2009

The assessment of and differences among intellectually disabled adults with Comorbid Autism Spectrum Disorders and epilepsy

Kimberly Robin Michelle Smith
Louisiana State University and Agricultural and Mechanical College, kimberlysmith15@gmail.com

THE ASSESSMENT OF AND DIFFERENCES AMONG INTELLECTUALLY DISABLED ADULTS WITH COMORBID AUTISM SPECTRUM DISORDERS AND EPILEPSY

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
Kimberly Robin Smith
B.A., University of California at Los Angeles, 2001
M.A., Louisiana State University, 2007
August 2009

ACKNOWLEDGEMENTS

I would like to thank my advisor and committee chair, Johnny L. Matson, Ph.D., for his expertise and guidance on this research project and for facilitating professional growth and development in clinical and research areas in psychology. I am especially appreciative to Rochelle Caplan, M.D., for providing me with a foundation in epilepsy research and support of my educational endeavors. I would also like to express gratitude to Wm. Drew Gouvier, Ph.D., Thompson E. Davis, III, Ph.D., Emily M. Elliott, Ph.D., and John R. Battista Ph.D., for their time, assistance, and knowledge as members of my dissertation committee. I would like to thank my colleagues for their help in collecting this data. Without them, this project may not have been possible. In addition, I would like to acknowledge the cooperation of participants and staff in this study in the hope that these results can provide a meaningful step in improving the lives of those with intellectual disabilities. Most of all, I am grateful to my parents and friends whose love, patience, and encouragement helped guide me throughout this process.

TABLE OF CONTENTS

LIST OF TABLES

ABSTRACT

The goal of this study was to systematically examine group differences among those with intellectual disabilities (ID) and comorbid Autism Spectrum Disorders (ASD) and epilepsy in an adult population through a detailed exploration of the characteristics that these disorders present in the areas of psychopathology, behavior problems, and impaired social behavior. Previous studies indicating that individuals with ID have comorbid ASD and neurological conditions tend to stop short of addressing these disorders' impact on the full range of psychosocial issues, particularly in adult samples. Assessments of psychopathology, behavior problems, and impaired social behavior were made with the ASD-Comorbidity-Adult Version and ASD-Behavior Problems-Adult Version batteries and the Matson Evaluation of Social Skills for Individuals with Severe Retardation. One hundred participants with ID were matched and compared across four equal groups comprising 25 participants with ID, 25 participants with epilepsy, 25 participants with ASD, and 25 participants with combined ASD and epilepsy. When controlling for age, gender, race, level of ID, and hearing and visual impairments, significant differences were found for psychopathology ($p < .05$), behavior problems ($p < .05$), and social skills ($p < .01$). A direct discriminant function analysis was also conducted to determine whether certain subscales could predict group membership. Overall, 63% of the participants in the sample could be reliably distinguished between groups on these measures. These data conclusively demonstrated that individuals with ID expressing combined comorbid ASD and epilepsy were significantly more impaired than the control group (ID only) or groups containing only a single comorbid factor with ID (ASD or epilepsy only) on measures of psychopathology, behavior problems, and social skills. Implications of these findings elucidate the nature of these disorders and their influence on patient care and management.

INTRODUCTION

Individuals that reside at state-run facilities represent some of the most difficult management cases faced by modern society. Many individuals who reside at state-run facilities are limited in their ability to care for themselves and require extended care for a single primary condition, but may be burdened with the complexity of multiple comorbidities (e.g., psychopathology, medical conditions, behavior problems, social skills impairment). This study examines in detail the comorbidity problem and applies a modern statistical approach to examine the effects of comorbidity from the perspective of the professional caregiver. In addition, this study offers a basis for applying quantitative metrics, which can be used to enhance an individual's management and may subsequently improve their quality of life.

Intellectual disabilities (ID) and Autism Spectrum Disorders (ASD) are often associated with genetic aberrations and are thus highly comorbid. The presence of genetic abnormalities also increases the likelihood of individuals expressing neurological conditions (Bolton & Griffiths, 1997). Therefore, it should not be surprising that ID, ASD, and epilepsy often present in combination, and that these individuals commonly reside at state-run facilities. However, research specifically examining these combinations in adult populations is extremely sparse despite the fact that comorbidity of these conditions is high and tends to present associated challenges among individuals in the more severe ranges of ID. Some of these challenges may include the presence or amplification of psychopathology, behavior problems, and impaired social skills, especially when epilepsy is present (Borthwick-Duffy, 1994; Bowley & Kerr, 2000; Coulter, 1983; Gabis, Pomeroy, & Andriola, 2005; Kanner, 2002; Lund, 1985; Matson, Bamburg, Mayville, & Khan, 1999; McGrother et al., 2006; Ring, Zia, Lindeman, & Himlok, 2007). Despite this, it is unknown whether individuals with ID, ASD, and epilepsy differ on

psychosocial measures of psychopathology, behavior problems, and impaired social skills, and whether the presence of ASD or epilepsy alone or in combination has a more negative impact on these ratings.

The present study applied selected scaling instruments to adults with ID, ASD, and epilepsy in an effort to provide quantitative details regarding these comorbid factors among adults residing at two state-run facilities. These scaling instruments were chosen because they were designed specifically for adults with more severe levels of ID who typically reside at state-run facilities. A set of working hypotheses predicted that instruments relating to the identification of psychopathology, behavior problems, and impaired social behavior would quantitatively reflect the interaction of comorbidities found in this institutional population in a meaningful way. Implications of these findings in the context of known issues in ID, epilepsy, and ASD, current assessment practices among these populations, and associated challenges among these populations are discussed.

REVIEW OF LITERATURE

Intellectual Disability

Definition and Classification. In the United States, sociopolitical forces have influenced the definition of ID, yet it has been universally documented in all races, genders and socio-demographic strata (Beirne-Smith Patton, & Ittenbach, 1994; Patton & Jones, 1994). Although formal diagnostic criteria for ID may vary, all definitions are operationally defined by difficulties in learning, impairments in social skills, daily functioning, and age of onset (Biasini, Grupe, Huffman, & Bray, 2001; Scheerenberger, 1987).

The official classification of ID was formulated by two organizations, the American Association on Mental Retardation (AAMR, 1992) and the American Psychiatric Association (APA, 2000). The AAMR, which was originally called the American Association on Mental Deficiency, and is now referred to as the American Association on Intellectual and Developmental Disabilities (AAIDD, 2007), defines ID as 'significantly sub-average intellectual functioning, existing concurrently with impairments in adaptive behavior and manifested before age 18' (AAMR, 1992). According to the AAIDD (2007) definition, sub-average intellectual functioning refers to intellectual quotient (IQ) scores below 70 on a test of general intelligence. Assessing a person's intellectual functioning is measured by the IQ (Eysenck, 1962), and is obtained by individually administering a standardized intelligence test (APA, 2000).

Assessment of Intellectual Functioning. In 1905, Alfred Binet developed the first instrument measuring judgment, comprehension, and reasoning (Eysenck, 1962). These measures were considered to encompass the definition of intelligence. The *Binet-Simon Individual Tests of Intelligence* adopted a psychometric approach that delineated degrees of ID and also differentiated between those who were intellectually disabled from those who had

behavior problems (Scheerenberger, 1987). Despite over a century's debate on the nature and exact definition of intelligence (Howard, 1993), intelligence tests have become a practical tool to aid in the diagnosis of ID (Eysenck, 1962). However, classifying severity levels of ID cannot always be made accurately by standardized intelligence tests alone. In some cases, test scores may be invalid or altogether unattainable. Therefore, alternative methods for assessing a person's functioning were developed as a supplement to IQ testing. This included a standardized measure of adaptive functioning, since no intelligence test can fully assess an individual's complete ability (Eysenck, 1962; Patton & Jones, 1994). Accordingly, in 1961, the AAMR revised the definition of ID to include impairments in adaptive functioning in addition to the previous criteria of sub-average intellectual functioning and onset prior to 18 years of age. The age criterion signifies that ID is considered a developmental disorder since age 18 is typically when critical psychosocial development and brain development have occurred.

The AAIDD definition of ID subdivides adaptive functioning into categories of communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work. This definition requires impairment in at least two of these areas (AAIDD, 2007). In an effort to develop a standardized approach to assessing adaptive functioning, the AAMR developed the Adaptive Behavior Scale (ABS) and defined adaptive functioning as behavior that is effective in meeting the natural and social demands in an individual's environment (Nihira, Foster, Shellhaas, & Leland, 1969). This definition takes into consideration that an individual's cultural environment and lifestyle may have a significant effect on their intellectual functioning and development of adaptive behaviors (Scheerenberger, 1987).

The ABS is a unique measure because it assesses social abilities rather than social deficits or inappropriate behavioral excesses (AAMR, 1992). Although the ABS was designed to capture

an individuals' global social ability (Marchetti & Campbell, 1990), critics argue that its clinical utility is limited because the instrument does not aid in identifying deficits and excesses necessary to create, modify, or refine treatment plans (Matson & Hammer, 1996). One of the first widely used measures of adaptive functioning was the Vineland Social Maturity Scale (Doll, 1953), developed to measure social competence in persons with ID or mental illness from birth to adulthood. Sparrow, Balla, and Cecchetti (1984a) developed their own methods to measure adaptive behavior in persons with and without handicaps from birth to adulthood. The Vineland Adaptive Behavior Scales (VABS) has three versions; the Interview Expanded Form (Sparrow, Balla, & Cecchetti, 1984a), the Interview Survey Form (Sparrow et al., 1984b), and the Classroom Edition (Sparrow, Balla, & Cecchetti, 1985) and are the most commonly used instruments to assess adaptive functioning. The authors suggest that the benefit of administering the VABS over the ABS is that the VABS identifies a person's strengths and weaknesses in the domains of Communication, Daily Living Skills, Socialization, and Motor Skills.

However, critics of these two measures argue that there is no empirical support for the divisions proposed by the AAMR or authors of the VABS (Perry & Factor, 1989), or that demonstrating impairments in at least two of these domains is necessary and sufficient for a diagnosis of ID (Zigler, Balla, & Hodapp, 1984). Irrespective of newer adaptive behavior measures (e.g., VAB-II; Sparrow et al. 2005), for persons that may be too impaired to complete intellectual testing, assessing an individual's adaptive ability in a quantifiable manner is important for determining social and occupational competence, and may influence the development, application, and modification of treatment plans (Greenspan, Switzsky, & Granfield, 1996).

The AAIDD played an integral part in the current classification system that defines ID. The American Psychological Association (APA, 2000) integrated portions of the AAIDD definition of ID to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders-Text Revision (DSM-IV-TR; APA, 2000). According to the DSM-IV-TR definition of ID, criteria include: 1) sub-average intellectual functioning, 2) concurrent impairments in adaptive functioning, and 3) onset before the age of 18 years. Sub-average intellectual functioning is defined as an IQ score of approximately 70 or below, which is two standard deviations below the average (APA, 2000). While the APA and AAIDD classification system share similar diagnostic features, the AAIDD definition emphasizes universal rights, service entitlement, social adaptation, and supported living for persons with ID (AAIDD, 2007), whereas the APA definition emphasizes service entitlement and identifies severity levels of ID based on intellectual functioning (Harris, 1995). Individuals with ID represent a heterogeneous group with a wide range of associated impairments. Levels of severity are associated with ranges of IQ scores and are classified into Mild, Moderate, Severe, and Profound (APA, 2000). Levels of severity that are not associated with IQ scores are classified into Severity Unspecified.

Characteristics of Intellectual Disability. Individuals who are classified as having Mild ID represent the majority of those with this disorder (i.e., 85%) and their IQ range is 50-55 to approximately 70 (APA, 2000). Individuals in this range typically experience minimal impairment in social, communication skills, and sensorimotor areas evident during the preschool years (APA, 2000). Impairments in these areas may not be noted until a later age when academic work is more heavily emphasized. During adulthood, these individuals usually live in the community unassisted or live with minimal assistance. These individuals can usually acquire adequate social and vocational skills necessary for employment (Harris, 1995).

Individuals who are classified as having Moderate ID represent the next largest group with this disorder (i.e., 10%) and their IQ range is 35-40 to 50-55 (APA, 2000). Individuals in this range typically experience impairments in social, academic and communication skills and require training in these areas. These individuals may enter the workforce but need supervision, in addition to living in the community with assistance (Harris, 1995).

Individuals who are classified as having Severe ID represent a smaller percentage than those with Moderate ID, with 3% to 4% of the ID population (APA, 2000). These individuals' IQ range is 20-25 to 35-40. Individuals with severe ID have impairments in social, communication, academic, and self-help skills. These individuals may have comorbid handicaps in sensorimotor areas or other areas (APA, 2000) and associated neurological conditions (e.g., seizures, epilepsy). These individuals typically live in the community with supervision, in group homes, or state-run facilities (Harris, 1995).

Individuals who are classified as having Profound ID represent a small percentage of those in the ID population, with 1% to 2% and IQ ranges below 20 or 25 (APA, 2000). These individuals have considerable limitations in social, communication, academic, and self-help skills and need highly structured environments with constant supervision. Since these individuals often have associated neurological conditions and impairments in sensorimotor areas, state-run facilities or community group homes that provide constant supervision are appropriate settings (Harris, 1995).

Lastly, individuals who are classified into the Severity Unspecified category are reserved for cases where ID is presumed but cannot be confirmed with standardized testing (APA, 2000). An IQ score may not be obtainable because the individual is too impaired (e.g., cannot attend to presented stimuli due to physical or sensory disabilities, such as blindness or deafness), may not

understand the directions, or may be non-compliant (Beirne-Smith, Ittenbach, & Patton, 1994; Singh, Oswald, & Ellis, 1998). These individuals should be classified based on the results of an adaptive behavior assessment, previous assessments, and clinical judgment (APA, 2000). These individuals typically reside at state-run facilities or community group homes that provide constant supervision (Harris, 1995).

Etiology of Intellectual Disability. According to the literature in the APA (2000), the etiology of ID can be divided into five categories with the estimated prevalence rates of each factor. They include 1) hereditary factors (5%), 2) early alterations of embryonic development (30%), 3) pregnancy and developmental problems during the perinatal period (10%), 4) general medical conditions acquired during infancy and childhood (5%), and 5) environmental teratogenic factors and other mental disorders (15%-20%).

Hereditary factors (e.g., genetic abnormalities) and developmental errors or teratogenic factors during prenatal, perinatal, and postnatal periods represent the majority of known etiologies of ID (Alexander, 1998; Harris, 1995). Genetic abnormalities may include Tay-Sachs disease, phenylketonuria, Rett's syndrome, fragile-X syndrome, and tuberous sclerosis, and can be definitively diagnosed during pregnancy by genetic testing and chromosome analysis (Bowley & Kerr, 2000; Dykens, Hodapp, & Finucane, 2000; Zaroff et al., 2006). These chromosomal abnormalities are associated with more severe levels of ID and are typically diagnosed during infancy whereas milder levels of ID tend to be diagnosed in early childhood (Alexander, 1998). Early alterations of embryonic development represent the largest group of known etiologies of ID and include causes like Down's syndrome and Angelman's syndrome (Crocker, 1992; Dykens et al., 2000).

Pregnancy and developmental problems during the perinatal and postnatal period such as hypoxia, hydrocephalus, premature birth, and neonatal seizures account for approximately 11% of those with more severe levels of ID (Bernes & Kaplan, 1994). General medical conditions acquired during infancy and childhood typically include head injuries, infections (e.g., meningitis or encephalitis), degenerative disorders, toxic metabolic disorders, and epilepsy (McLaren & Bryson, 1987).

Lastly, environmental teratogenic factors and other mental disorders may include toxins (e.g., lead and mercury exposure), malnutrition, and environmental deprivation (Dykens et al., 2000; Harris, 1995). Despite these five specific etiological categories, the etiology of ID is unknown for approximately 30-40% of individuals with ID (APA, 2000).

Prevalence of Intellectual Disability. Prevalence rates of ID differ depending on the classification system that is used to diagnose the disorder and sampling practices used in research (Baroff, 1991; McLaren & Bryson, 1987; Sheerenberger, 1987). For example, according to Singh et al. (1998), the prevalence of ID is approximately 3% of the general population when the diagnosis is based on IQ alone, whereas King, Hodapp, and Dykens (2005) argue that prevalence of ID appears to be closer to 1% of the general population when impairments in both intellectual and adaptive functioning are considered. Literature in the DSM-IV-TR also estimates that 1% of the general population has ID (APA, 2000). According to the United States National Library of Medicine and National Institutes of Health, prevalence of ID ranges from 1% to 3% of the general population when intellectual and adaptive functioning and age of onset are considered (NLM & NIH; 2007). Based on a review of the available literature, when intellectual functioning, adaptive behavior, and onset of age are applied, the prevalence of ID is most likely within the 1% to 3% range.

Autism Spectrum Disorders

Brief History. In 1943, the debate between the differential diagnosis of autism and ID was born when Leo Kanner described 11 male children ranging in age from 2 ½ years to 11 years old that displayed atypical patterns of behavior. Some of these atypical patterns of behavior included difficulties relating to people, deficits in communicative speech, circumscribed interests in objects, lacking a sense of humor, and insistence upon sameness (Frith, 1991). Kanner claimed that the children he observed did not have deficits in intellectual functioning despite having significant deficits in language, since rote memory was intact and these children had intelligent parents. However, 3 of the 11 children never acquired expressive language and 8 of the 11 children evinced aberrant communication patterns (e.g., immediate and delayed echolalia; Kanner, 1943). Based on the American Association of Mental Deficiency's original definition of ID in 1877, that defined it as a lack of natural development of the mental and moral or social powers, typically accompanied by physical defects (Scheerenberger, 1987), Kanner referred to these 11 children as mute rather than intellectually disabled (Kanner, 1943).

It was not until 1971 that Michael Rutter, an autism researcher, published an article attempting to clarify the practice of differentially diagnosing autism from ID. This was an important aspect of Rutter's work since he recognized that both individuals with autism and ID evinced significant deficits in communication and social skills. Like the importance of differentiating seizures from epilepsy, the ability to differentially diagnosis disorders such as autism from ID accurately has important implications in terms of treatment planning (Parks, 1988). Rutter argued that autism could be differentiated from ID because those with Kanner's autism had significantly more compulsive behavior or insistence upon sameness than those with

ID. Thus, Rutter believed any definition of autism must consider the individuals' intellectual functioning and developmental level (Kuhn & Cahn, 2004).

In addition to differentiating autism from ID, Rutter also attempted to delineate core features of autism and claimed that three features included: 1) impaired social relations, 2) delayed and/or abnormal language development, and 3) compulsive behavior or insistence on sameness (Rutter, 1978a). Rutter also argued that Kanner's autism could be differentiated from schizophrenia because those with Kanner's autism had a significantly higher prevalence of males than females, lacked delusions or hallucinations, and had relatively lower levels of intellectual functioning (Rutter, 1978b). In addition, the course of Kanner's autism was steady and progressive whereas the symptoms of schizophrenia waxed and waned (Rutter, 1978b).

Kanner's description of autistic features was influential in the identification of a new class of disorders. Likewise, Rutter's triad of autistic features was influential in the development of classifying and diagnosing autism and also differentiating the disorder from ID and schizophrenia (Kuhn & Cahn, 2004). Rutter and Kanner's description of autistic features were then integrated and classified into a new category of disorders termed Pervasive Developmental Disorders (PDD) in the DSM-III proposed by the American Psychological Association (APA, 1980) and ICD-10 proposed by the World Health Organization (WHO, 1992).

According to the DSM-III classification system, there were two categories of autism; Infantile and Residual. Criteria for Infantile Autism were met if a pervasive lack of social relationships and language deficits was evident before the age of 30 months (APA, 1980). On the other hand, Residual Autism was reserved for individuals who had met the criteria for Infantile Autism at one point, but no longer exhibited the symptoms (Volkmar, Bregman, Cohen, & Cicchetti, 1988). However, Volkmar, Cicchetti, Bregman, and Cohen (1992a; 1992b) contended

that the criteria for Infantile Autism was too narrowly focused on the "infantile" and "residual" aspects of the disorder and did not account for developmental changes. Thus, revisions to the DSM-III (DSM-III-R; APA, 1987) resulted in the elimination of "infantile" and "residual" in order to reflect the recognition that autistic behavior may be evident at any age or developmental level.

The DSM-III-R criteria for Autism encompassed a broader range of symptomatology but the revision was problematic because the broadening of criteria resulted in higher rates of false positives for PDD (Volkmar et al., 1992b). Therefore, field trials for the DSM-IV (APA, 1994) ensued in order to capture a more sensitive and specific working definition of autism (Volkmar et al., 1994). Another classification system used to classify individuals with PDD is the ICD-10. Like the DSM, the ICD-10 classification system requires the fulfillment of deficits in the areas of social interaction, communication, and restricted, repetitive behaviors and areas of interest. In addition, the ICD-10 states that developmental abnormalities must be present within the first three years of life for a diagnosis to be made.

Since there were two classification systems for diagnosing PDD, the DSM-IV field trials compared the DSM-III and DSM-III-R criteria with the ICD-10 criteria for PDD (WHO, 1992). Results of this comparison led Volkmar and his colleagues (1994) to conclude that the ICD-10 was superior to both the DSM-III and DSM-III-R with regard to the sensitivity and specificity over the age ranges and developmental levels. Though, critics of the ICD-10 argued that the criteria were too detailed because of the numerous items and criteria length.

Current Definition and Classification. The current APA classification system, using the DSM-IV-Text Revision (DSM-IV-TR; APA, 2000), categorizes PDD into Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder (CDD), Asperger's Disorder, and Pervasive

Developmental Disorder Not Otherwise Specified (PDDNOS). These five disorders are all characterized by significant impairments in social interaction usually manifesting first in childhood, and are currently referred to in the literature as Autism Spectrum Disorders (ASD; Lord, 1997).

Autistic Disorder (or autism) comprises three essential features, including qualitative impairment in social interaction, qualitative impairments in communication, and restricted, repetitive, and stereotyped patterns of behavior, interest, and activities prior to the onset of 36 months (APA, 2000). First, qualitative impairments in social interaction are defined by the endorsement of at least two of the following four symptoms: 1) marked impairment in use of multiple nonverbal behaviors, such as facial expression or eye-to-eye gaze; 2) failure to develop peer relationships that are appropriate to the individual's developmental level; 3) lack of spontaneous seeking to share achievements and interests; and, 4) lack of social or emotional reciprocity.

Second, qualitative impairments in communication are defined by the endorsement of at least one of the following four symptoms: 1) delay in, or total lack of, the development of spoken language; 2) in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others; 3) stereotyped and repetitive use of language or idiosyncratic language; and 4) lack of varied, spontaneous, make-believe play or social imitative play appropriate to developmental level. Third, qualitative impairments in restricted, repetitive, and stereotyped patterns of behavior, interest, and activities are defined by the endorsement of at least one or more of the following four symptoms: 1) an encompassing preoccupation with one or more stereotyped and restricted patterns of interest; 2) an apparently inflexible adherence to

specific nonfunctional routines or rituals; 3) the presence of stereotyped and repetitive motor mannerisms; and 4) persistent preoccupation with parts of objects (APA, 2000).

Criteria for autism cannot be met if the qualitative impairments are more consistent with the developmental trajectory of Rett's Disorder or CDD (APA, 2000). Lastly, criteria for PDDNOS is met when there is a severe and pervasive impairment in the development of reciprocal social interaction associated with either verbal or nonverbal communication deficits. This category includes 'atypical autism' in which presentations do not meet the three core criteria for autism because of late age of onset (i.e., symptoms present after 36 months) and atypical symptomatology is not as severe. Individuals with PDDNOS may also show the presence of stereotyped behavior, interests, and activities (APA, 2000), though; such loosely defined diagnostic criteria for PDDNOS underscores some limitations of the DSM (Matson & Boisjoli, 2007b).

Assessment of Autism Spectrum Disorders. The diagnosis of an ASD among individuals with ID is complicated by the presence and severity of ID. However, Ghaziuddin, Tsai, and Ghaziuddin (1992) argue that it is possible to make a diagnosis of autism in the presence of severe ID based on the characteristic pattern of social communication impairment and aberrant social behavior, such as smelling and touching of objects and self-injury. The additional clinical presentation of epilepsy may be complicated (Bowley & Kerr, 2000; Kanner, 2002; Ring et al., 2007) and present further taxonomic difficulties (Einfeld & Aman, 1995). For example, the behavioral manifestation of some epileptic seizures are stereotyped but of short duration. The behavior itself during this ictal period may not be directed, though, to the untrained eye, it may appear as though the individual is engaging in stereotyped behavior, consistent with one of the core features of autism as identified in the DSM-IV-TR (APA, 2000).

Such definitive diagnoses in the area of ASD are difficult to make because autism shares common features with other types of disorders, such as Landau-Kleffner syndrome (LKS; Landau & Kleffner, 1957; Park, 2003). Autism and LKS are similar because language regression occurs in both of these disorders and typically manifests during childhood (Park, 2003). However, Landau-Kleffner syndrome is not a developmental disorder but an acquired aphasia associated with clinical seizures or an epileptiform electroencephalogram (EEG). Language regression is attributed to brain damage in the cortical areas that contribute to language development (Landau & Kleffner, 1957). Early-onset cases of LKS may mimic the classic signs of early regression of autism; however, researchers know little about the effects of epileptic activity on the developing brain (Tuchman & Rapin, 2002) and in older adults. It is clear that based on the complex nature of individuals and heterogeneity within ASD, attempting to differentiate one ASD from another based solely on one feature is nearly impossible (Deisinger, 2001; Filipek et al., 1999; Frith, 2004; Howlin, 2003).

Ideally, the assessment process should include a multi-method approach and should be comprehensive. For adults with ID, this may include a detailed third-party informant (e.g. caregiver) interview, assessment of the individual using evidence-based measures, and multiple natural observations (Deisinger, 2001; Filipek et al., 1999). However, current evidence-based measures demonstrating both reliability and validity almost exclusively focus on the three core features of autism in pediatric populations (Filipek et al., 1999; Lord, 1997; Lord & Luyster, 2006; Matson, Wilkins, & González, 2008).

At present, the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al., 2000), and Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994), are considered the "gold standards" for diagnosing autism during childhood (Filipek et al., 1999,

p. 459). However, some disadvantages of using the ADOS-G and ADI-R include extensive training to administer these measures, length of administration, utilizing trained clinicians experienced with autism, and lack of diagnostic algorithms for other ASD ((Deisinger, 2001). Another commonly used measure for assessing autism is the *Childhood Autism Rating Scale* (CARS; Schopler, Reichler, & Renner, 1988), an instrument designed to differentiate between autism and ID in pediatric populations. However, it does not conform to DSM-IV criteria (Schopler, Reichler, & Renner, 1988), and does not distinguish between autism and other ASD (Filipek et al., 1999). Instead, scoring consists of cutoff scores that denote whether criteria for autism have been met or unmet (Schopler, Reichler, DeVellis, & Daly, 1980).

In adult populations, standardized instruments used to screen and confirm diagnoses of ASD demonstrating both reliability and validity are few in number (Matson & Boisjoli, 2007b; Matson, Wilkins, Smith, & Ancona, 2008; Towbin, 1997). Although, some assessment instruments designed specifically for diagnostic confirmation of ASD are available (Matson, Nebel-Schwalm, & Matson, 2007), such as the Autism Spectrum Disorders-Diagnostic-Adult Version battery (ASD-DA; Matson, Terlonge, & Gonzalez, 2006). The ASD-DA is an informant-based measure designed to aid in the diagnosis of autism and PDDNOS in adults with more severe levels of ID. It has also been designed to differentiate individuals with ASD from those with ID only. Items on the ASD-DA address the core features of autism as identified by the DSM-IV-TR (Matson, Terlonge, & Gonzalez, 2006), as well as items that address body posture, motor coordination, and repetitive and restricted areas of interest. According to a recent study based on a sample of 192 adults with ID residing at a state-run facility in the United States, the average inter-rater reliability was .40 and average test-retest kappa coefficient was .39 (ranging from .31 to .61; Matson, Wilkins, & Gonzalez, 2006).

Robust convergent and divergent validity has also been demonstrated (Matson, Wilkins, Boisjoli, & Smith, 2008), as well as established cut-off scores for differentiating autism, PDDNOS, and ID on the Socialization and Repetitive Behavior domain. No cut-off scores could be established for distinguishing autism versus PDDNOS on the Communication domain (Matson, Boisjoli, González, Smith, & Wilkins, 2007). A diagnosis of autism or PDDNOS is based on a team consensus of psychologists and physicians coupled with behavioral observations and are considered the gold standard for diagnosing ASD for adults among the lowest range of ID.

Assessment instruments that identify ASD and also identify comorbid psychopathology and psychosocial problems such as behavior problems are sorely lacking despite the fact that aggression, irritability, and other symptoms are common among individuals with ASD (Di Martino & Tuchman, 2001). For example, Bradley, Summers, Wood, and Bryson (2004) found that stereotypy, mania, mood, anxiety, schizophrenia, and organic symptoms were elevated among adults with ASD compared to adults without ASD. However, little is known with respect to individuals with combined ASD and ID or combined ASD and epilepsy on measures of psychopathology.

Etiology of Autism Spectrum Disorders. Autism Spectrum Disorders comprise a group of neurodevelopmental disorders whose etiology is predominantly unknown (Klinger et al., 2003; Steffenburg & Gillberg, 1989). However, numerous etiological theories of ASD that have been proposed include psychosocial, immunological, psychogenic, neurobiological, and genetic factors (Matson & Minshawi, 2006). Of these etiological theories, genetic studies present with the most compelling evidence, with numerous chromosomal anomalies that have been associated with autism (Gillberg, 1998).

Data from twin studies have also underscored the role of genetic inheritance in the development of autism (Bailey et al., 1995). For instance, early twin studies found a 95.7% concordance rate of autism among monozygotic twins and a 23.5% concordance rate among dizygotic twins in 40 twin pairs (Ritvo et al., 1985). More recent twin studies have documented a 60% to 90% concordance in monozygotic twins and a 6% concordance of autism in dizygotic twins (Bailey et al., 1995). Researchers conducting sibling studies have also documented a higher concordance rate of ASD ranging from 2% to 6% (Rutter, Silberg, O'Connor, & Simonoff, 1999), as well as the fact that ASD is a disorder that is predominated by males (Baron-Cohen, 2002; Spence, 2004; Wing, 1981b), further implicating the role of genetics.

Evidence from epidemiological research and high recurrence rates of neurological disorders among individuals with ASD and ID suggest a genetic pathogenesis (APA, 2000; Matsumoto et al., 2007; Wyllie, 1993; Zaroff et al., 2006). Although, epilepsy among those with autism is high regardless of intellectual functioning (Malhotra, Chakrabarti, Gupta, Kumar, & Gill, 2004; Rossi, Posar, & Parmeggiani, 2000; Roulet-Perez & Thierry, 2006; Tuchman & Rapin, 2002; Volkmar & Nelson, 1990), the risk of having autism increases when epilepsy is associated with ID (Tuchman & Rapin, 2002). For example, Bolton and Griffiths (1997) found that the risk of having autism and ID was positively correlated with the number of tubers (tumors) in the temporal lobes. Hunt and Dennis (1987) found a similar trend with a sample of 90 children with tuberous sclerosis in which 51% were classified as having autism and ID.

Furthermore, Hara (2007) found that individuals with tuberous sclerosis and fragile-X syndrome often had seizures even before autism symptomatology manifested. Tuchman, Rapin, and Shinnar, (1991) found a similar trend with an estimated 20% of individuals with ID having seizure onset before the age of 36 months. This suggests that the dysfunction accounting for the

signs and symptoms of ASD involves widespread neuronal networks (Asano et al., 2001; Curatolo et al., 2004), and that autism, epilepsy, and affective disorders may share a common neurochemical substrate (Di Martino & Tuchman, 2001; Parmeggiani et al., 2002). However, while genetic studies are compelling and genetic factors have been strongly implicated in the role of ASD (Rutter, 2005), there are no specific biological markers that have been identified at this time (Spence, 2004).

Prevalence of Autism Spectrum Disorders. The current perception is that prevalence of ASD, namely autism, is increasing. According to Wing and Potter (2002) that is because there is a heightened awareness of and concern about the development of the disorder. In addition, prevalence rates vary in the literature given the heterogeneity of development, changes in diagnostic criteria, differences in empirical sampling and methodology, and the development of more sensitive measures of ASD. For example, at one time comorbid rates of autism and ID had been reported to be as high as 75% (Fombonne, 1999), but a critical analysis of Fombonne's methodological practices and assessment of intellectual functioning suggest that it is much more likely that the prevalence of both autism and ID are estimated conservatively at approximately 40% to 50% (Edelson, 2006). Regardless of changing prevalence rates of autism, Rett's Disorder, CDD, Asperger's Disorder, and PDDNOS appear to be significantly less common than autism (Baird et al., 2006; CDC, 2007a, CDC, 2007b). However, autism and PDDNOS are the most frequently associated types of ASD within the adult ID population (Beadle-Brown et al., 2002; Bouras, Holt, Day, & Dosen, 1999; Howlin, Goode, Hutton, & Rutter, 2004).

Seizures and Epilepsy

Classification and Diagnosis. The most widely used classification system to differentiate seizure types and epileptic syndromes is the International Classification of

Epilepsies and Epileptic Syndromes (ICES) proposed by the Commission on Classification and Terminology of the International League Against Epilepsy (ILAE) in 1989 (Kammerman & Wasserman, 2001). According to the ICES, a seizure is defined as a sudden neurological occurrence whereas epilepsy is defined as a chronic neurological condition characterized by recurrent seizures (Gastaut, 1970; 1973). For a first seizure, it is important to distinguish between an isolated event (e.g., high fever, hypoglycemia) and an unprovoked neuronal event that may be the initial manifestation of a recurrent seizure because it influences the course of treatment. For instance, febrile seizures, seizures that occur during alcohol withdrawal or the course of acute trauma, infection, or metabolic illness are not classified as epilepsy and may not be treated with an anti-epileptic drug (AED; Adams, Victor, & Ropper, 1997; Kammerman & Wasserman, 2001).

In the event that the seizures are recurrent, epileptic syndromes are classified according to confirmed, suspected, or unknown etiology. EEG recordings are considered to be the gold standard for detecting seizures and diagnosing epilepsy because EEG patterns may identify the seizure type and also determine the presence of an epileptic focus (Cuthill & Espie, 2005). However, a 24-hour EEG may be necessary to confirm a diagnosis since a shorter duration of recording may produce a false negative (Kammerman & Wasserman, 2001). Thus, additional information such as an individual's behavioral manifestation during an episode, comorbid medical conditions, and response to treatment (ICES, 1989) may aid in classifying and diagnosing seizures and epilepsy (Sundaram, Sadler, Young, & Pillay, 1999).

In the general population, the aforementioned techniques represent the most reliable and valid method of classifying and diagnosing seizures and epilepsy. However, the assessment of epilepsy in adults with ID, especially new-onset cases, can be difficult because complex medical

procedures may be required. Due to the complexity of correctly classifying and diagnosing seizure types among individuals with ID (Bowley & Kerr, 2000), assessment of epilepsy typically includes evaluation from a neurologist and supplemental information of the individual including previous medical, developmental, and family history and clinical observation (Kerr, 2002; Mayville & Matson, 2004).

Seizure Types. Classifications of seizure types are differentiated by the source of the seizure within the brain and include localized (i.e., partial) seizure onset or distributed (i.e., generalized) seizure onset (Wyllie, 1993). Partial seizures are further divided into simple or complex and indicate whether consciousness is lost or retained. Simple partial seizures occur in approximately 15% of individuals with seizures and consciousness is not impaired. Depending on where the seizure activity occurs in the cerebral cortex, symptoms may be motor, cognitive, sensory, autonomic, or affective in nature. Conversely, complex partial seizures occur in approximately 35% of individuals with seizures and consciousness is partially or completely impaired. Clinical presentations are variable but individuals with complex partial seizures usually experience auras, automatisms, post-ictal confusion, or sedation. Individuals with complex partial seizures may also develop depression, anxiety, somatization, obsessive-compulsive behavior, and impaired social skills (Hermann et al., 2000; Goldstein & Harden, 2000; Kanner & Palac, 2000). Deficits in adaptive functioning are also common (Matson et al., 1999; McGrother et al., 2006).

For some individuals who experience partial seizures, seizure activity may then be distributed to other areas of the brain. This phenomenon is called secondary generalization. Distinguishing features of partial seizures with secondary generalization include a period of unresponsiveness and staring, aura, automatisms, and focal motor phenomena preceding a

seizure. Other individuals with partial seizures may not experience secondary generalization (Kwan, & Sander, 2004).

Although complex partial seizures comprise a larger percentage of those with seizures than simple partial seizures, generalized seizures account for the majority of individuals with epilepsy (i.e., 40%) and always involve loss of consciousnesses (Adamset al., 1997). Initial manifestations indicate involvement of both hemispheres, and most commonly impairment of consciousness with bilateral motor involvement. There are six types of generalized seizures (i.e., absence, myoclonic, clonic, tonic, tonic-clonic, atonic), each having their own characteristic signs. Absence seizures occur in 5% of individuals with seizures and are primarily observed in children. Characteristic signs of absence seizures include staring or eye flickering and some minor body movements may occur. Absence seizures are rare among individuals with ID. Myoclonic seizures include symmetric jerking of the extremities. Clonic seizures include rapid, repetitive motor activity, and tonic seizures include rigid posture. Tonic-clonic seizures include a combination of stiffening followed by flexion motions. Tonic-clonic seizures occur in 25% of all individuals with seizures and are the most common type of generalized seizures in adults. Other features of tonic-clonic seizures include labored respiration, cyanosis, incontinence, involuntary tongue biting, and post-ictal confusion, fatigue, and stupor. Atonic seizures include a sudden loss of postural tone (Adams et al., 1997).

Etiology of Epilepsy. Risk factors for epilepsy include a genetic predisposition or neurological abnormalities that may occur during prenatal, perinatal, and postnatal periods of development (Matsumoto, Miyazaki, Hayakawa, Komori, & Nakamura, 2007). Approximately 10% to 25% of epilepsy cases have a clear genetic basis whereas the majority of cases have an unknown etiology. Individuals with unknown etiological classifications, multiple seizure types

(i.e., Lennox-Gastaut), and status epilepticus (seizures lasting more than three minutes) often have a poor prognosis (Berg et al., 2001; Drislane & Schomer, 1994; Marcus, 1993; Rantakallio, von Wendt, & Koivu, 1987). Among adults with more severe ID, etiological classifications are often unknown and are also associated with a poor prognosis (Coulter, 1993; Lennox, 1960; Lombroso, 1983; Yamanouchi et al., 2005).

Prenatal causes of recurrent seizures may include alternations in neuronal and neurotransmitter metabolism, chromosomal abnormalities, and neuropathic changes (Adam et al., 1997). For instance, in individuals with Rett's Disorder, the specific X-linked genetic deficit that is strongly associated with mutations during perinatal development affect postnatal brain growth, and typically show severe ID and motor deficits. These individuals risk developing epilepsy in more than 90% in all cases (Steffenburg, Hagberg, & Hagberg, 2001).

Perinatal causes may include neonatal brain infections, inter-cranial hemorrhage, and anoxic encephalopathy. Lastly, postnatal causes may include congenital disorders, metabolic disorders, brain infections, and head trauma (Matsumoto, et al., 2007; Okumura et al., 2000). It is presumed that neurological deficits occur at higher rates in ID populations "through a common mechanism rather than a direct cause" (Wyllie, 1993, p. 160). For example, epilepsy in Angelman syndrome, Tuberos sclerosis, Fragile-X syndrome, Rett's Disorder, and Down's syndrome are all strongly linked (Bowley & Kerr, 2000).

According to Adams, Victor, and Ropper (1997), probable causes of recurrent seizures can generally be differentiated by age. For instance, probable causes of neonatal seizures may include congenital defects, birth injury, anoxia, and metabolic disorders. In early childhood, probable causes may include high fever, infections, trauma, and cortical dysgenesis, whereas in late adulthood probable causes may include vascular disease, tumor, degenerative disease, and

post-traumatic injury. Similar patterns of idiopathic seizures and epilepsy have been noted by Kammerman & Wasserman (2001), with post-traumatic injury, congenital disorders, and brain tumors as the most common cause of seizures and epilepsy in adolescents and young adults. In the geriatric adult ID population, individuals over 75 years of age are twice as likely to develop new-onset epilepsy (Hauser, Annegers, & Kurtland, 1993; Wyllie, 1993). Remote causes of seizures with no prior history may include anoxia or stroke (Kwan & Sander, 2004).

Prevalence of Epilepsy. Within the United States it is estimated that approximately 3% of the general population will develop epilepsy at some point in their lives (Kwan & Sander, 2004), but epilepsy typically manifests during childhood (Kammerman & Wasserman, 2001). The risk of having a neurological condition is highest among individuals with severe brain dysfunction such as cerebral palsy, and cognitive, motor, and receptive language deficits (Bowley & Kerr, 2000; Cole, 2002; Gabis et al., 2005; McDermott et al., 2005; Steffenburg & Gillberg, 1989), in addition to individuals with autism (Roulet-Perez & Thierry, 2006). Within the epilepsy literature, researchers have established that there is a significant positive correlation between the severity of the ID and the occurrence of epilepsy (Bowley & Kerr, 2000; Coulter, 1983; Heller, 1969; Ieshima & Takeshita, 1988; Kumada et al., 2005). For instance, in a sample of 1,479 children and adults, Forsgren and colleagues (1990) observed that 299 participants from the total sample had ID and epilepsy, with mild (14%), moderate (20.4%), severe (34%), and profound ID (30.8%). Among adults with ID, it has been estimated that prevalence of epilepsy is 22% in community samples (Welsh Office, 1995 as cited in Bowley & Kerr, 2000) with prevalence of epilepsy at approximately 40% to 60% in samples residing at state-run facilities (Mattson, 1996).

Epilepsy and Intellectual Disability. With respect to those with ID and epilepsy, a salient study conducted by Matson et al. (1999) investigated the effects of epilepsy on psychopathology, social and adaptive functioning, and behavior problems in 353 adults with severe and profound ID with and without epilepsy residing at a state-run facility. These researchers found that individuals with epilepsy had significant deficits in social and adaptive functioning but did not have significantly higher rates of psychopathology and behavior problems compared to those without epilepsy. On the contrary, Devinsky (2003) found that individuals with epilepsy had higher rates of depression, anxiety, cognitive disturbance compared to those without epilepsy. Additionally, according to Morgan, Baxter, and Kerr (2003, p.298), "the presence of epilepsy is related to likelihood of institutionalization and may be indicative of other factors, such as behavior problems."

While some researchers have found that mood disorders (e.g., depression, bipolar disorder) and behavior problems among adults with epilepsy are more frequent than among adults without epilepsy (Di Martino & Tuchman, 2001), no studies to date have expanded their research sample to investigate epilepsy and other comorbid disorders such as ASD and affective disorders, and behavior problems, which are common among adults with ID.

Epilepsy and Autism Spectrum Disorders. Although all seizure types are associated with ASD (Rossi et al., 2000), the most common are infantile spasms, severe myoclonic epilepsy, generalized epilepsy with febrile seizures, myoclonic astatic epilepsy, continuous spike-wave sleep, and Lennox-Gastaut syndrome (Peake, Nothgi, & Philip, 2006). Epilepsy occurs at significantly higher rates among individuals in the more impaired range of ID as well as the autistic spectrum (Gabis et al., 2005; Rossi et al., 1995). For example, Mouridsen, Rich, and Isager (1999) suggest that the risk of developing epilepsy may be as high as 70% in

individuals with CDD. Indeed, these researchers found that 76% of individuals with CDD developed epilepsy over an average follow-up period of 23 years. Furthermore, in a combined sample of 106 children and adults with autism and epilepsy, Rossi and colleagues (1995) found that all of the participants had ID. In a more recent study, Hara (2007) observed 130 participants with autism and found that 25% of the sample also had epilepsy and ID, with moderate (20%), severe (22%), and profound ID (56%) respectively.

Depending on whether samples come from the community or state-run facility, the frequency of epilepsy among individuals with autism ranges anywhere from 3% to 40% (Gabis et al., 2005; Rossi et al, 2000; Tuchman & Rapin, 2002). On the other hand, the frequency of epilepsy among individuals with PDDNOS is unknown because research in this specific area is virtually non-existent. Nevertheless, a study conducted by Parmeggiani et al. (2007) compared matched individuals with autism and PDDNOS and found no statistical differences with regard to epilepsy occurrence. Interestingly, these researchers also found that mild ID, neurological and genetic pathogenesis, and cerebral lesions were more common in the PDDNOS group than the autism group.

Dual Diagnosis

Definition and Classification. Currently, dual diagnosis (Borthwick-Duffy, & Eyman, 1990) is a term used to describe individuals with comorbid ID and psychopathology (Sturmey, Fink, & Sevin, 1993). Historically, the majority of mental health professionals widely held that individuals with ID were incapable of developing psychopathology (Reiss, Levitan, & Szyszko, 1982; Sovner & Hurley 1983). Thus, if an individual with ID engaged in behavior problems (e.g., aggression), the presence of the behavior problem was attributed to their intellectual impairment and not to the possible presence of a psychiatric disorder (Doll, 1953; Gardner &

Cole, 1990; Szymanski & Grossman, 1984). Accordingly, the term 'diagnostic overshadowing' was coined to refer to this phenomenon because it was believed that intellectual impairments "overshadowed" psychiatric disorders and thereby prevented the exploration for likely psychopathological symptoms (Reiss, & Szyszko, 1983).

It is now accepted that psychiatric disorders exist among individuals with ID (Gillberg, Persson, Grufman, & Themner, 1986). However, researchers suggest that individuals with ID may be more susceptible to developing psychiatric disorders than individuals without ID (Borthwick-Duffy, 1994; Di Nuovo & Buono, 2007; Rutter et al., 1976; Matson & Barrett, 1993). For example, based on a sample of 78,603 individuals with ID living in various residential settings throughout California, Borthwick-Duffy and Eyman (1990) determined that approximately 10% of the sample was dually diagnosed. This rate has been reportedly higher in institutionalized populations (Costello, 1982; Matson & Sevin, 1994). Based on a sample of adults with ID residing at an institution, 40% of the sample was dually diagnosed (Baumeister, Todd, & Sevin, 1993).

Among a sample of 940 individuals with mild to profound ID, Dudley et al. (1999) found that the most frequent psychiatric diagnosis were schizophrenic affective, organic, impulse, conducted, and adjustment disorders. In this sample, temper tantrums, screaming, threatening others, poor grooming, social withdrawal, and assault were the most commonly reported behavior problems. Interestingly, Dudley et al. (1999) reported that aggression, social withdrawal, and inappropriate behavior were not associated with a diagnosis of ID or level of ID.

According to the literature in the DSM-IV-TR, disorders that are most commonly associated with ID include Attention Deficit-Hyperactivity Disorder, Mood Disorders, ASD, Stereotypic Movement Disorder, and Mental Disorders Due to a General Medical Condition

(APA, 2000). Other psychiatric disorders that are common among individuals with ID include depression (McGillivray & McCabe, 2007; Tsiouris, 2001), schizophrenia, personality disorders (Di Nuovo & Buono, 2007), eating disorders (e.g., pica; Costello, 1982; Dudley et al., 1999; Iverson & Fox, 1989; La Malfa et al. 2007; Matson et al., 1996; Matson & Barrett, 1993). Individuals who are dually diagnosed also have associated behavioral problems, such as agitation, self-injurious behavior (SIB), and aggression (Kanner, 2002).

Within the area of dual diagnosis, Matson and Sevin (1994) suggest that there are four predisposing factors that impact psychiatric disorders that include organic (physiological, biochemical, genetic), behavioral (interactions between an individual and their environment), developmental (sequences of cognitive development), and socio-cultural (individuals' social experience). According to Di Martino and Tuchman (2001), at least 50-60% of patients with epilepsy develop psychiatric symptoms, particularly, depression, anxiety, and psychotic disorders. This suggests that there is evidence of a strong organic component. In addition, individuals with dual diagnosis also have significant impairments in adaptive skills relative to their peers without epilepsy (Espie et al., 1990). The relative risk of an individual with epilepsy developing a psychiatric illness is compounded by multiple factors, including additional brain injury, the type and severity of epilepsy, medication effects, psychosocial factors, and cognitive and temperamental attributes (Manchanda, 2002; March & Rao, 2002).

Assessment of Dual Diagnosis. Traditional standards for assessing psychiatric disorders in the general population often rely on self-report, interviews, and behavioral observations but may be inappropriate among individuals with more severe levels of ID (Myrbakk & von Tetzchner, 2008). For example, Kanner and Palac (2002) found that depression in epilepsy is high, but recognized that their data was based on self-rating scales on personality inventories and

standard psychiatric interviews. Arguably, assessment practices utilizing evidence-based measures should be a central component of service delivery (Matson et al., 2002), though referral, training, and diagnostic assessment practices vary depending on the residential setting (Borthwick-Duffy, 1994; Borthwick-Duffy & Eyman, 1990; Costello, 1982).

A review of the literature revealed that the functional level of the individual also plays a role in diagnostic practices (Myrbakk & vonTetzchner, 2008). In particular, some studies confirmed that individuals with mild ID were more likely to receive a psychiatric diagnosis while individuals with more severe levels of ID were less likely to receive a psychiatric diagnosis (Dudley et al., 1999; Reiss et al., 1982; Reiss, & Szyszko, 1983). In these studies, the presence of severe behavioral disturbances among individuals with more severe levels of ID was documented; however, their behavioral manifestations were not attributed to the presence of a possible psychiatric disorder. According to Holden and Gitlesen (2003), anxiety, depression, and psychosis are significantly more prevalent in individuals with moderate ID than in individuals with severe and profound ID.

Indeed, the clinical expression of psychiatric disorders and behavior problems in the ID population can be difficult to assess because of features associated with more severe levels of ID, such as limited social skills, minimal or delayed verbal communication, potential presence of behavior problems, and environmental factors (Aman, 1991; Di Nuovo & Buono, 2007; Enfield & Aman, 1995; Matson, Minshawi, Gonzalez, & Mayville, 2006; Myrbakk & vonTetzchner, 2008; Rojahn & Tasse, 1996). Additional challenges for clinicians may include differentially diagnosing disorders such as ID and ASD, since these individuals experience impairments in social and communication skills (LaMalfa et al., 2007).

According to Matson et al. (2002), assessing psychopathology in individuals with more severe levels of ID should include direct observation and care-giver report rather than self-report alone because the ability to reliably assess individuals using quantitative measures and observations increases the potential for the identification and treatment of psychiatric disorders (Matson et al., 2002). Some measures specifically designed for screening and/or diagnosing psychopathology among adults with ID include the Reiss Screen for Maladaptive Behavior (RSMB; Reiss, 1988), Psychiatric Assessment Schedule for Adults with Developmental Disabilities Checklist (PAS-ADD; Moss et al., 1998), Diagnostic Assessment for the Severely Handicapped-II (DASH-II; Matson, 1995b), and Assessment for Dual Diagnosis (ADD; Matson & Bamburg, 1998).

Behavior Problems

The most commonly observed behavior problems among individuals with ID include stereotypy, inflicting injury to the self (i.e., SIB), aggression, and disruptive behavior (Farrar-Schneider, 1992; Gardner; 1990; Matson et al., 1999). Myrbakk and vonTetzchner (2008) argue that the identification of behavior problems in the ID population seems to be associated with the functional level of the individual. However, individuals with behavior problems are much more likely to be placed at state-run facilities over community settings (Borthwick-Duffy, Eyman, & White, 1987; Rojahn & Tasse, 1996; Willer & Intagliata, 1982) and thus, have reportedly higher rates.

Stereotypes are one of the core features of autism as identified in the DSM-IV (APA, 2000) but are often observed among individuals with PDDNOS and ID (Militerni et al, 2002; Rojahn & Sisson, 1990; Symons et al., 2005). However, an individual may exhibit instances of unusual repetitive behaviors with epilepsy during an ictal state and it is important to delineate

seizures from stereotypies (Mayville & Matson, 2004). According to Helmstaeder (2001), individuals who experience neurological damage often have impaired executive functioning, working memory, emotional valence, and problems understanding consequences of behavior. Likewise, individuals with ID and ASD tend to share similar attributes. Learning, homeostatic, and biological theories represent potential explanations of why individuals engage in stereotyped behavior and exhibit behavior problems (Noll & Barrett, 2004).

Stereotypies are considered clinically significant when the behavior has the potential to harm himself or herself or someone else; interferes with the individual's potential to learn or work or interact adaptively with the environment; and/or is socially odd or bizarre that inhibits normalization within the community (Noll & Barrett, 2004). Some examples of common stereotyped behaviors include hand flapping, mouthing of objects, jumping up and down, making complex hand-finger movements, repeating phrases or vocalizations (Noll & Barrett, 2004), body rocking, head moving, and jerking (Bowley & Kerr, 2000). Militerni and colleagues (2002) found that the most common stereotyped behaviors among individuals with autism were abnormal blinking, finger movements, axial hyper kinetic movements, and rocking. Individuals with ID in this sample exhibited lip smacking and pouting.

Stereotyped behaviors may also include a repertoire of behaviors that are self-injurious. Researchers have found that the prevalence of both stereotypy and SIB are common among individuals with more severe forms of ID (Dawson, Matson, & Cherry, 1999; Einfeld & Aman, 1995; Matson & Bamburg, 1998; Rojahn et al., 2001). Frequently occurring forms of SIB among ID populations are self-hitting or banging directed at the head or face, and self-biting (Iwata et al., 1994). While it is well established that individuals with ID and autism may engage in stereotypy or SIB, it appears that individuals with autism are more impaired. In a large sample of

children with autism, Baghdadli and colleagues (2003) found that SIB was more common among individuals with more severe forms of autism than individuals with ID alone. In a previous study conducted by Bodfish et al. (2000), these researchers found that individuals with autism had significantly greater severity ratings for compulsions, stereotypy, and SIB than individuals with ID. However, no significant differences between the autism and ID group were found on measures of aggression and restlessness.

Additional aberrant behaviors that have been observed among the ID population in general include hyperactivity and inappropriate verbalizations (Di Nuovo & Buono, 2007; Einfeld & Aman, 1995). Some researchers also claim that the occurrence of these types of behaviors is much more likely among individuals who are dually diagnosed (Friedman et al., 1992; Hill & Furniss, 2006) and have neurological conditions (Coulter, 1983; Deb & Hunter, 1991; Goldstein & Harden, 2000; Kanner, 2002; Noeker, Haverkamp-Krois & Haverkamp, 2005). Other researchers believe these types of aberrant behaviors are non-verbal ways of communicating and exist separately from psychiatric disorders (Newsom & Hovanitz, 2003).

Assessment of Behavior Problems. Typically, the most common method of assessing behavior problems among individuals with ID include direct observation, identifying functions of behavior through a functional assessment interview (Gresham, Watson, & Skinner, 2001) or rating scale (Matson & Vollmer, 1995), conducting an experimental functional analysis (Iwata, Vollmer, & Zarcone, 1990), and administering rating scales based on informant information (Rojahn et al., 2001). However, there may be limited or unavailable resources to conduct an experimental functional analysis or it may be inappropriate altogether for some individuals with ID who engage in challenging behaviors with high frequency or have the potential to harm themselves or others (Sturmey, 1995).

Behavior problems are often measured by excessiveness, and behaviors that are considered excessive are behaviors that are disproportional to the provocation. The presence of behavior problems can be multi-factorial and complex and occur in approximately 50% to 60% of individuals with ID and epilepsy (Kerr, 2002). More specifically, characteristics of the individual (e.g., severity of ID, ASD, language deficits), coupled with behavioral manifestations (e.g., aggression, SIB, disruption), and mental and physical illness contribute to the complexity of the assessment process (Harris, Delmolino, & Glasberg, 1996; Mayville & Matson, 2004; McGrother et al., 2006) and could possibly be misinterpreted (Deb & Hunter, 1991).

Some available instruments designed specifically to assess behavior problems among adults with ID that rely on informant-based ratings include the *Aberrant Behavior Checklist* (ABC; Aman & Singh, 1986), *Behavior Problems Inventory* (BPI-01; Rojahn et al, 2001), and *Questions About Behaviorial Function* (QABF; Matson & Vollmer, 1995). They represent the most psychometrically sound instruments for adults with more severe levels of ID.

Social Skills

Social behaviors (i.e., verbal and nonverbal) are a repertoire of skills that are used to communicate and govern interactions with others (Bellack, 1979). The process of learning social skills is important for developing relationships, coping, conflict resolution, and promoting independence (Guralnick, 1986; Matson & Hammer, 1996). Socially appropriate and inappropriate behaviors are largely determined by society and are therefore culturally subjective in nature. In addition, there is no standard definition of what comprises social skills because they vary depending on a given situation and context (La Greca, Stone, & Bell, 1982). However, Bellack (1983) believed that social skills were observable, measurable, and involved interpersonal behaviors associated with quality of life issues. For example, individuals with adept

social skills are able to recognize social cues and respond appropriately in a given situation or context (Matson & Ollendick, 1988).

In general, some examples of socially appropriate behavior include, shows affection for familiar people, shares without being told to do so, and follows community rules (Matson et al., 1995). Some examples of socially inappropriate behavior include, avoids social and eye contact, does not follow simple instructions, and disrupts activities of others (Matson et al., 1995). Research has consistently shown that behavioral excess (e.g., SIB, aggression) and impairments in social skills are positively correlated with the functional level of the individual, with greatest impairment in those with severe and profound ID (Browder & West, 1991; Duncan et al., 1999).

Assessment of Social Skills. Typically, the most common method of assessing social skills include direct observation, observations of behavior in simulated situations, and behavioral observations during role-play situations (Bellack, 1979; Matson & Hammer, 1996). However, since individuals with ID may have additive handicaps (e.g., hearing, visual impairment) coupled with potential limited or unavailable resources to conduct these types of behavioral observations, checklists and rating scales that rely on informant-based information have been developed for individuals with more severe levels of ID (Browder & West, 1991). This method of assessing social skills is similar to screening and assessing psychopathology and behavior problems because it involves third party informant-based interviewing. Instead of assessing a "normal" range of symptoms, social behaviors are generally measured by a deficit or excess of behavioral repertoires.

Some available instruments designed specifically to assess social skills among adults with ID that rely on informant-based ratings include the VABS (Sparrow, Balla, & Cecchetti, 1984a), ABS (AAMR, 1992), Assessment of Social Competence (ASC; Meyer et al., 1985),

Social Performance Survey Schedule (SPSS; Lowe & Cautela, 1978), and Matson Evaluation of Social Skills for Individuals with sEvere Retardation (MESSIER; Matson, 1995a). However, some of these instruments only capture the mild-to-moderate ID population (Matson et al., 1983), or lack both reliability and validity.

The MESSIER (Matson, 1995a) represents one of the most extensively studied instruments assessing social skills for adults with more severe levels of ID. The MESSIER comprises clinically derived subscales designed to assess positive and negative verbal and non-verbal social behavior among adults with severe-to-profound ID. Internal consistency and inter-rater and test-retest reliability is high (Matson et al., 1999), with demonstrated convergent validity to the Socialization domain of the VABS (Matson, Carlisle, & Bamburg, 1989).

These types of rating scales are expedient, cost-effective, and comprise normative data (Lecavalier & Aman, 2004). Normative data allow clinicians and researchers to measure excesses and impairments with ease within this population (Browder & West, 1991). However, Marchetti & Campbell (1990) argue that informant-based instruments typically lack reliability and rely too heavily on informants, thereby increasing the chances of rater bias. Nevertheless, within the ID population, these type of instruments aid in assessing relevant behavioral aberrations and excesses.

Intellectual Disability and Autism Spectrum Disorders. A review of the literature suggests that adults with ASD and ID share characteristic impairments in social interactions, which are typically accompanied by deficits in communication and a repertoire of behaviors and activities that are restricted in interest (Beadle-Brown et al., 2002; Njardvik et al., 1999). Deficits in social skills are one of the most salient core features within populations residing at state-run facilities and have been observed among individuals with autism, PDDNOS, and ID, respectively

(Njardvik et al., 1999). Psychopathology, behavior problems, and social behavior are all associated with ID, epilepsy, and ASD, and influence each other. For example, Matson et al. (2009) examined behavior problems and social behavior among a large sample of adults with ID versus ASD (i.e., autism and PDDNOS) and found that individuals with ASD exhibited significantly more behavior problems and were more socially impaired than individuals with ID alone. In a previous study, Matson and colleagues (1998) found that endorsed ratings of psychopathological symptoms predicted increases in negative social behaviors (e.g., disturbs others, has temper tantrums) among 846 adults with severe and profound ID who were dually diagnosed). In addition to increased rates of psychopathology and impairment in social behavior, these individuals also evinced behavior problems. Another study conducted by Duncan and colleagues (1999) found similar patterns of social impairments, with a positive correlation between negative social skills (e.g., poor eye contact, holding onto others and not letting go) and increased behavior problems (e.g., aggression, SIB) compared to a control group.

RATIONALE

Previous studies indicating that ID, ASD, and neurological disorders are highly comorbid (Gabis et al., 2005; Kanner, 2002) tend to stop short of addressing these disorders' impact on the full range of psychosocial issues, particularly in adult samples. For instance, researchers often focus on depression and/or anxiety (Kanner & Palac, 2000; McGillivray & McCabe, 2007; Ross & Oliver, 2003) but do not examine other comorbidities such as conduct problems, behavioral excesses, ADHD, and social behavior. Although these behaviors have been researched among those who live in developmental settings, identifying differences in psychopathology, behavior problems, and social skills all together have not been compared to adults with a single disorder and adults with concomitant disorders.

Psychiatric disorders among this population are difficult to assess and may manifest into behavior problems and likely impact social behavior. Behavior problems such as aggression, destruction, and SIB are of primary concern because they can result in serious harm to the individual or to others. Furthermore, other behavior problems like disruptive and stereotyped behavior can cause significant interruptions to routines and can make socialization, integration, and normalization very difficult. Thus, the ability to assess psychopathology, behavior problems, and social behavior among adults with a single or concomitant disorder has important implications for treatment planning, monitoring, modification, and quality of life outcomes.

The purpose of this study was to study in detail comorbidity issues in the area of psychopathology, behavior problems, and social behavior within a specific population of adults residing at two state-run facilities. This was achieved by using multivariate analyses to compare groups of participants with ID alone, ID and epilepsy alone, ID and ASD alone, and finally, a combined group with ID, ASD, and epilepsy, which are disorders commonly found among

individuals residing at state-run residential facilities. Individuals with ID alone were considered as the control group and served as the group to which all other groups were compared. In addition, a direct discriminant function analysis was conducted to confirm whether groups could be correctly classified based on psychiatric disorders, behavior problems, and social behavior. This additional information was useful in determining which subscales could best predict a particular diagnostic population. This study represents an essential step towards illuminating some features of the larger ID population and its impact on comorbid issues while supporting the application of scaling methodology to identify psychiatric symptoms, behavior problems, and impairments in social behavior. Table 1 lists the dependent variables in this study.

Table 1
List of Dependent Measures and their Subscales

ASD-CA Subscales
Anxiety/Repetitive behavior
Conduct problems
Irritability/Behavioral excess
Attention/Hyperactivity
Depressive symptoms
ASD-BPA Subscales
Aggression/Destruction
Stereotypy
Self-injury
Disruptive behavior
MESSIER Subscales
Positive Verbal
Positive Nonverbal
General Positive
Negative Verbal
Negative Nonverbal
General Negative

- Hypothesis 1. Researchers in the field of ID, ASD, and epilepsy recognize that these individuals may express psychopathological symptoms similar to those with mild ID. Specifically, ADHD, negative emotion regulation, major depressive symptoms, anxiety, and mood changes have all been commonly associated with ID (Ross & Oliver, 2003), ASD (Matson & Boisjoli, 2008), and among individuals with epilepsy (Goldstein, & Harden, 2000; Kanner & Palac, 2002; Peake et al., 2006). Some researchers believe that these vulnerable populations are more susceptible to psychopathology than in the general population (Borthwick-Duffy, 1994; Sturmey et al., 1993). However, differences among individuals with ID, ASD, and epilepsy on measures of psychopathology have not been extensively examined, especially with respect to individuals on the lower functioning end of the ID spectrum. Based on a review of available literature on this topic, a working hypothesis was that the control group would have the lowest endorsed ratings on the ASD-CA subscales. Furthermore, since qualitative impairments in restricted, repetitive, and stereotyped patterns of behavior, interest, and activities is a core feature of autism and associated feature of PDDNOS according to the criteria in DSM-IV-TR (APA, 2000), it was also expected that the ASD group would have significantly higher ratings on the Anxiety/Repetitive behavior subscales than the control and epilepsy group. Given the dearth of research in this area, it was unknown whether the epilepsy or ASD alone group would have the highest ratings on the other four subscales. Thus, it was also unknown how subscale scores would differ among those in the combined group. Nevertheless, it was speculated that having additive handicaps would be more negatively impacting. These results will illuminate whether there are differences in clinical symptomatology among these groups and may elucidate the relationship between psychopathology and comorbid disorders. These results will also aid in our

understanding of whether epilepsy or ASD has a more negative impact on measures of psychopathology among lower functioning adults.

- Hypothesis 2. Previous research has shown that aggressive behavior, stereotypies, and SIB are most common among individuals with autism, PDDNOS, and ID respectively (Fee & Mason, 1992; Garner & Cole, 1990; Matson, Dempsey, LoVullo, & Wilkins, 2008; Rojahn & Sisson, 1990). Therefore, as a second working hypothesis, it was expected that the ASD group or combined group would have significantly more endorsed ratings on the Self-Injury subscale. Moreover, researchers have also demonstrated that individuals with epilepsy show more response inhibition, attention problems, irritability, aggression, and disruptive behavior than those without epilepsy (Besag, 2004; Devinsky, 2003; Hara, 2007). Therefore, it is expected that the epilepsy groups will have significantly more endorsed ratings on the Aggression/Destruction and Disruptive Behavior subscales than the ASD or control group. However, studies examining differences in behavior problems among adults with ID and comorbid ASD and epilepsy are sorely lacking. While it was hypothesized that that the control group would have the lowest endorsed ratings on the ASD-BPA subscales, it was unknown whether the presence of epilepsy versus ASD would more negatively influence behavior problem ratings on three of the four subscales. On the contrary, it was hypothesized that the ASD groups would have the highest endorsed rating on the Stereotypy subscale. Again, it was speculated that the combined group would have the highest behavior problem ratings compared to the control group.
- Hypothesis 3. Given that the literature suggests that behavior problems are associated with social skills impairments, and that deficits in socialization are among the three-core features of ASD, it was expected that the two ASD groups (ASD alone and combined) would have significantly more social skills impairments and less positive social behavior than the control or

epilepsy group. However, with respect to the two ASD groups, it was unknown if the presence of epilepsy would have any further significant impact on social behavior among adults that already had a neurodevelopmental disorder.

MATERIALS AND METHODS

Participants and Settings

Participants were residents from two state-run facilities located in the Southeastern region of the United States that provide 24-hour care to approximately 850 individuals with varying levels of ID, ages, genders, and races combined. An on-site licensed psychologist diagnosed all participants with ID based on DSM-IV-TR criteria (APA, 2000). Selection criteria for participants with and without ASD in this study were based on DSM-IV-TR and ICD-10 diagnostic criteria for Autistic Disorder and PDDNOS. Inter-rater agreement was required in order for a classification of ASD to be made. Diagnosis of epilepsy was made by a consulting licensed neurologist who determined classification of seizure type based on the ILAE criteria, clinical description of seizure activity, and available medical information (e.g., family history, age of onset, prior neurological trauma). Individuals who were excluded from participating in this study were those with non-epileptic seizures and no seizure activity for the past two years (ICES, 1989).

An a priori power analysis was conducted to determine the total sample size required for the present study. Using GPower 3 (Faul, Erdfelder, Lang, & Buchner, 2007) when alpha (α) is set at .05 (Cohen, 1965; 1988) with a medium effect size set at .50 and power set at .80 (Chase & Tucker, 1976; Cohen, 1992), it was determined that a total sample of 80 participants was required for a MANOVA with four groups (i.e., $n = 20$). Three hundred and thirty eight individuals with ID were recruited for this study across two centers. These individuals were divided into those with ID ($n = 131$), ASD ($n = 123$), epilepsy ($n = 33$), and combined ASD and epilepsy ($n = 51$). Participants were then matched across the four groups for age (within 10 years), gender, race, and level of ID. After matching practices were applied, the final total

sample included 100 participants divided into four groups. In each of the four groups, there were 25 participants with ID (control), 25 participants with epilepsy, 25 participants with ASD, and 25 participants with ASD and epilepsy (combined).

Among the four groups, participants' ages ranged from 29 to 72 years, with an average age of 48.58 years and standard deviation of 8.46 years. Length of stay at these two recruitment sites were divided into 2-6 months, 7-12 months, 13 months to five years, and more than 5 years. Ninety-five percent of the participants had resided at either of these two locations for more than five years. There were 72 (72%) males and 28 (28%) females with profound (n = 96) and unspecified (n = 4) ID. The majority of the participants were Caucasian (80%) while a smaller sub-sample was African American (20%). The majority of the participants did not have expressive language (74%) compared to those with expressive language (26%). Additionally, 97% of the participants did not have hearing impairments while 3% did have hearing impairments. Visual impairments in 8% of the total sample were also denoted. Demographic information for the final total sample population is presented in Table 2.

Table 2
Demographic Characteristics of Participants (N = 100)

	Group Membership[a]			
	Control n (%)	Epilepsy n (%)	ASD n (%)	Combined n (%)
Age at time of survey (years)				
22-45	10 (40)	10 (40)	10 (40)	10 (40)
46-65	14 (56)	14 (56)	14 (56)	14 (56)
66+	1 (4)	1 (4)	1 (4)	1 (4)

(Table 2 cont.)

Gender				
Male	18 (72)	18 (72)	18 (72)	18 (72)
Female	7 (28)	7 (28)	7 (28)	7 (28)
Race				
Caucasian	20 (80)	20 (80)	20 (80)	20 (80)
African American	5 (20)	5 (20)	5 (20)	5 (20)
Level of Intellectual Disability				
Profound	24 (96)	24 (96)	24 (96)	24 (96)
Unspecified	1 (4)	1 (4)	1 (4)	1 (4)
Verbal Ability				
Yes	8 (32)	7 (28)	7 (28)	4 (16)
No	17 (68)	18 (72)	18 (72)	21 (84)
Hearing Impairment				
Yes	2 (8)	2 (8)	1 (4)	2 (8)
No	23 (92)	23 (92)	24 (92)	23 (92)
Visual Impairment				
Yes	2 (8)	1 (4)	0 (0)	5 (20)
No	23 (92)	24 (92)	25 (100)	20 (80)

[a] $n = 25$ for each group.

The ASD only group comprised 18 participants with Autistic Disorder and 7 with PDDNOS. The combined comorbid ASD and epilepsy group comprised 16 participants with Autistic Disorder and 9 with PDDNOS. In both epilepsy groups (alone and combined), four

seizure types were noted in the sample. Overall, 34 out of 50 participants (i.e., 68%) had partial epilepsy evolving into secondary generalized seizures, with a smaller sub-sample of participants that had other seizure types that included 3 (6%) with partial, 11 (22%) with generalized, and 2 (4%) with Lennox-Gastaut syndrome. In the epilepsy only group, 6 (24%) had generalized, 1 (4%) had partial, 16 (64%) with partial with secondary generalization, and 2 (8%) with Lennox-Gastaut syndrome. In the combined comorbid ASD and epilepsy group, 5 (20%) participants had generalized, 2 (8%) had partial, and 18 (72%) had partial seizures with secondary generalization. These diagnoses were noted during chart review. Additional information regarding the participants' psychotropic medication regimen and Axis I diagnosis (APA, 2000) were also obtained during chart review (See Appendix A).

In order to assure that the four groups did not differ significantly on demographic characteristics, a chi-square statistical test was performed on age, gender, race, level of ID, verbal ability, hearing and visual impairment. A chi-square statistical test was also performed on seizure types across the two epilepsy groups (alone and combined ASD and epilepsy). A one-way ANOVA for age revealed that there were no significant differences across groups for age, $F(3, 96) = 0.14$, $p > .05$. The chi-square statistical test revealed that there were no significant differences across groups on gender, $\chi^2(3, N = 100) = .14$, $p > .05$, race, $\chi^2(3, N = 100) = .00$, $p > .05$, level of ID, $\chi^2(6, N = 100) = 4.00$, $p > .05$, verbal ability, $\chi^2(3, N = 100) = 1.87$, $p > .05$, hearing impairment, $\chi^2(3, N = 100) = .46$, $p > .05$, and visual impairment, $\chi^2(3, N = 100) = 7.61$, $p > .05$. Seizure type was not statistically significant, $\chi^2(3, N = 100) = 2.54$, $p > .05$.

Dependent Measures

Autism Spectrum Disorders-Comorbidity-Adult Version. The Autism Spectrum Disorders-Comorbidity Adult Version battery (ASD-CA; Matson, Terlonge, & Gonzalez, 2006)

was developed based on a review of the literature relating to psychopathology in individuals with ID, ASD, and minimal verbal communication. Initial development of the ASD-CA included 84 items. The following subcategories were clinically derived: Depression, Conduct Disorder, Attention Deficit Hyperactivity Disorder, Tic Disorder, Obsessive Compulsive Disorder, Phobia, and Eating Disorder. Following an item and factor analysis, the ASD-CA yielded the following factors: 1) Anxiety/Repetitive Behaviors, 2) Conduct Problems, 3) Irritability/Behavioral Excesses, 4) Attention, Hyperactivity, Impulsivity, and, 5) Depressive Symptoms (Matson & Boisjoli, 2008).

After an item and factor analyses were analyzed, the ASD-CA was reduced to 37 items (Matson & Boisjoli, 2008). Currently, the ASD-CA is an informant-based instrument designed to assess psychopathology in adults with ID and ASD and is based on DSM-IV-TR and ICD-10 criteria. The informant rates items on a binary scale according to symptom severity. A score of '0' indicates not a problem or impairment and a score of '1' indicate some problem or impairment for a given subcategory. Informants will be instructed to rate each item to the extent that an item had been a recent problem. Any endorsed item is then recorded and tallied to yield a profile score under the respective subcategories. Sample items include trembles or shakes in the presence of specific objects or situations, damages property, tantrums, has difficulty concentrating, and tearful or weepy. According to Matson and Boisjoli (2008), inter-rater reliability ranged from .30 to .77 with an average kappa for all the items of .43, and test-retest reliability with overall average kappa of .59 was found. Internal consistency for the overall scale was .91, with individual item subcategory coefficients ranging from .27 to .59.

Autism Spectrum Disorders-Behavior Problem-Adult Version. The Autism Spectrum Disorders-Behavior Problem-Adult Version battery (ASD-BPA; Matson, Terlonge, & Gonzalez,

2006) was developed and based on a review of the literature relating to behavior problems in individuals with ID, ASD, and minimal verbal communication. Initial development of the instrument included 20 items. After an item and factor analyses were analyzed, the ASD-BPA was reduced to 19 items (Matson & Rivet, 2008). Currently, the ASD-BPA is an informant-based instrument designed to measure behavior problems commonly associated with ASD, with the following four factors: 1) Aggression/Destruction, 2) Stereotypy, 3) Self-Injurious Behavior, and 4) Disruptive Behavior (Matson & Rivet, 2006). The informant rates items on a binary scale according to symptom severity. A score of '0' indicates not a problem or impairment/not at all, and a score of '1' indicates some problem or impairment. A demarcation of 'X' indicates that the item does not apply or the informant does not know the extent that it has been a recent problem. Any endorsed item is then recorded and tallied to yield a profile score under the respective subcategories. Examples of items on the ASD-BPA include aggression toward others, unusual play with objects, harming self by hitting, pinching or scratching, kicking objects, and yelling and shouting at others. According to Matson and Rivet (2008), inter-rater reliability ranged from .41 to .60 with an average kappa of .43, test-retest reliability for individual items ranged from .24 to .81, with an average kappa of .60, internal consistency was very good (KR = .81), and model fit was good, with 90% confidence intervals ranging from .009 to .051.

Matson Evaluation of Social Skills for Individuals with sEvere Retardation. The Matson Evaluation of Social Skills for Individuals with sEvere Retardation (MESSIER; Matson, 1995), as previously discussed, is an informant-based instrument designed specifically to assess positive and negative social skills in individuals with severe and profound ID. The measure comprises the following clinically derived subscales: 1) positive verbal, 2) positive nonverbal, 3) positive general, 4) negative verbal, 5) negative nonverbal, and 6) general negative. Informants

are asked to rate the appropriate frequency of each social behavior using a four-point Likert scale. A score of '0' indicates never, a score of '1' indicates rarely, a score of '2' indicates sometimes, and a score of '3' indicates often. Any endorsed item is then recorded and tallied to yield a profile score under the respective subscales (Matson, 1995a).

Procedure

Data collection for the study was conducted in accordance with the policies of the Human Rights Committee and approval from the Institutional Review Board was obtained prior to data collection. Data was previously gathered for each of the selected participants over a 12-month period by administering the ASD-CA, ASD-BPA, and MESSIER concurrently and according to manual specifications by doctoral-level graduate students. Informants were typically direct care staff members who had worked with the participant for a minimum of six months. During the assessment, the interviewer read the ASD-CA and ASD-BPA items verbatim and MESSIER items in a semi-structured interview format in a quiet area, free from distractions. The informants were asked to rate items based on symptom severity of the participant's observable behavior. Instrument administration took approximately 45 minutes to complete.

Data Analysis

A multivariate analysis using a one-way MANOVA was conducted to determine if there were significant main differences across groups (control, epilepsy, ASD, and combined ASD and epilepsy) on the three scaling instruments, the ASD-CA, ASD-BPA, and MESSIER. Using these data as background, follow-up data analysis was conducted for significant results using separate one-way univariate analysis of variance (ANOVA) to ascertain whether mean subscale scores on the ASD-CA, ASD-BPA, and MESSIER differed significantly across the four groups. After one-way ANOVAs were computed, post-hoc comparisons using Tukey's honestly significantly

difference (HSD) test were applied to further investigate pairs of significantly different means on these measures (Hinkle, Wiersma, & Jurs, 1998; Tabachnick & Fidell, 2007). Finally, a direct discriminant function analysis was performed in order to assess whether the present set of dependent measures could predict group membership and add complementary information to that provided by the multivariate analysis. The dependent measures (ASD-CA, ASD-BPA, MESSIER) served as the independent variable (i.e., predictor variable) while group membership served as the dependent variable (i.e., criterion variable) to investigate whether these set of predictors could be combined to predict group membership reliably.

RESULTS

In order to determine if groups differed on measures of psychopathology, behavior problems, and social skills, the vectors of means were compared for the four groups. When controlling for age, gender, race, level of ID, and visual and hearing impairment using Wilks's criterion (Λ) as the omnibus test statistic, results of the MANOVA revealed a significant overall main effect for group membership, Wilks's $\Lambda = .40$, $F(45, 244) = 1.99$, $p < .01$. The multivariate η^2 based on Wilks's Λ was .27. Therefore, the null hypothesis that there were no significant differences between groups on measures of psychopathology, behavior problems, and social skills was rejected. First, the vectors of means were compared on the ASD-CA. Significant differences were found among the four groups on the ASD-CA, Wilks's $\Lambda = .76$, $F(15, 254) = 1.82$, $p < .05$, $\eta^2 = .09$. A one-way ANOVA was conducted for each of the five subscales of the ASD-CA as follow-up tests to the MANOVA. The ANOVA on the Anxiety/Repetitive Behavior subscale was significant, $F(3, 96) = 2.93$, $p < .05$, $\eta^2 = .08$, in addition to the Irritability/Behavior excess subscale, $F(3, 96) = 4.74$, $p < .01$, $\eta^2 = .13$, Attention/Hyperactivity subscale, $F(3, 96) = 5.18$, $p < .01$, $\eta^2 = .14$, and Depressive Symptoms, $F(3, 96) = 3.73$, $p < .01$, $\eta^2 = .10$. The Conduct Problems subscale, $F(3, 96) = 1.22$, $p > .05$, $\eta^2 = .04$, was non-significant. The means and standard deviations on the ASD-CA for the four groups are presented in Table 3.

Table 3

Mean and Standard Deviation Subscale Scores for the ASD-CA Across Groups (N = 100)

Subscale	Score Range	Group Membership[a]				F	*p*
		Control M (SD)	Epilepsy M (SD)	ASD M (SD)	Combined M (SD)		
Anxiety/Repetitive Behavior	0.00 – 7.00	0.32 (1.22)	0.08 (.40)	0.76 (1.45)	1.24 (2.28)	2.93*	.04

(Table 3 cont.)

Conduct Problems	0.00 – 12.00	1.16 (2.19)	1.40 (1.58)	1.60 (2.45)	2.32 (2.67)	1.22	.31
Irritability/ Behavioral Excess	0.00 – 7.00	0.92 (1.89)	1.60 (2.06)	2.68 (2.21)	2.96 (2.51)	4.74**	.004
Attention/ Hyperactivity	0.00 – 8.00	1.00 (.1.41)	1.56 (1.66)	2.08 (2.06)	2.96 (2.09)	5.18**	.002
Depressive Symptoms	0.00 – 3.00	0.32 (.69)	0.44 (0.71)	0.52 (0.77)	1.04 (1.05)	3.73**	.01

[a] $n = 25$ for each group.
* $p < .05$. ** $p < .01$.

Post-hoc analyses using Tukey's HSD revealed that the combined group had significantly higher scores on the Anxiety/Repetitive behavior subscale in comparison to the epilepsy group ($p < .05$). Mean differences between groups for this subscale are presented in Table 4 below.

Table 4
Mean Differences between Groups on the Anxiety/Repetitive behavior subscale

	Control	Epilepsy	ASD	Combined
Control	--	.24	-.44	-.92
Epilepsy		--	-.68	-1.16*
ASD			--	-.48
Combined				--

* $p < .05$.

On the Irritability/Behavioral excess subscale, the combined ($p < .01$) and ASD group ($p < .05$) had significantly more endorsed ratings than the control group. Mean differences between groups for this subscale are presented in Table 5.

Table 5
Mean Differences between Groups on the Irritability/Behavioral excess subscale

	Control	Epilepsy	ASD	Combined
Control	--	-.68	-1.76*	-2.04**
Epilepsy		--	-1.08	-1.36
ASD			--	-.28
Combined				--

* $p < .05$. ** $p < .01$.

`On the Attention/Hyperactivity subscale, the combined group had significantly higher scores than the epilepsy ($p < .05$) and control group ($p < .01$). Mean differences between groups for this subscale are presented in Table 6.

Table 6
Mean Differences between Groups on the Attention/Hyperactivity subscale

	Control	Epilepsy	ASD	Combined
Control	--	-.56	-1.08	-1.96**
Epilepsy		--	-.52	1.40*
ASD			--	.88
Combined				--

* $p < .05$. ** $p < .01$.

On the Depressive symptoms subscale, the combined group had significantly higher scores than the control group ($p < .01$). The combined group also had higher scores than the epilepsy group ($p < .05$) but did not reach statistical significance. Mean differences between groups for this subscale are presented in Table 7.

Table 7
Mean Differences between Groups on the Depressive symptoms subscale

	Control	Epilepsy	ASD	Combined
Control	--	-.12	-.20	-.72*
Epilepsy		--	-.08	-.60
ASD			--	.52
Combined				--

* $p < .01$.

Second, the vectors of means were compared on the ASD-BPA. Significant differences were found among the four groups on the ASD-BPA, Wilks's $\Lambda = .79$, $F(12, 246) = 1.93$, $p < .05$. The multivariate η^2 based on Wilks's Λ was .08. A one-way ANOVA was conducted for each of the four subscales of the ASD-BPA as follow-up tests to the MANOVA. The ANOVA on the Aggression/Destruction subscale, $F(3, 96) = .79$, $p > .05$, $\eta^2 = .03$, and Stereotypy subscale, $F(3, 96) = 2.62$, $p > .05$, $\eta^2 = .08$, were non-significant while the Self-Injury subscale, $F(3, 96) = 3.34$, $p < .05$, $\eta^2 = .10$, and Disruptive Behavior subscale, $F(3, 96) = 3.38$, $p < .05$, $\eta^2 = .10$, were significant. The means and standard deviations on the ASD-BPA for the four groups are presented in Table 8.

Table 8
Mean and Standard Deviation Subscale Scores for the ASD-BPA Across Groups (N = 100)

Subscale	Score Range	Group Membership[a]				F	*p*
		Control M (SD)	Epilepsy M (SD)	ASD M (SD)	Combined M (SD)		
Aggression/ Destruction	0.00 – 6.00	0.56 (1.36)	0.68 (1.14)	0.76 (1.48)	1.16 (1.79)	.79	.50

(Table 8 cont.)

Stereotypy	0.00 – 3.00	0.52 (.92)	0.68 (.80)	1.24 (1.09)	92 (1.04)	2.62	.06
Self-Injury	0.00 – 2.00	0.08 (.28)	0.20 (0.50)	0.56 (0.87)	0.52 (.77)	3.34*	.02
Disruptive Behavior	0.00 – 4.00	0.44 (.96)	0.20 (.65)	0.72 (1.06)	1.00 (1.04)	3.38*	.02

[a] $n = 25$ for each group.
* $p < .05$.

Post-hoc analyses using Tukey's HSD revealed that the ASD group had significantly more SIB in comparison to the control group ($p < .05$). Mean differences between groups for the Self-injury subscale are presented in Table 9 below.

Table 9
Mean Differences between Groups on the Self-injury subscale

	Control	Epilepsy	ASD	Combined
Control	--	-.12	-.48*	-.44
Epilepsy		--	-.52	-.32
ASD			--	.04
Combined				--

* $p < .05$.

Post-hoc analyses using Tukey's HSD revealed that the combined group had significantly more disruptive behavior in comparison to the epilepsy group ($p < .05$). Mean differences between groups for the Disruptive behavior subscale are presented in Table 10 below.

Table 10
Mean Differences between Groups on the Disruptive behavior subscale

	Control	Epilepsy	ASD	Combined
Control	--	.24	-.28	-.56
Epilepsy		--	-.52	-.80*
ASD			--	.28
Combined				--

* $p < .05$.

Third, the vectors of means were compared on the MESSIER. Significant differences were found among the four groups on the MESSIER, Wilks's $\Lambda = .58$, $F(18, 257) = 3.05$, $p < .01$. The multivariate η^2 based on Wilks's Λ was .17. A one-way ANOVA was conducted for each of the six subscales of the MESSIER as follow-up tests to the MANOVA. The ANOVA on the Positive Verbal subscale was significant, $F(3, 96) = 3.70$, $p < .01$, $\eta^2 = .10$, in addition to the Positive Nonverbal, $F(3, 96) = 8.95$, $p < .01$, $\eta^2 = .22$, General Positive, $F(3, 96) = 7.30$, $p < .01$, $\eta^2 = .19$, Negative Nonverbal subscale, $F(3, 96) = 5.30$, $p < .01$, $\eta^2 = .14$, and General Negative subscale, $F(3, 96) = 3.16$, $p < .05$, $\eta^2 = .09$. No significant between-subjects effects were found for the Negative Verbal subscale, $F(3, 96) = 1.66$, $p > .05$, $\eta^2 = .05$. The means and standard deviations on the MESSIER for the four groups are presented in Table 11.

Table 11
Mean and Standard Deviation Subscale Scores for the MESSIER Across Groups (N=100)

		Group Membership[a]					
Subscale	Score Range	Control M (SD)	Epilepsy M (SD)	ASD M (SD)	Combined M (SD)	F	*p*
Positive Verbal	0.00 – 36.00	5.88 (6.91)	3.80 (5.78)	4.12 (4.38)	1.16 (1.46)	3.70**	.01

(Table 11 cont.)

Positive Nonverbal	0.00 – 49.00	24.48 (9.63)	20.40 (9.87)	17.08 (9.29)	11.96 (6.09)	8.95***	.001
General Positive	0.00 – 64.00	33.48 (18.57)	28.84 (15.72)	23.44 (16.08)	14.16 (9.74)	7.29***	.001
Negative Verbal	0.00 – 12.00	1.92 (2.60)	3.00 (3.27)	3.88 (3.23)	2.84 (3.31)	1.66	.18
Negative Nonverbal	0.00 – 29.00	7.76 (6.27)	8.52 (5.09)	12.48 (6.32)	13.44 (6.75)	5.30**	.002
General Negative	0.00 – 23.00	4.72 (4.76)	6.68 (5.90)	9.68 (6.63)	8.40 (6.73)	3.16*	.03

[a] $n = 25$ for each group.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Post-hoc analyses using Tukey's HSD revealed that the combined group produced significantly less positive verbal behavior in comparison to the control group ($p < .01$). The mean differences between groups for the Positive Verbal subscale are presented in Table 12 below.

Table 12
Mean Differences between Groups on the Positive Verbal subscale

	Control	Epilepsy	ASD	Combined
Control	--	2.08	1.76	4.72*
Epilepsy		--	-.32	2.64
ASD			--	-2.96
Combined				--

* $p < .01$.

On the Positive Nonverbal subscale, the combined ($p < .01$) and ASD ($p < .05$) group

produced significantly less constructive nonverbal behavior in comparison to the control group. In addition, the combined group ($p < .01$) produced significantly less constructive nonverbal behavior in comparison to the epilepsy group. Mean differences between groups for this subscale are presented in Table 13 below.

Table 13
Mean Differences between Groups on the Positive Nonverbal subscale

	Control	Epilepsy	ASD	Combined
Control	--	4.08	7.40*	12.52***
Epilepsy		--	3.32	8.44**
ASD			--	-5.12
Combined				--

* $p < .05$. ** $p < .01$. *** $p < .001$.

On the General Positive subscale, the combined group produced significantly less general positive social behavior in comparison to the epilepsy ($p < .01$) and control ($p < .01$) group. Mean differences between groups for the General Positive subscale are presented in Table 14 below.

Table 14
Mean Differences between Groups on the General Positive subscale

	Control	Epilepsy	ASD	Combined
Control	--	4.64	10.04	19.32***
Epilepsy		--	5.40	14.68**
ASD			--	-9.28
Combined				--

(Table 14 cont.)

$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

On the Negative Nonverbal subscale, the combined group produced significantly less general positive social behavior in comparison with the epilepsy ($p < .05$) and control ($p < .01$) group. The ASD group also produced significantly less general positive social behavior in comparison to the control ($p < .05$) group. Mean differences between groups on this subscale are presented in Table 15 below.

Table15
Mean Differences between Groups on the Negative Nonverbal subscale

	Control	Epilepsy	ASD	Combined
Control	--	-.76	-4.72*	-5.68**
Epilepsy		--	-3.96	4.92*
ASD			--	-.96
Combined				--

$^{*}p < .05$. $^{**}p < .01$.

On the General Negative subscale, the ASD group produced significantly more general negative social behavior in comparison to the control ($p < .05$) group. Mean differences between groups for the General Negative subscale are presented in Table 16 below.

Table16
Mean Differences between Groups on the General Negative subscale

	Control	Epilepsy	ASD	Combined
Control	--	-1.96	-4.96*	-3.68
Epilepsy		--	-3.00	-1.72

(Table 16 cont.)

ASD	--	-1.28
Combined		--

* $p < .05$.

Discriminant Function Analysis. This post-hoc analysis was performed to determine which scales contributed to the systematic variance distinguishing among the four groups. It was expected that this analysis would show which scales were the most sensitive for the population sampled. Using these data a recommendation could then be made to use only the best-performing scales when making decisions relating to participation and care. The overall Wilks's lambda was significant, Wilks's $\Lambda = .40$, χ^2 (45, $N = 100$) = 83.12, $p < .01$, indicating that overall the predictors differentiated among the groups. After partialling out the effects of the first discrminant function, the residual Wilks's lambda was not significant for the second discriminant function, Wilks's $\Lambda = .72$, χ^2 (28, $N = 100$) = 29.87, $p > .05$, and third discriminant function, Wilks's $\Lambda = .89$, χ^2 (13, $N = 100$) = 10.93, $p > .05$ indicating that the predictors of the second and third discriminant functions did not differentiate significantly among the four groups. Therefore, only the first discriminant function was interpreted.

The first discriminant function had an overall canonical correlation of .67 between the response variables and group membership, while the second discriminant function had a canonical correlation of .44 and the third discriminant function had a canonical correlation of .34. In Table 17, 10 out of the 15 scales showed significant correlations between the standardized predictors and group membership for the first discriminant function. Based on these coefficients, the MESSIER Positive Nonverbal subscale score demonstrates the strongest relationship within the first discriminant function. On the other hand, the MESSIER Negative Verbal, ASD-BPA

Stereotypy, and MESSIER General Negative scores showed a negative relationship with the second discriminant function, and the MESSIER Positive Verbal and ASD-BPA Disruptive Behavior showed the strongest relationship with the third discriminant function (See Table 17 below).

Table 17
Standardized Coefficients and Correlations of Predictor Variables with the Three Discriminant Functions

	Standardized Canonical Discriminant Function Coefficients			Correlation Coefficients with Discriminant Function		
Predictors	Function 1	Function 2	Function 3	Function 1	Function 2	Function 3
Positive Nonverbal[a]	-1.05	.38	.50	-.58*	-.02	.19
General Positive[a]	.25	-.40	-.74	-.52*	-.12	.12
Attention/ Hyperactivity[b]	.37	-.07	-.66	.44*	.06	-.13
Negative Nonverbal[a]	.15	.53	.31	.44*	-.08	.23
Irritability/ Behavioral excess[b]	.08	-.12	.06	.42*	-.16	.05
Depressive Symptoms[b]	.11	.47	-.10	.35*	.25	-.16
Self-Injury[c]	.20	-.20	.17	.33*	-.21	.19
Anxiety/Repetitive Behavior[b]	.03	.20	.66	.31*	.17	.25
Conduct problems[b]	-.00	.02	-.43	.21*	.10	-.80
Aggression/ Destruction[c]	-.26	.67	-.29	.16*	.10	-.08

(Table 17 cont.)

Negative Verbal[a]	-.36	-.77	-.42	.14	-.39*	.02
Stereotypy[c]	-.12	-.56	.48	.23	-.35*	.27
General Negative[a]	.57	-.59	-.04	.29	-.35*	.12
Positive Verbal[a]	-.02	.27	.72	-.34	-.12	.38*
Disruptive Behavior[c]	.30	.28	.49	.31	.19	.36*

[a] MESSIER
[b] ASD-CA
[c] ASD-BPA
* Largest absolute correlation between each variable and any discriminant function

The means on the discriminant functions are consistent with this interpretation. The combined group (M = 1.21) had the highest mean while the ASD (M = .41), epilepsy (M = -.53), and control group (M = -1.09) had the lower means on the first discriminant function. On the second discriminant function, the combined group (M = .48) had the highest mean while the control (M = .44), epilepsy (M = -.29), and ASD group (M = -.63) had the lower means. On the third discriminant function, the ASD group (M = .36) had the highest mean while the control (M = .28), combined (M = -.10), and epilepsy group (M = -.53) had the lower means. When the first discriminant function was used to predict group membership, 63% of the participants in the sample could be correctly classified. In order to take into account chance agreement, a kappa coefficient was computed and obtained a value of .51.

DISCUSSION

As predicted, these data conclusively demonstrated that individuals with ID expressing combined comorbid ASD and epilepsy were significantly more impaired than the control group (ID only) or groups containing only a single comorbid factor with ID (ASD or epilepsy only) on measures of psychopathology and behavior problems. Furthermore, based on the data from the vectors of means of the four groups, having additive handicaps increases the likelihood of having diminished and/or impaired social skills. The results of this study suggest that clinical presentations are observable and quantifiable even among those who are severely impaired. In fact, based on the results of the direct discriminant function analysis, the best predictors of group membership were the nonverbal subscales of the MESSIER. These results are not unexpected given that many adults residing at state-run facilities do not have expressive language. In particular, these results suggest that the ASD-CA, ASD-BPA, and MESSIER are useful scaling instruments in identifying issues related to a single primary condition or multiple comorbidities.

Taken as a whole, vectors of mean scores were incrementally poorer for the control, epilepsy, ASD, and combined ASD and epilepsy group respectively on almost all dependent measures. For instance, the data summarized in Tables 4 through 7 decisively show that, as a group, the combined group was rated as significantly more handicapped that the control group (ID only), ASD only group, and epilepsy only group on measures of psychopathological symptoms. It was hypothesized that the ASD groups (alone and combined) would be significantly more impaired than either the control group or epilepsy group alone. This hypothesis was supported based on the ratings of the Irritability/Behavioral Excess and Attention/Hyperactivity subscale, showing that the groups comprising individuals with ASD had a larger effect on subscale ratings than epilepsy. This finding suggests that having additive

handicaps increases the likelihood of having increased rates of psychopathological symptoms. Interestingly, the combined group means were significantly higher than the epilepsy group means on the Anxiety/Repetitive behavior, Attention/Hyperactivity, and Depressive symptoms subscales, underscoring the phenomenon that ASD is a significantly more serious complicating factor in managing mood symptoms among patient care than epilepsy for this sample population. These findings suggest that having a single primary condition or multiple comorbidities is additive among those with aberrant behavior but also interacts among those with mood symptoms.

In regard to behavior problems, it was hypothesized that the ASD groups (alone and combined) would be significantly more impaired than either the epilepsy alone group or control group on the Self-Injury and Stereotypy subscales. This hypothesis was partially supported based on the ratings of the Self-Injury subscale; though, no significant group differences were found on the Stereotypy subscale. For instance, Table 9 shows that the ASD group was significantly more impaired on the Self-Injury subscale than the control group; and that the additional effect of comorbid ASD and epilepsy was negligible. Mean scores on the ASD-BPA subscales also suggest that epilepsy itself is not always associated with the severity of the behavioral disorder like Morgan and colleagues (2003) would suggest.

While it was hypothesized that the epilepsy group would have significantly higher ratings on the Aggression/Destruction and Disruptive Behavior subscales of the ASD-BPA, on the Aggression/Destruction subscale, the variance was high; suggesting possible ambiguity in the way the subscale was applied. On the other hand, this finding may suggest that the comorbidity effect is insignificant with respect to this particular challenging behavior. This trend has been demonstrated in previous research in which non-significant differences between groups of

individuals with autism and ID on measures of aggression or restlessness were found (Bodfish et al., 2000). More evidence is needed to confirm whether behavioral challenges are correlated with psychopathological symptomatology in the ID population.

On the other hand, the Disruptive Behavior subscale (Table 10) shows the somewhat surprising finding that epilepsy contributed more to the disruptive behavior ratings than ASD. This finding is consistent with the literature in that other researchers have documented that individuals with epilepsy showed more response inhibition and disruptive behavior than those without epilepsy (Besag, 2004; Devinsky, 2003; Hara, 2007). One explanation for this phenomenon in the current study is that direct-care staff members consider the manifestation of seizures to have more disruptive potential than commonly occurring behaviors associated among those with ASD.

Although it is important to pinpoint specific behavior problems using standardized screening instruments, identifying environmental events that elicit behavior problems is also an important aspect of the assessment process (Gresham, Beebe-Frankenberger, & MacMillan, 1999). Since many individuals with dual diagnosis exhibit behavior problems across settings, conducting functional assessments designed to identify the maintaining function of a behavior should also be used (Applegate, Matson, & Cherry, 1999; Matson & Minshawi, 2006; Newsom & Hovanitz, 2003). It is important to note that the identification of behavior problems and their function may be difficult, especially when there are multiple maintaining variables (Matson & Boisjoli, 2007a).

Behavior problems can be an issue especially among individuals with ID and autism since a high proportion of these individuals have experienced long-term exposure to drug

treatment for behavioral problems (Aman et al., 1995), which may lead to tardive dyskinesia (involuntary body movements) and can also mimic the signs of stereotypy (Bowley & Kerr, 2000). For example, anti-epileptic drugs such as phenytoin have been known to induce hyperactivity, depression, psychomotor retardation, and aggression. Thus, clinicians should still be cautious and systematically screen for a wide range of observable/overt behaviors with the caveat that they may or may not be diagnostic indicators. Thus, the management of anti-epileptic drugs may need more frequent review among individuals with ID and comorbid epilepsy.

In addition to identifying rates of psychopathology and behavior problems among those with ID, identifying impaired social behavior is also a salient issue within this population. In regard to impaired social behavior, it was hypothesized that the ASD groups (alone and combined) would be significantly more impaired than either the epilepsy alone group or control group on measures of social skills. This hypothesis was supported based on the finding that the four groups differed significantly on all of the subscales of the MESSIER except for the Negative Verbal subscale. This result is not surprising given the preponderance of nonverbal individuals distributed throughout the groups in this study.

Specifically, the Positive Verbal subscale (Table 12) shows that individuals with combined ASD and epilepsy were the most severely impaired but that epilepsy contributed more to the effect than ASD alone. Clearly, both the Positive Verbal and Positive Nonverbal subscales show the combined group to be the most impaired. On the Negative Nonverbal subscale (Table 15), the ASD only group and combined ASD and epilepsy group had significantly more negative social behavior than the epilepsy alone group and control group. These results were expected given that items on this subscale addressed questions regarding isolation, rigidity, eye contact, odd mannerisms, and inappropriate physical contact. Since the ASD alone group and combined

ASD and epilepsy group had impaired nonverbal social skills (e.g., sharing interests, playing, smiling, and communicating using gestures), treatment interventions targeting these areas of behavior may be warranted.

While the MESSIER seems to characterize overall impairment correctly, the sensitivity of the 'General' subscales is asymmetrically biased against the General Negative subscales since only one significant group difference was found (i.e., ASD vs. control). This finding may possibly be attributed to the fact that this subscale had a smaller number of items relative to other MESSIER subscales. However, since the MESSIER is a clinically derived measure with known sensitivity among those with severe-to-profound intellectual impairment, the results of the current study may be considered robust and are consistent with the literature in the DSM-IV-TR (APA, 2000) that impairment in social skills is evident among those with ASD. Critics argue that one of the major issues with using the DSM-IV-TR is that many adults with severe and profound ID have atypical symptoms or clinical presentations (Kanner & Palac, 2002; Ross & Oliver, 2003). However, the results of the present study suggest that the direct-care staff were attuned to the issues of the present sample and that their ratings were sensitive to group differences. This finding is encouraging given the fact that clinical manifestations of can be difficult to assess among individuals with profound ID.

Given that group differences were not found on all subscales of psychopathology, behavior problems, and social skills impairments, the presence of behavior problems or impaired social skills is not necessarily symptomatic of a major psychiatric disorder or the converse among the ID population. Though, researchers argue that challenging behaviors are still largely associated among individuals with more severe intellectual impairments (Mybakk & Tetzchner, 2007; Pawlarcyzk & Beckwith, 1987; Sovner & Hurley, 1983; Tsiouris et al., 2003). Thus,

treatment interventions for individuals with ASD and ID may include functional communication training (Carr & Durand, 1985; Durand, 1990). Behavior modification and communication and social skills training are important elements of service delivery. Thus, it is unlikely that participants in the current study were not exposed to treatment at some point in their lives. These participants may have been receiving some form of treatment (e.g., psychotropic, behavioral, adaptive) prior to this study. Based on some trends of the ASD-CA and ASD-BPA subscales, it is possible that groups did not differ significantly on measures of Conduct Problems and Aggression/Destruction because of prior treatment effects. However, it was not possible to control for the effects of prior treatment, especially since participants were recruited from two state-run facilities and prevalence rates of these disorders are low.

Therefore, an individual's abilities and limitations need to be considered when treatment plans are developed and implemented. These treatment plans also need to be reviewed regularly and modified when appropriate. For example, treatment plans that contain goals and objectives addressing specific behavioral intervention strategies for individuals with dual diagnosis, behavior problems, and impaired social skills may be helpful in training them to learn appropriate behavioral alternatives, increase appropriate social interactions, and facilitate communication. By emphasizing the acquisition of other functional skills in addition to these behavioral intervention strategies, the goal is to decrease the likelihood of behavior problems among these individuals (Odom et al., 2003; Vollmer & Matson, 1995).

In the field of ID, assessment of individuals on the lower end of the ID range has traditionally been proven difficult. It is widely recognized that psychiatric disorders, behavior problems, and impaired social skills occur among individuals with ID; yet the development of and access to appropriate test instruments for this population still represents a challenge for

clinicians, physicians, caregivers, and the health care system in general. For instance, on the ASD-BPA Stereotypy scale, mean differences between the ASD and control group were notably different (M = -.72) but did not have a statistically significant effect (p = .06). The proportion of variance in these linear combinations of dependent variable scores associated with the group factor may have been stronger had there been more items. Since there were only four items comprising the Stereotypy subscale, the ASD-BPA may be insensitive or inappropriate for this population.

Furthermore, to the layman, the clinical manifestation of a seizure can mimic involuntary body movements or stereotypy, but they tend to be shorter in duration than stereotyped patterns of behavior (Bowley & Kerr, 2000). Direct-care staff may lack sufficient training to differentiate such subtleties. However, given the importance of accurate assessment and medical management, educating and training direct-care staff to identify and delineate seizures and/or pre-ictal automatisms from other behavioral manifestations is paramount and may aid these individuals in receiving appropriate care. Nevertheless, the application of informant-based rating scales have provided researchers and clinicians with a standardized means for assessing adults with ID and offers an important area of focus because it yields a simple and practical methodology for identifying and classifying aberrant behavior.

Although the use of indirect measures can yield valuable information for the ID population, definitive psychiatric diagnoses should not be made based on informant-based instruments alone. For example, on the ASD-CA Conduct Problems subscale, it is possible that an item such as 'Destroys others' property' may not have been selected by raters because of staff supervision and lack of opportunity for individuals to engage in this behavior. In addition, the majority of this sample was nonverbal so items such as, 'Talks excessively,' may also not have

been selected by raters. Ambiguous items or items on diagnostic measures that may be difficult for third-party informants to infer should be avoided. When available, it is important to consider the clinical history of the individual in making a diagnosis. It is also of paramount importance to know if family history is significant for psychiatric illness.

Ideally, a mental health clinician should have enough information about baseline behavior and mood levels to systematically observe and quantify departures from baseline. However, according to Ross and Oliver (2003), a major problem with validating psychiatric diagnoses based solely on presentations of behavior problems is that there is often a failure to confirm these using blind evaluations. For example, Matson and colleagues (1998) suggest that the labeling of a behavior problem may be associated more with an instance of a maladaptive behavior rather than the individuals' inclusion into a broader diagnostic group. Generally, however, such classifications may be expected to be helpful in streamlining assessment and treatment techniques if appropriate caution is used. Thus, evaluations of an individual should be comprehensive. In addition, early and accurate differential diagnosis and intervention may have profound impact on quality of life and the rehabilitative process.

There is a great need to have a reliable means of predicting the future emergence of behavior problems using, for example, quantitative estimates of ID and comorbidity with ASD and associated neurological deficits. Since epilepsy and other medical problems are associated with ASD, more research is needed to elucidate the relationship between behavior problems and adaptive skills deficits in this population. However, given that high rates of comborbid disorders and medical conditions exist in this population, diagnostic tools that are sensitive to many uncertainties in prognosis are needed but are not presently available. Unfortunately, as a group, the emotional, behavioral, and adaptive outcomes of individuals with profound cognitive deficits,

and comorbid autism and epilepsy are not favorable (Hara, 2007), given that two of the most important prognostic factors towards independence are cognition and behavior (Besag, 2004). Moreover, the presence of psychopathology, behavior problems, and impaired social behavior may also diminish the likelihood of being placed in a less restrictive environment (e.g., community group home, private residence). Thus, emphasizing the acquisition of appropriate social skills and mitigation of behavior problems for populations residing at state-run facilities may ultimately improve their quality of life by promoting some form of independence.

Since this population sample was non-random, these results cannot be generalized to the entire population of adults with ID. In spite of this, there are a limited number of studies that address these populations and also address the complexities and subtleties between psychopathology, behavior problems, and impaired social skills together. Understanding the psychosocial relationships among individuals residing at state-run residential settings is paramount because ID, ASD, and epilepsy are enduring disorders and may have life-long indirect costs related to the individual's ability to function and maintain a degree of independence (Besag, 2007).

The results of the present study are compelling because it appears that having an ASD has a more detrimental effect on psychosocial variables than having a neurological condition. This can be viewed as providing valuable information about individuals with profound deficits as well providing a model for future studies on different sub-populations. Ideally, a cross-sectional analysis of those residing in state-run facilities and community placements should be conducted in order to extend external validity. In addition, individuals who function within the mild-to-moderate range of ID should also be studied in order to determine if these findings and trends are consistent among those with a higher degree of intellectual functioning.

REFERENCES

Adams, R. D., Victor, M. & Ropper, A. H. (1997). *In principles of neurology,* (6th ed.). New York: McGraw-Hill.

Alexander, D. (1998). Prevention of mental retardation: Four decades of research. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 50-58.

Aman, G. (1991). *Assessing psychopathology and behavior problems in persons with mental retardation: A review of available instruments*. Rockville, M.D.: U.S. Department of Health and Human Services.

Aman, M. G., & Singh, N. N. (1986). *Aberrant Behavior Checklist: Manual*. Slosson Educational Publications, East Aurora, NY.

Aman, M.G., Van Bourgondien, M. E., Wolford, P. L., & Sarphare, G. (1995). Psychotropic and anticonvulsant drugs in subjects with autism: Prevalence and patterns of use. *Journal of the American Academy on Child and Adolescent Psychiatry, 34*, 1672-1681.

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

American Association on Intellectual and Developmental Disabilities (2007). *Intellectual and developmental disabilities: Definition, classification, and systems of support.* Washington, DC: Author.

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

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorder* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, D.C.: Author.

American Psychiatric Association, (2000). *Diagnostic and statistical manual of mental disorders-text revision* (4th ed., revised). Washington, DC: Author.

Applegate, H., Matson, J. M., & Cherry, K. E. (1999). An evaluation of functional variables affecting severe behavior problems in adults with mental retardation by using the Questions About Behavioral Function Scale (QABF). *Research in Developmental Disabilities*, *20*(3), 229-237.

Asano, E., Chugani D. C., Muzik, O., Behen, M., Janisse, J., Rothermel, R. et al. (2001). Autism in tuberous sclerosis complex is related to both cortical and subcortical dysfunction. *Neurology, 57*, 1269-1277.

Bailey, A., LeCouteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine, 25*, 63-77.

Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The special needs and autism project (SNAP). *Lancet, 368,* 210-215.

Baghdadli, A., Pascal, C., Grisli, S., & Aussilloux, C. (2003). Risk factors for self-injurious behaviours among 222 young children with autistic disorders. *Journal of Intellectual Disabilities Research, 47,* 622-627.

Baroff, G. S. (1991). *Developmental disabilities: Psychological aspects*. Austin, TX: Pro-Ed.

Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Science, 6*(6), 248-254.

Baumeister, A. A., Todd, M. E., & Sevin, J. A. (1993). Efficacy and specificity of pharmacological therapies for behavioral disorders in persons with mental retardation. *Clinical Neuropharmacology, 16,* 271–294.

Beadle-Brown, J., Murphy, G., Wing, L., Gould, J., Shah, A., & Holmes, N. (2002). Changes in social impairment for people with intellectual disabilities: A follow-up of the Camberwell Cohort. *Journal of Autism and Developmental Disorders, 32*, 196-206.

Beirne-Smith, M., Patton, J. R., & Ittenbach, R. (1994). Historical Perspectives (4th ed.). *Mental retardation* (pp. 26-53). New York: Macmillan College Publishing Company.

Bellack, A. S. (1979). A critical appraisal of strategies for assessing social skill. *Behavioral Assessment, 1,* 157-176.

Bellack, A. S. (1983). Recurrent problems in the behavioral assessment of social skills. *Behavior Research and Therapy, 21*(1), 29-41.

Berg, A. T., Shinnar, S., Levy, S. R., Testa, F. M., Smith-Rapaport, S., Beckerman, B., et al. (2001). Defining early seizure outcomes in pediatric epilepsy: The good, the bad and the in-between. *Epilepsy Research, 43*(1), 75-84.

Bernes, S. M., & Kaplan, A. M. (1994). Evolution of neonatal seizures. *Pediatric Clinic of North America*, *41*, 1069-1104.

Besag, F. M. C. (2004). Behavioral aspects of pediatric epilepsy syndromes. *Epilepsy & Behavior, 5*, S3-S13.

Biasini, F. J., Grupe, L., Huffman, L., & Bray, N. W. (2001). Comprehensive textbook of child

and adolescent disorders. In S. Netherton, D. Holmes, & C. E. Walker, (Eds.), *Mental retardation: A symptom and a syndrome*. New York: Oxford University Press.

Bodfish, J., Symons, F. J., Parker, D. E. & Lewis, M. H. (2000). Varieties if repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders, 30*, 237-243.

Bolton, P., F., & Griffiths, P. D. (1997). Association of tuberous sclerosis of temporal lobes with autism and atypical autism. *The Lancet, 349*(9049), 392-395.

Borthwick-Duffy, S. A. (1994). Epidemiology and prevalence of psychopathology in people with mental retardation. *Journal of Consulting and Clinical Psychology, 62*(1), 17-27.

Borthwick-Duffy, S. A., & Eyman, R. K. (1990). Who are the dually diagnosed? *American Journal on Mental Retardation, 94*(6), 586-595.

Bowley, C., & Kerr, M. (2000). Epilepsy and intellectual disability. *Journal of Intellectual Disability Research, 44*(5), 529-543.

Bouras, N., Holt, G., Day, K., & Dosen, A. (1999). *Mental health in mental retardation. The ABC for mental health, primary care, and other professionals.* WPA Section of Mental Retardation, London.

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.

Browder, D. M., & West, B. J. (1991). Assessment of social skills and interfering behavior. In D. M. Browder (Ed.), *Assessment of individuals with severe disabilities: An applied behavior approach to life skills assessment* (2nd ed., pp. 245-271). MD: Paul H. Brookes Publishing Co.

Carr, E. G., & Durand, M. (1985). Reducing behavior problems through functional communication training. *Journal of Applied Behavior Analysis, 18,* 111-126.

Centers for Disease Control and Prevention (2007a). Prevalence of autism spectrum disorders and developmental disabilities monitoring 14 sites, United States, 2002. *Morbidity and Mortality Weekly Report, 56,* 12-28.

Centers for Disease Control and Prevention. (2007b). Prevalence of Autism Spectrum Disorders: Autism and Developmental Disabilities Monitoring Network, Six Sites, United States, 2000. *Morbidity and Mortality Weekly Report, 56,* 1-11.

Chase, L. J & Tucker, R. K. (1976). Statistical power: Derivation, development and data-analytic implications. *Psychological Record, 26,* 473-486.

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: Erlbaum.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159.

Cole, A. J. (2002). Evaluation and treatment of epilepsy in multiply handicapped individuals. *Epilepsy & Behavior, 3*(6), 2-6.

Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes (1989). *Epilepsia, 30,* 389-399.

Costello, A. (1982) Assessment and diagnosis of psychopathology. In J. L. Matson, & R. P. Barrett (Eds.), *Psychopathology in the mentally retarded* (pp. 37-52). New York: Grune & Stratton.

Coulter, D. L. (1983). Epilepsy and mental retardation: An overview. *American Journal on Mental Retardation, 98*(11), 1-10.

Crocker, A. C. (1992). Data collection for the evaluation of mental retardation prevention activities: The fatal forty-three. *Mental Retardation, 30,* 303-317.

Curatolo, P., Portfirio, M. C., Manzi, B., & Seri, S. (2004). Autism in tuberous sclerosis. *European Journal of Paediatric Neurology, 8,* 327-332.

Cuthill, F. M., & Espie, C. A. (2005) Sensitivity and specificity of procedures for the differential diagnosis of epileptic and non-epileptic seizures: A systematic review. *Seizure, 14*(5), 293-303.

Dawson, J. E., Matson, J. L., & Cherry, K. E. (1999). An analysis of maladaptive behaviors in persons with autism, PDD-NOS, and mental retardation. *Research in Developmental Disabilities, 19*(5), 439-448.

Deb, S., & Hunter, D. (1991). Psychopathology of people with mental handicap and epilepsy. I. Maladaptive behaviour. *British Journal of Psychiatry, 159***,** 822–826.

Deisinger, J. A. (2001). Diagnosis and assessment of autistic spectrum disorders. In T. Wahlberg, F. E. Obiakor, S. Burkhardt, & A. F. Rotatori (Eds.), *Autistic spectrum disorders: Educational and clinical interventions* (pp. 181-209). London: Elsevier Press.

Devinsky, O. (2003). Psychiatric comorbidty in patients with epilepsy: Implications for diagnosis and treatment. *Epilepsy & Behavior, 4,* S2-S-10.

Di Martino, A., & Tuchman, R. F. (2001). Antiepileptic drugs: Affective use in autism spectrum disorders. *Pediatric Neurology, 25*(3), 199-207.

Di Nuovo, S. F., & Buono, S. (2007). Psychiatric syndromes comborbid with mental retardation: Differences in cognitive and adaptive skills. *Journal of Psychiatric Research, 41*, 795-800.

Doll, E. A. (1953). *The measurement of social competence: A manual for the Vineland social maturity scale.* Washington, DC: Educational Test Bureau.

Drislane, F W., & Schomer, D. L. (1994). Clinical implications of generalized electrographic status epilepticus. *Epilepsy Research, 19*(2), 111-121.

Dudley, J. R., Ahlgrim-Delzell, L., & Calhoun, M. L. (1999). Diverse diagnostic and behavioural patterns amongst people with a dual diagnosis. *Journal of Intellectual Disability Research, 43*(2), 70-79.

Duncan, D., Matson, J. L., Bamburg, J. W., Cherry, K. E., & Buckley, T. (1999). The relationship of self-injurious behavior and aggression to social skills in persons with severe and profound learning disabilities. *Research in Developmental Disabilities, 20,* 441-448.

Durand, V. M. (1990). *Severe behavior problems: A functional communication training approach*. New York, NY: Guilford Publications, Inc.

Dykens, E. M., Hodapp, R. M., & Finucane, B. M. (2000). *Genetics and mental retardation syndromes*. Baltimore: Paul H. Brookes Publishing Company.

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

Einfeld, S. L., & Aman, M. (1995). Issues in the taxonomy of psychopathology in mental retardation. *Journal of Autism and Developmental Disorders, 25*(2), 143-167.

Espie, C. A., Gillies, J. B., & Montgomery, J. M. (1990). Antiepileptic polypharmacy, psychosocial behavior and locus of control orientation among mentally handicapped adults living in the community. *Journal of Mental Deficiency Research, 34,* 351-360.

Eysenck, H. J. (1962). *Know Your I.Q.*. Maryland: Pelican Books.

Farrar-Schneider, D. (1992). *Assessment of individuals with severe disabilities: An applied approach to life skills assessment* (2nd ed.). D. M. Browder (Ed.), Baltimore: Paul H, Brookes Publishing Co.

Faul, F., Erdfelder, E., Lang, A.G., & Buchner, A. (2007). GPOWER 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-179.

Fee, V., & Matson, J. L. (1992). Definition, classification, and taxonomy. In J.K. Luiselli, J. L. Matson, & N. N. Singh (Eds.), *Assessment, analysis and treatment of self-injury*. New York: Springer-Verlag.

Filipek, P. A., Accardo, P. J., Baranek, G. T., Cook, E. H., Jr., Dawson, G., Gordon, B. et al. (1999). The screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders, 29*(6), 439-484.

Fombonne, E. (1999). The epidemiology of autism: A review. *Psychological Medicine, 2*9, 769-786.

Forsgren, L., Edvinsson, S., Blomquist, H. K., Heijbel J., & Sidenvall, R. (1990). Epilepsy in a population of mentally retarded children and adults. *Epilepsy Research, 6*(3), 234-248.

Friedman, L. D., Kastner, T., Plummer, A. T., Ruiz, M. Q., & Henning, D. (1992). Adverse behavioral effects in individuals with mental retardation and mood disorders treated with carbamazepine. *American Journal on Mental Deficiency, 96*, 541-546.

Frith, U. (1991). *Autism and Asperger syndrome*. Cambridge: Cambridge University Press.

Frith, U. (2004). Emanuel miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*(4), 672-686.

Gabis, L., Pomeroy, J., & Andriola, M. R. (2005). Autism and epilepsy: Cause, consequence, comorbidity, or coincidence? *Epilepsy & Behavior, 7*, 652-656.

Gardner, W. I., & Cole, C. L. (1990). Aggression and related conduct difficulties. In J. L. Matson (Ed.), *Handbook of behavior modification with the mentally retarded (*2nd ed., pp.225-248). New York: Plenum Press.

Gastaut, H. (1970). Clinical and electroencephalographic classification of epileptic seizures. *Epilepsia, 11,* 102-113.

Gastaut, H. (1973). *Dictionary of epilepsy.* Part 1: definitions. World Health Organization, Geneva: Author.

Ghaziuddin, M. Tsai, L., & Ghaziuddin, N. (1992). Comorbidity of autistic disorder in children and adolescents. *European Child and Adolescent Psychiatry, 1*, 209-213.

Gillberg, C. (1998). Chromosomal disorders and autism. *Journal of Autism and Developmental Disorders, 28*(5), 415-425.

Gillberg, C., Persson, E., Grufman, M., & Themner, U. (1986). Psychiatric disorders in mildly and severely mentally retarded urban children and adolescents: Epidemiological aspects. *British Journal of Psychiatry, 149,* 68-74.

Goldstein, M. A., & Harden, C. L. (2000). Epilepsy and anxiety. *Epilepsy & Behavior, 1*, 228-234.

Greenspan, S., Switzsky, H. N., & Granfield, J. M. (1996). Everyday intelligence and adaptive behavior: a theoretical framework. In J. W. Jacobson & J. A. Mulick (Eds.), *Manual of diagnosis and professional practice in mental retardation* (pp. 127-135). Washington DC: American Psychological Association.

Gresham, F. M., Beebe-Frankenberger, M. E., & MacMillan, D. L. (1999). A selective review of treatments for children with autism: Description and methodological considerations. *School Psychology Review, 28*(4), 559-575.

Gresham, F. M., Watson, T. S., & Skinner, C. H. (2001). Functional behavioral assessment: Principles, procedures, and future directions. *School Psychology Review, 30,* 156-172.

Guralnick, M. J. (1986). The peer relations of young handicapped and nonhandicapped children. In: P. S. Strain, M. J. Guralnick and H. M. Walker (Eds.), *Children's social behavior: Development, assessment, and modification*. Academic Press Inc: Orlando, FL.

Hara, H. (2007). Autism and epilepsy: A retrospective follow-up study. *Brain & Development, 29*(8), 486-490.

Harris, J. C. (1995). Mental Retardation. *Developmental neuropsychiatry* (pp. 91-126). New York: Oxford University Press.

Harris, S. L., Delmolino. L., & Glasberg, B.A. (1996). Psychological and behavioral assessment in mental retardation. *Child and Adolescent Psychiatric Clinics of North America, 5,* 797-808.

Hauser, W. A., Annegers, J. F., & Kurtland, L. T. (1993). Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota: 1935-1984. *Epilepsia, 34,* 453-467.

Heller, J. H. (1969). Human chromosome abnormalities as related to physical and mental dysfunction. *Journal of Heredity, 60*, 239-248.

Helmstaeder, C. (2001). Behavioral aspects of frontal lobe epilepsy. *Epilepsy & Behavior, 2*(5), 384-395.

Hermann, B. P., Seidenberg, M., Bell, B., Wodard, A., Rutecki, P., & Sheth, R. (2000). Comorbid psychiatric symptoms in temporal lobe epilepsy: Association with chronicity of epilepsy and impart on quality of life. *Epilepsy & Behavior, 1*, 184-190.

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

Hinkle, D. E., Wiersma, W., & Jurs, S. G. (1998). *Applied statistics for the behavioral sciences*. New York: Houghton Mifflin Company.

Holden, B., & Gitlesen, J. P. (2003). Prevalence of psychiatric symptoms in adults with mental retardation and challenging behavior. *Research in Developmental Disabilities, 24,* 323-332.

Howard, R. W. (1993). On what intelligence is. *British Journal of Psychology, 84*(1), 27-37.

Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry, 45,* 212-229.

Hunt, A., & Dennis, J. (1987). Psychiatric disorders among children with tuberous sclerosis. *Developmental Medicine in Child Neurology, 29*, 190-198.

Ieshima, A., & Takeshita, K. (1988). Chromosome abnormalities and epileptic seizures. *Japanese Journal of Human Genetics, 33,* 49-60.

Iverson, J. C., & Fox, R. A. (1989). Prevalence of psychopathology among mentally retarded adults. *Research in Developmental Disabilities ,10*, 77–83.

Iwata, B. A., Pace, G. M., Dorsey, M. F., Zarcone, J. R., Vollmer, T. R., Smith, R. G., et al., (1994). The functions of self-injurious behavior: An experimental-epidemiological analysis. *Journal of Applied Behavior Analysis, 27*, 215-240.

Iwata, B. A., Vollmer, T. R., & Zarcone, J. R. (1990). The experimental (functional) analysis of behavior disorders: Methodology, applications, and limitations. In A. C. Repp & N. N. Singh (Eds.), *Perspectives on the use of nonaversive and aversive interventions for persons with developmental disabilities* (pp. 301-330). Sycamore, IL: Sycamore Publishing Company.

Kammerman, S., & Wasserman, L. (2001). Seizure disorders: Part 1. Classification and diagnosis. *Best Practice, 175*, 99-103.

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

Kanner, A. M. (2002). Psychiatric comorbidity in patients with developmental disorders and epilepsy: a practical approach to its diagnosis and treatment. *Epilepsy & Behavior, 3*, 7-13.

Kanner, A. M.,& Palac, S. (2000). Depression in epilepsy: A common but often unrecognized comorbid malady. *Epilepsy &Behavior, 1*(1), 37-51.

Kerr, M. P. (2002). Behavioral assessment in mentally retarded and developmentally disabled patients with epilepsy. *Epilepsy & Behavior*, *3*(6), 1-17.

King, B. H., Hodapp, R. M., & Dykens, E. M. (2005). Mental retardation. In H. I. Kaplan & B. J. Sadock (Eds.)., *Comprehensive textbook of psychiatry: Vol. 2* (8th ed., pp. 3076-3106). Baltimore: Williams & Wilkins.

Klinger, L. G., Dawson, G., & Renner, P. (2003). Autistic Disorder. In E. J. Mash & R. A. Barkley, (Eds.), *Child psychopathology* (2nd ed., pp. 409-443). New York: The Guilford Press.

Kuhn, R., & Cahn, C. H. (2004). Eugen Bleuler's concepts of psychopathology. *History of Psychiatry, 15*(3), 361-366.

Kwan, P., & Sander, J. W. (2004). The natural history of epilepsy: An epidemiological view, *Journal of Neurology and Neurosurgery Psychiatry, 75*, 1376–1381.

Kumada, T., Ito, M., Miyajima, T., Fujii, T., Okuno, T., Go, T., et al. (2005). Multi-institutional study on the correlation between chromosomal abnormalities and epilepsy. *Brain & Development, 27*(2), 127-134.

La Greca, A. M., Stone, W. L., & Bell, C. R., (1982). Assessing the problematic interpersonal skills of mentally retarded individuals in a vocational setting. *Applied Research in Mental Retardation, 3*(1), 37-53.

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

Landau, W. M., & Kleffner, F. R. (1957). Syndrome of acquired aphasia with convulsive disorder. *Neurology, 7*, 523-530.

Lecavalier, L., & Aman, M. G. (2004). Rating Instruments. In J. L. Matson, R. B. Laud, & M. L. Matson (Eds.), *Behavior modification for persons with developmental disabilities: Treatment and support* (pp. 160-189). NADD Press: Kingston, NY.

Lennox, W. G. (1960). *Epilepsy and related disorders.* Boston: Little, Brown, and Company.

Lombroso, C. T. (1983). Prognosis in neonatal seizures. In: A. V. Delgado-Escueta, C. G. Wasterlain , D. M. Treiman, & R. J. Porter, (Eds.), *Advances in neurology: Status epilepticus* (pp. 101-113). New York: Raven Press.

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

Lord, C., & Luyster, R. (2006). Early diagnosis of children with autism spectrum disorders. *Clinical Neuroscience Research, 6,* 189-194.

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

Lowe, M. & Cautela, J. (1979). A self report measure of social skills. *Behavior Therapy, 9,* 535-544.

Lund, J. (1985). Epilepsy and psychiatric disorder in the mentally retarded adult. *Acta Psychiatrica Scandinavica, 72*, 557–562.

Malhotra, S. Chakrabarti, S. Gupta, N., Kumar, P., & Gill, S. (2004). Pervasive developmental disorders and its subtypes: Sociodemographic and clinical profiles. *German Journal of Psychiatry, 6*, 32-39.

Manchanda, R. (2002). Psychiatric disorders in epilepsy: Clinical aspects. *Epilepsy & Behavior, 3*, 39-45.

March, L., & Rao, V. (2002). Psychiatric complications in patients with epilepsy: A review. *Epilepsy Research, 49*, 11-23.

Marchetti, A. G., & Campbell, V. A. (1990). Social Skills. In J. L. Matson (Ed.), *Handbook of behavior modification with the mentally retarded* (2nd ed., pp. 333-355). New York and London: Plenum Press.

Marcus, J. C. (1993). Control of epilepsy in mentally retarded population: Lack of correlation with iq, neurological status, and electroencephalogram. *American Journal on Mental Retardation*, *98,* 47-51.

Matson, J. L. (1995a). *Manual for the Matson Evaluation of Social Skills for Individuals with Severe Retardation*. Baton Rouge, LA: Scientific Publishers, Inc.

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

Matson, J. L., Baglio, C. S., Smiroldo, B. B., Hamilton, M., & Packlowskyj, T. (1996). Characteristics of autism as assessed by the Diagnostic Assessment for the Severely Handicapped-II (DASH-II). *Research in Developmental Disabilities, 17*(2), 135-143.

Matson, J. L., Bamburg, J. W., Mayville, E. A., & Khan, I. (1999). Seizure disorders in persons with intellectual disability: An analysis of differences in social functioning, adaptive functioning, and maladaptive behaviors. *Journal of Intellectual Disability Research, 43,* 531-539.

Matson, J. L., & Bamburg, J. W. (1998). Reliability of the Assessment of Dual Diagnosis (ADD). *Research in Developmental Disabilities, 19*, 89-95.

Matson, J. L., & Barrett, R. P. (1993). *Psychopathology in the mentally retarded* (2nd ed.). Boston: Allyn and Bacon.

Matson, J. L., & Boisjoli, J. (2007a). Multiple versus single maintaining factors of challenging behaviors as assessed by the QABF for adults with intellectual disability (ID). *Journal of Intellectual and Developmental Disability*, *32*, 39-44.

Matson, J. L., & Boisjoli, J. (2007b) Differential diagnosis of PDDNOS in children. *Research in Autism Spectrum Disorders*, *1*(1), 75-84.

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

Matson, J.L., Boisjoli, J.A., González, M.L., Smith, K.R. & Wilkins, J. (2007). Norms and cut-off scores for the Autism Spectrum Disorders Diagnosis for Adults with Intellectual Disability. *Research in Autism Spectrum Disorders, 1*(4), 330-338.

Matson, J. L., Carlisle, C. B., & Bamburg, J. W. (1998). The convergent validity of the Matson Evaluation of Social Skills for Individuals with Severe Retardation (MESSIER). *Research in Developmental Disabilities*, *19*(6), 493-500.

Matson, J. L., Dempsey, T., LoVullo, S. V., & Wilkins, J. (2008). The effects of intellectual functioning on the range of core symptoms of autism spectrum disorders. *Research in Developmental Disabilities*, *29*(4), 341-350.

Matson, J. L., Fodstad, J. C., & Rivet, T. T. (2009). The relationship of social skills and problem behaviors in adults with intellectual disability and autism or PDD-NOS. *Research in Autism Spectrum Disorders, 3*(1), 258-268.

Matson, J. L., & Hammer, D. (1996). Assessment of social functioning. In: J. W. Jacobson & A. Mulick (Eds.), *Manual of diagnosis and professional practice in mental retardation*. American Psychological Association, Washington, DC.

Matson, J.L., Helsel, W.J., Bellack, A.S., & Senatore, V. (1983). Development of a rating scale to assess social skill deficits in mentally retarded adults. *Applied Research in Mental Retardation*, *4*, 399-408.

Matson, J. L., Kuhn, D. E., & Mayville, S. B. (2002). Assessment of psychopathology and behavior problems for adults with mental retardation. *The National Association of the Dually Diagnosed Bulletin, 5*, 19-21.

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

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

Matson, J. L., Minshawi, N. F, Gonzalez, M. L., & Mayville, S. B. (2006). The relationship of comorbid behavior problems to social skills in persons with profound mental retardation. *Behavior Modification, 30*, 507-525.

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

Matson, J.L., & Ollendick, T.H. (1988). *Enhancing children's social skills: Assessment and training.* New York: Pergamon Press.

Matson, J. L., & Rivet, T. T. (2008). Reliability and factor structure of the autism spectrum disorders-behavior problems for Adults (ASD-BPA) with intellectual disabilities and autism. *Journal of Mental Health Research in Intellectual Disabilities, 1*(1), 34-47.

Matson, J.L. & Sevin, J.A. (1994). Theories of dual diagnosis in mental retardation. *Journal of Consulting and Clinical Psychology, 62*, 6-16.

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

Matson, J. L., Terlonge, C., & Gonzalez, M. L. (2006). *Autism Spectrum Disorders-Behavior Problem-Adult Version*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J. L., Terlonge, C., & Gonzalez, M. L. (2006). *Autism Spectrum Disorders-Comorbidity- Adult Version*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J. L., Terlonge, C., & Gonzalez, M. L. (2006). *Autism Spectrum Disorders-Diagnostic-Adult Version*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J. L., & Vollmer, T. R. (1995). *Questions about behavioral functions (QABF)*. Baton Rouge, LA: Scientific Publishers Inc.

Matson, J.L., Wilkins, J., & Ancona, M. (2009). Autism in adults with severe intellectual disability: An empirical study of symptom presentation. *Journal of Intellectual and Developmental Disability, 33*(1), 36-42.

Matson, J. L., Wilkins, J., Boisjoli, J. A., & Smith, K. R. (2008).The validity of the Autism Spectrum Disorders-Diagnosis for intellectually disabled adults (ASD-DA). *Research in*

Developmental Disabilities, 29(6), 537-546.

Matson, J. L., Wilkins, J., & González, M. (2008). Early identification and diagnosis in autism spectrum disorders in young children and infants: How early is too early? *Research in Autism Spectrum Disorders*, *2*(1), 75-84.

Matson, J.L., Wilkins, J., Smith, K., & Ancona, M. (2008). PDD-NOS symptoms in adults with intellectual disability: Toward an empirically oriented diagnostic model. *Journal of Autism and Developmental Disorders, 38* (3), 530-537.

Matsumoto, A., Miyazaki, S., Hayakawa, C., Komori, T., & Nakamura, M., (2007). Epilepsy in severe motor and intellectual disabilities syndrome (SMIDS): A clinical and electroencephalographic study of epileptic syndromes. *Epilepsy Research, 77*(2-3), 120-127.

Mattson, R. H. (1996). The role of the old and the new antiepileptic drugs in special populations: mental and multiple handicaps. *Epilepsia, 37*, 45-53.

Mayville, E. A., & Matson, J. L. (2004). Assessment of seizures and related symptomatology in persons with mental retardation. *Behavior Modification, 28*(5), 679-693.

McDermott, S., Moran, R., & Platt, T., Wood, H., Issac, T., & Dasari, S. (2005). Prevalence of epilepsy in adults with mental retardation and related disabilities in primary care. *American Journal of Mental Retardation, 110*, 48-56.

McGillivray, J. A., & McCabe, M. P. (2007). Early detection of depression and associated risk factors in adults with mild/moderate intellectual disability. *Research in Developmental Disabilities, 28*, 59-70.

McGrother, C. W., Bhaumik, S., Thorp, C. F., Hauck, A., Branford, D., & Watson, J. M. (2006). Epilepsy in adults with intellectual disabilities: Prevalence, associations and service implications. *Seizure*, *15*(6), 376-338.

McLaren, J., & Bryson, S. E. (1987). Review of recent epidemiological studies in mental retardation: prevalence, associated disorders, and etiology. *American Journal of Mental Retardation, 92,* 243-254.

Meyer, L. H., Reiche, J., McQuarter, R., Cole, D., Vandercook, T., Evans, I., et al. (1985). *Assessment of social competence (ASC): A scale of social competence functions (revised).* Minneapolis: University of Minnesota Consortium Institute.

Militerni, R., Bravaccio, C., Falco, C., Fico, C., & Palermo, M. T. (2002). Repetitive behavior in autistic disorder. *European Child and Adolescent Psychiatry, 38*, 247-251.

Morgan, C. L., Baxter, H., & Kerr, M. P. (2003). Prevalence of epilepsy and associated health service utilization and mortality among patients with intellectual disability. *American Journal of Mental Retardation, 108*(5), 292-300.

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

Mouridsen, S. E., Rich, B., & Isager, T. (1999). Epilepsy in disintegrative psychosis and infantile autism: a long-term validation study. *Developmental Medicine in Child Neurology, 41*, 110-114.

Myrbakk, E., & vonTetzchner, S. (2008). Psychiatric disorders and behavior problems in people with intellectual disability. *Research in Developmental Disabilities, 29*(4), 316-332.

Newsom, C., & Hovanitz, C. A. (2003). Autistic spectrum disorders. In E. J. Mash & R. A. Barkley, (Eds.), *Treatment of childhood disorders* (3rd ed., pp. 455-491). New York: The Guilford Press.

Nihira, K., Foster, R., Shellhaas, M., & Leland, H. (1969). *AAMD Adaptive Behavior Scale.* Washington, DC: American Association on Mental Deficiency.

Noeker, M., Haverkamp-Krois, A., & Haverkamp, F. (2005). Development of mental health dysfunction in childhood epilepsy. *Brain Development, 27*(1), 5-16.

Noll, L. M., & Barrett, R. P. (2004). Stereotyped acts. In J. L. Matson, R. B. Laud, & M. L. Matson (Eds.), *Behavior Modification for Persons with Developmental Disabilities: Treatments and Supports: Vol. 1,* (pp.219-278). New York: National Association for the Dually Diagnosed.

Njardvik, U., Matson, J. L., & Cherry, K. E. (1999) A comparison of social skill in adults with autistic disorder, pervasive developmental disorder not otherwise specified, and mental retardation. *Journal of Autism and Developmental Disorders*, 29, 287–295.

Odom, S. L., Brown, W. H., Frey, T., Karasu, N., Smith-Canter, L. L., & Strain, P. S. (2003). Evidence-based practices for young children with autism: Contributions for single-subject design research. *Focus on Autism and Other Developmental Disabilities, 18(*3), 166-175.

Okumura, A., Watanabe, K., Negoro, T., Ishiguro, Y., Miura, K., Matsumoto, A., et al. (2000). MRI findings in patients with symptomatic localization-related epilepsies beginning in infancy and early childhood. *Seizure, 9*(8), 566-571.

Park, Y. D. (2003). The effects of vagus nerve stimulation therapy on patients with intractable seizures and either Landau-Kleffner syndrome and autism. *Epilepsy & Behavior, 4*, 286-290.

Parks, S. L. (1988). Psychometric instruments available for the assessment of autism. In E. Schopler & G.B. Mesibov (Eds.), *Diagnosis and assessment in autism* (pp. 123-136). New York: Plenum Press.

Parmeggiani, A., Posar, A., Antolini, C., Scaduto, M. C., Santucci, M., Giovanardi-Rossi, P. (2007). Epilepsy in patients with pervasive developmental disorder not otherwise specified. *Journal of Child Neurology, 22*(10), 1198-1203.

Patton, J. R., & Jones, E. (1994). Definitional Perspectives. *Mental retardation* (4th ed., pp. 26-53). New York: MacMillan College Publishing Company.

Pawlarczyk, D. & Beckwith, B. E. (1987). Depressive symptoms displayed by persons with mental retardation: A review. *Mental Disease,* 179**,** 609–613.

Peake, D., Nothgi, L. M., & Philip, S. (2006). Management of epilepsy in children with autism. *Current Paediatrics, 16,* 489-494.

Perry, A., & Factor, D. C. (1989). Psychometric validity and clinical usefulness of the Vineland Adaptive Behavior Scale and the AAMD Adaptive Behavior Scale for an autistic sample. *Journal of Autism and Developmental Disorders, 19*(1), 41-55.

Rantakallio, P., von Wendt, L., & Koivu, M. (1987). Prognosis of perinatal brain damage: A prospective study of a one year birth cohort of 12000 children. *Early Human Development, 15*(2), 75-84.

Reiss, S. (1988). *The Reiss Screen for Maladaptive Behavior test manual.* Worthington, OH: IDS Publishing Corporation.

Reiss, S., Levitan, G. W., & Szyszko, J. (1982). Emotional disturbance and mental retardation: Diagnostic overshadowing. *American Journal on Mental Deficiency 86*, 567–574.

Reiss, S., & Szyszko, J. (1983). Diagnostic overshadowing and professional experience with mentally retarded persons. *American Journal of Mental Deficiency,* 87, 396–402.

Ring, H., Zia, A., Lindeman, S., & Himlok, K. (2007). Interactions between seizure frequency, psychopathology and severity of intellectual disability in a population with epilepsy and a learning disability. *Epilepsy & Behavior, 11*, 92-97.

Ritvo, E. R., Freeman, B. J., Mason-Brothers, A., Mo, A., & Ritvo, A. M. (1985). Evidence for autosomal recessive inheritance in 46 families with multiple incidence of autism. *American Journal of Psychiatry, 142,* 187-192.

Rojahn, J. Matson, J. L., Lott, D. J., Esbensen, A. J., & Smalls, Y. (2001). The Behavior Problems Inventory: An instrument for the assessment of self-injury, stereotyped behavior and aggression/destruction in individuals with developmental disabilities. *Journal of Autism and Developmental Disorders, 31*, 577–588.

Rojahn, J., & Sisson, L. A. (1990). Stereotyped behavior. In: J. L. Matson, Editor, *Handbook of behavior modification with the mentally retarded* (2nd ed., pp. 181-213). Plenum Press, New York.

Rojahn, J., & Tasse, M. J. (1996). Psychopathology in mental retardation. In J. W. Jacobson & J. A. Mulick (Eds.), *Manual of diagnosis and professional practice in mental retardation* (pp. 147-156). American Psychological Association, Washington, DC.

Ross, E., & Oliver, C. (2003). The assessment of mood in adults who have severe or profound mental retardation. *Clinical Psychology Review, 23*, 225-245.

Rossi, P. G., Posar, A., & Parmeggiani, A. (2000). Epilepsy in adolescents and young adults with autistic disorder. *Brain & Development, 22*, 102-106.

Roulet-Perez, E., & Thierry, D. (2006). Autism, epilepsy and EEG epileptiform activity. In R. Tuchman & I. Rapin (Eds.), *Autism: A neurological disorder of early brain development-international review of child neurology series* (pp. 174-188). London, England: Mac Keith Press.

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

Rutter, M. (1978b). Diagnosis and definition. In M. Rutter & E. Schopler (Eds.), *Autism: A reappraisal of concepts and treatment (*pp. 25 1-267)*.* New York: Plenum Press.

Rutter, M. (2005). Genetic influences and autism. In F. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 425-452). New York: John Wiley.

Rutter, M., Le Couteur, A., & Lord, C. (2003). *ADI-R: The Autism Diagnostic Interview-Revised*. Los Angeles, CA: Western Psychological Services.

Rutter, M., Silberg, J., O'Connor, T., & Simonoff, E. (1999). Genetics and child psychiatry, II-empirical research findings. *Journal of Child Psychology and Psychiatry,* 40, 19–55.

Rutter, M., Tizard, J., Yule, W., Graham, P. & Whitmore, K. (1976). Research report: Isle of Wight studies, 1964–1974. *Psychological Medicine 6***,** 313–322.

Scheerenberger, R. C. (1987). *A history of mental retardation: A quarter century of promise.* London: Paul H. Brookes.

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

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

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

Singh, N. N., Oswald, D. P., & Ellis, C. R. (1998). Mental retardation. In T. H. Ollendick & M. Hersen (Eds.), *Handbook of child psychopathology* (3rd ed., pp. 91-116). New York: Plenum.

Sovner, R., *&* Hurley*,* A. D. *(*1983*).* Do the mentally retarded suffer from affective illness? *Archives of General Psychiatry, 40*, 61-67.

Sparrow, S., Balla, D., & Cicchetti, D. (1984a). *Vineland Adaptive Behavior Scales: Expanded Form Manual.* Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D.A., & Cicchetti, D. V. (1984b). *Vineland Adaptive Behavior Scales: Interview Survey Form*. Circle Pines, MN: American Guidance Service.

Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1985). *Vineland Adaptive Behavior Scales: Classroom Edition.* Circle Pines, MN: American Guidance Service.

Spence, S. (2004). The genetics of autism. *Autism and Autistic Spectrum Disorders, 11*(3), 196-204.

Steffenburg, U., Hagberg, G., & Hagberg, B., (2001). Epilepsy in a representative series of Rett's syndrome. *Acta Paediatrica, 90*, 34-39.

Steffenburg, S., & Gillberg, C. (1989). The etiology of autism. In: C. Gillberg (Ed.), *Diagnosis and Treatment of Autism* (pp. 63-82). New York and London: Plenum Press.

Sturmey, P. (1995). Analog baselines: A critical review of the methodology. *Research in Developmental Disabilities, 16,* 269-284.

Sturmey, P., Fink, C., & Sevin, J. (1993), The Behavior Problems Inventory: A replication and extension of its psychometric properties. *Journal of Developmental and Physical Disabilities, 5,* 327–336.

Sturmey, P., & Sevin, J. (1993). Dual diagnosis: An annotated bibliography of recent research. *Journal of Intellectual Disability Research, 37*, 437-448.

Sundaram, M., Sadler, R. M., Young G. B., & Pillay, N. (1999). EEG in epilepsy: Current perspectives. *Canadian Journal of Neurological Science, 26,* 255–262.

Symons, F. J., Sperry, L. A., Dropik, P. L., & Bodfish, J. W. (2005). The early development of

stereotypy and self-injury: A review of research methods. *Journal of Intellectual Disability Research, 49,* 144-158.

Szymanski, L. S., & Grossman, H. (1984). Dual implications of "dual diagnosis." *Mental Retardation,* 22, 155–156.

Tabachnick, B. G., & Fidell, L. S. (2006). *Using multivariate statistics* (5th ed.). Needham Heights, MA: Allyn and Bacon.

Towbin, K. E. (1997). Pervasive developmental disorders not otherwise specified. In: D. J. Cohen & F. R. Volkmar, (Eds.), *Handbook of autism and pervasive developmental disorders*. John Wiley & Sons, Inc., New York.

Tsiouris, J. A. (2001). Diagnosis of depression in people with severe/profound intellectual disability. *Journal of Intellectual Disability Research, 45*(2), 115-120.

Tsiouris, J. A., R. Mann, R., Patti, P. J., & Sturmey, P. (2003). *Challenging behaviours should* not be considered as depressive equivalents in individuals with intellectual disability, *Journal of Intellectual Disability Research,* 47, 14–21.

Tuchman R., F. & Rapin, I. (2002). Epilepsy in autism. *The Lancet, 1*, 352-358.

Tuchman, R. F., Rapin, I., & Shinnar, S. (1991). Autistic and dysphasic children, II: Epilepsy. *Pediatrics, 88*, 1219-1225.

United States National Library of Medicine & National Institutes of Health. (n.d.). *Medical encyclopedia: Mental retardation*. Retrieved March 1, 2007 from http://www.nlm.nih.gov/medlineplus/ency/article/ 001523.htm

Volkmar, F. R., Bregman, J., Cohen, D. J., & Cicchetti, D. V. (1988). DSM-III and DSM-III-R diagnoses of autism. *American Journal of Psychiatry, 145,* 1404-1408.

Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992a). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders*, *22,* 483-492.

Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992b). Developmental aspects of DSM-III-R criteria for autism. *Journal of Autism and Developmental Disorders*, *22,* 657-662.

Volkmar, F.R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M. et al. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry, 151,* 1361-1367.

Volkmar, F., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of American Academy of Child and Adolescent Psychiatry, 29,* 127-129.

Willer, B., & Intagliata, J. (1982). Comparison of family-care and group homes as alternatives to institutions. *American Journal of Mental Deficiency, 86,* 588-595.

Wing, L. (1981a). Asperger's syndrome: A clinical account. *Psychological Medicine, 11,* 115-130.

Wing, L. (1981b). Sex ratios in early childhood autism and related conditions. *Psychiatry Research, 5,* 129-137.

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. (1992). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research* (10th ed.). Geneva: Author.

Wyllie, E. (1993). *The treatment of epilepsy: Principles and practice***.** Philadelphia: Lippincott, Williams & Wilkins.

Yamanouchi, H., Imataka, G., Nakagawa, E., Nitta, A., Suzuki, N., Hirao, J., et al. (2005). An analysis of epilepsy with chromosomal abnormalities. *Brain and Development, 27*(5), 370-377.

Zaroff, C. M., Barr, W. B., Carlson, C., LaJoie, J., Madhavan, D., Miles, D. K. et al. (2006). Mental retardation and relation to seizure and tuber burden in tuberous sclerosis complex. *Seizure, 15*, 558-562.

Zigler, E., Balla, D., & Hodapp, R. (1984). On the definition and classification of mental retardation. *American Journal of Mental Deficiency, 89,* 215-230.

APPENDIX

STRATIFICATION OF PRESCRIBED PSYCHOTROPIC MEDICATIONS AND AXIS I DIAGNOSIS OF PARTICIPANTS

	Group Membership[a]			
	Control n (%)	Epilepsy n (%)	ASD n (%)	Combined n (%)
Psychotropic Medication				
Yes	0 (0)	25 (100)	10 (40)	23 (92)
No	25 (100)	0 (0)	15 (60)	2 (8)
Second Psychotropic Medication	0 (0)	8 (32)	6 (24)	7 (28)
Third Psychotropic Medication	0 (0)	2 (8)	2 (8)	3 (12)
Medication Type[b]				
Anti-depressant	0	0	0	1
Anti-psychotic	0	2	11	4
Anxiolytic	0	0	2	0
Mood stabilizer/AED	0	25	4	29
Psycho-stimulant	0	1	0	0
Axis I Diagnosis				
No Diagnosis	25	24	0	0
Autistic Disorder	0	0	18	16
PDDNOS	0	0	7	9
Anxiety Disorder NOS	0	0	1	0
ADHD NOS	0	0	0	1
Bipolar Disorder I	0	0	2	0

(Appendix cont.)

Dementia Alzheimer's	0	0	0	1
Major Depressive Disorder	0	0	0	1
Mood Disorder NOS	0	0	1	0
Specific Phobia	0	0	0	0
Pica	0	0	3	1
Psychotic Disorder NOS	0	0	1	0
Rumination Disorder	0	0	0	1
Stereotypic Movement Disorder with SIB	0	1	5	3
Total[c]	0	1	38	33

[a]$n = 25$ for each group.
[b]*Note*. Some participants were taking multiple psychotropic medications.
[c]*Note*. Some participants had more than one Axis I diagnosis.

VITA

Kimberly Robin Michelle Smith was born in Kwang Ju, South Korea, and lived in an orphanage until the age of five. At age five she was adopted and raised in Santa Monica, California. She earned a Bachelor of Arts degree in psychology and a minor in English from the University of California at Los Angeles, in June 2001. Kimberly earned her Master of Arts degree in psychology at Louisiana State University under the guidance of Johnny L. Matson, Ph.D., in May 2007. Her research and clinical areas of interest include individuals with intellectual and developmental disability, dual diagnosis, behavior problems, epilepsy, and psychopharmacology. She completed her pre-doctoral clinical psychology internship at the UCLA Semel Institute for Neuroscience in the Autism and Developmental Disabilities Track. After she receives her Doctor of Philosophy degree in clinical psychology from LSU in August 2009, Kimberly will make further contributions to the field of clinical psychology as a provisionally licensed psychologist in Nebraska where she will conduct assessments, provide treatments, and provide consultative services for adults with intellectual and developmental disabilities at a developmental center and children and their families in a private practice setting.