, these assessments were both modified resulting in the ADI-R and the PL-ADOS (Lord et al., 2000). The ADOS was developed for children with fluent speech, while the PL-ADOS was intended as an alternative for children who did not have adequate language skills (DiLavore et al., 1995). Despite an attempt at capturing children with a wide range of language skills, both instruments failed at assessing children with moderate language skills (Lord et al., 2000). Therefore, the ADOS-G, a synthesis of the ADOS and the PL-ADOS, was created to remedy this discrepancy.

The ADOS- G consists of four modules that are comprised of structured situations varying in the level of expressive language demands, which are intended to facilitate specific social behaviors. Module 1 is based on the PL-ADOS and is intended for children who do not consistently and spontaneously use phrase speech. Module 2 is designed for children who use

some phrase speech, but who are not fluent. Module 3 is based on the ADOS and is for children who are verbally fluent, and where the use of toys is age appropriate. Module 4 is partially based on the ADOS and is intended for adolescents and adults who are verbally fluent. While Modules 1 and 2 are typically conducted during interactions at various locations in a room (e.g., at a table, on the floor, etc.), Modules 3 and 4 are likely to occur while sitting at a table interacting in a conversational style. The modules are scored using a three-point scale that indicates the degree of abnormality related to autism. Similar to the ADI-R, the ADOS-G uses an algorithm specific to each module that computes a score for the socialization domain, communication domain, and the combination of the two. Autism or an ASD is indicated according to the thresholds obtained on each of the three indices.

According to Lord (2000), for inter-rater reliability among the four modules, mean kappa coefficients ranged from .65 to .78 and mean exact agreement ranged from 88.2% to 91.5%. Furthermore, The ADOS-G was able to consistently differentiate autism and PDD-NOS from non-spectrum disorders. However, it was not effective in discriminating between autism and PDD-NOS (Lord et al., 2000).

In conclusion, the ADOS-G is a reliable and valid instrument that provides an additional method for clinicians to assess ASD through direct observation and interaction. It provides a system that is flexible to the interaction style and language ability of the person being assessed. Drawbacks of this instrument include an extensive amount of training required for administration, scoring, and observation (Lord et al., 2000). In addition, the ADOS-G does not provide a method for assessing stereotyped and repetitive behaviors and therefore misses a significant portion of the criteria required for diagnosis. This measure is also limited in its ability to differentiate autism from other ASD, and it only provides a snapshot of current functioning rather than an assessment of behaviors over time.

In summary, there are several instruments available to clinicians to aid in the diagnosis of ASD. This is a welcome development because, historically, diagnoses were made using a "loose" interpretation of DSM criteria (Matson & Minshawi, 2006). The availability of assessment instruments provides a more systematic means of approaching diagnosis, and can improve the accuracy of decisions. In general, the reviewed scales have good psychometric properties; however, they are limited in their ability to discriminate among the various ASD. A recent trend is the creation of scales that can assess ASD at younger ages, as a means of beginning interventions at the first signs of impairment. Although early identification is a welcome trend, also needed are additional scales that can assess ASD in adults.

The importance of instruments that can aid in the diagnosis of ASD is highlighted by the increased prevalence of these disorders. One explanation for the rising prevalence is that the diagnostic category of ASD has broadened over time, with the effect of including a wider range of symptomotology. This has in turn increased the heterogeneity of ASD making instruments that are sensitive to each disorder of greater importance.

Prevalence

Within the last several decades there has been a progressive rise in the prevalence of ASD. The first attempt at establishing a prevalence rate for autism was conducted in the United Kingdom and documented in the pioneering work of Victor Lotter (1966). In his initial estimate, approximately 4.1 out of every 10,000 children were afflicted by autism. However, these numbers are substantially larger today. Current estimates by Fombonne (2005) show rates of 0.6% for all ASD, 13/10,000 for Autistic Disorder, 21/10,000 for PDD-NOS, 2.6/10,000 for Asperger Disorder, and 2/100,000 for Childhood Disintegrative Disorder. This dramatic increase has created widespread alarm among parents and professionals leading to unsubstantiated claims of causation. A partial list of proposed causal factors has included such things as: yeast, heavy

metal toxicity, chemical exposure, and gluten. However, the factor that has received the most attention is the measles, mumps and rubella (MMR) vaccine. This controversy began in reaction to a report by Wakefield (1998) that described 12 children with autism who were admitted to a hospital in London and who exhibited specific gastrointestinal symptoms. Wakefield suggested that, for these children, the MMR vaccine may have been responsible for gastrointestinal disturbances that led to the behavioral manifestations of autism. Contrary to his hypothesis and an associated lack of empirical evidence, numerous studies have discredited the link between the MMR vaccine and autism (Honda, Shimizu, & Rutter, 2005; Smeeth et al., 2004).

Although research does not exist to support environmental contaminants as the cause for the increasing prevalence of ASD, there is evidence that other factors may be at play. Wing (2002) offered more plausible explanations for the rising numbers, including the effects of broadening diagnostic criteria, greater public awareness, and the development of specialized services. In her analysis of 39 studies, the rates of ASD increased in synchrony with the changes to the diagnostic criteria. From Kanner's original description of autism through the current DSM-IV-TR and ICD-10 specifications, the definition of ASD has broadened, including individuals who would not have previously met criteria. Another reason is the increased public awareness of ASD, which was particularly low prior to the development of parent and professional interest groups in the 1960s. Wing (2002) argued that, presently, children with ASD have substantial access to specialized services, and this has created an increased demand for ASD diagnoses. These services may include specialized schools and classrooms, occupational and speech therapy, and intensive interventions.

A study by Croen, Grether, Hoogstrate, and Selvin (2002) illustrated the importance of diagnostic substitution, occurring when a new disorder becomes popular and is consistently diagnosed in place of a previous disorder (Matson & Minshawi, 2006). Croen et al. examined the

17

prevalence of autism in California between 1987 and 1994 as recorded by the Department of Developmental Services. Not only was there a dramatic increase in the prevalence of autism, but it coincided with a similar reduction in the prevalence of ID of unknown etiology. However, the authors suggested that the increase could have been as a result of better screening instruments and a greater public awareness of autism.

The hypothesis that diagnostic substitution accounted for the increased rates of autism is tenable due to the close relationship between autism and ID. Because ID occurs frequently with autism, it is possible, though not accurate, for autism to be a substituted diagnosis. Although the rate of comorbidity of autism and ID is thought to be overestimated in research, even conservative estimates support a substantial co-occurrence (Edelson, 2006). Due to this close relationship, any examination of ASD should also include a discussion of ID.

Intellectual Disability

ID, which was referred to as mental retardation (MR) prior to 2006, is currently defined by deficits in two areas: intellectual functioning and adaptive functioning. This dual-deficit approach began in 1959 (Heber) as part of the definition created by the American Association of Mental Retardation (AAMR). The AAMR, now known as the American Association of Intellectual and Developmental Disabilities (AAIDD), has been very influential in shaping definitions of ID, such as those established by the APA and World Health Organization (Greenspan, 1999).

A controversial definition of ID was provided in 9th edition of the AAMR (Luckasson et al., 1992). ID was defined as "substantial limitations in present functioning. It is characterized by significantly subaverage intellectual functioning, existing concurrently with related limitations in two or more of the following applicable adaptive skill areas: communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics,

leisure, and work. MR manifests before age 18." The notion of "present functioning" likely reflected the belief that ID was a condition that could be remediated. In addition, the 9th edition of the AAMR raised the intelligence quotient (IQ) ceiling of ID by five points from 70 to 75, possibly as a way of including scores of 70 when accounting for the standard error of measurement (Greenspan, 1999). Also, instead of using standard deviation as a way of classifying the severity of ID, the 1992 definition viewed severity as the degree of supports needed across adaptive behavior areas.

Likely in response to difficulties using the 1992 AAMR definition, the APA's 1996 definition of ID was considerably different (Cuskelly, 2004). ID was defined as "(a) significant limitations in general intellectual functioning; (b) significant limitations in adaptive functioning, which exist concurrently; and, (c) onset of intellectual and adaptive limitations before the age of 22 years." In addition, the concept of adaptive behavior limitations changed, reflecting a summary score of 2 standard deviations below the appropriate norming sample, or adaptive scores that are consistent with those who have ID. The 1996 definition also became more consistent with previous definitions of ID, classifying severity into four categories based on IQ scores and adaptive functioning.

The present definition of ID, according to the DSM-IV-TR is: "significantly subaverage general intellectual functioning (Criterion A) that is accompanied by significant limitations in adaptive functioning in at least two of the following skills areas: communication, self-care, home living, social/interpersonal skills, work, leisure, health, and safety (Criterion B). The onset must occur before the age of 18 years (Criterion C)." Significantly subaverage intellectual functioning represents an IQ of 70 or below. A score in this range is at least two standard deviations below the mean, and is assessed using one of the many standardized and individually administered intelligence instruments (APA, 2000). Adaptive functioning represents the extent to which a

person can "cope with life demands" and demonstrate independence relative to individuals with similar demographic characteristics. For the purposes of diagnosing ID, adaptive behavior can be assessed using a norm referenced instrument, such as the Vineland Adaptive Behavior Scales (Sparrow, Balla, & Cicchetti, 1984).

The DSM-IV-TR designates four levels of ID based on severity: mild ID (IQ between 50-55 and 70), moderate ID (IQ between 35-40 and 50-55), severe ID (IQ between 20-25 and 35-40), and profound ID (IQ below 20-25). Mild ID represents the largest group, approximately 85% of individuals with ID. Individuals in this group are typically able to obtain academic skills up to the sixth-grade level, and live with minimal assistance as adults. Ten percent of individuals with ID are in the moderate range of ID. People in this group usually do not acquire academic skills beyond the second grade. As adults, they may be able to perform semiskilled jobs, but usually require increased supervision. Individuals with severe ID consist of 3%-4% of individuals with ID. During their school years they may be able to learn basic academic skills, such as recognizing sight words or acquiring an understanding of number concepts. As adults they may be able to work performing simple tasks and live with assistance with their families or in the community. Individuals with profound ID account for 1%-2% of individuals with ID and are the most impaired. People in this group require highly structured environments and constant supervision. The last category of ID in the DSM-IV-TR is ID, severity unspecified. This designation is used when there is a strong suspicion of ID, but when IQ scores cannot be obtained (APA, 2000).

Prevalence and Etiology. Obtaining consistent estimates of the prevalence of ID can be difficult due to the many definitions of ID and the methodology that is used in research where prevalence rates are studied (Leonard, 2002). As already mentioned, there were historic differences in the criteria used to define subaverage intellectual and adaptive functioning. For

example, in the 5th revision of the AAMR, subaverage intellectual functioning was defined as one standard deviation below the mean, whereas currently it is defined as two standard deviations below the mean. This ambiguity allowed for variability in the operational definitions of ID that were used in research, leading to a wide range of estimated prevalence rates. Furthermore, according to Leonard and Wen (2002), adaptive behavior is frequently excluded in the epidemiological research of ID; instead, IQ is used as the sole criterion. In an extensive literature review, Leonard and Wen (2002) found average prevalence rates for severe ID to be approximately 3.8 per 1,000, and 35 per 1,000 for mild ID. In general, higher rates of ID were reported for children than for adults, and in males than in females. In addition, severe ID (IQ<50) is likely to co-occur with other neurological conditions, while ID without neurological impairments is more likely to occur in individuals who are of higher birth order, African-American descent, and lower maternal education (Drews, Yeargin-Allsopp, Decoufle, & Murphy, 1995).

For approximately 30%-40% of individuals that are evaluated in clinical settings, the cause of ID cannot be determined. Specific predisposing factors for ID include heredity, changes in embryonic development, environmental deprivation, mental disorders, problems occurring around the time of birth, and medical conditions that are acquired in childhood and infancy (APA, 2000). Predisposing factors related to heredity occur through "autosomal recessive mechanisms," such as Tay-Sachs Disease, and chromosomal anomalies, including Down Syndrome, which is the most common genetic disorder that occurs with ID (Cans et al., 1999). Early changes in embryonic development can include damage due to infections or prenatal toxins (e.g. fetal alcohol syndrome). Environmental deprivation may be the result of a lack of nurturing, social contact, or other forms of stimulation. In terms of mental disorders, ASD are predisposing factors for ID. There can also be complications during pregnancy, such as a lack of adequate

nutrition, prematurity, or inadequate oxygen at birth. Finally, ID can be acquired by medical

conditions that occur early in life, such as "infections, traumas, and poisonings" (APA, 2000).

COMORBIDITY

ASD co-occur with a variety of medical disorders including seizure disorders, fragile X syndrome, tuberous sclerosis, epilepsy, and hearing and visual impairments (Gillberg & Billstedt, 2000; Tsai, 1996). In addition, a significant proportion of individuals with ASD, also exhibit ID, which is associated with increased rates of comorbid psychopathology (Borthwick-Duffy & Eyman, 1990; Bregman, 1991). The reported prevalence rates of comorbidity in ID range from 10% to 40%, with the variability attributed to the instruments used, diagnostic criteria selected, populations sampled, and psychiatric disorders included (Deb, Thomas, & Bright, 2001). Although there is a substantial amount of research to support high rates of ID in autism (70-80%), this finding has been disputed in an extensive review by Edelson (2006). In this review, it was noted that many of the studies that provide prevalence estimates were either dated, used inappropriate measures of intelligence, made claims that were not based on empirical sources, or did not take into account the effects of autism, such as motivation, on the validity of test results.

Despite the contributions of ID in the expression of psychopathology in ASD, preliminary research is unclear as to whether the addition of ASD increases one's risk for psychiatric problems (Bradley et al., 2004; La Malfa et al., 2007). For instance, Bradley et al. (2004) found that adolescents and adults with autism and ID exhibited more psychiatric symptomotology than those with ID only. However, Tsakanikos, Bouras, Sturmey, and Holt (2006), in a large scale study comparing adults with autism and ID versus adults with ID only, found no differences in terms of the number of psychiatric diagnoses.

Currently, there is a lack of research on ASD and comorbidity especially in comparison with other disorders. Of the limited research that exists, the majority is geared towards individuals who are higher functioning and have the ability to articulate their symptoms (Bradley

et al., 2004). For the lower functioning, nonverbal individuals, symptoms of psychopathology may be expressed differently than what is seen in the general population. This poses an additional challenge for clinicians. Instead of being able to rely on a person's verbal account of symptoms, which requires expressive communication skills and, to an extent, the ability to reflect on one's internal states, the clinician must rely on observable behavior. For instance, when assessing for depression in low functioning individuals with autism, Ghaziuddin, Ghaziuddin, and Greden (2002) suggest relying on vegetative signs, such as changes in functioning and regression of skills in place of verbal reports. An additional difficulty in diagnosing comorbid psychopathology in people with ASD is that there are problems in conceptualizing comorbid conditions with ASD. There is controversy as to whether specific symptoms should be viewed as evidence of a coexisting psychiatric disorder or as a manifestation of the core features of ASD. Another complicating factor is that ASD are heterogeneous in nature, with an expression that is compounded by varying degrees of ID (Matson & Nebel-Schwalm, 2007). This further complicates the diagnostic picture and, in turn, blurs the line that separates ASD from evidence of comorbid symptomotology.

In addition to being focused on high-functioning verbal individuals, research in comorbidity in this population has been primarily limited to children. This is not due to ASD and comorbidity only occurring in children, but a focus on younger populations seems to be the nature of ASD research in general. Consequently, the following discussion of comorbidity will incorporate both research on adults and children with ASD. Of course, results of research that are currently limited to children should be generalized to adults with caution. In regards to specific disorders, many have been identified as comorbid with ASD, including: Anxiety Disorders, Obsessive-Compulsive Disorder (OCD), Specific Fears, Depression, Conduct Disorder, Tic Disorders, Attention Deficit Hyperactivity Disorder (ADHD), Eating and Feeding

Disorders, Catatonia, Selective Mutism, and Psychotic Disorders. Rather than elaborate on an exhaustive list of all disorders that have been presented in the literature, for the purposes of this paper and subsequent scale development, only those that are deemed most important will be discussed.

Anxiety

Anxiety has been frequently described as a common feature of ASD; however, it has been infrequently measured in a systematic fashion (Gillott, Furniss, & Walter, 2001). Mesibov, Shea, and Schopler (2005) hypothesized that, in ASD, anxiety is a consequence of an inability to understand the environment and anticipate unexpected changes. Other researchers have suggested that anxiety is manifested in stereotypical behaviors that serve anxiety reducing functions (Gillott et al., 2001). Consistent with research in the area, the study of ASD and anxiety has been focused on higher functioning groups, particularly children and adolescents. A consensus in the literature is that these individuals experience higher levels of anxiety, and at greater rates, than in the general population (Bellini, 2004; Gillott et al., 2001; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000).

      Obsessive-Compulsive Disorder. Of particular interest is the relationship between Obsessive-Compulsive Disorder (OCD) and ASD and whether these two conditions can co-occur. ASD consists of stereotyped behaviors that have been described as obsessive and ritualistic. For instance, Gillott et al. (2001) found that, in comparison to language impaired and nondisabled controls, high functioning individuals with ASD scored higher on measures of separation anxiety, social anxiety, and OCD. Although ASD consists of repetitive behaviors that resemble those seen in OCD, the underlying forces driving these behaviors may be difficult to determine. This factor is especially true for lower-functioning populations. In OCD, an obsession is defined as an image, impulse, or thought that is persistent and that causes marked distress. A

compulsion is a repetitive act that serves to reduce tension brought upon by obsessions. The inability to describe one's thoughts interferes with the process of determining if obsessions and compulsions are present in accordance with the DSM-IV-TR criteria. In an attempt to shed light on the topic, McDougle, Kresch, and Goodman (1995) compared the content and topography of obsessions and compulsions in adults with autism to those with OCD and found significant differences in the types of behaviors endorsed. However, the authors noted that the two groups were not matched by IQ and that cognitive and communicative differences could have contributed to the differences. Conversely, Russell, Mataix-Cols, Anson, and Murphy (2005) found that adults with high-functioning ASD had a similar frequency of obsessions and compulsions as compared to a gender matched group with OCD. In addition, 25% of the individuals diagnosed with autism also met ICD-10 criteria for OCD.

Specific Fears. While there is research identifying specific fears in individuals with ASD, it is predominantly limited to case studies. The first study to compare the fears of individuals with autism against non-impaired controls was Matson and Love (1990). They found quantitative and qualitative differences in the type of fears endorsed by the two groups. The children with autism exhibited a greater number of specific common fears, and for this group, the most frequently endorsed items were a fear of dark places, getting punished by their father, the dentist, thunderstorms, closed places, and crowds. However, the non-impaired controls were most afraid of getting injured, small animals, and criticism. Expanding upon the work of Matson and Love (1990), Canavera, Kleinpeter, Maccubbin, and Taga (2005) compared the fears of children with ASD, Down Syndrome, and non-impaired controls. The children with autism were rated as having more situational phobias and medical fears, but less fears of harm.

In summary, it appears that symptoms of anxiety and fear commonly occur in ASD; however, little research has been done in individuals with severe cognitive impairments or

adults. If anxiety is manifested as a function of an inability to understand the environment and anticipate change, it would be expected that this tendency be magnified in individuals with more severe forms of ASD and ID.

Depression

A disorder that commonly co-occurs with anxiety in the general population is depression (Bear, Connors, & Paradiso, 2007). Although large scale studies on the prevalence of depression in ASD have not been carried out, existing research suggests that depression may be the most common associated psychiatric disorder (Ghaziuddin et al., 2002). The prevalence rates of depression in Autistic Disorder as estimated by Ghaziuddin et al. (1992) are approximately 2%, while there are considerably higher rates for those with Asperger's Syndrome (Ghaziuddin, Weidmer-Mikhail, & Ghaziuddin, 1998). However, lower rates of depression in autism may be a reflection of impaired cognitive and verbal abilities rather than an indication of a true absence of psychopathology. Especially for lower functioning groups, behavioral symptoms are frequently used in place of subjective accounts of depressive symptoms. Stewart et al. (2006) found that in cases of depression reported in the literature, the majority were based upon third party accounts of symptoms, such as: a loss of interest in activities, decreased appetite, sleep disturbance, increase in maladaptive behaviors, and a decrease in self help skills. Consequently, as the severity of symptoms related to ASD increase, as well as the level of ID, so does the difficulty of assessing comorbid depression. As such, instruments that can reliably assess depression in ASD, that are not dependent on verbal abilities, are greatly needed.

A relatively large amount of research on depression co-occurring with ASD has been published to date; other disorders have received less attention. People with ASD often exhibit maladaptive behaviors such as noncompliance, aggression, and property destruction. These behaviors may be consistent with disorders of conduct.

Conduct Disorder

Conduct Disorder is defined in the DSM-IV-TR as a "persistent pattern of behavior" where societal norms are violated by the presence of behaviors that involve: aggression to people or animals, property destruction, theft or deceitfulness, or a serious violation of rules (APA, 2000). Although there is some evidence to support a relationship between ASD and Conduct Disorder, the body of research in this area is, at present, lacking. Nevertheless, Gilmour, Hill, Place, and Skuse (2004) found an overlap between children with Conduct Disorder and ASD in the extent to which they exhibit social communicative deficits. In addition, in this study, a significant proportion of the children with conduct disorder also had an undiagnosed ASD. Other research has been conducted that compared the psychiatric, cognitive, and social functioning of adolescents with Asperger's Syndrome to those with Conduct Disorder. Green, Gilchrist, Di Burton, and Cox (2000) found that adolescents with Asperger's Syndrome demonstrated greater deficits in independent functioning and greater levels of anxiety and obsessive-compulsive behaviors than a control group with Conduct Disorder. Green et al. (2000) found that adolescents with Conduct Disorder could be differentiated from those with Asperger's Syndrome and high-functioning autism by differences in IQ profile, and by patterns of specific communicative and social behaviors.

Tic Disorders

Another group of conditions that co-occur with ASD are Tic Disorders. A tic is defined as a "sudden, rapid, recurrent, nonrhythmic, stereotyped motor movement or vocalization" (APA, 2000). There are four Tic Disorders defined in the DSM-IV-TR: Transient Tic Disorder, Chronic Motor or Vocal Tic Disorder, Tourette's Disorder, and Tic Disorder Not Otherwise Specified. The most well known of these is Tourette's Syndrome, which may be the most severe condition of the group, as it requires both vocal and motor tics and a duration exceeding one

year. Although ASD and Tic Disorders are distinct, Baron-Cohen, Scahill, Izaguirre, Hornsey, and Robertson (1999) point out that they share several behavioral similarities, including repetitive verbalizations, obsessive-compulsive type behaviors, and abnormal motor movements. The similarities, particularly in terms of abnormal motor movements, can complicate differential diagnosis. However, a key difference is that the stereotypies commonly observed in ASD appear rhythmic and calming in nature, whereas tics are primarily involuntary, occur in clusters, and are in reaction to physical tension in a part of the body (APA, 2000; Comings, 1990).

Currently prevalence data for Tic Disorders in ASD is confined to Tourette's Syndrome. Baron-Cohen et al. (1999) found that in a large sample of children with autism, 6.5% also met DSM-III-R criteria for Tourette's Syndrome. The presence of comorbid tics can have important implications. Gadow and DeVincent (2005) discovered that children with ASD, who also have tics or ADHD, are more likely to exhibit other forms of psychopathology, be prescribed psychotropic medications, and have more severe symptoms of ASD.

Attention Deficit Hyperactivity Disorder

The primary features of Attention Deficit Hyperactivity Disorder (ADHD) are inattention and/or hyperactivity-impulsivity that are both problematic and exceed what is typical for one's developmental level (APA, 2000). According to the DSM-IV-TR, a diagnosis of ADHD requires the presence of six or more symptoms of either inattention or hyperactivity-impulsivity that are present for at least six months. In addition, the behaviors must have a significant impact on the person's functioning, and the disorder cannot be diagnosed in the presence of ASD, psychotic disorder, or be better explained by another psychiatric disorder.

The exclusion of a mutual diagnosis of ASD and ADHD in the DSM-IV-TR has sparked controversy. For one, symptoms of both inattention and hyperactivity frequently occur in ASD. Autistic Disorder is often characterized by hyperactivity, impulsivity, and a short attention span

(APA, 2000). In fact the majority of children with autism show abnormal activity levels (Gillberg & Billstedt, 2000). Furthermore, studies of people with high-functioning autism and Asperger's Syndrome have shown attentional deficits (Gillberg & Billstedt, 2000). Due to the ubiquitous nature of these symptoms, some believe that they should be considered a component of ASD, rather than symptoms of ADHD. In addition, there was a fear that a diagnosis of ADHD would result in the use of stimulant medications, which would exacerbate the symptoms of ASD. However, opponents argue that a comorbid diagnosis should be considered when the symptoms are severe, thus allowing for a more tailored treatment.

Eating and Feeding Disorders

Frequently associated with ASD are the Eating and Feeding Disorders of the DSM-IV-TR, which consist of: Pica, Rumination, and Feeding Disorder. Pica is defined as the persistent eating of nonnutritive substances for longer than one month that is inappropriate for one's developmental level (APA, 2000). This disorder is relatively common in institutionalized settings. Ali (2001), in an extensive literature review, found the prevalence of Pica in institutionalized individuals to be between 9.2- 2.5%. Although more evidence is necessary, these rates may be even higher in ASD than in other developmental disabilities. For instance, Kinnell (1985) found that adults with autism were more likely to engage in pica than those with Down's Syndrome. The identification and treatment of Pica is crucial because this behavior can lead to severe medical complications, including: intestinal parasites, infections, surgical complications from removing objects, toxicity, and death (Kinnell, 1985).

Rumination Disorder is defined as the repeated regurgitation and rechewing of food, with a pattern that occurs for longer than one month that is not attributable to a general medical condition, anorexia, or bulimia (APA, 2000). In addition, if the symptoms occur along with ID or ASD, then they must be severe enough to require separate clinical attention. Although

prevalence rates for the occurrence of rumination and ASD are currently not available, 6-10% of institutionalized individuals who have severe or profound ID ruminate on a regular basis (Fredericks, Carr, & Williams, 1998). Rumination can lead to both medical and social problems. Medically it can lead to weight loss, gastric disorders, dental problems, and an increased risk of aspiration (Clauser & Scibak, 1990; Fredericks et al., 1998). In addition, social isolation can result from the foul smell of regurgitation.

Finally, Feeding Disorder is defined as an ongoing failure to eat, which results in an inability to gain weight or a substantial loss of weight that occurs for at least one month. This cannot be caused by another medical condition, gastrointestinal disorder, or be better accounted for by a mental disorder or a lack of food. In addition the onset must occur before the age of 6 years. Schreck, Williams, and Smith (2004) provided evidence that children with autism are especially selective in their eating habits, as shown by an extreme preference for foods of particular types and textures. Selectivity in its extreme form can lead to serious consequences. Complications of food refusal include malnutrition, family stress, dehydration, and/or the need for tube feeding (Didden, Seys, & Schouwink, 1999).

Assessment of Comorbidity

It is apparent that individuals with ID and ASD are at increased risk for psychiatric disorders; however, most research is limited to children who are higher-functioning and able to express their symptoms. Instruments that can accurately identify psychopathology in adults with ASD and ID, who have limited verbal capacity, are greatly needed. Currently the available instruments, with the exception of a new measure, the ASD-CA, are confined to people with ID only, although some have been used with ID populations where autism is a common comorbid condition. The following is a discussion of four measures to assess psychopathology in adults with ID.

Psychopathology Inventory for Mentally Retarded Adults. One of the first scales to measure psychopathology in individuals with ID was the Psychopathology Inventory for Mentally Retarded Adults (PIMRA; Matson, Kazdin, & Senatore, 1984). The PIMRA is based on DSM-III criteria and consists of 56 items that measure symptoms of psychopathology in the following areas: Anxiety, Adjustment Disorder, Psychosexual, Affective, Schizophrenia, Somatoform, Personality, and Inappropriate Mental Adjustment. The PIMRA is available in self-report and informant based formats, and it has been translated into several international versions. In terms of psychometrics, both forms have good internal consistency, test-retest reliability, and acceptable levels of inter-rater reliability (Gustafsson & Sonnander, 2004; Matson et al., 1984; Senatore, Matson, & Kazdin, 1985). In addition, for the informant based measure, support of validity has been established for the Depression and Schizophrenia subscales (Kazdin, Matson, & Senatore, 1983; Swiezy, Matson, Kirkpatrick-Sanchez, & Williams, 1995).

Reiss Screen for Maladaptive Behavior. Another of the original scales, the Reiss Screen for Maladaptive Behavior (RSMB; Reiss, 1997), is an informant based screening instrument that is used to determine if an adult or adolescent with ID has a comorbid mental health problem. The RSMB employs a three-component screening method to decrease the occurrence of false negatives (Reiss, 1997). The first way the RSMB can be used to determine the need for further mental health assessment is through the instrument's total score, which reflects the severity of psychopathology. The second is through the six maladaptive items that identify serious risks, such as suicide. The third is through scores on subscales that correspond to specific types of psychopathology.

The RSMB items are based on DSM-III criteria and load onto seven subscales: Avoidant Disorder, Dependent Personality Disorder, Aggressive Behavior, Depression (Physical Signs), Depression (Behavioral Signs), Paranoia, and Psychosis. The RSMB has good inter-rater

reliability, internal consistency, and moderate test-retest reliability (Sturmey, Burcham, & Perkins, 1995). In addition, the instrument shows moderate to good concurrent agreement with the Psychopathology Inventory and the Aberrant Behavior Checklist (Sturmey & Bertman, 1994).

Psychiatric Assessment Schedule for Adults with Developmental Disabilities. The Psychiatric Assessment Schedule for Adults with Developmental Disabilities Checklist (PAS-ADD Checklist; Moss et al., 1998) is an informant based screening instrument used to help identify the presence of Axis I disorders in individuals with ID. The checklist is based on the PAS-ADD interview (Moss, Patel, Prosser, & Goldberg, 1993), which is a clinician implemented semi-structured interview. The checklist consists of 29 items that describe symptoms in the following areas: Autism, Psychoses, Obsessions and Compulsions, Phobia and Panic, Tension and Worry, and Appetite and Sleep. Items are rated along a four-point scale, and then, using a scoring algorithm, three total scores are calculated for the following dimensions: Affective or Neurotic Disorder, Possible Organic Condition, and Psychotic Disorder. Individuals with total scores exceeding thresholds on the specified scales are considered to be at risk for a psychiatric disorder, and are recommended to receive further psychiatric evaluation. In terms of psychometrics, the PAS-ADD Checklist has acceptable levels of inter-rater reliability for the total scores, and acceptable internal validity on all but the Autism and Psychosis scales (Moss et al., 1998). In addition, the instrument is able to effectively discriminate diagnostic conditions with 66 % sensitivity and 70% specificity (Sturmey, Newton, Cowley, Bouras, & Holt, 2005).

Diagnostic Assessment for the Severely Handicapped, Second Edition. The Diagnostic Assessment for the Severely Handicapped, Second Edition (DASH-II) is an 84 item, informant based scale that was derived from DSM-III-R criteria and is used to screen for psychopathology in individuals with severe or profound ID (Matson, 1995). Items are presented

33

in the form of symptoms that are rated according to their frequency, duration, and severity within the last two weeks. The DASH-II has 13 subscales: Impulse, Organic, Anxiety, Mood, PDD/Autism, Schizophrenia, Stereotypies, Impulse, Self-Injurious Behavior, Elimination, Eating, Sleep, and Sexual. An elevated scale on this measure indicates the need for an additional assessment to rule out psychopathology. Several of the DASH subscales have been validated, including: Stereotypies, SIB, Mania, and Autism/PDD (Matson et al., 1997; Matson & Smiroldo, 1997; Matson, Smiroldo, & Hastings, 1998). This instrument also has good reliability, with test-retest reliability between .85-.91, and inter-rater reliability between .91-.95 (Sevin et al., 1991). A factor analysis yielded 5 factors for the DASH-II: Emotional Lability/Antisocial, Language Disorder, Dementia/Anxiety, Sleep Disorder, and Psychosis (Sturmey, Matson, & Lott, 2004). While the DASH-II was not specifically normed for individuals with ASD, it has been used in this population to investigate psychopathology. Bradley et al. (2004) found that adolescents with autism and ID displayed a greater number of elevated subscales on the DASH-II as compared to ID controls. In a similar study using the DASH-II, (La Malfa et al., 2007) found increased rates of psychiatric disturbances in adults with ASD. Matson et al.(1996) conducted a study using the DASH-II with two groups: ASD and non-ASD group. The authors found elevations on the mania and stereotypies subscale.

PURPOSE

Sufficient literature exists to suggest that ASD co-occurs with other forms of psychopathology (Matson & Nebel-Schwalm, 2007). For instance, Russell et al. (2005) found that 25% of adults with high-functioning autism also met ICD-10 criteria for OCD. Additionally, ASD also co-occurs with depression (Ghaziuddin et al., 2002; Stewart et al., 2006), anxiety disorders (Bellini, 2004; Gillott et al., 2001; Kim et al., 2000), tic disorders (Baron-Cohen et al., 1999), and eating disorders (Fredericks et al., 1998; Kinnell, 1985). Furthermore, ASD frequently co-occurs with ID, which may put a person at even greater risk for comorbid psychopathology (Borthwick-Duffy & Eyman, 1990; Bregman, 1991). Nevertheless, the majority of research has been done with children, specifically those who are higher functioning and are able to verbalize their symptoms (Bradley et al., 2004). This is unfortunate when one considers that the majority of people with ASD are adults, and the symptoms of ASD are exacerbated as a function of increased cognitive impairment (Matson & Dempsey, 2008). It would then follow that adults with ASD and severe and profound ID are an underrepresented group in the assessment and treatment literature who deserve greater attention in regards to comorbid psychopathology.

Until recently, scales did not exist that specifically examined comorbid psychopathology in adults with ASD and ID. Most existing scales, such as the PAS-ADD Checklist, RSMB, DASH-II, and PIMRA are limited to adults with ID in general. Furthermore, with the exception of the DASH-II, these scales are better equipped to assess individuals with moderate and mild ID. While these measures may have some utility in the ASD population, what is needed is an instrument that is specifically normed for adults with ID and ASD. Scales that examine comorbidity in people with ID are focused on disorders that are common to ID populations. However, a scale that is sensitive to the range of comorbid disorders found in ASD is necessary.

Furthermore, due to the heterogeneous nature of ASD, such a scale could assist in the differentiation of the core characteristics of ASD from symptoms of psychopathology. In doing so, this can lead to more tailored treatments that are designed specifically for ASD, or to the unique symptoms that exist as part of another comorbid disorder. For instance, while ASD has been shown to respond best to behavioral treatments, other disorders may benefit from different techniques and/or the use of psychotropic medications (Matson & Boisjoli, 2008). A measure designed to assess this particular population is urgently needed. Fortunately, a recent development is the creation of the Autism Spectrum Disorders-Comorbidity for Adults (ASD-CA; Matson & Boisjoli, 2008), which is a new instrument designed to measure comorbid psychopathology in adults with ASD and ID.

There were two primary purposes for this study. One was exploratory, to use the ASD-CA to compare the frequency of symptom endorsements among three groups: participants classified as having ID only (ID); participants classified as having both ID and ASD (ID + ASD); and participants classified as having ID, ASD, and additional psychopathology (ID + ASD + psychopathology). For all subscales of the ASD-CA, it was hypothesized that the ID + ASD group would score higher than the ID group. This is because the ASD-CA was created specifically for ASD, likely making it better equipped to detect symptom variations in these individuals. The second hypothesis was that, across subscales, the ID + ASD + psychopathology group would have the highest scores because the primary goal of the ASD-CA is to detect psychopathology. Hypotheses were tested by comparing diagnostic group differences using a multivariate analysis of variance (MANOVA) and appropriate follow up tests. The results of these analyses provided information relevant to the second purpose of this study, which was to develop cutoff scores for the ASD-CA that will be used to identify individuals who are at an increased risk for psychopathology, necessitating further evaluation.

36

METHOD

Participants

Participants were selected from two state-operated developmental centers located in central and southern Louisiana that provide services for individuals with developmental disabilities. Participants were classified into one of three groups: ID, ID + ASD, or ID + ASD + psychopathology. A checklist with DSM-IV-TR and ICD-10 criteria was used to classify ASD (i.e., Autistic Disorder or PDD-NOS). Classification of ASD was based upon the agreement of two independent raters. Diagnoses of additional psychopathology were based upon the clinical consensus of a psychiatrist and licensed psychologist, and were defined as a non-ASD, Axis I disorder. Approval for the study was granted from LSU and the Louisiana Department of Health and Hospitals' research review boards. Informed consent was obtained from the participant's legal advocates. The sample consisted of 313 individuals primarily with severe and profound ID, differing in age, race, gender, and verbal abilities (see Table 1).

Measures

Autism Spectrum Disorders-Comorbidity for Adults. The Autism Spectrum Disorders-Comorbidity for Adults (ASD-CA; Matson & Boisjoli, 2008) is a recently developed scale used to identify psychopathology in adults with ASD. The scale consists of 37 items that were chosen to reflect comorbidity reported to frequently occur with ASD. Items were created based upon a review of literature, current diagnostic criteria, and in reference to other scales that measure comorbid psychopathology in ID populations (Matson & Boisjoli, 2008). The ASD-CA is an informant based measure with items scored on a two point scale: (0) not different, no impairment, or (1) different, some impairment. Items load onto 5 subscales that were empirically derived through factor analysis: (1) Anxiety/Repetitive Behaviors, (2) Conduct Problems, (3)

Table 1
Demographic Characteristics per Diagnostic Group

Demographic characteristics	Diagnostic group		
	ID (n = 151)	ID + ASD (n = 120)	ID + ASD + psychopathology (n = 42)
Age (in years), Mean (SD)	55 (14.1)	49 (11.5)	48 (11.8)
Male, no. (%)	80 (53.0)	67 (55.8)	26 (61.9)
Female, no. (%)	71 (47.0)	53 (44.2)	16 (38.1)
Level of ID, no. (%)			
Profound	95 (62.9)	108 (90.0)	36 (85.7)
Severe	36 (23.8)	6 (5.0)	1 (2.4)
Moderate	12 (7.9)	2 (1.7)	2 (4.8)
Mild	2 (1.3)	0 (0.0)	0 (0.0)
Unspecified	6 (4.0)	4 (3.3)	3 (7.1)
Race/ethnicity, no. (%)			
Caucasian	114 (75.5)	98 (81.7)	32 (76.2)
African American	37 (24.5)	22 (18.3)	9 (21.4)
Hispanic	0 (0.0)	0 (0.0)	1 (2.4)
Other	0 (0.0)	0 (0.0)	0 (0.0)
Verbal (yes), no. (%)	79 (52.3)	31 (25.8)	17 (71.4)
Psychotropic med. (yes), no. (%)	0 (0.0)	19 (15.8)	30 (71.4)

Irritability/Behavioral Excesses, (4) Attention/Hyperactivity/Impulsivity, and (5) Depressive Symptoms (Matson & Boisjoli, 2008). According to Matson and Boisjoli (2008), the ASD-CA has moderate to good reliability. Using the 37 retained items, kappa coefficients for inter-rater reliability ranged from .30 to .77 with an average kappa for all the items of .43. Kappa values were in the range of .35 to .92 for test-retest reliability with an overall average kappa of .59. The internal consistencies of the subscales were poor to adequate with KR-20 coefficients ranging from 0.44 to 0.85. However, the internal consistency of the entire scale was good with a KR-20 coefficient of 0.91 (Matson & Boisjoli, 2008).

Research Design

Subscale scores were created for each of the five factors of the ASD-CA, Anxiety/Repetitive Behaviors, Conduct Problems, Irritability/Behavioral Excesses, Attention/Hyperactivity/Impulsivity, and Depressive Symptoms. Subscale scores were calculated for participants by summing item scores contained within each factor. A Total subscale score was then created for participants by summing the five primary subscale scores. While the primary subscales were intended to provide a measure of psychopathology consistent with the respective subscale labels, the Total subscale score was intended to provide an overall measure of psychopathology.

Study 1 was conducted using multivariate analysis of variance (MANOVA) to determine if subscale scores differed as a function of diagnostic group. The multivariate test was followed by analyses of variance and post hoc tests, as needed, to further localize group differences.

Study 2 was conducted using a standard deviation approach to calculating cutoff scores, based upon the logic of Jacobson and Truax (1991) in their description of clinically significant change. Using scores on a hypothetical measure of psychopathology, clinically significant change was conceptualized as successful treatment moving a client's score either outside the

range of the dysfunctional distribution, or within the range of the functional distribution. This concept was operationalized in three ways: (1) a post treatment score two standard deviations away from the mean of the dysfunctional distribution in the direction of functionality; (2) a post treatment score two standard deviations within the mean of the functional distribution; or (3) a post treatment score closer to the mean of the functional distribution than the dysfunctional distribution. Opposite of Jacobson and Truax (1991), the goal of Study 2, was to calculate cutoff scores that indicate points at which an individual is at an increased probability of belonging to a dysfunctional distribution. In addition, because the ASD-CA was intended to detect a large number of individuals with potential comorbid psychopathology (at the expense of false positives), a more lenient cutoff criterion of one standard deviation was chosen. This criterion has been used in other scales that measure psychopathology in the ID population (Matson, Coe, Gardner, & Sovner, 1991; Matson, Fodstad, & Boisjoli, 2008). Despite the differences between Study 2 and Jacobson and Truax's (1991) concept of clinically significant change, the logic of both are the same in that they attempt to define cutoff points that help divide functionality from dysfunctionality.

Consistent with the logic of operational definition "(1)," as described above, cutoffs for the ASD-CA were defined as scores one standard deviation away from the ID + ASD group means in the direction of dysfunctionality. This criterion was chosen over criteria "(2)" and "(3)" because there were not enough participants to create adequately sized, matching dysfunctional distributions for the types of psychopathology implied by the subscales of the ASD-CA. The ID + ASD + psychopathology group was not used for developing cutoff scores, but was instead created for exploratory purposes, and used in Study 1 to determine if individuals with various types of psychopathology (see Table 2) would collectively score higher than those without psychopathology.

Table 2
Comorbid Disorders in ID + ASD + psychopathology

Disorder	n	%
Pica	15	35.71
Bipolar NOS	9	21.43
Mood NOS	5	11.90
Major Depressive	3	7.14
PTSD	3	7.14
Psychotic NOS	2	4.76
Tic NOS	2	4.76
Alzheimer's	1	2.38
Anxiety NOS	1	2.38
ADHD	1	2.38
Rumination	1	2.38

RESULTS

Prior to the analyses, data were examined for missing values, errors in data entry, outliers, and consistency with the assumptions of MANOVA. For all possible item values (11,951), five missing values (0.04%) were identified and replaced with the mean (Tabachnick & Fidell, 2007). Next, all remaining item values were inspected and were either 1 or 0, consistent with the scoring of the ASD-CA, and providing support for the accuracy of data entry. The remainder of data screening procedures were conducted by examining the dependent variables separately, according to diagnostic group (ID, ID + ASD, ID + ASD + psychopathology). Using a criterion of z scores greater than 3.29 (Tabachnick & Fidell, 2007), 10 participants (4 in ID; 2 in ID + ASD, and 4 in ID + ASD + psychopathology) had one subscale score identified as a univariate outlier. These participants were removed from the analysis. Finally, there were no multivariate outliers identified using Mahalanobis distance with a significance value of p < .001 (Tabachnick & Fidell, 2007).

Study 1

Study 1 was analyzed using SPSS 16.0 and a between-subjects MANOVA with diagnostic group (ID, ID + ASD, and ID + ASD + psychopathology) as the independent variable, and the subscale scores of the ASD-CA (Anxiety/Repetitive Behaviors, Conduct Problems, Irritability/Behavioral Excesses, Attention/Hyperactivity/Impulsivity, Depressive Symptoms, and Total) as the dependent variables. In order to determine the necessary sample size, an *a priori* power analysis was conducted using GPOWER (Erdfelder, Faul, & Buchner, 1996) with the following parameters: an effect size of 0.25 (Cohen's f^2), alpha of .05, power of 0.80, and 6 dependent variables. The power analysis resulted in a minimum total sample size of 42. In general, the robustness of MANOVA is influenced by several factors, including: discrepancy in sample sizes, multivariate normality, homogeneity of variance-covariance matrices, linearity,

and multicollinearity. Due to substantially uneven sample sizes, for Study 1 the numbers of participants per diagnostic group were made even by randomly deleting cases while matching by level of intellectual disability. In all, 109 participants were removed from the ID group, 78 from the ID + ASD group, and 0 from the ID + ASD + psychopathology group. This reduction resulted in an even number of participants (42) for all three diagnostic groups: 38 profound (86.4%), 1 severe (2.3%), 2 moderate (4.5%), 0 mild (0%), and 3 unspecified (6.8%).

In regards to the other assumptions of MANOVA, multivariate normality was ensured by adequate sample sizes, while homogeneity of variance-covariance matrices was ensured by an equal number of participants per group. Inspection of bivariate scatterplots revealed adequate linearity to proceed with the analysis. Finally, the presence of moderate correlations between dependent variables suggested the absence of multicollinearity.

Using Wilks' lambda, the multivariate test was significant for diagnostic group $F_{(12, 236)} = 2.25$, $p < .05$. In order to determine the nature of this effect, separate analyses of variance (ANOVAs) were performed for each dependent variable. Although Levene's test was significant for all subscales, homogeneity of variance was protected by equal sample sizes (Field, 2005). Significant univariate main effects were present for the following subscales: Anxiety/Repetitive Behaviors, $F_{(2, 123)} = 7.61$, $p < .01$; Irritability/Behavioral Excesses, $F_{(2, 123)} = 6.19$, $p < .01$; Attention/Hyperactivity/Impulsivity, $F_{(2, 123)} = 6.53$, $p < .01$; Depressive Symptoms, $F_{(2, 123)} = 3.23$, $p < .05$; and Total, $F_{(2, 123)} = 7.00$, $p < .01$. For subscales with significant univariate results, post hoc tests, using Games-Howell as the test statistic, were conducted to better localize group differences. In general, significant pairwise differences existed between ID and ID + ASD, and ID and ID + ASD + psychopathology (see Table 3).

Table 3

Means, Standard Deviations, and Significant Group Differences per Subscale of the ASD-CA

Subscales	Diagnostic group		
	ID only	ID and ASD	ID, ASD, psychopathology
Anxiety/Repetitive Behaviors			
Mean (S.D.)	0.05 [a, b] (0.22)	0.83 [a] (1.34)	0.38 [b] (0.85)
Conduct Problems			
Mean (S.D.)	1.57 (2.36)	2.86 (3.69)	2.38 (2.52)
Irritability/Behavioral Excesses			
Mean (S.D.)	1.36 [a, b] (1.82)	2.90 [a] (2.45)	2.60 [b] (2.08)
Attention/Hyperactivity/Impulsivity			
Mean (S.D.)	1.17 [a, b] (1.48)	2.40 [a] (2.40)	2.55 [b] (1.78)
Depressive Symptoms			
Mean (S.D.)	0.26 [a] (0.63)	0.74 [a] (1.04)	0.50 (0.86)
Total subscale			
Mean (S.D.)	4.40 [a, b] (4.92)	9.81 [a] (9.06)	8.40 [b] (5.94)

Note: For each row, means sharing superscripts are significantly different at *p < .05*. For all subscales, higher means indicate higher symptom endorsements.

Study 2

Although it was necessary to use reduced the sample size for Study 1 in order to ensure the robustness of the MANOVA and the univariate tests, removed participants were reinserted back into the sample for Study 2. Cutoff scores were determined by calculating values one standard deviation above the respective ID + ASD subscale means and then rounding to the nearest whole or half number depending on the mean and standard deviation of the particular subscale. For instance, because Depressive Symptoms had a small group mean (0.53) and small

standard deviation (0.87), a cutoff score of 1.5 was selected. This was done because a cutoff score of 1 would have been equivalent to 0.54 standard deviations above the mean, while a cutoff score of 2 would have been equivalent to 1.70 standard deviations above the mean. Resulting means, standard deviations, and cutoff scores are presented in Table 4.

Table 4
Subscale Means, Standard Deviations, and Cutoff Scores

Subscale	M	SD	Cutoff Score
Anxiety/Repetitive Behaviors	0.4	0.8	1.5
Conduct Problems	1.99	2.57	5.0
Irritability/Behavioral Excesses	2.44	2.19	5.0
Attention/Hyperactivity/Impulsivity	2.16	1.99	5.0
Depressive Symptoms	0.53	0.87	1.5
Total	7.52	6.11	14.0

DISCUSSION

The hypotheses set forth met with mixed results. In general the ID + ASD group scored significantly higher than the ID group on most subscales except for Conduct Problems. As mentioned, a probable explanation for this significant finding was that the ASD-CA was created specifically for ASD, making it more sensitive to symptom variations in these individuals. It was surprising that the same result did not hold true for the Conduct subscale, especially when you consider that autism is a risk factor for challenging behaviors (Dominick, Lainhart, Tager-Flusberg, & Folstein, 2007; Hill & Furniss, 2006; Macdonald et al., 2007). This non-significant finding may be partially explained by the relationship between challenging behaviors and the use of psychotropic medications in persons with ID (Matson, Bamburg, et al., 2000; Matson, Bielecki, Mayville, & Matson, 2003). Although evidence for the effectiveness of psychotropic medications for challenging behaviors is currently inconclusive (Matson & Neal, in press), it is interesting to note that for the entire sample, 15.8% of persons with ID + ASD were prescribed psychotropic medications as opposed to 0% of persons with ID.

It was also expected that the ID + ASD + psychopathology group would have the highest subscale scores, because the ASD-CA was designed specifically for detecting comorbid psychopathology in this population. As reported, this was not the case, likely the result of using a generic psychopathology group instead of ones specific to each subscale. Potentially, if each subscale had its own psychopathology group, such as a comorbid anxiety group for Anxiety/ Repetitive Behaviors, there would be significant group differences. Again, another plausible explanation for this unexpected result was the substantial difference in psychotropic medication use between ID + ASD (15.8%), and ID + ASD + psychopathology (71.4%). Psychotropic medication can have an overall suppressive effect on behavior (Baumeister & Sevin, 1990;

Matson, Bamburg, et al., 2000), and its use may have been at least partially responsible for the lower scores of the psychopathology group.

Although Study 1 was conducted using a reduced sample size with groups that were matched by level of ID, the results of the analyses portrayed an overall pattern among diagnostic groups in relation to the subscales of the ASD-CA, and highlighted the inadequacy of the generic psychopathology group in producing the predicted score differences. In Study 2, cutoff scores were created for the ASD-CA to help identify individuals with ID + ASD who are more likely to have comorbid psychopathology. Although the creation of cutoff scores was an important step in the development of the ASD-CA, studies are needed to further examine the scale's usefulness. For instance, the convergent and discriminate validity of the ASD-CA should be investigated by correlating its subscales with other scales that measure related constructs. Also, with larger multi-site investigations it may be possible to evaluate aspects of concurrent validity by examining the extent to which the ASD-CA is able to discriminate among individuals with various types of comorbid psychopathology. It will also be important to investigate the effects of base rates on the accuracy of the ASD-CA. Although reliability and validity are often considered to be the ultimate yardsticks in evaluating the accuracy of a psychological test, their importance can be dwarfed by discrepant base rates in the population (Gouvier, 2001). Higher ratios of individuals with psychopathology to those without psychopathology will result in a higher number of false positives when using the ASD-CA. Base rates discrepancies can greatly impact the interpretation of the ASD-CA and should therefore be evaluated in future studies.

Because the ASD-CA is the only scale designed to measure psychopathology in adults with ID and ASD, its further development is urgently needed. The creation of this scale has been long awaited considering the evidence that ASD co-occurs with other forms of psychopathology, and that, until recently, adults with ID and ASD have been long neglected in the assessment and

treatment literature. While existing scales measure psychopathology in adults with ID, the ASD-CA's specific focus of measuring psychopathology in adults with ID and ASD should be of increased utility for this population. The present study was intended to be exploratory and to further develop the ASD-CA by developing cutoff scores for its subscales. It is hoped that this study represents a seminal step in the development of a scale that will aide in the identification of comorbid psychopathology in adults with ID and ASD, resulting in more tailored and effective treatments.

REFERENCES

Ali, Z. (2001). Pica in people with intellectual disability: A literature review of aetiology, epidemiology and complications. *Journal of Intellectual & Developmental Disability, 26*, 205-215.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, D.C.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, D.C.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, D.C.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th, text rev ed.). Washington, DC.

Asperger, H. (1944). Die "autistichen Psychopathen" im Kindersalter. *Archive fur Psychiatrie und Nervenkrankheiten, 117*, 76-136.

Asperger, H., & Frith, U. (1991). *'Autistic psychopathy' in childhood.* New York, NY, US: Cambridge University Press.

Baron-Cohen, S., Scahill, V. L., Izaguirre, J., Hornsey, H., & Robertson, M. M. (1999). The prevalence of Gilles de la Tourette syndrome in children and adolescents with autism: A large scale study. *Psychological Medicine, 29*, 1151-1159.

Baumeister, A., & Sevin, J. (1990). Pharmacologic control of aberrant behavior in the mentally retarded: Toward a more rational approach. *Neuroscience & Biobehavioral Reviews*, *14*, 253-262.

Bear, M. F., Connors, B. W., & Paradiso, M. A. (2007). *Neuroscience: exploring the brain.* Batlimore, MD: Lippincott Williams & Wilkins.

Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities, 19*, 78-86.

Borthwick-Duffy, S. A., & Eyman, R. K. (1990). Who are the dually diagnosed? *American Journal on Mental Retardation, 94*, 586-595.

Bradley, E. A., Summers, J. A., Wood, H. L., & Bryson, S. E. (2004). Comparing rates of psychiatric and behavior disorders in adolescents and young adults with severe intellectual disability with and without Autism. *Journal of Autism and Developmental Disorders, 34*, 151-161.

Bregman, J. D. (1991). Current developments in the understanding of mental retardation: II. Psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry, 30*, 861-872.

Cans, C., Wilhelm, L., Baille, M. F., du Mazaubrun, C., Grandjean, H., & Rumeau-Rouquette, C. (1999). Aetiological findings and associated factors in children with severe mental retardation. *Developmental Medicine & Child Neurology, 41*, 233-329.

Clauser, B., & Scibak, J. W. (1990). Direct and generalized effects of food satiation in reducing rumination. *Research in Developmental Disabilities, 11*, 23-36.

Comings, D. E. (1990). *Tourette Syndrome and human behavior*. Duarte, CA: Hope Press.

Creak, M. (1964). Schizophrenia syndrome in childhood: Progress report of a working party. *Cerebral Palsy Bulletin, 3*, 501–504.

Croen, L. A., Grether, J. K., Hoogstrate, J., & Selvin, S. (2002). The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders, 32*, 207-215.

Cuskelly, M. (2004). The evolving construct of intellectual disability: Is everything old new again? *International Journal of Disability, Development and Education, 51*, 117-122.

Deb, S., Thomas, M., & Bright, C. (2001). Mental disorder in adults with intellectual disability. I: Prevalence of functional psychiatric illness among a community-based population aged between 16 and 64 years. *Journal of Intellectual Disability Research, 45*, 495-505.

Didden, R., Seys, D., & Schouwink, D. (1999). Treatment of chronic food refusal in a young developmentally disabled child. *Behavioral Interventions, 14*, 213-222.

DiLavore, P. C., Lord, C., & Rutter, M. (1995). Pre-linguistic autism diagnostic observation schedule. *Journal of Autism and Developmental Disorders, 25*, 355-379.

Dominick, K., Davis, N., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities*, *28*, 145-162.

Drews, C. D., Yeargin-Allsopp, M., Decoufle, P., & Murphy, C. C. (1995). Variation in the influence of selected sociodemographic risk factors for mental retardation. *American Journal of Public Health, 85*, 329-334.

Edelson, M. G. (2006). Are the majority of children with autism mentally retarded? A systematic evaluation of the data. *Focus on Autism and Other Developmental Disabilities, 21*, 66-83.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments & Computers, 28*, 1-11.

Evans, D. W., Canavera, K., Kleinpeter, F. L., Maccubbin, E., & Taga, K. (2005). The fears, phobias and anxieties of children with autism spectrum disorders and Down Syndrome: Comparisons with developmentally and chronologically age matched children. *Child Psychiatry & Human Development, 36*, 3-26.

Field, A. (2005). *Discovering statistics using SPSS (2nd ed.)*. Thousand Oaks, CA US: Sage Publications, Inc.

Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities, 18*, 281-294.

Fredericks, D. W., Carr, J. E., & Williams, W. L. (1998). Overview of the treatment of rumination disorder for adults in a residential setting. *Journal of Behavior Therapy and Experimental Psychiatry, 29*, 31-40.

Gadow, K. D., & DeVincent, C. J. (2005). Clinical significance of tics and Attention-Deficit Hyperactivity Disorder (ADHD) in children with Pervasive Developmental Disorder. *Journal of Child Neurology, 20*, 481-488.

Ghaziuddin, Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research, 42*, 279-283.

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

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

Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism, 5*, 277-286.

Gilmour, J., Hill, B., Place, M., & Skuse, D. H. (2004). Social communication deficits in conduct disorder: A clinical and community survey. *Journal of Child Psychology and Psychiatry, 45*, 967-978.

Gouvier, W. (2001). Are you sure you're really telling the truth? *NeuroRehabilitation*, *16*, 215-219.

Green, J., Gilchrist, A., Burton, D., & 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.

Greenspan, S. (1999). What is meant by mental retardation? *International Review of Psychiatry, 11*, 6-18.

Gustafsson, C., & Sonnander, K. (2004). A psychometric evaluation of a Swedish version of the psychopathology inventory for mentally retarded adults (PIMRA). *Research in Developmental Disabilities, 26*, 183-201.

Heber, R. (1959). *A manual on terminology and classification on mental retardation* (Rev. ed.). Washington, DC: American Association on Mental Deficiency.

Hill, J., & Furniss, F. (2006). Patterns of emotional and behavioural disturbance associated with autistic traits in young people with severe intellectual disabilities and challenging behaviours. *Research in Developmental Disabilities*, *27*, 517-528.

Honda, H., Shimizu, Y., & Rutter, M. (2005). No effect of MMR withdrawal on the incidence of autism: a total population study. *Journal of Child Psychology and Psychiatry, 46*, 572-579.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*, 12-19.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2*, 217-250.

Kazdin, A. E., Matson, J. L., & Senatore, V. (1983). Assessment of depression in mentally retarded adults. *American Journal of Psychiatry, 140*, 1040-1043.

Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism, 4*, 117-132.

Kinnell, H. G. (1985). Pica as a feature of autism. *British Journal of Psychiatry, 147*, 80-82.

Klinger, L. G., & Renner, P. (2000). Performance-based measures in autism: Implications for diagnosis, early detection, and identification of cognitive profiles. *Journal of Clinical Child Psychology, 29*, 479-492.

Kolvin, I., Ounsted, C., Humphrey, M., & McNay, A. (1971). Studies in the childhood psychoses: II. the phenomenology of childhood psychoses. *British Journal of Psychiatry, 118*, 385-395.

Krug, D. A., Arick, J., & Almond, P. (1980). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. *Journal of Child Psychology and Psychiatry, 21*, 221-229.

La Malfa, G., Lassi, S., Salvini, R., Giganti, C., Bertelli, M., & Albertini, G. (2007). The relationship between autism and psychiatric disorders in Intellectually Disabled Adults. *Research in Autism Spectrum Disorders, 1*, 218-228.

Le Couteur, A., Rutter, M., Lord, C., & Rios, P. (1989). Autism Diagnostic Interview: A standardized investigator-based instrument. *Journal of Autism and Developmental Disorders, 19*, 363-387.

Leonard, H., & Wen, X. (2002). The epidemiology of mental retardation: Challenges and opportunities in the new millennium. *Mental Retardation and Developmental Disabilities Research Reviews, 8*, 117-134.

Lord, C. (1997). Diagnostic instruments in autism spectrum disorders. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders (2nd ed.)* (pp. 460-483). Hoboken, NJ, US: John Wiley & Sons Inc.

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The Autism Diagnostic Observation Schedule--Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30*, 205-223.

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

Lord, C., Rutter, M. L., Goode, S., & Heemsbergen, J. (1989). Autism Diagnostic Observation Schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders, 19*, 185-212.

Lotter, V. (1966). Epidemiology of autistic conditions in young children: Prevalence. *Social Psychiatry, 1*, 124-127.

Luckasson, R., Coulter, D. L., Polloway, E. A., Reese, S., Schalock, R. L., Snell, M. E., et al. (1992). *Mental retardation: Definition, classification, and systems of supports* (9th ed.). Washington, DC: American Association on Mental Retardation.

MacDonald, R., Green, G., Mansfield, R., Geckeler, A., Gardenier, N., Anderson, J., et al. (2007). Stereotypy in young children with autism and typically developing children. *Research in Developmental Disabilities*, *28*, 266-277.

Matson, J. L. (1995). *The Diagnostic Assessment of the Severely Handicapped –Revised (DASH-II)*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J. L., Baglio, C. S., Smiroldo, B. B., Hamilton, M., Packlowskyj, T., Williams, D., et al. (1996). Characteristics of autism as assessed by the Diagnostic Assessment for the Severely Handicapped-II (DASH-II). *Research in Developmental Disabilities, 17*, 135-143.

Matson, J., Bamburg, J., Mayville, E., Pinkston, J., Bielecki, J., Kuhn, D., et al. (2000). Psychopharmacology and mental retardation: A 10 year review (1990-1999). *Research in Developmental Disabilities*, *21*, 263-296.

Matson, J., Bielecki, J., Mayville, S., & Matson, M. (2003). Psychopharmacology research for individuals with mental retardation: Methodological issues and suggestions. *Research in Developmental Disabilities*, *24*, 149-157.

Matson, J. L., & Boisjoli, J. A. (2008). Autism spectrum disorders in adults with intellectual disability and comorbid psychopathology: Scale development and reliability of the ASD-CA. *Research in Autism Spectrum Disorders, 2*, 276-287.

Matson, J.L., Fodstad, J.C., & Boisjoli, J.A. (2008). Cutoff scores, norms and patterns of feeding problems for the Screening Tool of fEeding Problems (STEP) for adults with intellectual disabilities. *Research in Developmental Disabilities*, *29*, 363-372.

Matson, J., Coe, D., Gardner, W., & Sovner, R. (1991). A factor analytic study of the Diagnostic Assessment for the Severely Handicapped scale. *Journal of Nervous and Mental Disease*, *179*, 553-557.

Matson, J. L., & Dempsey, T. (2008). Stereotypy in adults with autism spectrum disorders: Relationship and diagnostic fidelity. *Journal of Developmental and Physical Disabilities, 20*, 155-165.

Matson, J. L., Dempsey, T., LoVullo, S. V., & Wilkins, J. (2008). The effects of intellectual functioning on the range of core symptoms of autism spectrum disorders. *Research in Developmental Disabilities, 29,* 341-350.

Matson, J. L., Hamilton, M., Duncan, D., Bamburg, J., Smiroldo, B., Anderson, S., et al. (1997). Characteristics of stereotypic movement disorder and self-injurious behavior assessed with the Diagnostic Assessment for the Severely Handicapped (DASH-II). *Research in Developmental Disabilities, 18*, 457-469.

Matson, J. L., Kazdin, A. E., & Senatore, V. (1984). Psychometric properties of the psychopathology instrument for mentally retarded adults. *Applied Research in Mental Retardation, 5*, 81-89.

Matson, J. L., & Love, S. R. (1990). A comparison of parent-reported fear for autistic and nonhandicapped age-matched children and youth. *Australia & New Zealand Journal of Developmental Disabilities, 16*, 349-357.

Matson, J. L., & Minshawi, N. F. (2006). Early Intervention for autism spectrum disorders, volume 1: A critical analysis. Amsterdam, the Netherlands: Elsevier Science.

Matson, J.L., & Neal, D. (in press). Psychotropic medication use for challenging behaviors in persons with intellectual disabilities: An overview. *Research in Developmental Disabilities.*

Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities, 28*, 341-352.

Matson, J. L., Nebel-Schwalm, M. S., & Matson, M. L. (2007). A review of methodological issues in the differential diagnosis of autism spectrum disorders in children. *Research in Autism Spectrum Disorders, 1*, 38-54.

Matson, J. L., & Smiroldo, B. B. (1997). Validity of the Mania subscale of the Diagnostic Assessment for the Severely Handicapped-II (DASH-II). *Research in Developmental Disabilities, 18*, 221-225.

Matson, J. L., Smiroldo, B. B., & Hastings, T. L. (1998). Validity of the Autism/PDD Subscale of the DASH-II. *Journal of Autism and Developmental Disorders, 28*, 77-81.

McDougle, C. J., Kresch, L. E., & Goodman, W. K. (1995). A case-controlled study of repetitive thoughts and behavior in adults with autistic disorder and obsessive-compulsive disorder. *American Journal of Psychiatry, 152*, 772-777.

Mesibov, G. B., Shea, V., & Schopler, E. (2005). *The TEACCH approach to autism spectrum disorders*. New York, NY: Kluwer Academic/Plenum Publishers.

Moss, S., Patel, P., Prosser, H., & Goldberg, D. (1993). Psychiatric morbidity in older people with moderate and severe learning disability: I. Development and reliability of the patient interview (PAS-ADD). *British Journal of Psychiatry, 163*, 471-480.

Moss, S., Prosser, H., Costello, H., Simpson, N., Patel, P., Rowe, S., et al. (1998). Reliability and validity of the PAS-ADD Checklist for detecting psychiatric disorders in adults with intellectual disability. *Journal of Intellectual Disability Research, 42*, 173-183.

Perry, A., Condillac, R. A., Freeman, N. L., Dunn-Geier, J., & Belair, J. (2005). Multi-site study of the Childhood Autism Rating Scale (CARS) in five clinical groups of young children. *Journal of Autism and Developmental Disorders, 35*, 625-634.

Reiss, S. (1997). Comments on the Reiss Screen for Maladaptive Behaviour and its factor structure. *Journal of Intellectual Disability Research, 41*, 346-354.

Ritvo, E. R., & Freeman, B. J. (1978). National Society for Autistic Children definition of the syndrome of autism. *Journal of Pediatric Psychology, 2*, 142-145.

Russell, A. J., Mataix-Cols, D., Anson, M., & Murphy, D. G. M. (2005). Obsessions and compulsions in Asperger syndrome and high-functioning autism. *British Journal of Psychiatry, 186*, 525-528.

Rutter, M. (1978). Diagnosis and definitions of childhood autism. *Journal of Autism & Childhood Schizophrenia, 8*, 139-161.

Rutter, M., & Schopler, E. (1988). In E. Schopler & G. B. Mesibov (Eds.), *Diagnosis and Assessment in Autism*. NY: Plenum Press.

Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood Autism Rating Scale (CARS). *Journal of Autism and Developmental Disorders, 10*, 91-103.

Schreck, K. A., Williams, K., & Smith, A. F. (2004). A comparison of eating behaviors between children with and without autism. *Journal of Autism and Developmental Disorders, 34*, 433-438.

Senatore, V., Matson, J. L., & Kazdin, A. E. (1985). An inventory to assess psychopathology of mentally retarded adults. *American Journal of Mental Deficiency, 89*, 459-466.

Sevin, J. A., Matson, J. L., Coe, D. A., & Fee, V. E. (1991). A comparison and evaluation of three commonly used autism scales. *Journal of Autism and Developmental Disorders, 21*, 417-432.

Smeeth, L., Cook, C., Fombonne, E., Heavey, L., Rodrigues, L. C., Smith, P. G., et al. (2004). MMR vaccination and pervasive developmental disorders: A case-control study. *Lancet, 364*, 963-969.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984). *Vineland Adaptive Behavior Scales*. Cirle pines, MN: American Guidance Service.

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.

Sturmey, P., & Bertman, L. J. (1994). Validity of the Reiss Screen for Maladaptive Behavior. *American Journal on Mental Retardation, 99*, 201-206.

Sturmey, P., Burcham, K. J., & Perkins, T. S. (1995). The Reiss Screen for Maladaptive Behaviour: Its reliability and internal consistencies. *Journal of Intellectual Disability Research, 39*, 191-195.

Sturmey, P., Matson, J. L., & Lott, J. D. (2004). The factor structure of the DASH-II. *Journal of Developmental and Physical Disabilities, 16*, 247-255.

Sturmey, P., Newton, J., Cowley, A., Bouras, N., & Holt, G. (2005). The PAS-ADD Checklist: Independent replication of its psychometric properties in a community sample. *British Journal of Psychiatry, 186*, 319-323.

Swiezy, N. B., Matson, J. L., Kirkpatrick-Sanchez, S., & Williams, D. E. (1995). A criterion validity study of the schizophrenia subscale of the Psychopathology Instrument for Mentally Retarded Adults (PIMRA). *Research in Developmental Disabilities, 16*, 75-80.

Tabachnick, B., & Fidell, L. (2007). Using multivariate statistics (5th ed.). Boston, MA : Allyn & Bacon/Pearson Education.

Towbin, K. E. (1997). Pervasive Developmental Disorder Not Otherwise Specified. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders (2nd ed.)* (pp. 123-148). Hoboken, NJ, US: John Wiley & Sons Inc.

Tsai, L. Y. (1996). Breif Report: Comorbid psychiatric disorders of autistic disorder. *Journal of Autism and Developmental Disorders, 26*, 159-163.

Tsakanikos, E., Bouras, N., Sturmey, P., & Holt, G. (2006). Psychiatric co-morbidity and gender differences in intellectual disability. *Journal of Intellectual Disability Research, 50*, 582-587.

Volkmar, F. R., Cicchetti, D. V., Dykens, E., & Sparrow, S. S. (1988). An evaluation of the Autism Behavior Checklist. *Journal of Autism and Developmental Disorders, 18*, 81-97.

Volkmar, F. R., Klin, A., & Cohen, D. J. (1997). Diagnosis and classification of autism and related conditions: consensus and issues. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders (2nd ed.)* (pp. 5-40). Hoboken, NJ, US: John Wiley & Sons Inc.

Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., & Malik, M. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet, 351*, 637-641.

Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews, 8*, 151-161.

World Health Organization. (1992). *International Statistical Classification of Diseases and Related Health Problems* (10th ed.). Geneva: Author.

APPENDIX

Items of the ASD-CA

1   Easily becomes upset.

2   Concentration problems.

3   Repetition of actions or words to reduce stress.

4   Restless.

5   Interrupts the activities of others.

6   Has difficulty making decisions.

7   Sudden, rapid, repetitive movements or vocalizations that is not associated with a physical disability

8   Crying.

9   Runs and climbs more than others his/her age.

10 Destroys other's property.

11 Always "on-the-go."

12 Intrudes upon the activities of others.

13 Chokes on food or drink.

14 Spiteful, vindictive, revengeful, or wants to get back at others.

15 Tearful or weepy.

16 Damages property.

17 Steals.

18 Loses belongings (e.g., books, toys).

19 Avoids specific objects, persons, or situations causing interference with normal routine

23 Deliberately annoys others.

24 Easily becomes angry.

25 Tantrums.

26 Low energy or fatigue.

27   Persistent or recurring impulses that interfere with activities (e.g. impulse to shout).

30   Bullies, threatens, or intimidates others.

31   Irritable mood.

33   Noisy while playing.

34   Waits for his/her turn.

36   Eats too quickly.

37   Talks excessively.

VITA

Santino LoVullo was born in Montebello, California, in 1975. He earned a Bachelor of Arts degree in psychology in 1997. In 1998, he began an eight year career as an elementary school teacher for children with ASD. To further his knowledge of ASD, he enrolled in a doctoral program in clinical psychology at Louisiana State University, where he is currently completing his third year of studies. His research and clinical interests focus on the assessment and treatment of individuals with ASD and severe problem behaviors.