2009

Psychopathology in person with and without co-morbid autism

Martin Nicholas Ancona
Louisiana State University and Agricultural and Mechanical College, martinnicholasancona@live.com

PSYCHOPATHOLOGY IN PERSON WITH AND WITHOUT CO-MORBID AUTISM

A Dissertation

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
Doctor of Philosophy

in

The Department of Psychology

by
Martin Ancona
B.A., University of North Texas, 2001
M.A., Louisiana State University, 2005
December 2009

TABLE OF CONTENTS

ABSTRACT

Over the last 20 years, substantial gains have been made in the understanding of autism spectrum disorders (ASD) and intellectual disability (ID). This increased understanding has brought with it the knowledge that the intellectual, social, and communication deficits present in those with ASD and/or ID by no means grant immunity to psychiatric disorders present in the general population. Although ASD and ID often co-exist throughout the lifespan, most studies have examined these two disorders in the context of separate disorders in children. As researchers have demonstrated that the dually diagnosed have unique assessment and treatment needs compared to those with a single diagnosis, the need for research examining the sequelae present when ASD and ID co-occur is needed. This study utilized multivariate analyses of variance (MANOVA) to determine whether mean differences exist across three diagnostic groups: ID only, ID and autism, and ID and pervasive developmental disorder not otherwise specified (PDD-NOS) on the linear combination of subscales present on the Diagnostic Assessment of the Severely Handicapped-II (DASH-II), a psychometrically sound measure that assesses psychopathology in those with ID. The results of this study supported an association between the presence of secondary ASDs and the presence of psychiatric and behavioral disorders with those with ASDs displaying more psychopathology than those ID

INTRODUCTION

In the last 20 years, research in the fields of pervasive developmental disorders and intellectual disability (ID) has progressed substantially. Psychological, pharmacological, and genetic studies have increased both our understanding and our ability to manage these disorders (Piven, 2002; Research Units of Pediatric Psychopharmacology Autism Network, 2002). At the heart of many of these advances is the increased capability of reliably identifying these disorders. However, correct diagnosis of these conditions is often obfuscated by many factors, including the heterogeneity of these disorders, high levels of symptom overlap, the fluid nature of the diagnostic criteria, and the lack of psychometrically sound instruments (Fitzgerald & Corvin, 2001; LeCavalier, 2006). Progress in the area of assessment and measurement is essential for diagnostic purposes, for a better understanding of behavioral phenotypes and for further delineation of the relationship between brain and behavior. The purpose of this study is to compare and contrast the frequency and type of comorbid psychopathology present in three groups: those with ID only, those with ID and autism, and those with ID and pervasive developmental disorder – not otherwise specified (PDD-NOS).

AUTISM SPECTRUM DISORDERS

History

Leo Kanner first described autism in 1943 while observing 11 children who exhibited eccentric patterns of behavior. Among the symptoms reported by Kanner were abnormal speech with echolalia, pronominal reversal, literalness of speech, inability to use language for communication, monotonous and repetitive behaviors, and a desire for routines and rituals. Autism was seen as a complex psychiatric disorder and was originally viewed on a continuum with severe mental illness, particularly schizophrenia (Fish, Shapiro, Campbell, & Wile, 1968). Although incorrect, the comparison to schizophrenia and other psychotic disorders is due, in part, to Kanner's unfortunate choice of the word "autism," which was originally a term used by Bleuler to describe schizophrenic withdrawal (Cohen, Donnellan, & Paul, 1987).

The complex clinical profile of autism has served as both a blessing, stimulating a large amount of research, and a curse, confounding the process of arriving at a final definition of the disorder (Charman & Baird, 2002). The large amount of research devoted to autism has led to significant changes in Kanner's (1943) original definition of the disorder. Several landmark findings that occurred after Kanner's original definition of autism have helped us arrive at our current clinical conceptualization of the disorder (Rutter & Garmezy, 1983). One of the earliest and most significant of these revisions occurred when studies revealed that a large percentage of persons with autism had co-occurring intellectual disabilities and associated medical problems (Kolvin, 1971).

Although the vast majority of research greatly aided in the understanding of autism, some research had actually hindered progress into the understanding of the disorder. In 1944, Bruno Bettelheim, director of the Chicago-based Orthogenic School for Children with Psychiatric Problems, attributed the etiology of autism to parents who were unresponsive to their children

during the first few years of life (Wing, 1988). Although later studies of the family systems of autistic children debunked Bettelheim's theories, his research consumed the scientific community for many years (Charman & Baird, 2002).

Emotional withdrawal was once believed to be a primary symptom in persons with autism (Matson & Minshawi, 2006). This lack of emotional development was judged as a major cause of many of the eccentric behaviors present in persons with the disorder. However, present research suggests that these impairments seen in persons with autism may be due to the deficits in the understanding and responding to social information rather than impeded emotional development (Leekam & Moore, 2001; Matson & Minshawi). In fact, a growing number of researchers believe that persons with autism have normal or near-normal levels of emotional development, but the deficits in developmental areas such as social imitation, joint attention, orienting to social stimuli, and perception of faces obfuscate this (Klinger, Dawson & Renner, 2003; Matson & Minshawi).

According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR), a diagnosis of autistic disorder is warranted when there is a qualitative impairment in social interaction, as manifested by two or more of the following symptoms:

1. Marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction.

2. Failure to develop peer relationships appropriate to a person's developmental level.

3. A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people.

4. A lack of social or emotional reciprocity.

In addition, the person must evidence qualitative impairments in communication as shown by one of the following:

1. A delay in, or total absence of, spoken language.

2. Stereotyped and repetitive use of language or idiosyncratic language.

3. Lack of spontaneous make-believe or social imitative play appropriate to the person's developmental level.

Additionally, the person must evidence restricted and repetitive behavior patterns as evidenced by one or more of the following:

1. Fixation with one or more stereotyped and restricted patterns of interest that are inordinate in either intensity or focus.

2. Unyielding adherence to specific, non-functional routines or rituals.

3. Stereotyped and repetitive motor mannerisms.

4. Persistent preoccupation with parts of objects.

Delays or abnormal functioning in social interaction, language, or symbolic play must also appear before the age of 3 years and the disturbance cannot better be accounted for by Rett's disorder or Childhood Disintegrative disorder (APA, 2000). By looking at the above criteria, some may conclude that autism is a rather straightforward disorder. However, symptoms of autism go beyond the diagnostic criteria listed to produce deficits in joint attention, orientation to stimuli, face perception, and perception of emotion.

Not all symptoms of autism are present at birth. Rather, the number of symptoms shows a positive correlation with age: As the person matures, more symptoms arise. These time-related increases in symptomatology are due to the fact that core autistic symptoms often cause accessory symptoms (also called "secondary symptoms"). One primary symptom, deficits in

joint attention, is believed to be responsible for below average levels of imitative behavior. Joint attention is defined as the ability to "coordinate attention between interactive social partners with respect to objects or events in order to share an awareness of objects or events." Deficits in joint attention are one of the first manifest symptoms of autism and are predictive of later expressive language deficits (Dawson, & Klinger, 2003). Although most infants with autism evidence impaired levels of joint attention, the extent of the impairment appears to be a function of the infant's intellectual level (Leekam & Moore, 2001). In a recent study, 83% of autistic preschoolers with an IQ greater than 70 followed an adult's gaze while 25% of autistic preschoolers with an IQ below 70 did the same. On the other hand, preschoolers with developmental disability evinced in-tact gaze following regardless of level of intelligence (Leekam & Moore).

Compared to children without autism,children with autism show deficient ability to orient themselves to all environmental stimuli. Although these children demonstrate a global impairment in this area, the deficit is particularly pronounced in the domain of orientation to social stimuli (e.g., people laughing, people calling the child's name, hand clapping; Matson & Minshawi, 2006). This deficit in orientation skills is true for both in vivo and imaginary situations. For example, Ruffman, Garnham, and Rideout (2001) found that children with autism showed below average levels of anticipatory looking during a story that involved human characters but evidenced average levels during a story that involved non-social objects (e.g., inanimate

Most newborns begin forming social relationships within the first few days of life. The ability to recognize faces, present within 2-3 days after birth, is essential to socialization (Bushnell, Sai, & Mullen, 1989), Mounting evidence suggests that persons with autism

experience difficulty in facial processing that, in turn, impedes their ability to understand and respond to social information (Klinger, Dawson, & Renner, 2003). As with the ability to orient to environmental stimuli, severe deficits in perception appear to be limited mainly to faces (Klinger et al). For example, Boucher and Lewis (1992) found that children with autism were impaired in comparison to normally developing individuals on both a picture-matching task and a picture-recognition task consisting mostly of faces (Klinger et al). However, when the focus of the task was recognition of non-social objects (e.g., buildings), no statistically significant impairment was observed (Boucher & Lewis, 1992).

Some researchers believe that persons with autism do not possess deficits in facial recognition but rather process facial features in an idiosyncratic style (Celani, Battacchi, & Arroocidiano, 1999). These researchers cite research studies which conclude that persons with autism tend to focus on the mouth region, rather than focus on the eye region as persons without autism typically do (Langdell, 1978). Citing evidence that children, adolescents, and adults with autism fail to show the normal decrement in facial processing when shown inverted pictures of faces, many scientists believe that this style of facial processing may be adaptive rather than maladaptive (Hobson, Ouston, & Lee, 1988). Others posit that this unusual processing style may develop over time as a compensation strategy for intrinsic facial processing deficiencies (Klin, Sparrow, deBidt, Cicchetti, Cohen, & Volkmar, 1999).

Evidence of an aberrant facial processing style is also supported by both structural and electrical studies of the brain. In a recent study, three- and four-year-old children with an ASD, with a developmental delay, and with normal development were shown two groups of pictures: One group of pictures contained a picture of their mother's face and that of a stranger's face, while the second group contained pictures of the child's favorite toy and an unfamiliar toy.

Typical developmentally delayed children displayed different event-related potentials (ERPs) when shown their mother's face as compared to a stranger's face. However, subjects in the ASD group showed similar ERPs in response to both pictures. Furthermore, subjects in all three groups each showed different ERPs in response to the picture of their favorite versus the unfamiliar toy (McPartland, Dawson, & Webb, 2004). Comparison and contrast of the exact ERP patterns of persons matched for chronological age in all three groups, when exposed to toys, revealed no significant differences in ERP patterns (i.e., greater P400 and Nc amplitude at the lateral scalp locations in response to the unfamiliar toy) when persons with autism spectrum disorders were compared to the normal group and those with delays (Dawson, Munson, Estes, Osterling, McPartland, Toth, Carver, & Abbot, 2002).

Other electrophysiological studies revealed that ERPs in persons with autism also differed in pattern and physiological location. In a landmark study, Dawson (2002) and colleagues found that high-functioning adolescents and adults with autism exhibited longer latency of the face-specific N170 ERP component when compared to IQ-matched adolescents and adults. In addition, the group with autism did not show a differential response to inverted faces nor did they show the typical right-lateralized ERP found in individuals with normal development. Schultz et al. (2000) conducted a functional IDI (fIDI) study of face processing in autism and reported that persons with autism used the region of the brain typically associated with the processing of objects, the inferior temporal gyri, when they were shown pictures of faces. In contrast, they showed less brain activation associated with face processing (i.e., the fusiform gyrus). These studies suggest that individuals with autism are less efficient in the processing of faces, lack specificity in firing patterns when processing faces, and facial processing is abnormally represented in the brain (McPartland et al., 2004).

Perception of emotion is one of the primary avenues through which people understand their social world, and one of the main ways in which people recognize and perceive the emotions of others is through the reading of facial expressions (Grossman, Klin, & Carter, 2000). The utility of reading facial expressions in order to perceive the emotions of others has been present since the beginning of time and is nearly constant throughout all cultures (Grossman et al.). In fact, researchers since Charles Darwin have noted that persons from vastly different cultures use similar facial expressions to reveal emotions such as anger and happiness (Ekman & Friesen, 1971).

Deficits in emotional perception and emotional expression were once believed to be a primary symptom of autism (Klinger, Dawson, & Renner, 2003). Although these deficits were once believed to be global, debate now exists as to the exact nature and extent of this impairment (Klinger et al., 2003). The idea that persons with autism were impaired in regards to the perception of emotion can be traced back to Hobson's (1989) picture sorting study in which participants were asked to sort pictures of people into discrete groups based on some salient characteristics of the picture. While most participants sorted pictures according to facial expressions (e.g., happy, sad, frustrated, angry), persons with autism compiled the pictures according to articles of clothing. Further supporting the idea of emotional deficits were other studies that documented disabilities in picking out pictures with particular emotional expressions, matching pictures based on the emotion being expressed in them, and the lack of an affective priming effect when shown affect-laden faces before being shown a neutral stimulus (Celani, et al, 1999).

Epidemiology of Autism Spectrum Disorders

Since its inclusion in the DSM-III (APA, 1980), our understanding of autistic disorder has greatly evolved. While this evolution has been helpful in understanding and treating the disorder, it has greatly complicated epidemiological studies (Tidmarsh & Volkmar, 2003; Wing & Potter, 2002; Charman, 2002). The research literature reviewed for this study examined the epidemiology of autistic disorder in two distinct ways: through incidence studies and the more commonly reported method of prevalence studies.

When researchers chart occurrence of disease using incidence studies, they examine how many cases of a disorder occur beginning within a certain time period, such as a year. Incidence studies are useful for examining outbreak of illness within a well-defined onset (e.g., influenza). In contrast, prevalence studies are used to estimate the number of people with a particular illness or disorder at a certain point in time, such as one day during a particular year. These studies are typically used with illnesses that do not have a definite beginning or end (e.g., most psychiatric disorders; Wing & Potter, 2002). Probably because of the difficulty determining a definite time of onset for autistic disorder, which may be different from person-to-person, most studies reviewed for this study utilized prevalence studies (Charman & Baird 2002). Regardless of which type of study was used (incidence or prevalence), there was little consensus regarding the number of persons with either autistic disorder or autism spectrum disorders.

As mentioned earlier, only a small proportion of studies surveyed for this paper utilized incidence studies. This is most likely because the exact date of onset for autistic disorder is rarely known (Wing & Potter, 2002). Therefore, examining the number of persons with autistic disorder using incidence studies is very difficult. Additionally, the studies examined were based on second-hand reports of autistic disorder utilizing subjects' medical charts. The studies that

did use incidence numbers were conducted in many different countries (Powell, Edwards, & Edwards, 2000). All the incidence studies did agree on one fact: The numbers of those with autism spectrum disorders were steadily increasing.

With the lack of a clear time of onset (due to both a general lack of knowledge about the disorder and the provisional findings that age of onset may differ from person-to-person; Charman, 2002), prevalence studies are the preferred method of studying most psychiatric disorders, including autistic disorder. As with incidence studies, the majority of prevalence studies on autistic disorder document a linear increase in cases (Charman; Wing & Potter, 2002; Tidmarsh & Volkmar, 2003; Sadock & Sadock, 2003). Lotter conducted the initial prevalence study in 1966 and found the prevalence to be approximately .4/1000 cases for strict autistic disorder and approximately 2.0/1,000 for autism spectrum disorders (Fombonne, 1999; Gillberg & Wing, 1999; Wing & Gould, 1979). Many epidemiological studies conducted from 1966 to the late 1990s corroborated Lotter's (1966) prevalence studies (Charman, 2002). Of particular note is a meta-analytic study conducted by Fombonne, DuMazabrun, & Cans, (1997) and Gillberg and Wing. Both presented time analyses. Both studies corroborated Lotter's prevalence findings of approximately .4/1,000 (95% CI=.84-1.08) for studies conducted between 1990-1997 while Fombonne found a rate of .72/1,000 (95% CI = +/- .97). It is important to note that these meta analyses are a combination of diverse studies using distinct methodology, criteria, and populations.

More recent prevalence studies have documented rates of approximately 1.7/1,000 (95% CI = 1.1-2.5) and 4.0/1,000 (95% CI = 2.8-5.6) and rates for autism spectrum disorders of approximately 5.8/1,000 (95% CI = 4.3-7.7) and 6.7/1,000 (95% CI = 5.1-8.7), significantly higher than the ones published in the aforementioned meta analyses (Bertrand, Mars, Boyle,

Bove, Yeargin-Allsopp, & Decoufle, 2001; Charrabarti & Fombonne, 2001). There is much debate as to what these new numbers mean. Do they reflect a truly increased prevalence or are they simply artifacts? There could be many reasons for these numbers including a broader diagnostic criteria, increased public knowledge about the disorder, differences in methods used in research studies , and recognition that autistic disorder can be associated with other psychiatric and medical conditions (Wing & Potter, 2002).

As mentioned earlier, retrospective examination of previous epidemiological studies that examined the prevalence of autism, a trend showing that each successive diagnostic criteria set led to an increase in the mean rate of autism in the population (Wing & Potter, 2002). In other words, while Kanner and Eisenberg's (1958) original criteria set (i.e., aloofness and elaborate, repetitive routines) produced the smallest mean rate for autism (approximately 3.9/10,000 out of six studies), DSM-IV/ICD-10 criteria produced the highest number of cases of the disorder (Wing & Potter, 2002)

This positive linear trend may be due to a broadening of diagnostic criteria over time. In other words, Kanner's and Eisenberg's (1958) criteria were difficult to apply in the general population. Aloofness is usually found in persons with severe or profound ID (IQ < 35), while the performance of repetitive routines requires higher-level cognitive abilities to organize and carry out (Wing & Gould, 1979). However, DSM-IV and ICD-10 criteria (the most recent criteria set used to diagnose the disorder) allow for a vast range of social and communicative impairments that are more inclusive than the original criteria set (Wing & Potter, 2002; Charman, 2002; Tidmarsh & Volkmar, 2003).

Autistic disorder was paid little attention to by either the public or the scientific community prior to the 1960s (Matson & Minshawi, 2006). However, the emergence of both

parental and scientific organization caused more interest to be drawn to the disorder. These organizations largely pushed for educational, research, and treatment funding. Scientific interest was stimulated by the development of research-based organizations with the objective of discovering the biological and psychological causes of the disorder. Additionally, controversies about a supposed link between the measles, mumps, and rubella (MMR) vaccine and autism brought about increased media coverage of the disorder (Wing & Potter, 2002).

Some of the increase in cases in autistic disorder and autism spectrum disorders is attributable to the different sizes and type of populations used as a sampling pool and/or the different methods used to identify cases (Fombonne et al., 2001).

Size of sample used in studies appears to be related to the rate of autism reported. In a retrospective study on sample sizes in autism research, Honda, Shimizu, & Misumi (1996) reported that rates of autism found were higher when smaller sample sizes were used (i.e., rates of 10.0/10,000 persons were reported more frequently when studies involved fewer than 50,000 subjects). More recently, Wing and Potter (2002) corroborated this result when they conducted a retrospective examination of autism prevalence studies.

Partly responsible for this sample-size rate of occurrence relationship is due to the fact that studies that utilize smaller samples tend to use more rigorous and comprehensive screening methods (Charman, 2002; Fombonne, 2001). To achieve optimal estimates of prevalence, it is recommended that multi-phase detection process (i.e., a screening phase followed by a more specific diagnostic phase) that targets a medium-size population (using smaller size populations decreases the width of the confidence interval; Charman) be used.

Additionally, many studies contain a subject pool with an artificially restricted range, consisting primarily of samples from restricted environments, such as the special education

system. As a result, researchers often artificially exclude cases in the community (Charman, 2002; Wing & Potter, 2002). The highest rates of autism were found when researchers used participants who were having routine developmental check-ups for pre-school (Wing & Potter; Fombonne, 1999; Fombonne & Chakrabarti, 2001; Halsey & Hyman, 2001; Kadesjo, Gillberge, & Hagberg, 1999; Kerr, Witt & Engerstrom, 2001).

Autism Spectrum Disorders and Comorbid Psychopathology

Researchers have defined comorbidity as the occurrence of two or more mental disorders in the same person in the same time period (Matson & Nebel-Schwalm, 2005). So far, little research has addressed the types and rates of psychiatric comorbidity in persons with in those with ASDs (Borthwick-Duffy, 1994). While researchers do agree that the full range of psychiatric disorders co-occur in persons with ASDs, there fails to be a consensus on the exact epidemiology of co-occurring psychopathology in this population (Borthwick-Duffy). In fact, in a review of articles for this study, the estimated prevalence for mental disorders differed among all the articles surveyed, from a low of 4 % to a high of 38% with an average of 8%. (Wing, 1981; APA, 1994; Borthwick-Duffy; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). With the exception of psychosis, researchers also disagree on rates of individual psychiatric disorders.(Ketelaars, Hortwitz, & Systema, 2008). For example, while some researchers have found an association between the diagnosis of an ASD and increased incidence of a comorbid mood or anxiety disorders (Howlin & Moore, 1997; Stewart et al., 2006; Lainhart), others have failed to replicate this finding (Ketelaars et al., 2008).

Mood disorders fall into one of two broad categories: unipolar mood disorders and bipolar mood disorders (Ghaziuddin, Ghaziuddin, & Greden , 2002; Matson & Nebel-Schwalm, 2005; APA, 2000). While the cardinal feature of unipolar mood disorders involves depressed

mood for more days than not for at least a two-week period, the main features of bipolar mood disorders involve both depression and a manic or mixed state for at least a seven-day period (APA, 2000). While it is widely agreed upon that the lifetime prevalence of depression in the general population is approximately 17%, (Angst, 1999), there is no consensus on the epidemiology of mood disorders in those with an ASD.

Although the exact prevalence of depression in those with ASDs is unknown, it is believed that depression is the most common comorbid psychiatric disorder in those with an ASD (Ghaziuddin et al., 2002; Wing, 1981; Matson & Nebel-Schwalm, 2005). However, it is unclear whether the reports of increased prevalence of comorbid depression relative to other psychiatric disorders are due to the relatively large amount of attention given to mood disorders, as compared to other psychiatric illness, by researchers studying ASD or whether depression truly is the most prevalent disorder in those with an ASD (Ghaziuddin et al.).

Along with depression, anxiety was the first recognized psychiatric disorder occurring in those with an ASD (Wing, 1981). In fact, anecdotal reports of what was believed to be symptoms of unipolar depression were observed in the first series of persons with autistic disorder (Kanner, 1943; Rutter & Garmezy, 1983). Although no large scale case controlled epidemiological studies have been conducted to ascertain the prevalence of major depression in those with an ASD, researchers have amassed an increasing body of evidence confirming the hypothesis that unipolar mood disorders, such as major depressive disorder, are extremely common in those with ASDs (Wing, 1981).

While the first formal study of depression in those with ASD were simply case reports and case studies (Ghaziuddin & Tsai, 1991), later studies contained higher sample sizes and utilized more statistically advanced methods to determine prevalence. In one of the first large-

scale studies of co-occurring psychopathology in those with ASD, Wing (1981) found that major depression was present in 30% of adults with Asperger's disorder. In one of the first hospital-based studies, Ghaziuddin, Ghaziuddin, and Tsai (1992) found that major depression was present in two percent of those with an ASD, a higher rate than that of any other psychiatric disorder. However, a major methodological flaw in this study was the fact that direct clinical interviews probing for specific disorders were not utilized. In one of the first studies to employ semi-structured clinical interviews to screen for psychiatric disorders in the ASD population, Ghaziuddin, Wiedmer-Mikhail, & Ghaziuddin (1998) found equivalent rates of depression in those with Asperger's as Wing did in 1981. In another study, Tantam (2003) found similar results, reporting that major depression was the most prevalent psychiatric disorder in those with an ASD. In a meta-analytic study on the literature focusing on depression on those with an ASD, Lainhart and Folstein (1994) concluded that unipolar mood disorders were not only the most prevalent comorbid psychiatric disorders in the ASD population but also were largely under-diagnosed.

Although most researchers believe that major depression is the most common co-occurring disorder in those with an ASD, it is also being learned that bipolar mood disorders are present as well. One of the earliest reports of bipolar disorder came from Gillberg (1985), who described a patient with Asperger's disorder who went on to develop bipolar disorder during the course of treatment for his developmental disability. Furthermore, Realmuto and August (1991) described three adult patients with Asperger's disorders and co-occurring bipolar depression. Unfortunately, because the few studies that examine bipolar disorder in those with an ASD are case studies, no prevalence figures are currently available. However, these case studies do serve to corroborate the finding that the full range of DSM-IV mood disorders are most likely present

in those with ASDs (Matson, Baglio, Smiroldo, Hamilton, & Packlowskyj, 1996).

The lack of psychometrically sound tests and measures for use with this population, the overlap of symptom profiles for depression and ASD, the lack of verbal skills to communicate changes in sad mood that is characteristic of depression, and the possibility that ASD symptoms may affect the clinical presentation of a mood disorder are all reasons for disagreement regarding the prevalence of co-occurring mood disorders in those with an ASD. First of all, there is currently a lack of measures meant for use on an ASD population (Matson & Nebel-Schwalm, 2005). For example, in the research surveyed for this study, the most commonly used measures to assess unipolar and bipolar mood disorders in the ASD literature were the Beck Depression Inventory (Beck, Ward, & Mendelson, 1961), Hamilton Depression Rating Scale (Hamilton, 1960), and the YMRS (Simon, Unutzer, & Young, 1978). These were designed and constructed well before the current surge of interest in ASDs and, hence, included no persons with an ASD in the norming sample (Reiss, 1990). In fact, some studies reported specific problems with these scales during their research protocols, including the fact that respondents with ASDs could not answer questions on the forms that asked about subjective feelings of guilt and apprehension (Ghaziuddin & Tsai, 1991). To address the incompatibility of the BDI in persons with an ASD, authors made cursory revisions of the measure for use in persons with autism, allowing questions about feelings to be answered by third-party informants familiar with the subject. Authors have criticized these revisions saying that those with ASDs may have difficulty answering questions and that informants may also have trouble answering the questions for the person, especially since one of the chief symptoms of ASD is an mismatch between facial expression and experienced emotions (Stewart et al; Macdonald, Rutter, & Howlin, 1989).

A second factor complicating the detection of psychiatric disorders in those with an ASD is the effect that the ASD syndrome, itself, has on the appearance of comorbid mental disorders. Persons with an ASD evidence difficulty in expressing their emotions. Researchers have determined that this deficiency in emotional expression that persons with ASD experience often alters the outward symptoms of psychiatric disorders that may be co-occurring. The main pathway responsible for this handicap in emotional expression is the deficient affective and cognitive systems present in those with an ASD (Hobson, 1989; Rutter & Garmezy, 1983). These deficits were first noted as far back as Kanner (1943) who stated that autistic children "come into the world with the innate inability to form the usual, biologically provided affective contact with people." These emotional impairments impact the autistic person's ability to communicate clearly through producing deficits in a variety of areas including: integrating emotion with attention, effectively expressing the full range of emotions through facial expressions and body language/physical gestures, hindering their ability to describe complex emotions, and effectively understanding nuanced social cues (Hobson, 1986). Because there is no biological test used to diagnose depression, the diagnosis is typically made through observable behaviors, such as information gleaned from a clinical interview or behavioral observation. As a result, the ability of the patient to clearly communicate emotions and feelings through verbal and non-verbal methods is paramount to making an accurate diagnosis. Because verbal and non-verbal expression is handicapped in those with an ASD, it is easy to see why psychiatric diagnoses are often missed. For example, the chief criterion for major depressive disorder is the experiencing of sadness (APA, 2000). In order to optimize the chances of detection of this, the patient must be able to express how they are feeling to the clinician through words and body gestures. Researchers have shown that person with an ASD have difficulty

doing this. For example, in a seminal study demonstrating autistic persons' inability to successfully express emotions they are experiencing and to integrate them with outward physical expression, MacDonald et al. (1989) showed subjects with an ASD experienced difficulty matching their facial expression to the emotion they were feeling at the time. This mismatch between feelings and facial expressions led investigators to incorrectly deduce what type of emotion the subject was experiencing. At times the person's facial expression might have been completely different to what they were feeling at the time (e.g., person was feeling sad but had a happy facial expression) where at other times, the person may have shown no facial expression at all.

Further evidence of the inability to elaborate on emotions was shown by Capps, Yamira and Sigman (1992) during which subjects with an ASD exhibited difficulty elaborating on multi-faceted emotions such as loss. Although many persons other than those with ASDs may experience impoverished verbal abilities that allow them to describe how they inwardly feel, they can often compensate with non-verbal communication such as hand gestures. However, many with ASDs also experience deficits in the use of non-verbal communication, such as using hand and body gestures (APA, 2000; Attwood, Frith, & Hermelin 1988). Thus, while the general population may have trouble with one modality of communication, they can use a different modality to compensate. Those with an ASD typically experience deficits in both verbal and nonverbal forms of communication. Thus, the tendency of the outward appearance of persons with autism to belie actual experienced emotions can alter how symptoms of depression to something other than described in the DSM (Ghaziuddin, et al., 2002). It is via the deficiencies in verbal and nonverbal communication that symptoms which are hallmark symptoms in the population are masked by deficiencies in communication present in those with ASDs.

Further masking symptoms of mood disorders in those with autism is the fact that depression often aggravates maladaptive behaviors present in autism. Behaviors described by authors include self-injurious acts, aggression, incontinence, agitation, decrease in self-care/grooming, refusal to attend functions, and excessive food and drink intake (Matson & Gonzalez, 2006). Many researchers hypothesize that the behavioral deterioration often seen in adolescents, young adults and older adults with an ASD could be due to the emergence of depression that typically occurs when people enter different stages of their lives (Howlin & Moore, 1997). Researchers have shown that an increase in self-injury and aggression can signify the presence of a mood disorder, especially in populations with impoverished verbal abilities. In support of this hypothesis, they point to the fact that the incidence of maladaptive behaviors typically decrease in frequency, intensity, and duration after treatment of depression (Matson, 1991). Despite the association between maladaptive behaviors and the occurrence of mood disorders, the APA, World Health Organization [WHO], and the American Association on Mental Retardation [AAMR] have not yet altered their diagnostic criteria to coincide with research findings on symptomatology of mood disorders in this population (APA, 2004; WHO, 2007, AAMR, 2006). Many authors suggest that since depression has a different phenotype for those with deficits in communication ability, the formulation of a separate diagnostic criteria for these populations would increase detection of mood disorders and may lead to increase detection and more effective treatments of depression in those with an ASD (Tantam, 2003).

As mentioned earlier, the lack of large scale research examining morbidity rates of co-occurring mood disorders in the ASD population, hinders determination of the exact prevalence rates of depression in this population is not yet known (Ghaziuddin, et al., 2002; Wing, 1981; Matson & Nebel-Schwalm, 2005). Despite the lack of data on those with both depression and

ASD, certain epidemiological trends in the existing research are important to note. First of all, it appears that females with ASDs have a higher incidence of depression than males with ASDs. However, because females with an ASD are most often more severely impaired than their male counterparts, more research should be done to determine whether or not the occurrence of depression is associated to the severity of the ASD. In other words, it is unknown whether the prevalence is depression is higher in females with an ASD because they, in general, have more severe ASD symptoms, or if there is truly a main effect for gender on depression in ASD.

Attempting to compile a comprehensive listing of symptoms that adults with ASD present with when experiencing clinical depression, Stewart et al. (2006) examined the existing ASD depression research conducted on adults. Because of the lack of research on this subject, the authors commented that they were unable to utilize as stringent exclusion criteria as they wanted. The only exclusion criteria they utilized were that the article could not include persons with depression secondary to bipolar disorder and that the research study had to specifically mention persons with autism, Asperger's, and/or unipolar depression (Stewart et al.). The authors noted that throughout the studies, depressed mood was the most common diagnostic feature of depression cited in the studies which they reviewed. Somewhat expectedly, however, the authors also noted that only in one case did the person actually report that they were feeling sad (Ghaziuddin and Tsai, 1991). The most common methods employed by the studies to determine that the person with an ASD was experiencing sad or depressed mood were to note behavioral manifestations of depressions, such as noting that the subject had a sad facial expression or that the person cried easily (Stewart at al.). In only one study did the persons actually voice suicidal thoughts (Wing, 1981). Other symptoms of depression noted in the ASD population were: loss of interest in activities (n = 7), decreased appetite (n = 8), insomnia (n = 1)

and hypersomnia (n = 10; Sovner, 1988b). In only two cases was there noted any psychomotor retardation (Clarke, Baxter, Perry, & Prasher, 1999). This was particularly surprising in light of the fact that psychomotor retardation is often present in depressed persons in the general population (APA, 2000). One theme uncovered in the author's review of studies on ASD depression, was that symptoms of depression were uncovered more through third party observation of behaviors than self-report of subjects (Stewart et al.). Many limitations with this meta-analytic review were uncovered, namely that study authors used symptoms that could be representative of an ASD, itself, such as social withdrawal or abnormal speech patterns, to be diagnostic of depression (Stewart et al.).

Anxiety disorders have a high rate of overlap with mood disorders in both the ASD and the non-ASD population (Barlow, 2002; Matson & Nebel-Schwalm, 2005). As with mood disorders, anecdotal reports of anxiety-related behaviors were observed in the first studies of persons with autism. In fact, in one of his first writings on autism, Kanner (1943) noted that several of his original patients demonstrated a phobic-like fear of certain objects and social situations. Although initial reports alluded to the fact that anxiety disorders were present in those with autism, the hypothesis was not systematically explored until relatively recently. This is likely due to the fact that many common anxiety disorders, such as social anxiety disorder (SAD), and especially obsessive-compulsive disorder (OCD), share many characteristics and symptoms with ASD, and thus are often overlooked (APA, 2000; Rumsey, Rapoport, & Sceery, 1985). As knowledge of ASDs has increased, more attention has been given to the diagnosis and treatment of comorbid psychiatric disorders in this population. One area of study is interplay between anxiety and mood disorders. Interestingly, researchers have noted many parallels in the dynamics of comorbid mood and anxiety disorders in the general population and in those with an

ASD. Of particular note is finding the presence of an escalatory effect, in which one type of disorder can exacerbate the frequency, intensity and duration of the other disorder. For example, researchers have noted that depression in the ASD population appears to increase the frequency and severity of symptoms of OCD and SAD, respectively (Ghaziuddin, Alessi, & Greden, 1995).

As mentioned earlier, symptoms of ASDs often overlap with those of anxiety disorders. For example, some key symptoms of OCD, including the performance of repetitive actions or verbalizations, and the desire to keep surroundings the same are sometimes facets of ASDs. (APA, 2000). The overlap between manifestations of OCD and of ASDs appear to be closer to the rule that the exception. Researchers examining the relationship between the two have documented that approximately 86% of persons with autism demonstrate repetitive behaviors similar to those exhibited in those with OCD, and 20% of those with OCD desire constancy in their environment, a common feature of those with ASD (Bejerot, Nylander, & Lindstrom, 2001). Because such a high percentage of persons with an ASD also engage in repetitive behaviors that resemble those of their OCD counterparts, the ability to differentially diagnose the two disorders or tell when both disorders are present, is very important. Since behavioral manifestations of both classes of disorders are so similar, one must examine the underlying motivation for the actions. Specifically, persons with OCD feel apprehension in regards to their compulsions while those with ASD do not (Matson & Boisjoli, in press). While the discernment of an ASD from OCD when compulsions are present has to do with understanding the motivation for behavior, teasing apart the two disorders when a desire for sameness is present is done by examining the constellation of symptoms that are occurring alongside this distress over many types of change (Matson & Boisjoli, in press; Rumsey et al., 1985).

Because of the high degree of symptom overlap between autism and anxiety disorders, particularly OCD, much debate as to whether anxiety disorders are, in fact, separate syndromes from ASD. In an attempt to help understand this issue, McDougle, Kresch, Goodman and Naylor (1995) conducted factor analytic studies of both disorders. These studies found that certain behaviors/symptoms tended to load highly on the OCD construct while failing to load on ASD. These factors included: the presence of hoarding, touching, tapping and self-damaging behaviors, and the lack of checking, counting, and aggressive symmetry-related behaviors. A major drawback of this study was that persons in the OCD group had IQs in the normal range while those in the ASD group had an average IQ of 67.1, a difference that may explain the between-group differences in symptoms (McDougle et al.). A second study aimed at determining key differences between ASD and OCD documented that those with OCD-only tended to exhibit higher levels of obsessive thought and higher frequencies of somatically-based compulsions and repeating rituals than those with ASD-only (Russell, Mataix,-Colls, & Ansom, 2005).

Swiss psychiatrist Eugene Bleuler originally coined the term *schizophrenia* to describe the fragmented mental faculties present in those with the disorder. Although he recognized that all schizophrenic symptoms wax and wane over the course of the illness, he viewed some symptoms as more central to the disordered thought processes present in the disorder than others. He labeled symptoms essential to abnormal thought processes as fundamental symptoms and symptoms caused by these thought disturbances as accessory symptoms. The fundamental symptoms are now referred to as the "four A's": affective flattening, associative loosening, ambivalence and autism (Foster-Greene, 2002; Flaum, 1995).

In a debate similar to the one surrounding the anxiety disorders and ASD, autism and schizophrenia have been conceptualized as disorders lying on the same continuum. In fact, until

relatively recently the disorders were viewed as one and the same. (Matson & Nebel-Schwalm, 2005; APA, 2000). Part of the reason that ASDs and schizophrenia were viewed as synonymous terms is that both disorders share a large number of symptoms. For example, persons with both autism and psychosis sometimes engage in grossly disorganized behaviors, demonstrate flat affect, display an incongruence between mood and affect and seem to appear apathetic to social interaction. Confounding the diagnostic picture even more is the fact that the clinical picture of ID, the disorder that most frequently co-occurs with autism, also shares symptoms with schizophrenia (Johnstone & Frith, 1996). Specifically, both disorders share the positive symptoms of hallucinations, delusions, repetitive and stereotyped behavior and disorganized or incoherent speech (Cherry, Penn, Matson, & Bamburg, 1999). Although there is consensus estimating the prevalence of schizophrenia in those with ID at 3%, a figure three times higher than that of the general population, there is vast disagreement about the prevalence of comorbid ASD and schizophrenia (Bouras, Martin & Leese, 2004; Turner, 1989).

As with other disorders that co-occur with ASD, the reasons for disagreements about prevalence are many: specifically a lack of measures that are appropriate for use with the population, patients' lack of verbal skills to express symptoms, and the fact that when ASD and psychosis co-occur, the disorders interact. First of all, measures used to detect psychosis, such as the MMPI-2, presume that the test taker has the ability to introspect. Obviously these tests would be difficult for most people with an ASD to answer (Stewart et al., 2006). Since these tests were based on samples of the population who were not formally diagnosed with an ASD, the use of these tests to those with a disability would be impossible (Volkmar & Cohen, 1991).

Not only does the lack of appropriate measures hinder obtaining data on the co-occurrence of psychotic spectrum disorders and ASD, the lack of verbal skills characteristic of

persons with ASD stymies appropriate diagnosis. Out of the five DSM-IV criteria that contribute to a diagnosis of schizophrenia, data on three of the symptoms are expressed primarily through verbal communication (experiencing of hallucinations, delusions, and disorganized speech) and may be difficult for third party informants to document (APA, 2000).

Lastly, as mentioned earlier, researchers have corroborated that the symptoms of ASD often alter the presentation of co-occurring psychotic spectrum disorders. For instance, symptoms of schizophrenia, such as hallucinations, delusions and grossly disorganized behavior, may be masked by the repetitive behaviors found in many persons with autism. Further complicating matters is the fact that psychosis often aggravates symptoms of autism, such as self-injurious behaviors and aggression (Walsh, 2006) and, like with treatment of depression, their incidence usually decreases after treatment. While it is widely accepted that an increase in these behaviors can be indicative of the experiencing of a psychotic spectrum disorder in a person with severe autism, they are not listed as diagnostic criteria for any of the psychotic disorders (APA, 2000).

PSYCHOPATHOLOGY IN THOSE WITH ID-ASD

As interest in those with ID-ASD diagnoses is relatively new, the number of studies examining psychopathology in those with ID-ASD diagnoses is relatively small in number. Keeping this fact in mind, it should be noted that the majority of studies support the idea that psychopathology is more common in those with secondary ASD diagnoses than in those with ID only (Bradley, Summers, Wood, & Bryson, 2004; LaMalfa, Lassi, Bertelli, Salvini, & Plaudi, 2004). Although most studies of this subject further the notion that ASDs confer unique vulnerability to psychopathology above that contributed to ID, many of the studies are marked by methodological weaknesses that undermine the amount of confidence that can be drawn from these studies' conclusions.

In the first study to directly examine psychopathology in adults with ID-ASD diagnoses, Morgan, Roy, and Chance (2003) concluded that ASDs were in fact a risk factor for additional psychopathology in those with primary diagnoses of ID. In particular, they cited that those with both cognitive impairments and ASDs were more likely to also have a concurrent mood disorder, particularly major depression (Morgan et al.). Although this study galvanized research in the area of ASDs in those with ID, it was marked by a failure to control for a variety of potentially confounding variables, most notably that of degree of intellectual impairment. As mentioned previously, level of intellectual and adaptive impairment has been well documented to be a risk factor for additional psychiatric diagnoses (Sadock & Sadock, 2003; Matson & Frame, 1986; Tsakanikos, Costello, Holt, Sturmey, & Newton, 2005). The method of data collection in this study may have also influenced the prevalence rates of psychiatric comorbidity in this study, and led to skewed results. Researchers in the field of ID research recommend that research in this population should involve either direct observation of participants under study or the questioning

of caretakers familiar with the individual when screening for either ID, ASDs or psychiatric comorbidity (Kerker, Owens, Zigler, & Horwitz, 2004). The rationale for this is predicated on past research where the prevalence of psychiatric disorders in those with ID was found to be much lower when chart reviews were used rather than other methods (Reiss, 1990). For example, Reiss found a 12% prevalence of psychiatric disorders among those with ID attending a community-based day program using chart reviews to assess for psychopathology and a 39% prevalence rate using standardized instruments meant for the assessment of psychopathology in this population. Thus, although this study served to galvanize research in the area of psychopathology in those with ID-ASD diagnoses, it was difficult to determine whether differences in psychiatric comorbidity were due to the ASD diagnoses, themselves, or whether these differences were due to uncontrolled variables, particularly level of ID. This is very important to note because other researchers have demonstrated that those with ID have increased levels of pathology, especially psychiatric (Sadock & Sadock; Matson & Frame; Tsakanikos, Costello, Holt, Sturmey, & Newton) . Therefore, this could have contributed to the findings that Morgan, Roy and Chance found.

A later study conducted by Tsakanikos et al. (2005) stands in direct contradiction to past studies that found an association between ASD and additional psychiatric disorders (Bradley et al., 2004; Morgan, et al., 2003; LaMalfa et al., 2004). The reason for these different findings may lie in the fact that sampling methods used by the studies' authors failed to adequately represent all persons with ID. This is because the authors sampled from the community at large rather than from inpatient and developmental centers, where those with more severe intellectual disability are likely to reside (Kerker et al., 2004). Additionally, persons residing in the community are much less likely to be as psychiatrically impaired as those who reside in inpatient

and developmental centers. While both of these studies served to forward research in the area of psychopathology in those with ID-ASD diagnoses, they both relied on data gathered from small sample sizes and information recorded in participants' medical records or registry data from clinics. As with studies that rely on community samples, Kerker et al. (2004) have voiced concerns that reliance on this type of outpatient data fails to adequately represent those with more severe levels of cognitive and adaptive impairment, most of whom live in developmental centers, inpatient centers or group home, and, hence, would not traditionally present to an outpatient clinic or hospital.

Those with ID-ASD are a relatively under-studied population. Hence, the number of studies examining psychopathology in those with ID-ASD diagnoses is relatively small in number. Although the majority of studies support the idea that psychopathology is more common in those with secondary ASD diagnoses than in those with ID only (Bradley et al., 2004; Morgan et al.). Although most studies of this subject support the idea that ASDs confer unique vulnerability to psychopathology above that contributed to ID, many of the studies are marked by methodological weaknesses that undermine the amount of confidence that can be drawn from these studies' conclusions.

Surprisingly enough, even in the developmental disabilities research, no studies could be located which studied this topic. Therefore, conclusions drawn from previous research are weak at best. This presents a significant impediment to future research in the area of psychiatric disorders in this area and represents somewhat of a deficiency in the preexisting psychiatric and psychological literature regarding those with comorbid ASD-ID diagnoses. Because both are associated with comorbid conditions, both psychiatric and physiological, research should extend previous finding on this important topic.

Those with ID-ASD are a relatively under-studied population. Hence, the number of studies examining psychopathology in those with ID-ASD diagnoses is relatively small in number. Although the majority of studies support the idea that psychopathology is more common in those with secondary ASD diagnoses than in those with ID only (Bradley et al., 2004; Morgan et al.). Although most studies of this subject support the idea that ASDs confer unique vulnerability to psychopathology above that contributed to ID, many of the studies are marked by methodological weaknesses that undermine the amount of confidence that can be drawn from these studies' conclusions.

The present-day construct of ID has been known by a variety of names throughout history. These frequent name changes are more than just cosmetic; they serve as a barometer by which both scientific and public understanding of the disorder is measured (Hodapp & Dykens, 2003). Intellectual disability failed to attract attention in the United States until approximately 1844 when reformist Samuel Gridley Howe, galvanized by an article about the treatment, education, and rehabilitation of the intellectually disabled in France, formed a state commission to investigate the number and condition of the intellectually disabled in the United States (Trent, 1982). The commission's investigation revealed a large population of persons with ID whose needs for education and treatment were being largely unmet. Following this commission's recommendations, the first United States' public and private training schools formed for persons with intellectual disability. Quickly thereafter, facilities were started throughout the United States (Haskell, 1944). The immediate goal of these schools was to serve as surrogate families for persons with ID while the long-term goal was to educate persons with ID and return them to the community (Hodapp & Dykens, 2003; Trent, 1994; Berg). However, due to various social and political forces at work both internally, within the schools, and externally, in the country's political and social climate, by the mid 20th century the mission of these schools changed from caretaker to warehousing facility (Hodapp & Dykens, 2003).

Despite its tumultuous past, many advances have been made in the area of intellectual disability, particularly in the past 50 years. These advances include the discovery of many of the factors responsible for the etiology of many types of ID, improvements in both treatment and management strategies for ID, and growing public acceptance of the disorder. Despite this progress, there is conflicting knowledge in many areas of intellectual disability. These areas

include the precise role of pharmacotherapy in the treatment of the disorder and what nosological system should be used to classify persons with intellectual disability (APA, 2000).

A diagnosis of ID carries with it serious consequences regarding one's social and occupational future. As a result, the diagnostic criteria set forth by the DSM-IV-TR (APA, 2000) to warrant a diagnosis of ID are stringent. In order to receive a diagnosis, three criteria must be met. First, the person's IQ must be 70 or lower. Second, the person must demonstrate deficits in adaptive behavior, meaning deficits in the ability to successfully carry out "daily activities required for personal and social self-sufficiency" (Sadock & Sadock, 2003). These abilities include communicating one's needs to others, carrying out daily living skills such as eating and bathing, and the ability to understand and follow social rules and norms. Finally, in most states, the person must be below the age of 18 years. The APA lists ID on a discrete axis to ensure that the possible presence of personality disorders and/or ID will not be overshadowed by more obvious axis I disorders (APA). The diagnosis is made whether or the not the person has a comorbid axis III disorder causing the three criteria to be met (Sadock & Sadock). The requirement that all three criteria be met in order to receive a diagnosis of ID is not arbitrary. Rather, it stops the diagnosis from being given in cases of adult-onset cognitive degenerating diseases (e.g., Dementia of the Alzheimer's Type) or adult-onset head trauma that causes a decrement in IQ (APA; Emerson, Hatton, Bromley, & Caine, 1998).

CURRENT STATUS

Due to its impracticality, lack of empirical support, and psychometric limitations, the 1992 AAMR definition has been widely criticized by researchers and clinicians alike (MacMillan et al., 1993). The AAMR's 1992 definition shifted the IQ criterion warranting a diagnosis of ID upwards from 70 to 75. Although this five-point shift may seem inconsequential on the surface, on the Gaussian bell curve it makes a tremendous amount of difference because it qualifies twice as many persons for a diagnosis of ID (Reschly, 1972).

Definitions of mental illness and developmental disabilities are empirically derived, usually through factor analytic studies that show what particular symptoms cluster together to form a syndrome (APA, 1994). The AAMR's definition of ID specifies 10 domains of adaptive functioning, including such rarely tested areas as leisure, health, and safety, community use, and self-direction. This specification contradicts factor analytic studies of adaptive functioning which routinely show two to seven factors of adaptive behavior, with a single higher-order primary factor accounting for much of the variance (APA, 2000; McGrew & Bruininks, 1989). In response to these factor-analytic studies demonstrating that some of their factors impossible to be measured, the 1992 AAMR definition allows for clinical judgment, rather than quantitative data, to be used in determining whether the person is deficient in many of these areas. In fairness, all other diagnostic manuals – including the 1987 DSM-III-R and the 1983 AAMR guideline – allow the use of clinical judgment in the evaluation of deficiencies in adaptive functioning. However, the chances for an error in clinical judgment increase exponentially with the increase in the number of areas of adaptive functioning to be judged. Exacerbating the situation is the finding that many of the domains specified by the AAMR are interdependent and that many are new to clinicians (Hodapp & Dykens, 2003).

Irrelevance of Support Specifiers

Additional confusion and controversy were created by the 1992 AAMR definition's use of support specifiers (e.g., intermittent, limited, extensive, and pervasive) rather than severity specifiers (e.g., mild, moderate, severe, and profound). The AAMR's objective in using these support specifiers was to indicate that the difficulties arising in the ID population are due to a defective interaction between the person and his/her environment. Good intentions aside, these specifiers obfuscated the fact that the person's level of disability is not always associated with the degree of support services needed. For example, in a study done by Ross, Begab, Dondis, Giampiccolo, & Meyer (1985), persons diagnosed with mild ID varied widely in the amount of support services required with 64 percent functionally independent and 24 and 12% either partially or totally dependent on others. The move away from level of impairment thus generates unnecessary confusion (Hodapp & Dykens, 2003).

DIFFERENTIATION BETWEEN ID AND ASD

The number of similar symptoms that occur causes problems (Kraijer, 2003). Because of the large number of similarities between the two disorders, clinical observation is not always sufficient. Unfortunately, research aimed at differentiating ID when it occurs in isolation in adult populations from co-occurring ID and autism in adults is sparse. In the only paper on the topic of symptoms of autism and ID in adults versus the symptoms of ID-only in adults, it was concluded that symptoms of co-ocurring autism and ID included active withdrawal from others and restricted patterns of behavior. In particular, those with co-occurring autism engaged in behaviors such as injuring self to avoid contact with others and isolating self, lacked interest in group activities (e.g., sports), and having a high frequency of stereotyped behaviors (Matson, Wilkins, & Ancona, 2008). To aid in differential diagnosis between the two disorders, researchers have often incorporated psychological testing in their studies to discern differences between ASDs and ID. In general, these studies have documented that, when compared to those with ID, persons with ASD exhibit larger discrepancies between Performancere and Verbal score indices on IQ tests, greater deficits in expressive language and socialization, higher frequencies of challenging behaviors, and fewer and less advanced adaptive behaviors (DeMyer, 1974; Ando & Yoshimuro, 1979; Ando, Yoshimuro, & Wakabayashi, 1989).

As mentioned earlier, autism and ID can co-occur with one another or, less commonly, by themselves. As a result, clinicians and researchers often need to determine if patients need to be diagnosed with either both disorders or with solely autism or solely ID. As a result, there is a need for research focusing on determining the differences between symptom profiles of those with both autism and ID as opposed to those with only ID or only an ASD. Although research in

this area is sparse and focused almost entirely on the children, lead researchers in the field of developmental disabilities have shown that a key area to examine when making a differential diagnosis is adaptive behavior over time. Specifically, those with comorbid autism and ID make substantive gains in adaptive behaviors over time, symptoms of those with ID and/or ASD persist largely unchanged into adulthood and difference among the three groups (ASD-only, ID-only, comorbid ASD and ID) remain stable over time (Matson et al.,1996). Thus, there is reason to believe that research in this area can be extended to the adult population.

The primary differences between those presenting with both ASD and ID as opposed to those presenting with either ID-only or an ASD-only have been determined through the use of tests of intelligence and scales of adaptive behavior. For example, researchers have noted that the intelligence score profiles of persons with "pure" ID are markedly different from those who present with both autism and ID (Kraijer, 2003). One of the first studies to demonstrate this performance discrepancy was conducted by DeMyer (1972) that compared 54 children with comorbid autism and ID with 29 children with ID only. On all intelligence test indices (e.g., motor, perceptual-motor, perceptual, and verbal performance), those with co-occurring ID and autism were less advanced that the those with ID only. In particular, the greatest disparity in scores was shown on indices of verbal performance.

A second study by DeMyer, (1974) compared the Wechsler Intelligence Scales for Children (WISC) score profiles for children with ID only to those of children with both ID and autism. Although both groups showed higher Performance IQ indices as compared to Verbal IQ indices, the groups with comorbid autism showed a much larger Verbal IQ-Performance IQ split (up to 30 points). Additionally, the researchers' results showed remarkable stability six-years later with correlations of .58, .63, and .70 for Verbal IQ, Performance IQ, and Full Scale IQ).

A third study conducted by Ando and Yoshimura (1979) compared persons with comorbid ID and autism with persons with ID only. All children were functioning at the moderately or severely ID range. The persons with autism showed significantly greater deficits in receptive and expressive language skills, showed higher levels of maladaptive behaviors (e.g., self-injury, aggression, destruction, hyperactivity, withdrawal, avoidance of eye contact, and stereotypic behaviors). In many social skills domains, persons with autism and ID functioned at lower levels than those with ID only.

Generally, with regards to stability over time, the differences between the two groups remained significant. At the same time, persons with ID and autism showed significant improvement in adaptive behaviors while showing only minor improvement in academic skills when re-tested three years later. Generally, however, the differences between the two groups continued to be significant (Ando & Yoshumura, 1979). Following from Ando and Yoshimura's seminal study, psychologists and psychiatrists have been advised to use the differences in adaptive behavior as a crucial point in the differential diagnosis of the two disorders as is listed in the DSM-IV-TR (APA, 2002). Unfortunately, most studies did not utilize this in the formation of their research groups.

PURPOSE

In recent years there has been an increase in the amount of research examining ASD and ID. Although this research has advanced knowledge about these disorders, most findings have limited generalizability for two reasons:

1. Researchers have primarily examined these disorders as separate constructs, rather than in their more common presentation as co-occurring disorders.

2. The vast majority of the research has focused on child, rather than adult, populations.

Researchers have documented that children and adolescents with ID bear an increased risk of Axis I psychopathology with an increased chance of more serious psychopathology present in those with more profound levels of cognitive impairment (Sadock & Sadock, 2003). In addition, there is emerging evidence that persons with ASD are more likely to have co-occurring psychiatric disorders, especially stereotypies (Wing, 1996), depression and anxiety (Ghaziuddin et al., 2002; Wing, 1981; Matson & Nebel-Schwalm, 2005), and schizophrenia (Bouras et al., 2004; Turner, 1989). Although estimates of autism-ID comorbidity are as high as 70 percent, the majority of these studies examined ASD and ID in isolation, not as co-occurring entities. Thus, the utility of these previous studies in determining if, in fact, ID or ASD was responsible for the positive relationship between ID and psychiatric illness is limited (APA, 2002). Although there are some studies which examine the relationship between adults with both ID and ASD by looking at both disorders together, these studies have been criticized for their failure to control for factors, such as level of ID, age, and gender, which are known to affect rates and severity of psychopathology (Tsakanikos et al., 2005; Sadock & Sadock). In addition, all of these studies examined psychiatric illness in adults as a whole, not taking into account that many psychiatric disorders are affected by stage of life (Sadock & Sadock, 2003; APA, 2002). Thus, more

statistically sound research examining the association between those dually diagnosed with ID and an ASD is needed.

The purpose of this study is to extend the current research by determining which, if any, psychiatric and behavior disorders are more common in adults with a co-occurring ASD and ID and to determine whether the presence of either PDD-NOS or autistic disorder in those with ID is predictive of increased psychopathology over ID alone. In order to control for variance contributed by level of ID (e.g., moderate, severe, and profound) confounding and complicating factors, in particular factors of the following: gender (male/female), years of age, dementia and disorders that result in dementia as a secondary or long-term effect (e.g., Down's syndrome, primary dementia), and the statistical error variance contributed by these factors will be statistically controlled for . In addition, in order to gain an increase understanding of how age affects the severity of psychiatric symptoms, the sample will be examined by age group to determine if an increase in age is associated with an increase or decrease in symptoms. This should help elucidate the relationship between ASD, ID and comorbid psychiatric and behavioral pathology when it occurs in conjunction with moderate, severe and profound ID.

Additionally, because the participants in this study are selected from developmental centers, rather than sampled from the community at large, the results will be more applicable to the population of those with comorbid ID-ASD diagnoses and give researchers a better idea of the extent of the relationship between co-occurring ID, ASD and Axis I psychiatric disorders. As a result, this study will help clarify many of the deficiencies in past studies on this topic while also helping to extend previous researchers' findings on this topic, and will help strengthen past findings in the literature.

RATIONALE

Autism and ID often co-exist (Matson & Rivet, 2008). Although there is increased risk for other psychiatric disorders in persons with sole diagnoses of ID (Deb, Mathews, Holt & Bouras, 2001) it is unclear whether people with ID and autism are more prone to psychiatric disorders than people with ID only.

There is some evidence to suggest that persons with autism are more vulnerable to psychiatric disorders other than ID. For example, social and cognitive impairments in autism and schizophrenia have historically contributed to a controversy on whether the two disorders were on the same continuum (Kanner, 1943) or whether autism per se is part of a prodromal phase signaling later psychosis (Foster-Greene, 2002). However, this hypothesis has received no empirical support (APA, 2002). In addition, there is some emerging evidence that affective disorders, especially depression, are the most common psychiatric disorders in people with autism. A number of researchers have demonstrated a higher prevalence rate of both major depression and anxiety among immediate relatives of people with autism when compared to people with Down's syndrome (Bolton, Murphy, Higgins, Griffith & Pickles, 2002).

Although some there is some evidence that people with ID and autism are at greater risk for psychopathology, especially depression, than people with ID alone, the findings of these studies are limited because of small sample sizes and failure to control for potential confounding variables such as level of ID and age. This research will attempt to extend emerging findings by examining whether the presence of either autism or PDD-NOS is associated with increased psychopathology when level of ID is controlled for while controlling for confounding variables such as age and level of ID.

RESEARCH QUESTIONS AND HYPOTHESES

Based on the aforementioned data, the following specific hypotheses are made: Questions:

Hypothesis One: Psychiatric and behavioral disorders will be over-represented in those with a secondary diagnosis of an ASD as compared to those with a diagnosis of ID-only.

Hypothesis Two: The severity of psychiatric and behavioral disorders will follow a severity continuum, with autism being associated with most severe levels of psychopathology, PDD-NOS being associated with moderate levels and ID-only being associated with the least severe levels of psychopathology.

Hypothesis Three: The presence of an ASD will serve as a predictor of the overall level of overall psychopathology as well as psychiatric disorders individually.

METHOD

Participants

Participants for this study were selected from Pinecrest Developmental Center (PDC) and Hammond Developmental Center (HDC) in Louisiana. Both are state-run facilities that consist of individual homes that are under 24-hour supervision. Both centers provide medical, nursing, dental and mental health services to their residents. While PDC houses 499 residents, HDC serves 384 residents. The mean age of residents at PDC is 47.2 years of age and the mean age of residents at HDC is 49.2 years of age. Both facilities predominately serve persons with diagnoses of moderate, severe and profound ID.

There were several inclusion and exclusion criteria stipulated. In order to be included in this study, participants had to be currently diagnosed with ID and had to have resided at either PDC or HDC for at least six months. In addition, if direct care staff familiar with the participant for six months were not able to be contacted, the participant was excluded from the study. The total sample was originally made up of 337 persons. One person with a primary diagnosis of dementia and two persons with Down's syndrome, which features dementia as a common occurrence, were excluded from the study. This resulted in a final sample of 334 persons. The following are the demographics of the sample in regards to ID diagnosis: 77 % profound ID (n = 256), 13.9 % severe ID (n = 47), 5.3 % moderate ID (n = 18), and 0.6 % mild ID (n = 2). Approximate 4.2 % of the sample was comprised of those with an ID diagnosis of unspecified severity (n = 14). Of the total sample, 49.2 % (n = 165) was diagnosed with ID only, 30.7 % (n = 104) were diagnosed with PDD-NOS, and 20.4 % were diagnosed with autism (n = 68). At the time the data was being collected, 38.9 % (n = 130) of the participants were on psychotropic

medications. Additionally, 43% (*n* = 145) of the sample had documented seizure activity per the medical records. Out of these individuals, 70.3% (*n* = 102) have a diagnosis of ID-only and 29.7% (*n* = 43) had ID-ASD diagnoses. The 334 participants selected for this study ranged from 16 years of age to 82 years of age, with a mean age of 48.59 and a median age of 48 years. Diagnoses of ID were made previous to this study by licensed psychologists using psychometrically sound tests of intellectual functioning and adaptive behavior scales. The intelligence tests used were the Stanford-Binet Intelligence Scales, Fifth Edition (Roid, 2003a) and the Leiter International Performance Scale (Leiter, 1969; Roid & Miller, 1997). Choice of which intelligence scale to be used was based on whether or not the client was verbal or non-verbal. Because the Stanford-Binet measures IQ down to 10, some consider the Stanford-Binet to be the preferred instrument for those with profound levels of ID. While the Stanford-Binet measures IQ as low as 10, the Leiter-Revised measures IQ as low as 30. Therefore, if the client was non-verbal and was deemed to have an IQ less than 30, no exact IQ could be attained. Both the Leiter-R and Stanford-Binet – Fifth Edition utilize composite IQ scores with a mean of 100 and a standard deviation of 15. Additionally, both tests derive subscale scores with a mean of 10 and a standard deviation of 3. Although both the Stanford Binet- Fifth Edition and the Leiter-R utilize composite IQ scores with the same means and standard deviations that allow quantitative comparison of the scores, there is some debate if these both tests qualitatively measure the same constructs (Tylenda, Beckett, & Barrett, 2007). This is because the Stanford-Binet – Fifth Edition global IQ score incorporates both verbal and non-verbal abilities (called the Verbal IQ index and the Non-Verbal IQ index), the Leiter R's composite emphasizes only fluid intelligence. Thus, while the Stanford-Binet – Fifth Edition produces a Full Scale IQ score that is partially influenced by socioeconomic status and culture, the Leiter-R is relatively free from

cultural influence. This method of diagnosis is considered the standard for a diagnosis of ID (Lecavalier, Tasse, & Levesque, 2001). DSM-IV-TR (APA, 2000) and ICD-10 (WHO, 2004) diagnostic criteria were used for this study. In addition to diagnoses of ID, 84 participants were diagnosed with autistic disorder and 69 wer, and Bare diagnosed with PDD-NOS. Two raters were required to be in agreement on diagnostic criteria in order for a classification of ASD to be made. In addition, to ensure that the autistic behaviors occurred across time, ratings were obtained at different times. The participants were classified as meeting criteria for Autistic Disorder or Pervasive Developmental Disorder Not Otherwise Specified (PDDNOS). In addition to the classification of ASD, 84 of the participants had at least one additional Axis I diagnosis and 16 of these participants had more than one Axis I diagnoses. These diagnoses were made previous to the current study by a licensed psychologist in consensus with the habilitation team. This method is considered optimal in ASD diagnostic research (Matson & Nebel-Schwalm, 2005). The most frequent comorbid Axis I diagnosis was Stereotypic Movement Disorder, with or without self-injurious behavior (n = 29); other Axis I conditions included Pica (n = 25), Bipolar Disorder (n = 26), Mood Disorder NOS (n = 5), Major Depressive Disorder (n = 12), Tic Disorder (n = 2), Rumination Disorder (n = 1), Psychotic Disorder (n = 13), Anxiety Disorder NOS (n = 1), Attention Deficit Hyperactivity Disorder (n = 1), Phobia (n = 1), and OCD (n = 1).

Power

In order to determine the sample size needed for the study, an a priori power analysis was conducted. GPower 3 (Faul & Erdfelder, 2007) was used with a medium effect size of $f^2 = .25$, an alpha level of .05, and power of .95 (Cohen, 1965).

Measure

The measure is an informant-based screening tool designed to assess psychopathology in individuals with severe and profound intellectual disability (Matson, 1995). The DASH-II utilizes third-party informants who have known the patient-in-question for a period of at least six months. The DASH-II utilizes a Likert scale approach to indicate that the presence and severity of a particular disorder. A composite score is derived as well as a subscale score corresponding to each disorder assessed by the measure. The scale is heavily grounded in empirically-supported behaviorism and, as such, assesses the frequency, severity, and duration of the problem behaviors.

The DASH-II is comprised of 13 separate subscales: (a) impulse control disorders, (b) organic brain disorders, (c) anxiety disorders, (d) depression, (e) mania, (f) PDD, (g) schizophrenia, (h) stereotypies, (i) eating disorders, (j) sexual disorders, (k) dyssomnias, (l) self-injury, and (m) elimination disorders. Each of the three separate dimensions is rated from 0-2. For the frequency dimension, the options are: 0 (not a problem/absent in the past two weeks), 1 (1-10 times in the previous two weeks), or 2 (more than 10 times in the past two weeks). For the severity dimension, information is requested from the informant regarding how much disruption and/or the extent of damage the behavior caused in the past two weeks: 0 (no damage/problems), 1 (no damage but some disruption) or 2 (behavior caused injury or property damage at least one time). Additionally, for the duration dimension, data is requested from the informant regarding how long the behavior has been occurring: 0 (less than one month,), 1 (1-12 months), and 2 (more than 12 months). Scores that indicate clinical significance vary across the range of the disorders assessed by the DASH-II (Matson).

Although the subscales do not share a one-to-one correspondence to DSM diagnostic criteria, diagnoses established by the DASH-II based on two criteria: (a) endorsement of at least half of the items on the depression, anxiety, mania, PDD/autism, or schizophrenia subscale, or (b) endorsement of at least one item at a level of 1 or 2 on the severity subscale for tics, self-injurious behavior, behavior disorders, elimination disorders, eating disorders, sleep disorders, sexual disorders, organic syndrome, and impulse control disorders. The rationale underlying the use of different criteria for the first five disorders assessed by the DASH-II as compared to the last eight disorders assessed by it is that the first five scales of the DASH-II represent syndromes and the last eight scales measure simple disorders. In other words, the initial five subscales of the DASH-II consist of more complex disorders which are comprised of a constellation of symptoms, while the last eight subscales assess uni-dimensional disorders that require just one symptom be present to warrant a diagnosis (Matson et al., 1991).

The DASH-II is comprised of items from the DSM and from previous measures and studies which examined psychiatric problems common in those with ID (APA, 1983). Items were selected for the scale based on two criteria:

1. How relevant and appropriate the item was for those with severe and profound levels of ID.

2. How understandable items would be to those who lacked formal knowledge of psychiatric assessment.

The second criterion was deemed especially important because many informants were residential assistants and, as such, lacked training in the field of mental illness (Matson et al., 1991).

Normative sample characteristics for the DASH-II were normed and validated on a heterogeneous sample that comprised 506 residents of developmental facilities (n = 247 females, n = 259 males). Racially, 88.6% of the sample was Caucasian whereas the remainder of the sample was African American. Approximately 97% of the norming group had lived in one of four state institutions dedicated to those with intellectual disability located either in Louisiana or in Wisconsin for a period of at least one year (Matson et al., 1991). The participants were diagnosed with either severe ID (32.2%) or profound ID (62.7%) according to criteria set forth by the American Association on Mental Retardation (1983) and the DSM-III-R (APA, 1983) that specifies severe deficits in intellectual functioning and in adaptive behavior before the person reached 18 years of age (APA, 1987). The norming sample of the DASH also represented the range of physical disorders frequently encountered in those with ID the most common represented in the norming sample were the inability to walk (35%), and seizure activity (18%; Matson et al.).

Standardization procedures/data collection for ratings for each item of the DASH were gathered from one-on-one interviews administered by psychology undergraduate and graduate student with direct care staff members who had been familiar with the subject in question for at least one month. In the overwhelming majority of cases (78%), the staff member had known the patient for at least one year. As a result, they could provide first-hand information about the duration of a behavior. If a staff member who knew a particular subject for one year was not available, staff members were encouraged to make duration estimations based on medical record review and data from other direct-care staff members (Matson et al., 1991).

Approximately 14.5 items were endorsed for each subject. Scales with the highest average were elimination disorders and PDD/autism. Scales with the lowest means were

schizophrenia, anxiety, and eating disorders. Item-wise, items related to problems regarding elimination of waste and compliance with direct care staff were highest across all three dimensions of the DASH-II. Items that were the least frequently endorsed were items assessing pyromanic behaviors and the experiencing of hallucinations.

The internal consistency for each subscale ranges from .84 for elimination disorders to .20 for the Schizophrenia subscale with an average of .76. The subscales of the DASH-II were shown to be non-overlapping/not orhogonal, with scores on one scale not affecting scores on any other scale (Matson et al., 1991). Therefore, internal consistency was deemed to be appropriate for use in this study.

Reliability on the entire scale is good with a test- retest reliability of .84 and an inter-rater reliability of .86 (Matson, 1995). Inter-relater reliability for each item contained in the measure was high with the severity dimension being .96, duration being .95, and frequency being .91 (Matson et al., 1991). In addition, many of the subscales of the DASH-II have been validated , including the PDD/dutism, depression, and mania subscales. The mania subscale correctly differentiated between patients with bipolar disorder in the manic phase and non-bipolar patients and had high convergent validity with the Young Mania Rating Scale, a widely used measures used to assess mania symptoms in psychiatric populations (Matson & Gonzalez, 2004; Matson & Smiroldo, 1988). The depression/mood subscale was validated through data obtained on individual patients' pre-existing diagnoses that were either given by licensed psychologists or psychiatrists or were previously placed in their respective medical charts by licensed clinical psychologists or clinical psychiatrists at the facilities in which the participants resided at the time of the norming and validating of the DASH norming study. .

PROCEDURE

Data Collection

The study used archival data for which data had been collected in 2007. Doctoral students, enrolled in a clinical psychology program, trained in administration and scoring of the DASH-II, administered this measure to direct care staff at PDC and HDC. As students who conducted the DASH-II interviews were practicum students who worked with direct-care staff of both developmental centers, direct-care staff was familiar with the persons conducting the interviews. Interview times ranged from 30 to 45 minutes. The variance in interview time was primarily attributable to how well staff member understood the questions being asked of them. Interviews were conducted within the client homes at PDC and HDC. Interviews followed a structured format with direct-care staff being read questions from the DASH-II and asked for a response. The informants' names and shifts were documented on the front of each assessment packet. Direct-care staff members were not given any rewards or inducements to participate in the study.

Data Security

Data was secured per the recommendations of Simon, Unutzer, Young, and Jurgen (2000). These recommendations include storing data on a password-protected computer database. Each participant's data was de-identified and assigned a code number. Only the primary investigator and the consulting psychologist had access to identifying demographic information. The graduate student assistants had access to raw data for data entry and verification. All hard-copy data, such as assessment protocols, were stored in a locked, private office.

DATA ANALYSIS

This study utilized a quasi-experimental design, where participants were assigned to various levels of the independent variable not randomly but based on their pre-existing disorders. First, descriptive statistics were used to report the frequency and the percent of persons with co-occurring psychiatric disorders across the three experimental groups: those diagnosed with ID and autism, those diagnosed with ID and PDD-NOS, and those diagnosed with ID only. Participants were assumed to have a particular disorder if their score on the DASH-II is in the clinically elevated range. Next, these same statistics were reported for the specific types of psychopathology per the seven relevant subscales of the DASH-II relevant to this study: a) Impulse control disorders, (b) Organic neurological disorders, (c) Anxiety disorders, (d) Affective disorders, (e) mania, (f) schizophrenia, and (g) stereotypies. Chi square tests for independence were run to examine whether a particular disorder (presence or absence) was overrepresented in one group or another.

Because previous research has shown a relationship between severity of ID and type and severity of psychiatric illness (Sadock & Sadock, 2003), this study divided the sample into three separate subsets according to the level or severity of intellectual disability (e.g., moderate, severe, and profound). This was done to maximize the chances that effects are due to an ASD diagnosis rather than to severity of ID. Disorders over-represented in one group or another were included in the multivariate analyses while others were excluded. Originally, multivariate analyses of covariance (MANCOVA) were proposed to determine, for each subset (i.e., moderate ID, severe ID, profound ID), whether there were mean differences across the three diagnostic groups on the linear combination of the seven relevant DASH-II subscale scores, as well as on each scale individually. However, the proposed covariate, age, was not statistically

significant with the dependent variables. Thus, the proposed covariate was not contributing additional variance. As a result, multiple analyses of variance (MANOVA) were utilized.

Finally, in order to determine whether a diagnosis of PDD-NOS or autistic disorder was predictive of certain psychiatric disorders after level of ID is controlled for, a series of hierarchical regression analyses were conducted. A separate hierarchical regression was used for each separate subscale of the DASH-II of interest: impulse control disorder, organic disorder, anxiety disorders, affective/mood disorders, mania, schizophrenia, mania, and stereotypies. This analysis allowed for the assessment of the impact of a predictor variable (PDD-NOS or autistic disorder) on a criterion variable (psychopathology per the DASH-II) while statistically controlling for the continuous variable (Full Scale IQ scores). Thus, IQ scores, based on level of ID recorded in the patient's chart, were entered in the first block to predict subscale scores on the DASH-II. In the second block, the participant's diagnostic category (PDD-NOS or autistic disorder) was entered. A statistically significant increase in R^2 will suggest that diagnostic category is predicting co-occurring psychopathology over and above level of ID.

RESULTS

The sample was originally made up of 337 persons. Three persons with a diagnosis which features dementia as a common occurrence were excluded from the study. This resulted in a final sample of 334 persons. The following are the demographics of the sample in regards to ID diagnosis: 77 % profound ID (*n* = 256), 13.9 % severe ID (*n* = 47), 5.3 % moderate ID (*n* = 18), and 0.6 % mild ID (*n* = 2). Approximately 4.2 % of the sample was comprised of those with an ID diagnosis of unspecified severity (*n* = 14). Of the total sample (including those with unspecified and mild ID), 49.2 % (*n* = 165) was diagnosed with ID only, 30.7 % (*n* = 104) were diagnosed with PDD-NOS, and 20.4 % were diagnosed with autism (*n* = 68). At the time the data was being collected, 38.9 % (*n* = 130) of the participants were on psychotropic medications. Additionally, 43% (*n* = 145) of the sample had documented seizure activity per the medical records. Out of these individuals, 70.3% (*n* = 102) have a diagnosis of ID-only and 20.4% (*n* = 65) had ID-ASD diagnoses.

Of the total sample, 45.1 % (*n* = 152) had clinical elevations on the psychiatric disorders measured by the DASH-II and 32.3% (*n* = 109) did not. The remainder of the sample could not be examined due to missing ratings on the DASH-II. Out of those with significant elevations on the DASH-II, 32.89 % (n = 50) were diagnosed with ID, autism, and a psychiatric disorder; 32.89% (*n* = 50) were diagnosed with ID, PDD-NOS and a psychiatric disorder, and 31.52 % (*n* = 52) with ID only and a psychiatric disorder. Furthermore, 76.92 % (*n* = 50) of individuals with autism had a clinical elevation on the DASH-II; 48.08 % (*n* = 50) of individuals with PDD-NOS had a clinical elevation on the DASH-II, and 42.24 % (*n* = 70) of individuals with ID only had an elevation on the DASH-II. Statistics on individual DASH-II scales per each independent variable are listed earlier.

Chi square tests for independence were run to determine whether there was an association between the presence or the absence of a secondary ASD (e.g., autism, PDD-NOS) and the presence or absence of psychiatric disorders as measured by clinically significant elevations on the relevant DASH-II subscale. For moderate ID, results indicated significant relationships for stereotypies, $\chi^2(2) = 3.29$, $p < .05$ and mood disorders, $\chi^2(2) = 2.03$, $p < .05$. In regards to severe ID, significant relationships were found for stereotypies, $\chi^2(2) = 10.96$, $p < .05$ and mania, $\chi^2(2) = 4.0$, $p < .05$. Finally, in regards to profound ID, significant relationships were found for four disorders: mania, $\chi^2(2) = 11.91$; schizophrenia, $\chi^2(2) = 8.52$, $p < .05$; stereotypies, $\chi^2(2) = 19.58$, $p < .05$ and impulsecontrol disorder, $\chi^2(2) = 9.14$, $p < .05$. Only the significant subscales were included in the MANOVA analyses for each subsample (i.e., moderate, severe, and profound ID).

For moderate ID, a between subjects MANOVA was conducted to determine the influence of ASD diagnosis on the stereotypies and mania subscales of the DASH-II. The independent variable was ASD diagnosis (i.e., moderate ID only, moderate ID and autism, and moderate ID and PDD-NOS). The dependent variables were the stereotypies and mood subscales of the DASH-II. A bivariate, zero order correlation evidenced a non-significant relationship between age and the dependent variables of mood, $r = .72$, $p = .06$ and stereotypies. $r = .00$, $p > .05$.,Thus, age was not entered as a covariate One person with a primary diagnosis of dementia and two persons with Down's Syndrome who earned IQs in the moderate ID range were excluded from the analyses. There were no univariate or multivariate cell outliers at $\alpha = .001$. Results of assumptions of homogeneity of variance, linearity and multicollinearity were satisfactory.

Using the Wilks' lambda criterion, the combined dependent variables were not significantly related to age, $F(2, 113) = 0.34$, $p > .05$ but were significantly related to ASD, approximate $F(4, 226) = 3.41$, $p < .01$. Univariate between-subjects tests indicated that ASD diagnosis was not significantly related to obtained scores on the DASH-II mood subscale ($p = .13$; partial eta squared = .26). However, an ASD diagnosis was significantly related to obtained scores on the DASH-II stereotypies subscale ($p = .01$, partial eta squared = .52). Tukey's HSD post-hoc tests revealed that those with autism differed significantly from those with PDD-NOS (*Md* = 2.5) and those with PDD-NOS differed significantly from those with ID-only (*Md*= 1.84) and those with autism had higher scores on the DASH-II stereotypies subscale than did those with ID only (*Md* = 4.54).

For severe ID, a between-subjects MANOVA was conducted to determine the influence of ASD diagnosis on the stereotypies and mania subscales of the DASH-II. The independent variable was ASD diagnosis (i.e., severe ID only, severe ID and autism, and severe ID and PDD-NOS). The dependent variables were the stereotypy and mania subscales of the DASH-II. A bivariate correlation revealed that age, the prospective covariate, was not significantly associated to either dependent variable: Mania, $r = -.25$, $p > .05$ or Stereotypies, $r = .00$, $p > .05$. No persons with primary diagnoses of dementia or Down's syndrome were present in this subset. There were no univariate or multivariate cell outliers at $\alpha = .001$. Results of assumptions of homogeneity of variance, linearity and multicollinearity were satisfactory.

Using the Wilks' Lambda criterion, the combined dependent variables of stereotypies and mania were not statistically significantly related to age, approximate $F(2, 40) = 2.16$, $p > .05$ but were statistically significantly related to ASD, approximate $F(4, 80) = 2.50$, $p < .05$. Univariate between subjects tests indicated that a diagnosis of ID-only, autism and ID or PDD-NOS and ID

were significantly related to obtained scores on the DASH-II stereotypies subscale (p = .05, partial eta squared = .14). In addition, the diagnosis of ID-only, autism and ID, and PDD-NOS and an ID were not significantly related to obtained scores on the DASH-II mania subscale (p = .13, partial eta squared = .09). Tukey's HSD post-hoc tests revealed that those with PDD-NOS obtained significantly higher scores than those with PDD-NOS ($M\ d$= 2.1, p < .05) and those with PDD-NOS obtained significantly higher scores than those with ID-only (Md= 1.03, p < .05) but although those with autism obtained higher scores on the DASH-II stereotypies subscale than did those with ID only, the difference in means did not reach significance (Md = .03, p > .05).

For profound ID, a between-subjects MANOVA was conducted to determine the influence of ASD diagnosis on the stereotypies and mania subscales of the DASH-II. The independent variable was ASD diagnosis (i.e., profound ID only, profound ID and autism, and profound ID and PDD-NOS). The dependent variables were the schizophrenia/psychosis, stereotypies, impulse control disorders, and mania subscales of the DASH-II. A bivariate correlation revealed that age was not significantly correlated to the dependent variables of mania, r = -.30, p > .05, impulse control disorders, r = .08, p > .05; stereotypies, r = -.00, p > .05 or psychosis/schizophrenia, r =.04, p > .05. Because of the lack of association between age, the prospective covariate, and the dependent variables, age was not entered as a covariate. There were no univariate or multivariate cell outliers at α = .001. Results of assumptions of homogeneity of variance, linearity and multicollinearity were satisfactory.

Using the Wilks' Lambda criterion, the combined dependent variables of stereotypies, schizophrenia, impulse control disorders and mania were not statistically significantly related to age, approximate F (4, 220) = 1.13, p > .05 but were statistically significantly related to ASD,

approximate F(8, 440) = 2.50, $p < .01$. Univariate between subjects tests indicated that a diagnosis of ID-only, autism and ID or PDD-NOS and ID were significantly related to obtained scores on the DASH-II stereotypies subscale (p = .00, partial eta squared = .14). However, the diagnosis of ID-only, autism and ID, and PDD-NOS and an ID were significantly related to obtained scores on the DASH-II mania subscale ($p = .04$, partial eta squared = .04), the schizophrenia subscale ($p = .00$, partial eta squared = .05) and Impulse Control Disorder ($p = .00$. partial eta squared = .05). A follow-up Tukey's HSD test conducted on those with stereotypies revealed that those with autism obtained significantly higher means than those with ID-only (*Md* = 2.49, $p < .05$). and those with autism had significantly higher scores on the DASH-II subscale than those with PDD-NOS (*Md* = 1.30, $p < .05$). Additionally, those with PDD-NOS obtained higher scores than did those with ID-only (*Md* = 1.18, $p < .05$). A second follow-up Tukey's HSD test conducted on those with mania revealed that those with autism and those with PDD did not differ significantly from each other (*Md* = 0.35, $p > .05$) while those with autism obtained significantly higher means than those with ID-only (*Md* = 1.21, $p < .05$) and those with PDD-NOS obtained significantly higher means than those with ID-only (*Md* = 0.87, $p < .05$). A third follow-up Tukey's HSD test conducted on those with impulse control disorders revealed that although those with autism obtained higher means than those with PDD-NOS, it was not significantly different (*Md* = 0.15, $p > .05$) while those with autism did have significantly higher scores than those with ID-only (*Md* = 1.81, $p < .05$), and those with PDD-NOS also obtained significantly higher scores than those with ID-only (*Md* = 1.65, $p < .05$). A fourth Tukey's HSD test conducted on those with schizophrenia revealed that although those with autism obtained higher means than those with PDD-NOS, they did not differ significantly from one another (*Md* = 0.26, $p > .05$). Those with autism obtained significantly higher scores than those with ID-only

(*Md* = 0.76, *p*<.*05*). Those with PDD-NOS obtained significantly higher means than those with ID-only (*Md* = 0.50, $p < .05$).

A multiple hierarchical regression then was performed in order to determine whether a diagnosis of PDD-NOS or autism is predictive of psychiatric disorders in general and individual disorders specifically. The entry of the variables was predicated on the widely accepted finding that level of cognitive impairment and adaptive functioning in inversely related to amount and severity of psychiatric symptoms. In other words, more severe levels of ID are associated with increased numbers and severity of psychiatric and behavioral disorders (Matson & Frame, 2006; Sadock & Sadock, 2003). Thus, participants' level of ID was entered on the first block and ASD diagnosis was entered on the second block. The dependent variable was total level of psychopathology per total score on the DASH-II.

Initial inspection of data for linearity using scatter plots demonstrated a linear relationship between the predictor and dependent variables. Plots of standardized residuals were inspected and revealed a scatter around zero, meeting the assumption of a relatively even distribution.

Residual plots of standardized residuals by predicted values revealed no violations of the assumptions of linearity, normality, or homoscedasticity. The points in the plot formed a relatively straight line relationship (linearity), evidenced an even distribution above and below the line (normality), and were dispersed evenly about the reference line (homoscedascticity). As such, interpretation of regression could proceed with confidence.

For overall psychopathology per the DASH-II, level of ID was not significantly related to the dependent variable, $F(1, 285) = 2.96$, $p = .01$, and explained 1% of the variance in overall DASH-II psychopathology scores ($R^2 = .01$). In Step 2, the addition of ASD diagnosis added

significantly to the overall prediction of the dependent variable, explaining an additional 7% of the variance, ΔF (1, 284) = 21.83, *f* $p < .05$ Thus, the overall model demonstrated statistical significance in the prediction of psychopathology, explaining 7.1% of the total variance, *F(* 2, 284) = 12.50, p < .01.

For the DASH-II mood disorders subscale, hierarchical regression analysis results indicated that IQ failed to significantly predict scores on the DASH-II mood disorders subscale, $R^2 = .00$, $F(1, 292) = 1.36$, $p > .05$. However, the addition of ASD diagnoses as a predictor added significantly to the model's predictive value, $\Delta R^2 = .02$, $F(2, 293) = 3.20$, $p < .05$. The accompanying

For DASH-II scores on the mania subscale, results indicates again that IQ failed to significantly predict scores on this subscale, $R^2 = .00$, $F(1, 295) = 1.24$, $p > .05$. However, the addition of ASD as a predictor added significantly to the model's predictive value, $\Delta R^2 = .05$. $F(2, 293) = 8.32$, $p < .05$.

For scores on the DASH-II, Neurological/Organic subscale, results again indicated that IQ, alone, failed to reach statistical significance in the prediction of participant scores on this subscale, $R^2 =$,.00 ,$F(1, 293) = 4.58$, p > .05 while the addition of ASD diagnoses added significantly to the model's predictive value, $\Delta R^2 =$ ..02, $F(2,293) = 6.71$, $p < .05$.

For scores on the DASH-II stereotypies subscale, results indicated that IQ alone was a significant predictor of scores on this subscale, $R^2 = 1.90$, F (1,294) = 5.78, $p < .05$. Additionally, the addition of ASD diagnoses added significantly to the model's predictive value, $\Delta R^2 = 8.40$, $F(2,294) = 16.95$, $p < .05$.

For Impulse Control Disorders subscale, results indicated that IQ alone was not a significant predictor of scores on this subscale, $R^2 = .00$, $F(1, 290) = 0.64$, $p > .05$ while the addition of

ASD diagnoses added significantly to the model's predictive value, ΔR^2 = ..02, F (2,289) = 2.78. Thus, ASD was significant.

A multiple hierarchical regression analysis was run for age. Results indicate that IQ failed to significantly predict overall scores on the DASH-II, R^2 = .00, $F(1, 295) = 1.24$, $p > .05$ and the addition of age as a predictor failed to add significantly to the model's predictive value, ΔR^2 = .001. $F(1, 282) = 1.46$, $p < .05$.

DISCUSSION

The presence of a secondary ASD diagnosis was associated with the occurrence of additional psychopathology in those with primary diagnoses of ID. This was especially true for psychiatric and behavioral diagnoses characterized by symptoms that were often associated with high degrees of externalizing symptoms. Thus, the results of this study supported the first hypothesis that a secondary ASD diagnosis would be associated with the presence of psychiatric and behavioral disorders. In other words, participants with a secondary ASD diagnosis would obtain higher mean differences on the DASH-II subscales and the measure as a whole when compared to those with ID only. This was true for some of the DASH-II subscales across all three diagnostic groups (e.g., moderate ID, severe ID, and profound ID) on the stereotypies, mania, schizophrenia, and impulse control disorder subscales. The second hypothesis stated that mean scores on the DASH-II subscales would follow a severity continuum, with those with autism obtaining the highest means and those with ID only obtaining the lowest. Results showed that, as a group, those with ASDs obtained higher overall mean scores on relevant DASH-II subscale than those with ID only. However, the presence of autism, considered the most severe type of ASD (APA, 2003; Matson & Rivet, 2008), generally was not associated with higher levels of psychiatric disorders relative to the less severe ASDs, such as PDD-NOS, per scores on the DASH-II. Thus, support for the second hypothesis was not found. As with hypothesis one, the third hypothesis was also supported by the data. Specifically, a diagnosis of either autism or PDD-NOS was predictive of psychiatric and behavioral disorders above and beyond IQ based on the participant's previously diagnosed level of ID.

When studying the prevalence of psychotropic medication usage among participants in this study, additional support for an increased association between psychopathology and a

secondary ASD diagnosis was found: While 38.9% (*n* = 130) of the total sample were on psychotropic medication, almost all of these [40.2% (*n* = 52)] were diagnosed with autism . Further support of the association between secondary ASD diagnoses and psychopathology was provided by the rate of seizure disorder in the sample. As mentioned earlier, studies of children and adolescents with autism have shown the onset of psychiatric difficulties around the time of puberty and early adolescence (Gillberg & Schaumann, 1981). Citing evidence that the highest frequency of seizures occurs during the adolescent years (Sillanpaa, 1999), some have associated the beginnings of these psychiatric disorders in those with autism to this increase in seizure frequency around adolescence (Bradley et al., 2003). In the sample examined in this study, 29.7% (*n* = 43) who had a documented history of seizure activity were diagnosed with an ASD while 70.3% (*n* = 102) were diagnosed with ID only. Thus, although seizure activity in this sample is less prevalent in those with a secondary ASD, those with ASDs demonstrate a higher number of some psychiatric disorders. As a result, it could be that the finding of an increased prevalence in psychiatric and behavioral disorders in those with secondary ASD diagnoses indicates that this increase in psychiatric disorders in comparison to those with ID is likely not related to seizure activity in this group.

While the results of this study replicate the general findings of the vast majority of past studies citing increased levels of psychopathology in those with secondary ASD diagnoses (Bradley et al., 2004; Morgan et al., 2003; LaMalfa et al., 2004), they also contradicted the sole study that failed to find this relationship (Tsakanikos et al., 2005). Because this lack of consensus among studies may diminish the confidence in the conclusions that can be drawn from this study, as well as past research on this topic as a whole, further examination of the results of this study, and how they compare and contrast to past studies, is warranted.

Testing the same general hypothesis as this study, Tsakanikos et al. (2005) concluded that psychiatric disorders were no more prevalent in those with ID-ASD than in those with diagnoses of ID only. While both looked at similar populations, methodological differences involved in determining participants' diagnoses could have led to different conclusions between the studies. As mentioned earlier, behaviors present in those with developmental disabilities often mimic symptoms of co-existing psychiatric disorders (Matson & Frame, 1986). Following from this, researchers in the field of developmental disabilities discovered that symptoms of ID can overshadow symptoms of comorbid psychiatric disorders, resulting in under diagnosis of comorbid psychopathology in those with ID (Gabriel, 1994; Reiss et al., 1982). In an effort to avoid this phenomenon, called diagnostic overshadowing, it has been recommended that assessments for psychopathology and developmental disabilities be conducted at separate times (Kerker et al., 2004; Gabriel). The fact that a separate assessment was done for co-occurring psychopathology for participants in this study helped to avoid diagnostic overshadowing. The fact that assessments were done at separate times may have contributed to the different results found in this study as compared to past studies which failed to find a significant relationship between secondary ASDs and comorbid psychopathology.

Different sampling techniques among studies also may have led to disparate results. Past researchers, one group of whom found no difference in psychopathology between the two groups (Tsakanikos et al., 2005), utilized data drawn from the community at large to the exclusion of developmental centers (Morgan et al., 2003; Bradley et al., 2004). While the practice of community sampling often increases the external validity of findings when conducting research on the general population, Kerker et al. (2004) have argued that research on those with severe developmental disabilities predominantly reside in developmental or inpatient centers and should

at least partially sample from these settings to increase external validity. Because two of the past studies examining psychopathology in those with ID relied on outpatient samples, it is difficult to generalize from their samples. However, this study, which examined those with more severe cognitive impairments, used a sample from developmental centers, which maximized external validity.

Another reason for contradictory conclusions could be that the research design of past studies failed to take into account variance introduced by ID itself. Although a well documented relationship has been established between level of ID and frequency and severity of psychopathology (Matson & Frame, 1986; Matson & Gonzalez, 2006; Mash & Dozois; 2003; Sadock & Sadock, 2003 Matson et al., 1991), most studies located for this paper failed to control for the effects of ID. As a result, it is difficult to draw definitive conclusions from them about the association between a secondary ASDs and psychiatric morbidity in a population with ID. By controlling for the effects of ID, this study extends previous findings that ASD does in fact increase the risk of psychopathology above and beyond the risk imposed by ID alone (Bradley et al., 2004 ; Morgan et al., 2003; Piven & Palmer, 1999).

While this study utilized a period estimate to assess for co-occurring disorders, some previous studies assessed for comorbid psychopathology using point prevalence methods (Tsakanikos et al., 2005). Demonstrating fleeting symptoms of depression or anxiety in reaction to internal or external factors is a normal phenomenon, not necessarily a sign of psychopathology. Rather, only when these reactions are sustained over time does the person likely have a formal psychiatric disorder. Additionally, even those with formally diagnosable psychiatric disorders often exhibit a fluid course of symptoms, often worsening and improving over time in response to a variety of factors (APA, 2002). Although no studies have examined

the course of psychiatric and behavioral disorders in those with ID and autism, it is assumed that mental disorders in those with ID follow the same fluctuating course of those in the general population. Because point estimates provide only a snapshot of the participant at one point in time, researchers have argued that reliance on them is dangerous, especially when they are utilized in non-verbal populations who cannot give longitudinal reports of their symptoms (Kerker et al., 2004). Despite the argument against the use of this type of data, some previous studies utilized point prevalence data which could have led to either under-diagnosis or over-diagnosis of psychiatric disorders if they were assessed at a time of an acute stressor or routine stress (Reiss, 1990; Tsakanikos et al.).

Additionally, discrepancies in findings among studies may be secondary to confounding environmental and social factors present in the individual's surroundings. Previous research has demonstrated that the presentation, onset, maintenance, and exacerbation of mental illness is influenced by biological, psychological and social factors (Taylor, 1990). Following from this, Kerker et al. (2004) have suggested that different environments, each with its unique factors that predispose and protect from mental illness, may impact the frequency and presentation of mental illness seen in those with ID. Thus, while some previous studies used samples residing in group homes or at homes with families, others utilized samples that resided in inpatient centers. Each of these is marked by protective and risk factors in regards to the development and presentation of psychopathology. For example, while familial supports sometimes provide more individual attention than an inpatient center, inpatient settings often allow for more opportunities to interact with peers (Kerker et al.). This could have influenced whether participants presented with particular psychiatric disorders or not.

Differences in results may have also resulted in varying methods used between studies to determine co-occurring mental health diagnoses. As mentioned earlier, previous research has shown that the heavy reliance on verbal report of inner experiences makes the diagnostic systems in the DSM-IV-TR (APA, 2002) and ICD-10 (WHO, 1992) difficult for use in those with severe ID (King, DeAntonio, McCracken, Forness, & Ackerland, 1994). Despite this, some of the previous studies based mental health diagnoses on criteria set forth in these manuals (LaMalfa et al., 2004). While some studies relied heavily on manuals with questionable applicability in this population, others relied on notes made in participants' medical charts to infer the diagnosis of psychiatric and behavioral disorders (Morgan et al., 2003). As with using existing diagnostic manuals for those with ID, the utilization of medical chart data has also proven to be controversial (Kerker et al. 2004). In a seminal study of this phenomenon, the prevalence of co-occurring psychiatric and behavioral disorders in those with ID was approximately 12% when using chart reviews and approximately 40% when appropriate diagnostic measures for this population were utilized (Reiss, 1990).

Although the general findings of this study are in-line with most researchers' conceptualizations that a secondary ASD diagnosis confers additional, unique vulnerability to the risk of psychiatric comorbidity above that which is contributed by ID alone, the results of this study do diverge from past studies on some factors (Morgan et al., 2003; Bradley et al., 2004; Ghaziuddin & Tsai, 1991). First, following from past studies, it was expected that depression, along with psychosis, would be the most likely to occur in those with a secondary ASD diagnosis (Morgan et al). Second, the findings of the current study failed to replicate the hypothesized positive relationship between severity of ASD and the number of secondary psychiatric and behavioral disorders (Matson & Rivet, 2008).

Although the number of studies still is small in number, some researchers have begun to hypothesize which types of co-occurring psychopathology are most likely to occur in those with ID-ASD diagnoses. While there fails to be complete agreement on the prevalence of different disorders in those with ASDs, previous studies suggest that psychotic disorders and mood disorders, particularly major depression, are among most prevalent (Ghaziuddin & Tsai, 1991; Morgan et al., 2003; LaMalfa et al., 2004; Bradley et al., 2004). While the findings of the current study do replicate that psychosis, particularly schizophrenia, was more common in those with ASDs, unipolar depression was not more prevalent in any of the three diagnostic groups in this study. In addition to the methodological differences mentioned earlier in this section (e.g., different samples, failure to control for the effects of ID), other specific reasons that depression failed to reach significance, and that the severity continuum hypothesized to exist by some but not evident in this study, ought to be explored.

One possible explanation for the failure of mood disorders to reach significance in this sample could be due to the distribution of ID in this sample as compared to those in other studies. As mentioned earlier, while increased levels of cognitive impairment are associated with increased levels of psychopathology, there also is some evidence to suggest that depression itself actually is more common in those with mild and moderate level of ID than in those with severe and profound ID (Jacobson, 1982; Kerker et al., 2004). Many past studies that have found depression to be most common in those with ID and ASD diagnoses either failed to control for the influence of ID altogether or utilized samples in which mild and moderate ID were much higher in number than those with severe and profound ID, or both (Morgan et al., 2003; LaMalfa et al., 2004). This study consisted of an opposite distribution, in which the frequency of

intellectual impairment was skewed towards those with severe and profound ID. This may, in turn, account for the fact that depression was not prevalent in the sample.

Aside from differences in the level of ID present in the sample and consequent comorbid disorders, subjectivity of item content may have led to difficulties in obtaining the exact prevalence rate of internalizing disorders, such as depression, in this population. Although most types of psychopathology are accompanied by behavioral symptoms, complete assessment of some disorders relies on the ability of the individual to verbalize emotions and feelings so that the assessor can understand inner emotional states. Because the majority of participants in this study either were non-verbal or had only rudimentary communication abilities, particularly when compared to past studies which contained higher numbers of persons with less severe ID (Morgan et al., 2003), the assessment of these inner emotional states was up to inference by the direct care staff. As a result, the exact prevalence of disorders with symptoms of a more internalizing nature may be under-detected.

Also, differences in the times of day in which staff had contact with the client, and variations in the amount of time the care worker had been caring for the client may have led to an under-estimate of internalizing disorders in this population. Although the literature supports variability in the responses of staff (McGill et al., 2001), every effort was made in the collection of data for this study to utilize direct care staff, who have more contact with clients, rather than home managers or other professionals, who often have a more supervisory role, resulting in less contact with residents of the facility. When direct care staff was interviewed about the participant in question, the worker was required to have worked with the client for a minimum of six months. However, even when this criterion was met, variability in the amount of time each direct care staff member had spent with each specific client still differed from day-to-day. This is

because while the direct care worker was usually assigned to a group of clients, she/he had to spend one-on-one time with some of them when delivering particular services that are part of activities of daily living such as bathing and feeding. Thus, the staff member is more likely to observe certain behaviors than others. The behaviors that they are more likely to be observed and noted in the patient's chart for other staff members to see, are likely those that are more disruptive or externalizing in nature and are represented by the items from many of the scales that were significant in the initial analyses. Particular items that may have been affected by the amount of time spent with the patient include: sleepwalks, speech in harder to understand that it used to be, is cranky, and takes another's belongings.

Finally, the exact shifts the direct care staff member interacted with the client in question may have affected the significance of certain scales. Although all persons interviewed about clients had extensive involvement in the care of the client in question, many direct care staff members may have only interacted with the client at certain times of the day or night. Because symptoms of some disorders may be more noticeable at certain times of the day than others, there is a possibility that the behavior in question went unnoticed. Examples of items that may have been affected include: sleepwalks and has difficulty falling asleep.

As researchers attempt to determine which psychiatric disorders are most common in those with ID-ASD diagnoses, many also have hypothesized the existence of a "severity continuum" in which autism, considered to be the most severe form of an ASD as measured by the amount of social and communication impairment (Matson & Rivet, 2008), is associated with increased frequency and severity of psychopathology as compared to more moderate forms of ASD, such as PDD-NOS. However, while the findings of this study support that those with ASDs have higher levels of psychopathology than those with ID only, the results failed to

support that those with autism have higher levels of psychopathology than those with PDD-NOS. While post-hoc tests on DASH-II subscales significant in the MANOVA indicated that those with a general secondary ASD diagnosis obtained higher means than those with ID only, those with autism obtained higher scores on only half of these subscales when compared to those with PDD-NOS. Thus, the results of this study suggest that while those with ASDs appear to have higher levels of psychopathology than those with ID only, those with autism do not necessarily demonstrate higher levels of psychopathology than those with less severe ASDs, such as PDD-NOS. One of the reasons the severity continuum was not observed in the results of this study could be due to the effects of psychotropic medication on participants in this study. Although medications participants in the sample were taking are likely currently changing and it is impossible to gauge the specific effects of medications they are taking, records for this study demonstrate that approximately 21.6% (n = 73) of participants in this study were taking medications; 69.9% of these were diagnosed with autism (n = 51). It would make sense that these medications would control for any psychiatric symptoms in these participants and obscured a severity continuum.

Limitations

Several limitations of the current study should be addressed. While the relationship between a secondary ASD diagnosis and additional psychopathology reached significance, the effect sizes of the relationship were small when objectively evaluated using Cohen's (1988) criteria. Thus, while the differences between the various groups were not likely to have occurred by chance, the diagnosis of a secondary ASD was only modestly related to additional psychopathology. In recent years, concerns have been noted in the intervention literature regarding whether statistical significance testing and the reporting of effect sizes by themselves

are sufficient for evaluating the results of a study (Campbell, 2005). These authors argue that while effect sizes are important to consider, it is also important to take into account the clinical significance or the importance of the findings in applied psychology (Campbell; Thompson, 1999; Cohen, 1994). While in isolation the results of the study fail to be clinically significant, they serve as an indication of the need for further research on psychopathology in general as it occurs in this population and specifically on the base rates of psychiatric disorders in this population. Partially due to the increased complexity of assessing psychopathology in those with ID only, our knowledge of base rates of mental disorders in this population is incomplete (Sadock & Sadock, 2003). While the effect sizes in this study suggest that persons with a secondary ASD are more likely to have certain disorders, the results do not indicate how much more common additional psychopathology is when compared to those with sole diagnoses of ID. Thus, as they currently stand, the results simply inform us that those with ID-ASD are more likely than those with ID only to have certain disorders. Knowing the base rates of various psychiatric disorders in the ID only and those with ID-ASD diagnoses will help to evaluate the effectiveness of measures designed to assess co-occurring psychopathology in this population. Because determination of the base rates without effective measures is impossible and determining the effectiveness of measures is hard to do without knowing the base rates of disorders, this problem is a sort of dilemma. Additionally, knowing the base rates of psychopathology in this population will help practitioners to decide if using a particular measure to assess for psychopathology is clinically worthwhile or not. Therefore, unless we first definitively determine the base rates of psychopathology in those with ID only, future attempts at prediction may be fraught with false positives.

Because both pervasive developmental disorders and schizophrenia are both characterized by forms of purposeless behaviors (APA, 2002), some items found on the Schizophrenia/Psychosis subscale may also be characteristic of autistic disorder or PDD-NOS. Specifically, in pervasive developmental disorders, these behaviors are operationally defined as stereotyped, repetitive patterns of behavior while in psychotic disorders; they are operationally defined as grossly disorganized behaviors (APA). Thus, elevations found on the Schizophrenia/Psychosis subscale may actually be partially due overlapping symptoms of a pervasive developmental disorder to which the DASH-II was sensitive. In order to determine this, a post-hoc item analysis was conducted on participants with profound ID who placed in the clinically elevated range on the DASH-II Schizophrenia subscale. Using the *DSM-IV-TR* criteria for a diagnosis of Schizophrenia or Schizoaffective Disorders, criteria were identified that overlapped with a diagnosis of either autistic disorder or PDD-NOS. The only criteria in the *DSM-IV-TR* that this applied to both and were included in the DASH-II under the Autism/PDD subscale were grossly disorganized/catatonic behavior. Item analysis was then conducted on persons with profound ID who were in the clinically significant range for schizophrenia. Only one person (0.2% of the persons significant on this subscale) significant on the Schizophrenia subscale had this item endorsed. Most of the symptoms of schizophrenia in this group fell under the larger rubrics of reality distortion. The most prevalent symptoms in the sample were hallucinations (68.2%, n = 21). Negative symptoms occurred, but at a lower rate than those involving reality distortion.

Several limitations present in this study should be kept in mind when interpreting the results. First, one may find fault with the validity of the measure of the dependent variable. As mentioned earlier, most psychiatric and behavioral disorders do not follow a stable course but

rather fluctuate in response to a variety of internal and external factors such as environmental stressors, medication side effects, and the presence of a general medical condition. Most of the disorders assessed in the study are no exception to this. In order to meet formal diagnostic criteria for disorders present in the DSM or ICD, individuals are required to exhibit symptoms of a particular disorder for a specified period of time (APA, 2002, WHO, 1992). Although the DASH-II is an improvement in that it gathers period estimate rather than point estimate data, the time periods it inquires about (i.e.,14 days) do not match criteria required for a formal DSM diagnosis. Thus, it is possible for a person who may exhibit symptoms of a disorder to be judged as having the disorder without meeting formal DSM criteria. Although impossible due to limited time and financial resources, a more accurate measure of whether or not patients have the psychiatric disorders measured in this study might have been to interview direct care staff daily during the entirety of the time period required for a proper DSM diagnosis (six months, in most cases).

Second of all, restriction of range might have skewed the rates of psychiatric disorders in the sample. As stated earlier, the sample examined in this study consisted entirely of persons in a residential/inpatient setting. Although this is the gold standard for research on patients with severe cognitive impairment (Kerker et al., 2004), in order for this to be generalizable to all persons with ID, future studies should aim at acquiring adequate proportions of this population residing in both inpatient and outpatient centers. Because statistical significance tests only supply information about the sample, as opposed to the population, and because those in an inpatient setting are likely more severely ill than their outpatient counterparts, the rates of some select psychiatric disorders may be misrepresented in this sample in comparison to the entire ASD-ID population. While some disorders may be overrepresented in this sample, it also is possible that

certain disorders are underrepresented or managed well enough that they are harder to detect. The residential center where the patients reside has several psychologists on staff that treat psychiatric disorders in this population. Thus, it is logical to expect that persons who reside in the residential center receive more mental health care than persons with ID who reside in group homes or with guardians. As a result, the rates of certain treatable disorders may be better managed and be harder to detect than in persons with ID who reside outside the residential setting.

Third of all, alpha inflation, particularly on later statistical analyses, may have led to Type 1 error. Although one criterion of statistical analysis selection was to choose analyses that limited the scope of Type 1 or Type 2 statistical error, an inevitable consequence of any type of statistical analysis is inflation of Type 1 error. According to Cohen (1988), this is the biggest danger on later steps of the statistical analysis. However, it should be noted that effect sized below .10 are usually indicative of Type 1 error (Cohen) and effect sizes found in later analyses are well above this figure.

Finally, the sample under study contained primarily those with moderate, severe and profound ID. In order for the results of this study to be generalizable to the all persons with ID, more persons with mild ID should be included in future studies. In addition, because a person's environment can introduce protective factors and risk factors in regards to psychiatric comorbidity, more persons living in a community environment should be included.

Overall, the presence of a secondary ASD diagnosis was associated with the occurrence of additional externalizing psychopathology in those with primary diagnoses of ID. Although secondary ASDs were associated with higher levels of psychopathology, autism was not associated with increased levels of psychopathology compared to those with PDD-NOS.

Although the general findings of this study support the majority of past studies that have documented an increased association between ID-ASD diagnoses and additional psychopathology, it also diverged with some studies on exactly which disorders were more common in this population. However, differences, such as the level of ID contained in the sample and the setting itself, may have led to differences in these results. In order for the results of this study to be of value to applied psychology, definitive base rates for the amount of specific types of psychopathology in this population should be determined.

The results of this study highlight the need for further research into psychopathology as it occurs in both those with ID diagnoses and those with secondary ASDs. In particular, this study demonstrates the importance of continuing to refine assessment methods in this population to attempt to separate out what part is ID related and what part is related to pure psychopathology.

REFERENCES

American Academy of Pediatrics Committee on Children with Disabilities (2001). The pediatrician's role in the diagnosis and management of autistic spectrum disorder in children. *Pediatrics, 197,* 1221-1226.

American Association on Mental Retardation (AAMR, 1992). *Mental retardation: Definition,* classification, and systems of supports. Washington, DC: Author.

American Psychiatric Association (APA, 1980). *Diagnostic and statistical manual of mental disorders*, third edition- revised. Washington DC: American Psychiatric Association.

American Psychiatric Association (APA, 2000). *Diagnostic and statistical manual of mental disorders*, fourth edition – text revised. Washington DC: American Psychiatric Association.

Ando, H., & Yoshimura, I. (1979). Effects of age on communication skill level prevalence levels and prevalence of maladaptive behaviors in autistic and mentally retarded children. *Journal of Autism and Developmental Disorders, 9,* 83-93.

Ando, H., Yoshimura, I., & Wakabayashi, S. (1980). Effects of age on adaptive levels and academic skill levels in autistic and mentally retarded children. *Journal of Autism and Developmental Disabilities, 10,* 173-184.

Angst. J. (1999). A dimensional view of today's classification of depressive and anxiety states. *Salud Mental, 22,* 42-47.

Asubel, D., & Sullivan, E. (1970). *Theory of problems of child development*. New York: Grune.

Attwood, A., Frith, U, & Hermelin, B. (1988). The understanding and use of inter-personal gestures by autistic and Downe's syndrome children. *Journal of Autism and Developmental Disorders*, 18(2), 241-257.

Bailey, D.B., Hatton, D.D., & Skinner, M. (1998). Earl development trajectories of males with fragile-X syndrome. *American Journal on Intellectual Disability, 103,* 29-39.

Barlow, D.H. (2002). *Anxiety and its disorders.* New York: Guilford Publications.

Baron-Cohen, S., Allen, J., Gillberg, C. (1992). Can autism be detected at 18 months? The need, the haystack, and the CHAT. *British Journal of Psychiatry,* 161, 839-843.

Baron-Cohen, S., Mortimore, C., Moriarty, J., Izaguire, J., & Robertson, M. (1999). The prevalence of Gilles de la Tourette Syndrome in children and adolescents with autism. *Journal of Child Psychology and Psychiatry, 40,* 213-218.

Beck, A.B., Ward, C.H., & Mendelson, M. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561-571.

Beirne-Smith, M., Patton, J. R., & Kim, S. (2006). *Mental Retardation* (7th ed.). Upper Saddle River, NJ: Prentice Hall/Merrill.

Bejerot, S., Nylander, S. & Lindstrom, L. (2001). Autistic traits in obsessive-compulsive disorder. *Nordic Journal of Psychiatry, 55*(3), 169-176.

Bernheimer, C., & Keogh, B. (1988). Stability of cognitive performance of children with developmental delays. *American Journal of Mental Deficiency, 92,* 539-542.

Bertrand, J., Mars, A., Boyle, C., Bove, F., Yeargin-Allsopp, M., & Decoufle, P. (2001). Prevalence of autism in a United States population: The Brick Township, New Jersey investigation. *Pediatrics, 108,* 1155-1161.

Birch, H., Richardson, S.A., Baird, D., Horobin, G., Illsley, R. (1970). *Mental subnormaility in the community: A clinical and epidemiological study.* Williams & Wilkins: Baltimore.

Bolton, P.F., Murphy, M., Higgins, N., Griffiths, P.D., & Pickles, A. (2002). Neuroepileptic determinants of autism spectrum disorders in tuberous sclerosis complex. *Brain, 125,* 1247-1255.

Bondy, A.S., & Frost, L.A. (1995). Educational approaches to preschool: Behavior techniques n a public school setting. In E. Schopler & G.B. Mesibov (Eds.). *Learning and cognition in autism* (pp. 311-333). New York: Plenium Press.

Borthwick-Duffy, S.A. (1994). Epidemiology and the presence of psychopathology in people with mental retardation. *Journal of Consulting and Clinical Psychology, 62,* 17-27.

Boucher, J., & Lewis, V. (1992). Unfamiliar face recognition in relatively able autistic children. *Journal of Child Psychology and Psychiatry, 33*(5), 843-859.

Bouras, N., Martin, G., & Leese, M. (2004). Schizophrenia-spectrum psychoses in people without intellectual disability. *Journal of Intellectual Disability Research,* 48(6), 548-555.

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.

Broman, S., Nichols, P.L., Shaughnessy, P., & Kennedy, W. (1987). *Retardation in children: A developmental study of cognitive deficit.* Hillsdale, NJ: Erlbaum.

Bushnell, I.W., Sai, F., & Mullin, J.T. (1989). Neonatal recognition of the mother's face. *British Journal of Developmental Psychology, 7,* 3-15.

Campbell, T.C. (2005). An introduction to clinical significance. *Journal of Early Intervention,* 27, 3, 210-227.

Capps, L, Yamiria, N., & Sigman, M. (1992). Understanding of simple and complex cues in non-retarded children with autism. *Journal of Child Psychology and Psy*chiatry, 33, 1169-1182.

Carr, J. (1970). Mental and motor development in young Mongul children. *Journal of Mental Deficiency, 14,* 205-220.

Cath, D., Ran, N., & Smith, J.H. (2008). Symptom overlap between autism spectrum disorder, generalized anxiety disorder, social anxiety disorder, and obsessive compulsive disorder in adults: A preliminary case-controlled study. *Psychopathology,* 41(2), 101-110.

Celani, Battacchi, & Arcodoacono (1999). The understanding of the emotional meaning of facial expressions in people with autism. *Journal of Autism and Developmental Disorders,* 29, 1, 57-66.

Charrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association, 285,* 3093-3099.

Charman, T. (2002). The prevalence of autism spectrum disorders. *European Child & Adolescent Psychiatry,* 11, 249-256.

Charman, T., & Baird, G. (2002). Practitioner review: Diagnosis of autism spectrum disorders. *Journal of Child Psychology and Psychiatry, 4,* 289-361.

Cherry, K.E., Penn, D, Matson, J.L, & Bamburg, J.W. (2000). Characteristics of schizophrenia among persons with severe or mental retardation. *Psychiatric Services, 51*(7), 922-924.

Chicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development,* 55(1), 1-7.

Clarke, D., Baxter, M., Perry, D., & Prasher, V. (1999). The diagnosis of affective and psychotic disorders in adults with autism.: Seven case reports, *Autism, 3,* 149-164.

Clarke, A.M., & Clarke, A.B. (1988). The adult outcome of early behavioral abnormalities. *International Journal of Behavioral Development, 11*(1), 3-19.

Cohen, J. (1965). Some statistical issues in psychological research. In B.B. Wolman (ed.), *Handbook of Clinical Psychology* (pp. 95-121). New York: McGraw-Hill.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

Cohen, D.J., Donnellan, A.M., & Paul, R. (1987). *Handbook of autism and pervasive developmental disorders*. Oxford, England: John Wiley & Sons.

Cohen, J. (1994). The earth is round ($p < .05$). *American Psychologist, 49,* 997-1003.

Coie, J. D., Watt, N. F., West, S. G., Hawkins, J. D., Asarnow, J. R., Markman, H. J., Ramey, S. L., Shure, M. B. & Long, B. (1993). The science of prevention. A conceptual framework and some directions for a national research program. *American Psychologist, 48,* 1013-1022.

Committee on Educational Interventions for Children with Autism (2001). *Educating Children with Autism.* Washington, D.C.: National Academy Press.

Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., Drew, A., & Wheelwright, S. (1999). Autism spectrum disorders at 20 and 42 months of age: Stability of clinical and ADI-R diagnosis. *Journal of Clinical Psychology and Psychiatry,* 40, 719-732.

Creak, M. (1961). Schizophrenia syndrome in childhood: Progress report of a working party. *Cerebral Palsy Bulletin, 3,* 501-504.

Crillien, C.M., Jameson, S., Wilkinson, E.M. (1966). Studies in mental handicap. I. Prevalence and distribution by clinical type and severity of deficit. *Archives of Dis Child,* 41, 528-538.

Cromwell, R.L., Palk, B.E., & Foshee, J.G. (1961). Studies in activity level V. *American Journal of Mental Deficiency, 65,* 744-748.

Dawson, G., Munson, J., Estes, A., Osterling, J., McPartland, J., Toth, K., Carver, L., & Abbot, R. (2002). Neurocognitive function and joint attention ability in young children with autism spectrum disorder. *Child Development, 73,* 345-358.

Deb, S., Matthews, T., Holt, G., & Bouras, N. (2001). Practice Guidelines for the Assessment and Diagnosis of Mental Health Problems in Adults with Intellectual Disability. Brighton: Pavillion.

DeMyer, M.K. (1974). The measured intelligence of autistic children. *Journal of Autism and Schizophrenia,* 41(1), 42-60.

DeMyer, M.K., Alpera, G.D., Barton, S., DeMyer, W.E., Churchill, D.W., & Hintgen, J.J. (1972). Imitation in autistic, early schizophrenic and non-psychotic subnormal children. *Journal of Autism and Childhood Schizophrenia, 2,* 264-287.

Denning, C.B., Chamberlain, J.A., & Polloway, E.A. (2000). An evaluation of state guidelines

for mental retardation: Focus on definition and classification practices. *Education and Training in Mental Retardation & Developmental Disabilities*, 35(2), 226-232

.

Donovan, C.L. & Spence, S.H. (1990). Prevention of childhood anxiety disorders. *Clinical Psychology Review*, 20(4), 509-531.

Dykens, E.M., Hodapp, R.M., & Evans, D.W. (1994). Profiles and development of adaptive behavior in children with Down syndrome. *American Journal of Mental Retardation, 98,* 580-587.

Dykens, E.M., Ort, S., Cohen, I., Finucane, B., Spiridigliozzi, G., Lachiewicz, A., Reiss, A., Freund, L., Hagerman, R., O'Connor, R. (1996). Trajectories and profiles of adaptive behavior in males with fragile-X syndrome: Multi-center studies. *Journal of Autism and Developmental Disorders*, 26, 286-301.

Eaves, R.C., & Williams, T.O. (2006). The reliability and construct validity of rating for the autism behavior checklist. *Psychology in the Schools, 43,* 129-142.

Edelson, M. (2006). Are the majority of children with autism mentally retarded? A systematic evaluation of the data. *Focus on Autism and Other Developmental Disabilities*, 21(2), 66-83.

Einfield, S.L., & Tonge, B.J. (1996). Population prevalence of psychopathology in children and adolescents with intellectual disability: II. Epidemiological findings. *Journal of Intellectual Disability Research*, 40, 99-109.

Einfeld, S.L. & Tonge, B.L. (1999), Observations on the use of ICD-10 guide for mental retardation. *Journal of Intellectual Disability Research, 43,* 408-412.

Ekman, P., & Friesen, W.V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, 17(2), 124-129.

Ellis, N.R., Barnett, C.D., & Pryer, M.W. (1960). Operant behavior in mental defectives: Exploratory studies. *Journal of the experimental analysis of behavior, 3,* 63-69.

Emerson, E, Hatton, C, Bromley, J. & Caine. (1998). *Clinical psychology and those with intellectual disability*. New York: John Wiley and Sons.

Emerson, E., & Hatton, C. (2007). Mental health of children and adolescents with intellectual disabilities in Britain. *British Journal of Psychiatry, 191*. 493-499.

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

Evans, D.W. & Gray, F.L. (2000). Compulsive-like behavior in individuals with Down's

syndrome: Its relation to mental age level, adaptive and maladaptive behavior. *Child Development*, 71(2), 288-300.

Fewell, R.R. (1991), *Play Assessment Scale (5th Revision)*. Unpublished manuscript, University of Miami School of Medicine.

Fischer, K., & Rose, S. (1994). Dynamic development of coordination of components in brain and behavior: A framework for theory. In G. Dawson & K. Fischer (Eds.). *Human behavior and brain development* (pp. 3-66). New York: Guilford.

Filipek, P.A., Accardo, P.J., Ashwal, S., Baranek, G.T., Cook, E.H., Jr., Dawson, G., Gordon, B., Gravel, J.S., Johnson, C.P., Kallen, R.J., Levy, S.E., Minshew, N.J., Ozonoff, S., Prizant, B.M., Rapin, I., Rogers, S.J., Stone, W.I., Teplin, S.W., Tuchman, R.F., Volkmar, F.R. (2000). Practice parameter: Screening and diagnosis of autism: Report of the American Academy of the American Academy of Neurology and Child Neurology Society. *Neurology*, 55, 468-479.

Fish, B. (1977). Neurobiological antecedents of schizophrenia in children: Evidence for an inherited, congenital, neurintegrative defect. *Archives of General Psychiatry, 34,* 1297-1313.

Fish, B., Shaprio, T., Campbell, T., Campbell, M., & Wile, R. (1968). A classification of schizophrenic children under five years. *American Journal of Psychiatry, 124*, 1415-1423.

Fitzgerald, M., & Corvin, A. (2001). Diagnosis and differential diagnosis of Asperger syndrome. *Advances in Psychiatric Treatment, 7*, 310-318.

Flaum, M. (1995). The diagnosis of schizophrenia. In Shiriqui, C.L, & Narsallah, H.A. (eds.) *Contemporary issues in the diagnosis and treatment of schizophrenia.* (pp. 83-108). Washington, DC: American Psychiatric Association.

Folstein, S., & Rosen-Sheidley, B. (2001). Genetics of autism: Complex etiology for a heterogeneous disorder. *Nature Reviews: Genetics, 2*, 943-955.

Fombonne, E. (1999). The epidemiology of autism: A review. *Psychological Medicine,* 29, 769-786.

Fombonne, E., (2001). What is the prevalence of Asperger disorder? *Journal of Autism and Developmental Disorders*, 31, 361-364.

Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: an update. *Journal of Autism and Developmental Disorders*, 33, 365-382.

Fombonne, E., & Chakrabarti, S. (2001). No evidence for a new variant of measles-mumps-rubella induced autism. *Pediatrics, 108:* e 58.

Fombonne, E., DuMazabrun, C., & Cans, C. (1997). Autism and associated medical disorders in a French epidemiological survey. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(11), 1561-1569.

Foster-Greene, M. (2002). *Schizophrenia revealed: From neurons to social interactions.* New York: W.W. Norton.

Gabriel, S.R. (1994). The developmentally disabled, psychiatrically impaired client. *Journal of Psychosocial Nursing*, 32, 35-39.

Garmezy, N. & Streitman, S. (1974). Children at risk: The search for the antecedents of schizophrenia. *Schizophrenia Bulletin, 8,* 14-90.

Ghaziuddin, M., Alessi, N. & Greden, J.F. (1995). Life events and depression in children with pervasive developmental disorder. *Journal of Autism and Pervasive Developmental Disorders*, 25(5), 495-502.

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

Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research in clinical care. *Journal of Autism and Developmental Disorders*, 32(4), 299-306.

Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome. *Journal of Intellectual Disability Research, 42*(4), 279-283.

Gitta, M.Z. & Goldberg, B. (1995). Dual diagnosis psychiatric and physical disorders in a a clinical sample, part II. *Clinical Bulletin of Developmental Disabilities Program, 6,* 1-2.

Gillberg, C. (1985). Asperger's syndrome and recurrent psychosis. *Journal of Autism and Developmental Disorders*, 15(4), 389-397.

Gillberg, C. & Schaumann, H. (1981). Infantile autism and puberty. *Journal of Autism and Developmental Disorders*, 11, 365-371.

Gillberg, L, & Wing, R. (1999). Autism. *Acta Psychiatrica, 99,* 399-466.

Gilllott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism, 5*(3), 277-286.

Gitta, M.Z. & Goldberg, B. (1995). Dual diagnosis of psychiatric and physical disorders in a clinical sample, Part II. *Clinical Bulletin of Developmental Disabilities Program, 6,* 1-2.

Goodman, J.F., & Cameron, J. (1978). The meaning of IQ constancy in young retarded Children. *Journal of Genetic Psychology, 132*, 109-119.

Grimm, L.G., & Yarnold, P.R. (1995). *Reading and understanding multivariate statistics.* Washington, D.C.: American Psychological Association Press.

Grossan, J., Klin, A., & Carter, A. (2000). Verbal bias in recognition in facial emotions in children with Asperger syndrome. *Journal of Child Psychology and Psychiatry, 41*, 3, 369-379.

Groth-Marnat, G. (2003). *Handbook of psychological assessment*. Hoboken, NJ: John Wiley & Sons, Inc.

Halsey, N.A., Hyman, S.L., the Confederate Writing Panel (2001). Measle-mumps-rubella vaccine and autism spectrum disorders: report from the new challenges in childhood immunizations conference convened in Oak Brook, Illinois, June 12-12, 2000. *Pediatrics, 107:* e84.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23, 56-61.

Harris, S.L., Handleman, J.S., Gordon, R., Kristoff, B., & Fuentes, F. (1991). Changes in cognitive and language functioning of preschool children with autism. *Journal of Autism and Developmental Disorders*, 21, 281-290.

Haskell, R.H. (1944). Mental deficiency over a hundred years. *American Journal of Psychiatry*, 100(6), 107-118.

Helsel, W., & Matson, J (1988). Relationship of depression to social skills and intellectual functioning in mentally retarded adults. *Journal of Mental Deficiency, 32*(5), 411-418.

Hobson, R. (1986). The autistic child's appraisal of expression of emotion. *Journal of Child Psychology and Psychiatry,* 27(5), 671-680.

Hobson, R., Ouston, J., & Lee, A. (1988). What's in a face? The case of autism. *British Journal of Psychology, 79*,4, 441-453.

Hobson, R. (1989). *Autism, Nature, diagnosis and treatment.* New York: Guilford Press.

Hodapp, R.M., & Dykens, E.M. (2003). Mental retardation. In E.J. Mash & C. Barkley (eds.), *Child psychopathology (2nd ed.)* (pp. 486-519). New York: Guilford Press.

Holden, R.H. (1972). Prediction of mental retardation in infancy. *Mental Retardation,* 10(1), 28-30.

Honda, H., Shimizu, Y., & Misumi, K. (1996), Cumulative incidence of childhood autism in Japan. *British Journal of Psychiatry, 169*(2), 228-235.

Howlin, P., & Moore, A. (1997). Diagnosis in autism. *Autism,* 1, 135-162.

Hoyson, M., Jamieson, B., & Strain, P.S. (1984). Individualized group instruction of normally developing and autistic-like children: the LEAP curriculum model. *Journal of the Division for Early Childhood, Summer,* 157-172.

Hulley, S.R., & Cummings, S.R. (1988). *Designing clinical research.* Baltimore: Williams & Wilkins.

Huttenlocher, P.R. (1994). Synaptogenesis in the human cerebral cortex. In G. Dawson & K.W. Fischer (Eds.). *Human behavior and the developing brain* (pp 137-152). New York: Guilford.

Jacobson, J.W. (1982). Problem behavior and psychiatric impairment within a developmentally delayed population: I. Behavior frequency. *Applied Research in Intellectual Disability*, 3, 121-139.

Jacobson, J.W., & Mulick, J.A. (2006). Ten years later: Two AAMR tales of a condition. in Switzky, H. & Greenspan, S. (eds.) *What is mental retardation: Ideas for the evolving disability in the 21st century*. Washington D.C.: AAMR.

Johnstone, E.C. & Frith, C.D. (1996). Validations of three dimensions of schizophrenic symptoms in a large unselected sample of patients. *Psychological Medicine, 26*(4), 669-679.

Kadesjo, B., Gillberg, C., & Hagberg, B. (1999). Brief report: Autism and Asperger syndrome in seven-year-old children. *Journal of Autism and Developmental Disorders*, 29, 327-332.

Kaijer, D.W. (1997). *Autism and autistic-like conditions in mental retardation.* Washington, D.C. Garland Science.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2,* 17-250.

Kerker, B.D., Owens, P.L., Zigler, E., & Horwitz, S.M (2004). Mental health disorders among individuals with mental retardation: Challenges to accurate prevalence estimates. *Public Health Reports, 119,* 409-417.

Ketelaars, C., Horwitz, E., Systema, S. (2008). Brief report: Adults with mild autism spectrum disorders: Scores on the Autism Spectrum Quotient and comorbid psychopathology. *Journal of Autism and Developmental Disorders, 38*(1), 176-180.

King, B.H, DeAntonio, C., McCracken, J.T., Forness, S.R., & Ackerland, V. (1994). Psychiatric Consultation in severe and profound mental retardation. *American Journal of Psychiatry,* 151, 1801-1808.

Klinger, L.G., Dawson, G., & Renner, P. (2003). *Child psychopatholgy*. New York: Guilford Press.

Klinger, L.G., Lee, J.M., Bush, M.D., Klinger, M.R., & Crump, S.E. (2001, April). *Implicit learning in autism: Artificial grammar learning*. Paper presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Klin, A., Sparrow, S.S., deBidt, A., Cicchetti, D.V., Cohen, D.J., & Volkmar, F.R. (1999). A normed study of face recognition in autism and related disorders. *Journal of Autism and Developmental Disorders*, 29, 499-508.

Klinger, L.G., Dawson, G., & Renner, P. (2003). Autistic Disorder. In E. Mash & R. Barkley (Eds.), *Child psychopathology* (2nd edition). New York: Guilford Press.

Klinger, L., & Renner, P. (2000). Performance-based measures in autism: Implications for diagnosis, early detection, and identification profiles. *Journal of Clinical Psychology*, 29 (4), 479-492.

Kolvin, I. (1971). Studies in childhood psychoses I. *British Journal of Psychiatry*, 118(545). 385-395.

Kolvin, I., Ounsted, C., & Humphrey, M. (1971). Studies in childhood psychoses II. *British Journal of Psychiatry, 118*(545), 396-402.

Krug, D., Arick, & Almond, P. (1993). Autism Screening Instrument for Educational Planning. Austin, Texas, Pro-Ed.

LaMalfa, G., Lassi, S., Bertelli, M., Salvini, R., & Placidi, G.F. (2004). Autism and intellectual disability: A study of prevalence on a sample of the Italian population. *Journal of Intellectual Disability Research*, 48(3), 262-267.

Lainhart, J.E., & Folstein, S.E. (1994). Affective disorders in people with autism. *Journal of Autism and Developmental Disorders*, 24(5), 578-601.

Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry*, 19(3), 255-268.

Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders. *Journal of Autism and Developmental Disorders,* 36(8), 1101-1114.

Lecavalier, L., Tasse, M., & Levesque, S. (2001). Assessment of mental retardation by school psychologists, *Canadian Journal of School Psychology, 17*(1), 97-10.

Lee, J.H. & Dunner, D.L. (2008). The effect of anxiety disorder comorbidity on treatment resistant bipolar disorders. *Depression and Anxiety, 25*(2), 91-97.

Leekam, S., & Moore, C. (2001). T*he development of autism*. Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Leiter, R.G. (1969). General instructions for the Leiter International Performance Scale. Chicago, IL: Stoelting.

Leyfer, O.; Folstein, S.E.; Susan E. Folstein; Bacalman, S., Davis, No.O.; Dinh, El; Morgan, J.; Tager-Flusberg, H.E. (2006). Comorbid psychiatric disorders in children with autism. *Journal of Autism and Developmental Disorders, 36,* 849-861.

Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry,* 6, 1365-1382.

Lord, C. & Paul, R. (1997). Language and communication in autism. *Handbook of autism and developmental disorders,* 2nd *edn.* (pp. 195-225). New York: Wiley.

Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social Psychiatry,* 1, 124-137.

Lovaas, O.I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology,* 55, 3-9.

McClintock, K. Hall, S., & Oliver, C. (2003). Risk markers associated with challenging behaviors in people with intellectual disabilities: A meta-analytic study. *Journal of Intellectual Disability Research,* 47, 405-416.

McEachin, J.J., Smith, T., & Lovaas, O.I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal of Intellectual Disability,* 97, 359-372.

Maisto, A.A., & German, M.L. (1986). Reliability, predictive validity, and intercorrelationships of early assessment indices used with developmentally delayed infants and children. *Journal of Clinical Child Psychology, 15,* 327-332.

Macdonald, H., Rutter, M., & Howlin, P. (1989). Recognition and expression of emotional cues by autistic and normal adults. *Journal of Child Psychology and Psychiatry,* 30, 865-877.

MacMillan, D.L., Hendrick,, I.G., & Watkins, A. (1988). Impact of Diana, Larry P., and P.L. 94-142 on minority students. *Exceptional Children, 54,* 24-30.

Mash, E.J. & Dozois, D.J. (2003). Childhood Psychopathology: A Developmental-Systems Perspective. In Mash E.J. & Barkley, R.A. (Eds.), *Childhood Psychopathology,* (pp. 3-71). New York: Guilford Press.

Matson, J.L. (1994). *The Diagnostic Assessment of the Severely Handicapped – II (DASH-II). User's Guide*. Baton Rouge, LA; Scientific Publishers, Inc.

Matson, J.L., Baglio, C.S., Smiroldo, , B.B., Hamilton, M., & Packlowskyz, T. (1996). Characteristics of autism as assessed by the Diagnostic Assessment of the Severely Handicapped (DASH-II). *Research in Developmental Disabilities, 17*(21), 135-143.

Matson, J.L., & Barrett, R.P. (Eds.), (1993). *Psychopathology in the mentally retarded, Second edition*. Boston: Allyn and Bacon.

Matson, J.L, & Frame, C.L. (1986). *Psychopathology among mentally retarded children and* adolescents. Beverly Hills, CA: Sage.

Matson, J.L., Gardner, W.I., Coe, D.A., & Sovner, R. (1991). A scale for evaluating emotional disorders in severely and profoundly mentally retarded persons: Development of the Diagnostic Assessment of the Severely Handicapped (DASH-II). *Research in Developmental Disabilities, 17, 135-143.*

Matson, J.L., & Gonzalez, M. (2006). Mania and intellectual disability: The course of manic symptoms in persons with intellectual disability. *American Journal on Mental Retardation*, 111(5), 378-383.

Matson, J.L., & Minshawi, N.F. (2006). *Early intervention for autism spectrum disorders: A critical analysis*. Oxford, England: Elsevier Sciences, Inc.

Matson, J.L., LeBlanc, L.A., Weinheimer, B., & Cherry, K.E. (1999). Reliability of the Matson Evaluation for Social Skills for Individuals with Severe Retardation (MESSIER). *Behavior Modification, 23(4),* 647-661.

Matson, J.L., & Nebel-Schwalm, M. (2005). Comorbid psychopathology with autism spectrum disorders: An overview. *Research in Developmental Disabilities, 28*(4), 341-352.

Matson, J.L., & Rivet, T. (2008). Characteristic of challenging behavior in adults with autism, PDD-NOS, and intellectual disability. *Journal of Intellectual and Developmental Disability*, 33(4), 323-329.

Matson, J.L. & Smiroldo, B.B. (1988). Validity of the mania subscale of the diagnostic assessment for the severely handicapped-II (DASH-II). *Research in Developmental* Disabilities, 18(3), 221-225.

Matson, J.L. Smiroldo, B.B., & Hastings, T.L. (1998). Validity of the autism, pervasive developmental disorders subscale of the Diagnostic Assessment for the Severely Handicapped-II. *Journal of Autism and Developmental Disorders, 28,* 77-81.

Matson, J.L., Terlonge, C., & Gonzalez, M. (2006a). *Autism Spectrum Disorders – problem behavior*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J.L., Wilkins, J. & Ancona, M. (2008). Autism in adults with severe intellectual disability: An empirical study of symptom presentation. *Journal of Intellectual and*

Developmental Disability, 33(1), 36-42.

McCall, R.B., Appelbaum, M.I., & Hogarty, P.S. (1973). Developmental changes in mental performance. *Monographs of the Society for Research in Child Developments*, 38(3, Serial No. 150), 1-83.

McCall, R.B., Hogarty, P.S., & Hurlburt, N. (1972). Transitions in infant sensorimotor development. *Child Development, 52*, 1-12.

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*(5), 772-777.

McGrew, K. & Bruininks, R.H. (1989). The factor structure of adaptive behavior. *School Psychology Review*, 18(1), 64-81.

McPartland, J., Dawson, G., Webb, S.J. (2004). Event-related potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. Developmental *Sciences*, 9(1), 1235-1245.

Medical Research Council (2001). *IDC Review of Autism Research: Epidemiology and Causes*. IDC, London.

Menolascino, F.J. (Ed.) (1970). *Psychiatric approaches to intellectual disability.* New York: Basic Books.

Morgan, S. (1988). Diagnostic assessment of autism: A review of objective scales. *Journal of Psychoeducational Assessment, 6*, 130-151.

Morgan, C.N, Roy, M., & Chance, P. (2003). Psychiatric co-morbidity and medication use in autism. *Psychiatric Bulletin, 27*, 378-381.

Mouridsen, S.E., Rich, ., & Isager, T. (1999). Psychiatric morbidity in disintegrative psychosis and infantile autism; a long-term follow-up study. *Psychopathology, 32*, 177-183.

Mundy, P., & Crowson, M. (1997). Joint attention and early social communication: Implications for research on intervention with autism. *Journal of Autism and Developmental Disorders*, 27, 653-676.

Myers, B.A., & Pueschel, S.M. (1987). Pseudodementia in the mentally retarded. *Clinical Pediatrics*, 26(5), 275-277.

Nelson, K.B. (1991). Prenatal and Perinatal factors in the etiology of autism. *Pediatrics*, 87, 761-766.

Ollendick, T. H., Oswald, D. P., & Ollendick, D. G. (1993). Anxiety disorders in mentally retarded persons. In J. L. Matson & R. P. Barrett (Eds.),

Psycho-pathology in the mentally retarded, 2nd Edition. New York: Grune & Stratton, Inc., pp. 41-85.

Opitz, J. (1996, March). *Historiography of the causal analysis of intellectual disability.* Speech to the 29th Annual Gatlinburg Conference on Research and Theory in Mental Retardation, Gatlinburg, TN.

Ort, S., & Cohen, I. (1996). Trajectories and profiles of adaptive behavior in measles with fragile-X symdrome: Mulitcenter studies. *Journal of Autism and Developmental Disorders*, 26(3), 287-301.

Perkins, D.N., & Grotzer, T.A. (Teaching intelligence. *American Psychologist*, 52(10), 1125-1133.

Piven, J. (2002). Genetics and personality: The example of the broad autism phenotype. In J. Benjamin, R.P. Epstein, & R.H. Belmaker (Eds.), *Molecular genetics and the human personality* (pp. 43-62). Washington, D.C.: American Psychiatric Publishing.

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

Polloway, E.A., Smith, D.J., & Patton, J.R. (1996). Historic changes in mental retardation and intellectual disabilities. *Education and Training in Mental Retardation and Developmental Disabilities*, 31(1).

Powell, J.E., Edwards, A., & Edwards, M. (2000). Changes in the incidence of childhood autism and other autism spectrum disorders. *Developmental Medicine and Childhood Neurology*, 42(9).

Ranitkallio, P. (1987). Social class differences in intellectual disability and subnormality. *Scandanavian Journal of Social Medicine*, *15*, 63-66.

Realmuto, G.M., & August, G.J. (1991). Catatonia in autistic disorder: A sign of comorbidity or variable expression? *Journal of Autism and Developmental Disorders*, 21(4), 517-528.

Reiss, S. (1990). Prevalence of dual diagnosis in community-based day programs in the Chicago area. *American Journal of Mental Retardation. 94*, 578-585.

Reiss, S, Levitan, G.W., & Szysko, J. (1982). Emotional disturbance and mental retardation *American Journal of Mental Deficiency*, *86*,567-574.

Reschly, D. (1972). Technical Report: EPDA Summer Institute. Tuscon, AZ: Arizona Center for Educational Success.

Research Units on Pediatric Psychopharmacology Autism Network. (2002). A double

blind, placebo-controlled trial of risperidone in children with autistic disorder. *New England Journal of Medicine*, 347, 314-321.

Robinson, N.M. & Robinson, H.B. (1965). A follow-up study of children of low birth weight and control children at school age. *Pediatrics, 35*(3), 425-433.

Rodier, P. & Hyman, S. (1998). Early environmental factors in autism. *Mental Retar*dation *and Developmental Disabilities Research Reviews*, 4, 121-128.

Rogers, S.J. & Lewis, H. (1989). An effective day treatment model for young children with pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28. 207-214.

Roid, G.H. (2003a). *Standford-Binet Intelligence Scales.* Fifth Edition. Itasca, IL: Riverside Publishing.

Roid, G.H. & Miller, L.J. (1997). *Leiter International Performance Scale – Revised: Examiner's manual.* Wood Dale, IL: Stoelting Co.

Rolevield, N., Zielhuis, G.A., & Gabreels, F. (1997). The prevalence of mental retardation: a critical review of recent literature. *Developmental Medicine and Child Neurology*, 39, 125-132.

Ross, R.T., Begab, M.J., Dondis, E.H., Giampiccolo, J., & Meyer, C.E. (1985). *Lives of the retarded: A forty-year follow-up study*. Stanford, CA: Stanford University Press.

Ruffman, T., Garnham, W., & Rideout, P. (2001). Social understand in autism: Eye gaze as a measure of core insights. *Journal of Child Psychology and Psychiatry, 42,*8. 1083-1094.

Rumsey, J.M., Rapoport, J.L., & Sceery, W.L. (1985). Autistic children as adults: Psychiatric, social, and behavioral outcomes. *Journal of the American Academy of Child Psychiatry*, 24(4), 465-473.

Russell, A.J., Mataix,-Colls, P., & Ansom, M. (2005). Obsessions and compulsions in Asperger syndrome and high-functioning autism. *British Journal of Psychiatry, 186,* 6, 525-528.

Rutter M., & Garmezy, N. (1983). Developmental Psychopathology. In E.M. Hetherington (Ed.), *Handbook of child psychology.* New York: Wiley.

Ryan, R. (1994). Posttraumatic stress disorders in persons with developmental disabilities. *Networker,* 1-5.

Sadock, B.J., & Sadock, V.A. (2003). *Synopsis of psychiatry.* Washington, D.C.: Lipincott, Williams, & Wilkins Publishers.

Schopler, E., Brehm, S.S., Kinsbourne, M., & Reichler, J. (1971). Effect of treatment structure on development in autistic children. *Archives of General Psychiatry, 24,* 415-421.

Schoppler, 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,* 19, 91-103.

Schoppler, E., Reichler, R.J., & Renner, B.R. (1988). *The Childhood Autism Rating Scale (CARS).* Los Angeles: Western Psychological Services.

Schuerger, J.M., & Witt, A.C. (1989). The temporal stability of individually tested intelligence. *Journal of Clinical Psychology, 45,* 294-302.

Sevin, J.A., Matson, J.L., Coe, D.A., Fee, V.E., & Sevin, B.M. (1991). A comparison and evaluation of three commonly used autism scales. *Journal of Autism and Developmental Disorders*, 21, 417-423.

Share, D.L. ,& Silva, P.A. (1980). Gender bias in IQ-discrepancy and post discrepancy definitions of reading disability. *Journal of Learning Disorders, 36*(1), 4-14.

Siegel, B., Pilner, C., Eschler, J., & Elliot, G.R. (1988). Stone, W.L., & Ousley, O.L. (1997). *STAT Manual: Screening tool for autism in two year-olds*. Unpublished manuscript.Vanderbilt University.

Silk, J.S., Nath, S.R., Siegel, L.R., & Kendall, P.C. (2000). Conceptualizing mental disorders in children: Where have we been and where are we going? *Development and Psychopathology,* 12(4), 713-735.

Sillanpaa, M. (1999). Definitions of epidemiology. In M. Sillanpaa, L. Gram, S.I. Johannessen, & T. Tomson (Eds.), *Epilepsy and mental retardation* (pp. 1-5). Petersfield, Hampshire: Wrighton Biomedical.

Silverstein, A.B. (1982). Note on the constancy of IQ. *American Journal of Mental Deficiency, 87*(2), 227-228.

Simon, G.E., Unutzer, J., Young, B.E. (2000). Large medical databases, population based research, and patient confidentiality. *American Journal of Psychiatry, 157,* 11, 1731-1737.

Soares, J.C., Gershon, S, & Soares, S.C. (2003). *Handbook of medical psychiatry.* Informa Healthcare: Indianapolis.

Sparrow, S.S. , Bella, D.A., & Cicchetti, D.V. (1984). Vineland Adaptive Behavior Scales. Pearson Assessments: New York.

Stavrakaki, C., Antochi, R., & Emery, P. (2004c). Obsessive compulsive disorders in adults with Down syndrome and other developmental disabilities. *Psychiatric Annals, 34,* 196-

200.

Stavrakaki, C., & Lunsky, Y. (2007). Depression, anxiety, and adjustment disorders in people with intellectual disabilities. In Bouras & G. Hold (Eds.). Psychiatric and Behavioral Disorders in intellectual and developmental disorders (pp 113-130). Cambridge, UK: Cambridge University Press.

Stavrakaki, C. & Mintsoulis, C. (1997). Implications of a clinical study of anxiety disorders in persons with mental retardation. *Psychiatric Annals, 27,* 182-189.

Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism: Autism and Asperger's: A review. *Autism, 10*(1), 103-116.

Stone, W.L., Ousley, O.Y., & Littleford, C.D. (1997). Motor imitation in young children with autism: What's the object? *Journal of Abnormal Psychology, 25*, 4 75-485.

Stone, W.L., Ousley, O.Y., Yoder, P.J., Hogan, K.L., & Hepburn, S.L. (1997). Nonverbal communication in two and three-year-olds with autism. *Journal of Autism and Developmental Disabilities*, 27. 677-696.

Stone, W.L., Coonrod, E.E., & Ousley, O.Y. (2000). Screening tool for autism in two-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, 30, 607-612.

Stone, W.L., Coonrod, E.E., Turner, L.M., & Pozdol, S.L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34(6), 691-701.

Strain, P.S., Hoyson, M., & Jamieson, B. (1985). Normally developing preschoolers as intervention agents for autistic-like children: Effects on class deportment and social interaction. *Journal of the Division for Early Childhood,* 105-115.

Stromme, P., & Magnus, P. (2000). Correlations between socioeconomic status, IQ, and aetiology in intellectual disability: A population-based study of Norwegian children *Developmental Medicine and Child Neurology, 42*, 76-86.

Sturmey, P., & Sevin, J.A. (1994). Defining and assessing autism. In J.L. Matson (Ed.), *Autism in children and adults: Etiology, assessment, and intervention* (pp. 13-36). Pacific Grove, CA: Brooke/Cole.

Sturmey, P., Matson, J.L., & Sevin, J.A. (1992). Analysis of the internal consistency of three autism scales. *Journal of Autism and Developmental Disorders, 22,* 321-328.

Tabachnick, B.G., & Fidell, L.S. (2001). *Using Multivariate Statistics: Fourth Edition*. Needham Heights, MA: Allyn & Bacon.

Tantam, D. (2003). Psychological disorder in adolescents and adults with Asperger syndrome. *Autism, 4*(1), 47-62.

Taylor, R.L. (1990). The Larry P. decision a decade later: Problems and future directions. *Intellectual Disability, 28*(1), iii-vi.

Teal, M.B., & Wielbe, M.J. (1986). A validity analysis of selected instruments used to assess autism. *Journal of Autism and Devlopmental Disorders, 16,* 485-494.

Thompson, B. (1999). Journal of editorial policies regarding statistical significance tests: Heat is to fire as *p* is to importance. *Educational Psychology Review, 11,* 157-169.

Tidmarsh, L., & Volkmar, F.R. (2003). Diagnosis and epidemiology of autism spectrum disorders. *Canadian Journal of Psychiatry, 48,* 517-525.

Trent, J.W. (1982). Reasoning about mental retardation: A study of the American Association of Mental Deficiency, *Dissertation Abstracts International, 43*(5), 1714.

Tsakanikos, E., Costello, H., Holt, G., Bouras, N., Sturmey, P., & Newton, T. (2005). Psychopathology in adults with autism and intellectual disability. *Journal of Autism Intellectual Disability,* 36, 1123-1129.

Tu, J.B., & Zellweger, H. (1965). Blood-serotonin deficiency in Down's syndrome. *Lancet, 2,* 715-716.

Turner, T.H. (1989). Schizophrenia and mental handicap. *Psychological Medicine,* 19(2), 301-314.

Tylenda, B., Beckett, J., & Barrett, R.P. (2007). Assessing mental retardation using standardized intelligence tests. *International Review of Research in Mental Retardation, 34,* 27-54.

Vernon, P. (1979). *Intelligence: Heredity and environment.* San Francisco: Freeman.

Volkmar, F.R., Carter, A., Sparrow, S.S., & Cicchetti, D.V. (1993). Qualifying social development in autism. *Journal of the American Academy of Child and Adolescent* Psychiatry, 32, 627-632.

Volkmar, F.R. & Cohen, D.J. (1991). Co-morbid association of autism and schizophrenia. *American Journal of Psychiatry, 148,* 1705-1707.

Walsh, B.W. (2006). *Treating self-injury: A practical guide.* New York: Guilford Press.

.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine,* 11(1), 115-129.

Wing, L. (1988). *Diagnosis and assessment in autism.* New York: Plenium Press.

Wing, L. (1996). The Autistic Spectrum: A Guide for Parents and Professionals. London: Constable.

Wing, L., & Gould, J. (1979). Severe impairment of social interaction. *Journal of Autism and Developmental Disorders*, 9(1), 11-19.

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

World Health Organization (2004). *International Classification of Diseases (10th ed.)*. Geneva, Switzerland: Author.

Young, R.C., Briggs, J.T., Ziegler, V.E., & Meyer, D.D. (1978). A rating scale for mania. *British Journal of Psychiatry, 133*, 429-435.

VITA

Martin Ancona was born in Irving, Texas. A graduate of University of North Texas in Denton, Texas, he went on to earn his doctorate in clinical psychology at Louisiana State University. He completed his pre-doctoral internship at Wayne State University School of Medicine in Detroit, Michigan.