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# Factors that affect sleep in adults with developmental disability

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FACTORS THAT AFFECT SLEEP IN ADULTS WITH DEVELOPEMENTAL DISABILITY

A Thesis

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

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by

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## **ABSTRACT**

Sleep problems are a common occurrence in the typically developing population. These problems are even more frequent in those with developmental disabilities; however, sleep disorders are often under diagnosed in this population in clinical populations. Currently, there is a lack of research that examines the rate of sleep problems in adults with Autism Spectrum Disorders (ASD). The purpose of this study is to examine differences in the endorsements of sleep problems between three groups: 71 adults with Autistic Disorder (AD) and intellectual disability (ID), 71 adults with Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) and ID, and 71 adults with ID only, as assessed by the Diagnostic Assessment for the Severely Handicapped- Second Edition (DASH-II). The DASH-II, which includes a sleep scale, was designed to screen for psychopathology in those with severe to profound ID. Participants among the three groups did not differ significantly on total sleep scores or on individual items within the subscale. Follow up regression analysis examining what factors (i.e., ASD group, age, presence of psychotropic medications, presences of a non ASD Axis 1 diagnosis, and level of ID) predicted sleep problems found that the only significant predictor was presence of psychotropic medications. Implications and limitations of the study are discussed.

## INTRODUCTION

Sleep is one of the basic activities that all humans require to function. Sleep has been hypothesized to serve many functions such as energy conservation, body restoration, and memory consolidation (Breedlove, Rosenzweig, & Watson, 2007). Therefore, when sleep is disrupted, consequences can result in anything from poor general physical health (Tibbitts, 2008) to deteriorated ability to complete daily tasks (Roth & Ancoli-Israel, 1999). The importance of sleep holds true for all humans. However, there are some people for whom disrupted or disordered sleep may have even greater consequences. One such group is adults with developmental disabilities, such as autism.

Leo Kanner first described autism in 1943. He noted three main defining characteristics that are still considered the core features of autism: impairment in socialization, impairment in communication, and abnormal behavior. Now viewed as a spectrum of disorders known as Autism Spectrum Disorders (ASD), researchers also note that comorbidity with other psychological disorders is common. For example, some individuals with ASD also have diagnoses of Attention-Deficit/Hyperactivity Disorder (ADHD), anxiety disorders, Intellectual Disability (ID), feeding disorders and sleeping disorders (Matson, et al., 1996). Comorbid disorders in those with ASD are sometimes difficult to examine as the topography of disorders may be different in this population. In addition, as is the case with sleep disorders, symptoms may be categorized as behavior problems instead of a disorder in and of itself.

In regards to sleep problems in individuals with ASD, research has shown that disordered sleep is a fairly common problem (Nebel-Schwalm & Matson, 2008; Richdale & Schreck, 2009). Unfortunately, a lack of research exists examining the intricacies of these problems, particularly in adults with ASD. For example, since autism is on the spectrum with several other related

disorders such as PDD-NOS, we do not currently know if sleep problems differ between different ASDs. In addition, the effect of the presence of comorbidity and psychotropic medications in those with ASD is unknown.

The purpose of the current study was to examine the rate of sleep problems in three groups of individuals: adults with Autistic Disorder (AD) and ID, adults with Pervasive Developmental Disorder- Not Otherwise Specified (PDD-NOS) and ID and adults with ID only. The goal was to determine if sleep problems vary between these groups while controlling for other factors such as comorbidity and the presence of psychotropic medications. We will begin with an overview of the history, core features, and diagnostic criteria for ASD which will be followed by a discussion of sleep disorders, specifically in those with ASD along with the effects of sleep problems in this population.

## **AUTISM SPECTRUM DISORDERS**

### **History of AD and PDD-NOS**

In 1943, Leo Kanner published his first article concerning autism which described the cases of 11 children who all exhibited an abnormal pattern of symptoms. Upon being brought to Kanner, many of the children were thought to have been deaf due to their lack of interaction and response to their environments while others were classified as having schizophrenia or being feeble-minded. All of these children showed similar behaviors that did not fit with previously existing syndromes and disorders. Kanner noted that these children had three main common characteristics: deficits in communication, the desire to maintain sameness and routine, and difficulties in social relationships.

The degree of language ability varied in Kanner's sample. Within the 11 children that Kanner observed, three never developed language, while the others had varying language abilities. However, when language was present, it often was not meaningful or functional. One example of atypical communication exhibited was echolalia (i.e., repeating what was previously heard). Echolalia also occurs in typically developing children but only for a brief period of time, until around age three (Sharma, O'Sullivan, & Baird, 2008). Kanner (1943) also found that these children exhibited what he described as an "anxiously obsessive desire for the maintenance of sameness" (p. 245). This desire refers to both consistency in the child's physical environment (e.g., placement of furniture) as well as the child's daily routine. Kanner described instances of children throwing tantrums when their routines changed even in the slightest of ways.

In Kanner's (1943) sample, this obsession with sameness also resulted in the children's ability to relate better to objects than to people. Objects are often static and unchanging, whereas



people are dynamic. Therefore, these children had difficulties forming social relationships with people, regardless of whether the individual was a stranger or a parent. For example, Kanner noted that the children showed little to no reaction to the return of a parent after a long absence whereas a typical child would likely show happiness and go to greet the parent.

Aside from the three major features noted above, Kanner (1943) identified other similarities within his sample. Kanner noted that many of the children exhibited feeding and sleeping difficulties and aversions to loud noises and moving objects. Additionally, many of the children exhibited challenging behaviors such as aggression or self-injurious behavior. Kanner also noted that while some of the children in the sample had cognitive deficits, a subgroup exhibited extensive vocabularies and excellent memory. Another similarity between children in Kanner's sample was that there was no period of typical development. That is, the children showed deficits from a very early age. This differed from children with schizophrenia who displayed typical development for a period of several years before symptoms emerged. With these similarities in addition to the three core features, Kanner began to delineate this new disorder.

After Kanner (1944) made his initial observations, he coined the term "early infantile autism" to describe the pattern of symptoms exhibited in these children. As the children aged, Kanner (1951) continued to follow their development and noted changes. In response to his observations, other researchers began examining this new syndrome. For example, Eveloff (1960) found distinguishing characteristics between those children with only intellectual disability or brain damage and those with autism. He noted that repetitive behaviors in children with autism are more complex and more solitary than in those with intellectual disabilities.

Eveloff also stated that “the defective child uses his limited resources to adapt to reality, while the autistic child ignores his potentialities and reality” (p. 68).

While Kanner began to define what he meant by the term autism, confusion developed. This situation occurred because Kanner was not the first to use the term “autism.” In 1913, Bleuer used the term “autistic thinking” to describe thoughts that are “divorced both from logic and reality” (p. 874) as is seen in schizophrenia. He described autistic thinking as a withdrawal from reality. On the other hand, Kanner (1943) used the term autism to describe a set of symptoms in which the child never behaved typically; therefore, no withdrawal ever occurred. From this confusion, a debate in the field ensued regarding whether autism and childhood schizophrenia were separate disorders (Rutter, 1972).

Confusion between the diagnoses of autism versus schizophrenia was evident in Creak’s (1961) description of childhood psychosis. According to Creak, childhood schizophrenia could be identified by nine characteristics: 1) pervasive impairments in forming emotional relationships; 2) lack of understanding of personal identity; 3) abnormal fixation of parts of objects while ignoring general function of the object; 4) desire for consistency and sameness in the external environment; 5) abnormally high anxiety, specifically in response to changes in the environment; 6) distorted perceptual experiences, either over-sensitive or under-sensitive; 7) language impairments such as an absence of language development or presence of echolalia; 8) distorted ability to move spontaneously or independently, as evidenced by abnormal gait or rocking and spinning; and, 9) possible deficits in intellectual functioning. Within Creak’s description of childhood psychosis, there were considerable overlaps with the criteria which Kanner (1943) described for autism.

While some researchers believed there was no reason to differentiate between autism and childhood schizophrenia, others began to examine the differences between the two disorders. One of the trends identified was that the social class of parents of children with autism tended to be higher than those with schizophrenia (Lotter, 1967). That is, parents of children with autism tended to be of above average intelligence and of higher socioeconomic status. A second difference between autism and childhood schizophrenia was family history of schizophrenia. Rutter (1972) observed that those with autism rarely had a family member with schizophrenia whereas individuals with schizophrenia had a parent or sibling with the same disorder approximately 10% of the time. Third, sex ratios were found to be more similar in schizophrenia than in autism; those with autism were more likely to be male, while sex ratios in schizophrenia were roughly equal.(Rutter, 1967). A fourth difference between autism and childhood schizophrenia was that individuals with autism were more likely to have an intellectual disability while those with schizophrenia tended to be of normal intelligence (Pollack, 1960). A fifth distinction between the disorders was that people with schizophrenia were more likely to experience hallucinations and delusions than those with autism (Mayer-Gross, Slater, & Roth, 1955). Finally, Rutter, Greenfield and Lockyer (1967) pointed out that remission and relapse periods were fairly common in schizophrenia but rarely seen in those with autism. Thus, autism is a more chronic disorder than schizophrenia.

Based on the aforementioned distinctions between autism and childhood schizophrenia, Rutter (1972) stated that the term “childhood schizophrenia” was no longer applicable. Instead, Rutter (1968) suggested that age of symptom onset be used to distinguish disorders in children. Based on this notion, if symptoms were evident from infancy a diagnosis of autism as described by Kanner (1943) would be warranted. A second subtype, currently known as Childhood

Disintegrative Disorder, was diagnosed when development was typical for the first 3-5 years and then regressed. Finally, when onset of psychosis occurred in adolescence, the disorder would be described as schizophrenia. Based on this evidence, it was determined that autism was a separate entity from childhood schizophrenia.

Although Kanner first made his observations in 1943 and Rutter made his distinctions about autism in 1968, autism did not become an official diagnosis until 1980. In the *Diagnostic and Statistical Manual, First Edition*<sup>1</sup> (DSM-I; American Psychological Association [APA], 1952) and the *Diagnostic and Statistical Manual, Second Edition* (DSM-II; APA, 1968), no ASD diagnoses existed. As a result, individuals who exhibited autism-like symptoms would have likely been classified as having schizophrenia as this was the diagnosis that would have most closely fit their symptoms (Rutter, 1968).

It was not until the *Diagnostic and Statistical Manual, Third Edition* (DSM-III; APA, 1980) that the term autism was first introduced as a diagnosis under the category of Pervasive Developmental Disorders. Referred to as Infantile Autism, a diagnosis required the presence of Kanner's three main observations (i.e. socialization deficits, communication difficulties, and other odd behaviors) as well as an onset by the age of 30 months. For those individuals who exhibited the autistic symptoms after the age of 30 months and before the age of 12 years, a child-onset diagnosis of autism was also created. Further, a diagnosis of Atypical Autism was created for those individuals who did not meet the full criteria for infantile autism but still exhibited autistic-like symptoms.

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<sup>1</sup> The *Diagnostic and Statistical Manual, Forth Edition, Text Revision* (DSM-IV-TR; APA, 2000) and the *International Statistical Classification of Diseases and Related Health Problems, 10<sup>th</sup> Edition* (ICD-10; World Health Organization, 1992) are two commonly used systems for diagnosing psychological disorders. Due to the two systems sharing many similarities, the DSM-IV-TR, the more widely used tool, will be used in the discussion on diagnosis.

The subsequent editions of the *DSM* offered several other diagnostic changes for ASD. The *Diagnostic and Statistical Manual, Third Edition, Revised (DSM-III-R; APA, 1987)* changed the term Infantile Autism to Autistic Disorder, as well as changed the term Atypical Autism to PDD-NOS. The descriptor “not otherwise specified” is seen in many other diagnostic categories in the *DSM*, such as Eating Disorder-NOS, Anxiety Disorder- NOS, Mood Disorder-NOS and Learning Disorder-NOS, to describe problems which do not completely meet criteria for the primary disorder. The most recent revisions of the *DSM*, the *Diagnostic and Statistical Manual, Forth Edition (DSM-IV; APA, 1994)* and the *DSM-IV-TR (APA, 2000)*, differentiate five diagnoses under PDD which include AD, Asperger’s Syndrome, Rett’s Disorder, Childhood Disintegrative Disorder, and PDD-NOS. While all of the disorders have differences, the main diagnostic criteria for these disorders are built around the three main features that Kanner described over 60 years ago.

### **Core Features of ASD**

When Kanner (1943) first described autism, he described a triad of core features which were typically observed in those with the autism: deficits in socialization, language/communication, and abnormal behavior. These three features are still thought to be the core characteristics of autism and the other ASD diagnoses.

**Socialization.** Individuals with ASD have difficulties making and maintaining social relationships. One of the social impairments seen in autism is a lack of social relatedness which is defined by Plauche and Meyers (2008) as the “inherent drive to connect with others and share complimentary emotional states” (p. 531). Deficits in joint attention are one of the results of impairments in social relatedness. Joint attention (i.e., the act of two people sharing the experience of attending to the same object through the use of gestures and gaze) plays an

important role in socialization and language (Jones & Carr, 2004). For example, when a parent points to a dog and the child also looks to see the dog, there is an opportunity for learning language and sharing the experience is present. Joint attention develops between the ages of 9 to 18 months in typically developing children (Butterworth & Jarrett, 1991); however, individuals with ASD develop joint attention later and are not as easily able to shift their attention (Adamson, McArthur, Markov, Dunbar, & Bakeman, 2001).

Individuals with autism also exhibit abnormalities in forming attachments with others. Kanner (1943) originally noted that a lack of attachment existed between parent and child, as he stated the child would not react when the parent returned after a period of absence. Currently, it is not believed that people with ASD lack the ability to form attachments (Sturmev & Sevin, 1994), but that they form abnormal attachments that are qualitatively different from their peers (Dissanayake & Crossley, 1996). For example, children with ASD may not become upset when separated from the parent (Sigman & Ungerer, 1984). The differences in attachment are also seen in adults with ASD. In a sample of adults with high-functioning autism, rates of secure attachment were less than what is typically seen in the general population; however, the authors note that the rates of secure attachment were similar to what is observed in other clinical samples (Taylor, Target, & Charman, 2008). In general, individuals with better social functioning form more secure attachments with others (van IJzendoorn, et al., 2007).

Aside from problems in joint attention and in forming attachment, socialization abnormalities can be seen several other ways in those with ASD. Adults with ASD have a tendency to prefer being alone, avoiding eye contact, and displaying odd mannerisms in public (Matson, Dempsey, & LoVullo, 2009). An individual's lack of interest in forming relationships, along with other skill deficits, has negative implications for adults with ASD as they attempt to

function in educational and work environments (LeBlanc, Riley, & Goldsmith, 2008). Though treatment programs have shown that socialization can improve over the lifespan, overall, impairments in social functioning persist throughout development and show little improvement without intervention (Beadle-Brown, et al., 2002). Generally speaking, adults with higher cognitive functioning show the greatest improvement in socialization over time (Piven, Harper, Palmer, & Arndt, 1996)

**Language and Communication.** The second major core feature observed in those with ASD is impairment in language and communication. These deficits are evident in both verbal and nonverbal forms of communication. In Kanner's (1943) original description of autism, three of the children never developed verbal language while others exhibited varying degrees of language abilities. In some cases, limited language skills may be acquired and then a regression in ability may occur in later childhood. For example, Lord, Rutter, and Couteur (1994) found that 25% to 30% of children with autism or PDD-NOS developed the use of some words between 12 and 18 months, then regressed. While some individuals have no verbal language, other individuals have verbal language abilities, but exhibit language abnormalities. One such abnormality is echolalia, the non-functional repetition of what another person says, also referred to as "parroting" (Eveloff, 1960). A second abnormality is pronoun reversal in which the individual substitutes the incorrect pronoun while speaking (Lord & Paul, 1997), such as saying "You don't want to go" instead of "I don't want to go."

In addition to verbal language, nonverbal language abnormalities are also evident. Lord and Paul (1997) noted several common impairments in those with an ASD including lack of eye contact, decreased nonverbal responses to the other person, such as nodding, and decreased

awareness of facial expression and body language of others. As a result, those with an ASD also have nonverbal deficits in expressive and receptive nonverbal language.

These communication difficulties also have implications for social interactions. Language is closely linked with socialization in pragmatics, the social use of language. For example, knowing what topic to discuss, understanding turn taking while talking, and knowing when others are uninterested through body language and facial expression, all involve pragmatics. Individuals with autism have difficulty understanding pragmatics and interpreting social cues. As such, this contributes to their deficits in socialization (Plauche Johnson & Myers, 2008). For example, an individual with Asperger's Syndrome may only talk about topics which he is interested and not understand when the other person wants to change the topic.

Similar to the findings in the socialization domain, mixed results exist regarding changes in language ability in adulthood. In some cases, findings support that regardless of cognitive ability most individuals showed improvement in language from childhood to adulthood (Ballaban-Gil, Rapin, Tuchman, & Shinnar, 1996). For some, this improvement was in both language expression and comprehension while for others improved communication was the result of learning to use sign language. On the other hand, Shattuck and colleagues (Shattuck, et al., 2007) found that language abilities were relatively stable over time.

**Behavior.** The third core feature common to ASD is abnormalities in behavior which Kanner (1943) defined as an insistence on sameness, restricted interests and repetitive behaviors. In regard to insistence on sameness, people with an ASD often follow a strict routine in which the same tasks are carried out the same way in the same order. For example, in Kanner's sample he found that when taking a walk, the individual would follow the same route every time.



Plauche Johnson and Myers (2008) noted that when these routines are broken, individuals with autism are likely to exhibit challenging behaviors such as throwing a tantrum.

Many individuals with ASD also exhibit stereotyped behaviors. Stereotypies are defined as “repetitive, seemingly driven and nonfunctional motor behaviors” (APA, 2000, p.80) and can also include repetitive vocalizations (Matson, Kiely, & Bamburg, 1997). Common stereotypies are hand flapping, spinning, and body rocking. In addition, a sub-class of stereotypies known as self-injurious behavior (SIB) exists. SIB is a repetitive physical behavior where if the behavior was not blocked, bodily harm to the person engaging in SIB would likely result. Examples of SIB include head banging, hand biting, or excessive scratching. In some instances, stereotypies can occur at such high frequencies that they hinder the learning of new skills (Morrison & Rosales-Ruiz, 1997). For many individuals, these repetitive behaviors are found to persist through childhood and into adulthood (Cunningham & Schreibman, 2008). While some adults do show decreases in repetitive behaviors, reductions have not been reliably correlated with level of intelligence (Ballaban-Gil, et al., 1996; Seltzer, et al., 2003).

Additionally, people with ASD often have restricted interests (APA, 2000). Individuals with autism may spend hours of their day fixated on one activity. Examples of restricted interests are preoccupation with parts of objects (e.g., wheels of a truck) or specific historical event. These interests are abnormal in their intensity when compared to the interests of typically developing peers. Although all five ASDs share these three core symptoms (i.e., impairments in communication and socialization, as well as restricted, stereotyped patterns of behavior, activities or interests), it is important to understand how these core features differ across ASDs.

## **Current Diagnostic Criteria**

Within the ASD diagnoses, the disorders share the same core features (Nebel-Schwalm & Matson, 2008), but vary in symptom severity or age of onset. As shown by the diagnostic changes in the different editions of the *DSM*, autism went from being subsumed in one diagnosis (i.e., childhood schizophrenia) to being considered a spectrum of disorders (i.e., Pervasive Developmental Disorders). According to the *DSM-IV-TR* (APA, 2000), the ASD spectrum is composed of five different disorders that begin in childhood: AD, Asperger's Syndrome, Rett's Syndrome, Childhood Disintegrative Disorder, and PDD-NOS. As autism and PDD-NOS are two of the more common ASDs (Matson & Boisjoli, 2007) and since they are the disorders to be focused on in this study, they will be the center of the discussion on differential diagnosis.

**Autistic Disorder.** The definition of Autistic Disorder has been fairly consistent since its introduction in the *DSM-III* (APA, 1980). The three core features as described by Kanner are still evident in the current diagnostic criteria. According to the most recent criteria from the APA, the *DSM-IV-TR*, the first criterion for the diagnosis of autism is the presence of two deficits in social interactions. Impairments may be exhibited as an individual's lack of nonverbal behavior such as facial expression, failure to develop peer relationships typical for the individual's developmental level, lack of social or emotional reciprocity, or lack of spontaneously seeking to share things with others (APA, 2000).

The second criterion is impairment in communication. This impairment is indicated by at least one of the following: a lack or delay in language development; an inability to begin and hold a conversation with someone; or stereotyped or repetitive uses of language, lack of age-appropriate make-believe play or social imitation.

The third criterion for Autistic Disorder is repetitive and stereotyped interests or behaviors. Impairments may be exhibited as a “preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal in intensity or focus; apparently inflexible adherence to specific, nonfunctional routines or rituals; stereotyped and repetitive motor mannerisms; and persistent preoccupation with parts of objects” (APA, 2000, p. 75). The final criterion is that these symptoms must be present by the age of three and cannot be explained by a different ASD.

**Pervasive Developmental Disorder-- Not Otherwise Specified.** Currently, the *DSM-IV-TR* specifies that a diagnosis of PDD-NOS should be given when there is “a severe and pervasive impairment in the development of reciprocal social interaction associated with impairment in either verbal or nonverbal communication skill or with the presence of stereotyped behaviors, interests or activities” (APA, 2000, p. 64). In addition, an individual who receives a diagnosis of PDD-NOS will not meet the diagnostic criteria for a different ASD, Schizophrenia, Schizotypal Personality Disorder or Avoidant Personality Disorder.

### **Differential Diagnosis**

As seen by the fairly vague criteria in the *DSM-IV-TR* (APA, 2000) for PDD-NOS, room for interpretation exists about what constitutes a diagnosis of AD versus a diagnosis of PDD-NOS thus questions arise about differential diagnosis. One question clinicians have been faced with answering is whether PDD-NOS should be defined as a lesser degree of symptoms of AD or whether PDD-NOS is another distinct disorder (Towbin, 1997). Currently the *DSM-IV-TR* (APA, 2000) defines PDD-NOS as a catchall category for individuals who have some impairments seen in other ASDs, but who not do meet full criteria. Therefore, the diagnosis of PDD-NOS is relative and dependent on ruling out other ASDs. Yet, ruling out other diagnoses is

not an objective process as no concrete cutoff points or operational definitions have been established for determining what constitutes which diagnoses (Towbin, 1997). As a result, distinguishing between AD and PDD-NOS can be based on arbitrary, subjective criteria (Wing, 1997). Further, people with ASD often do not present with symptoms that neatly fit into diagnostic criteria. This may cause the majority of individual who fall on the ASD spectrum to be classified as having PDD-NOS. (Towbin, 1997).

To help resolve the problem of differential diagnosis, research has attempted to determine which characteristics best differentiate individuals with autism and those with PDD-NOS. Making these distinctions is most difficult with people with high-functioning autism and low-functioning PDD-NOS as these two groups present with the most similar symptomatology (Towbin, 1997). In general terms, Willemsen-Swinkels and Buitellar (2002) pointed out four instances in which a diagnosis of PDD-NOS would be warranted. The first instance is when onset of symptoms occurs after the age of 3 years. Secondly, a diagnosis of PDD-NOS may be warranted when the individual exhibits atypical symptoms which fall outside of the main criteria for autism. Thirdly, when symptoms are not of a high enough severity, a diagnosis of PDD-NOS may be warranted. And finally, individuals who do not exhibit the pattern of symptoms (i.e., less than two social impairments or no communication or behavioral impairments) would be diagnosed with PDD-NOS. Looking at more specific symptomatology, Mayes et al. (1993) described seven reliable criteria have been determined which distinguish PDD-NOS and AD: choosing solitary activities over group activities, poor social signals, abnormal comfort seeking, a lack of social usage in communication, impaired make-believe, impaired conversation, and interest in non-functional aspects of objects. People with AD show more impairment in these areas relative to people with PDD-NOS. Furthermore, comparisons have shown that persons

with PDD-NOS have better cognitive, communication, and social relatedness skills relative to those with autism (Sevin, Knight, & Braud, 2007). Individuals with PDD-NOS also demonstrated higher levels of functioning than those with autism, as well as lower rates of autistic behaviors such as stereotypies (Walker, et al., 2004). Overall, these findings demonstrate that there are differences between those with AD and those with PDD-NOS. However, researchers have yet to determine consistent differences between studies. Accurate differential continues to be difficult.

Though typically thought of as being a childhood disorder, ASD is considered a chronic, persistent disorder in which symptoms, while varying in severity, are present throughout a person's entire life (Bristol & Schopler, 1983). In general, however, the vast majority of research on ASD focuses on children while research involving adults has been largely neglected. In regards to assessment for ASD in adults, Matson and Neal (2009) note that assessment should be an ongoing process due to the fact that symptoms can change throughout a person's life. In studies which reassessed adults who had previously been diagnosed with autism, 13-45% of individuals no longer met criteria for autism (Piven, et al., 1996; Seltzer, et al., 2003). While some individuals may no longer qualify for a diagnosis, others may be diagnosed with ASD for the first time in adulthood (Ritvo, Ritvo, Freeman, & Mason-Brothers, 1994). In this case, retrospective data needs to be collected from caregivers and informants to gain information regarding onset of symptoms.

### **Prevalence of ASD**

The prevalence of ASD has increased in recent years (Wing & Potter, 2002). Once thought to occur in only 3 or 4 per 10,000 people (Howlin, 2006), current conservative prevalence estimates are approximately 36.4 per 10,000 people (Fombonne, 2005). There are

several possible reasons presented by Wing and Potter (2002) for this sudden increase in prevalence. These are: changes in diagnostic criteria as evidence by changes in the *DSM*; methodological differences between studies; increased physician and public awareness; increased understanding that intellectual disability, physical disorders, normal intelligence, or other psychological disorders can co-occur with autism; the development of clinicians specifically for the area of autism; attention to age of onset and its relation to ASD; and the possibility of a true increase in the number of occurrences of autism. Most likely, some combination of these factors has led to an increase in the diagnosis of ASD. Regardless of the reason for increases in prevalence rates in recent years, ASD is becoming a more frequent occurrence. PDD-NOS, the most frequent of the ASDs, occurs in approximately 20.8 per 10,000 people while AD occurs in approximately 13 per 10,000 people (Fombonne, 2005).

## INTELLECTUAL DISABILITIES

When discussing ASD, it is important to also address ID. As noted by Kanner in 1943, not all individuals who have ASD also display deficits in cognitive functioning; however, a subgroup of individuals have comorbid diagnoses of ASD and ID. The Centers for Disease Control and Prevention report that ID affects 12-16 per 10,000 individuals; however, prevalence is even higher in the ASD population. Approximately 70% of those with autism have a comorbid diagnosis of ID (Fombonne, 2003; Lang, Bouma, Sytema, Kraijer, & Minderaa, 2006). More specifically, Fombonne (2003) found that 30% of those with autism have mild to moderate ID while 40% have severe to profound ID.

As defined by the *DSM-IV-TR* (APA, 2000), the first criterion for a diagnosis of ID requires a score of 70 or less on an intelligence assessment. Based on the IQ score obtained, four levels of ID can be described. Mild ID is associated with an IQ between 50-55 and 70. Those with IQ scores between 35-40 and 50-55 are classified as having moderate ID. Severe ID is associated with an IQ in the range of 20-25 and 35-40 and finally an IQ in the range of 20-25 or below constitutes profound mental retardation. In addition to a low IQ score, an individual needs to exhibit impairments or deficits in at least two of the following: communication, health, leisure time, safety, school, self-care, taking care of a home, work. Finally, impairments must be present by the age of 18. As evidenced by the range of IQ scores which would warrant a diagnosis of ID, two individuals with ID could present very differently in skill level and abilities.

Aside from the diagnostic criteria described in the *DSM-IV-TR*, research has shown other behavior tendencies in those with ID. Similar to what is seen in those with ASD, stereotypies occur at a high rate in those with ID (Matson, et al., 1997). This is also the case with challenging

behaviors such as verbal and physical aggression, self-injurious behavior, and property destruction (Emerson, et al., 2001).

In addition, comorbid psychopathologies are fairly common in those with ID (Matson & Shoemaker, 2009). The most commonly diagnosed comorbid disorder with ID is ASD; however, it is not the only mental disorder observed in those with ID. Some of the more common psychological diagnoses include mood and anxiety disorders, ADHD, and sleep disorders (Plauche Johnson, Walker, Palomo-Gonzalez, & Curry, 2006; Santosh & Baird, 1999). Unfortunately, identifying these disorders proves more difficult as assessment instruments are often not designed for this population (Matson & Shoemaker, 2009). Regardless of the difficulty in assessing for these diagnoses, identifying comorbidity is important so that the individual's quality of life may be increased.



## **SLEEP DISORDERS**

### **Diagnostic Criteria**

Most people will experience sleep problems during their lifetime. Often, these problems will be mild and not interfere with daily functioning. For an estimated 10% of the general population, however, sleep difficulties will reach a level of clinical significance and will require a diagnosis (Partinen & Hublin, 1994).

There are two main references which classify sleep disorders, the *DSM-IV-TR* (APA, 2000), and the *International Classification of Sleep Disorders-Second Edition (ICSD-2)* which is published by the American Academy of Sleep Medicine (2005). Both references are similar in the disorders included, as well as the criteria for those disorders. For the purposes of this paper, the *DSM-IV-TR* (APA, 2000) criteria will be used to briefly describe each of the sleep disorders. The *DSM-IV-TR* separates sleep disorders into two main categories. The first category includes the dysomnias which concerns problems with the sleep process (i.e., amount, quality or timing of sleep), and consists of primary insomnia, primary hypersomnia, narcolepsy, breathing related sleep disorders and circadian rhythm sleep disorders. Nightmare disorder, sleepwalking disorder and night terror disorders make up the second category of sleep disorders, the parasomnias which address problems that occur during sleep or sleep-wake transitions.

**Insomnia.** The first of the dysomnias is insomnia which is generally described as having difficulty falling asleep or maintaining sleep (APA, 2000). These difficulties can occur when initiating sleep, maintaining sleep or awakening early (Brotini & Luigi Gigli, 2004; Estivill, et al., 2003) and can last for varying durations (i.e., transient refers to less than 4 weeks; short-term refers to more than 4 weeks but less than 3 to 6 months; chronic refers to longer than 3 to 6 months; Estivill, et al., 2003). Prevalence studies estimate that 7.5% of the population has

insomnia (Bixler, Vgontzas, Lin, Vela-Bueno, & Kales, 2002). In addition, risk factors for insomnia have been identified such as hyperarousal (Bonnet & Arand, 1997; Roth & Drake, 2004), a comorbid psychological disorder (Lichstein, Wilson, & Johnson, 2000), a comorbid medical condition (Katz & McHorney, 1998; Smith, Huang, & Manber, 2005) and being older (Brotini & Luigi Gigli, 2004; Kamel & Gammach, 2006).

**Hypersomnia.** While insomnia is the difficulty falling or maintaining sleep, other people suffer from excessive daytime sleepiness known as hypersomnia. According to the *DSM-IV-TR*, the main characteristic of this disorder is “excessive sleepiness for at least one month (or less if recurrent) as evidenced by either prolonged sleep episodes or daytime sleep episodes that occur almost daily” (APA, 2000, p. 268). More specifically, those with hypersomnia have sleep that is not refreshing and have difficulty waking up early in the morning or after a nap (Bassetti, Pelayo, & Guilleminault, 2005). In many cases, it is important to identify the source of what is causing the excessive sleepiness, as there are several possible contributing factors. For example, hypersomnia may be caused by neurological disorders (e.g., epilepsy, stroke, or tumor), other sleep disorders (e.g., insomnia, breathing related sleep disorders, circadian rhythm disorder or restless leg syndrome), behavioral disorders (e.g., mood disorder, chronic fatigue syndrome), or medical disorders (e.g., infection, muscle disease, or metabolic disorders; Kohrman, 2005). In addition, a subtype of hypersomnia exists which is called idiopathic hypersomnia. This subtype is diagnosed when all other causes have been ruled out and no pathophysiology can be determined (Bassetti, et al., 2005). Since hypersomnia is often not the primary issue, it is often discussed in the context of the primary disorder.

**Narcolepsy.** The third of the dysomnias is narcolepsy. Narcolepsy is characterized by “irresistible attacks of refreshing sleep that occurs daily over at least three months” (APA, 2000,

p. 269). A person with narcolepsy may feel severely sleepy for the majority of his day but then will have sleep episodes that typically last for 10-20 minutes and awake feeling refreshed (Thorpy, 2001). During these naps, cataplexy (the sudden loss of muscle tone), hypnopompic or hypnagogic hallucinations (auditory and visual hallucinations experienced while falling asleep or waking up), or sleep paralysis (the conscious awareness of the inability to move one's body during sleep and wake transitions) are also experienced (Hishikawa & Shimizu, 1995). This situation becomes problematic because the naps are involuntary and can occur at anytime and during any activity, such as driving (Green & Stillman, 1998). Though the specific etiology of narcolepsy is unknown, it is generally accepted that an imbalance of neurotransmitters and genetic factors both play a role in the disorder (Dauvilliers, Billiard, & Montplaisir, 2003).

**Breathing-Related Sleep Disorders.** According to the *DSM-IV-TR* (APA, 2000), Breathing-Related Sleep Disorders (BRSD) are the forth dysomnia and are estimated to effect 2% to 4% of people (Young, et al., 2007). In general terms, BRSD is diagnosed when insomnia or excessive sleepiness occurs due to the total cessation of airflow (i.e., apnea) or the partial cessation of airflow (i.e., hypopnea) which leads to low blood oxygen levels during sleep. Due to the lack of oxygen, the individual becomes aroused and wakes up which in turn leads to low quality and/or quantity of sleep. Apnea and hypopnea episodes typically last from 10 to 20 and can occur at varying frequencies during the night (McCoy, Koopmann, & Taussig, 1981). Being older, male, and obese are all risk factors which increase in individual's risk of developing a BRSD (Bliwise, et al., 1987).

**Circadian Rhythm Sleep Disorders.** The final of the dysomnias is Circadian Rhythm Sleep Disorders. With these disorders, disturbances occur due to a mismatch of one's sleep-wake cycles with the environment thereby causing excessive sleepiness or insomnia (APA,

2000). In general, Circadian Rhythm Sleep Disorder has two main types: those in which the physical environment changes in relation to the individual's internal rhythm, and those in which the internal mechanisms do not match with the external environment (Okawa & Uchiyama, 2007). The first subtype includes jetlag (i.e., when sleep cycles become disrupted due to the traveling across time zones) and problems associated with night shift work. In these situations, the individual's circadian rhythm is no longer in sync with external cues such as light (Minkel & Dinges, 2009). The second subtype consists of delayed sleep phase type, and non 24-hour sleep-wake type. In delayed sleep phase type a person may not feel sleepy until the early morning hours and then may sleep until late morning or early afternoon. In non-24-hour sleep-wake type, which is often observed in blind individuals, a person's internal clock is on a 25-hour schedule instead of a 24-hour schedule (Uchiyama & Lockley, 2009). In general, jet lag and shift work types of circadian disturbances are not chronic problems whereas the delayed type tends to be more persistent (Arendt, Stone, & Skene, 2005).

**Nightmare Disorder.** Nightmare Disorder, a parasomnia, occurs when an individual awakens from a period of sleep and recalls detailed and vivid dreams of a threatening or frightening nature. These dreams occur during the REM stage of sleep (Fisher, Bryne, Edwards, & Kahn, 1970). Upon awakening, the person is alert and aware of his surroundings (APA, 2000). Though nightmares are a common occurrence, experienced by 100% of the population, a diagnosis of this disorder requires the nightmares to be severe and frequent enough to cause impairment and distress for the individual (Nielsen & Zadra, 2005). For most individuals, nightmares occur more often in childhood and tend to become less frequent into adulthood (Nielsen & Zadra, 2000).

**Sleep Terrors.** Individuals with Sleep Terror Disorder awake from sleep screaming along with increased autonomic arousal as evidenced by increased breathing, heart rate, and sweating. During the episode, the person will not be oriented or responsive to others who attempt to comfort them. After the episode, the individual will not recollect dreaming or any part of the screaming episode (APA, 2000). Unlike nightmares, sleep terrors occur during the non-REM phase of sleep and are experienced less frequently. Only 3% of children 4 to 12 years of age experience sleep terrors (Panossian & Avidan, 2009), while the prevalence is closer to 1% in adults (Robinson & Guilleminault, 2003). Additionally, genetic and developmental factors contribute more to the occurrence of sleep terrors in children whereas psychological factors such as anxiety and depression may contribute more to sleep terrors in adults (Kales, Kales, et al., 1980).

**Sleepwalking.** The next of the parasomnias is Sleepwalking Disorder, also known as somnambulism. During sleepwalking episodes, the individual, while still in a deep stage of sleep, will get out of bed and begin walking but will be unresponsive and not easily awoken. Similar to sleep terrors, the person experiences amnesia for the event upon awakening. Sleepwalking is fairly common in children with prevalence rates between 10-15% but only 2-4% in adults (Hublin, Kaprio, Partinen, Heikkila, & Koskenvuo, 1997). Typically onset of sleepwalking occurs before age 10 and ceases to occur by age 15 (Kales, Soldatos, et al., 1980). Normally, somnambulism does not cause harm; however, cases have been reported in which injury was the result of violent or odd behavior, such as jumping out of a window (Bassetti, 2009).

**Parasomnia-- Not Otherwise Specified.** The final sleep disorder is Parasomnia-Not Otherwise Specified which accounts for abnormal sleep behavior not explained by the other

parasomnias. The *DSM-IV-TR* (APA, 2000) gives the example of REM Sleep Behavior Disorder. Typically, during the REM stage of sleep, no bodily movement occurs as the motoneurons have been inhibited. However, in REM Sleep Behavior Disorder the motoneurons are not inhibited, thereby leading to a person acting out his dreams. These movements can be dramatic, sometimes violent movements which can lead to injury of the individual or the other person in bed.

### **Assessments of Sleep Problems**

With having reviewed what differentiates different sleep disorders and what may contribute to sleep disorders, one must then consider the best way to collect information about the individual's sleep. Several different methods for assessment are polysomnography, direct observation, and informant report. Each of these methods offers its own advantages as well as disadvantages.

**Polysomnography.** Polysomnography is an objective measure used to assess sleep. This assessment utilizes several data collection methods including tracking blood oxygen levels, body position, breathing rate, muscle activity, eye movement, heart rate, and brain activity (Chez, Memon, & Hung, 2004). From this data, information can be gained about sleep patterns and stages, as well as seizure activity. In order to conduct a polysomnography the individual needs to spend the night in a sleep lab so that they can be monitored. Therefore this method, though thorough, can be time consuming and expensive relative to other methods (Nixon & Brouillette, 2002).

**Actigraphy.** Actigraphy is another objective measure of sleep which has shown high correlations with results of polysomnography (Ancoli-Israel, et al., 2003). In this method, the individual typically wears a band around his wrist which detects and records movements to

indicate levels of activity (Westermeyer, et al., 2007). While actigraphy has the advantage of not interfering with the individual's routine, it highly useful for detecting all sleep disorders such as BRSD (Ancoli-Israel, et al., 2003). In addition, actigraphy would not be appropriate for those who often have calm, quite wakefulness as these periods would be interpreted as sleep due to the lack of movement (Sadeh & Acebo, 2002). This weakness of actigraphy would be especially problematic for the ID population as they have decreased levels of activity (Ancoli-Israel, et al., 2003).

**Direct Observation.** Another method of gaining information on sleep habits is to use direct observation. This method is often utilized in residential and in-patient facilities through the use of sleep logs since staff can easily collect the data. Data collected may include if the individual is awake or asleep, in bed or out of bed, or if any odd behaviors were exhibited during sleep, such as screaming. Smith, Nowakowski, Soeffing, Orff, and Perlis (2003) suggest 30 minute intervals or less be used when using direct observation of sleep since data becomes less reliable as the intervals become shorter.

**Informant Report.** Several assessments exist to measure sleep problems that use parents or caregivers as informants to gain information about the individual. In general, informant reports can provide much data in a short amount of time and are inexpensive (Nixon & Brouillette, 2002). Though many sleep measure have been developed, only the measures typically used for those with ASD will be reviewed here.

One measure used to examine sleep problems in children with an ASD is the Children's Sleep Habits Questionnaire (Owens, Spirito, & McGuinn, 2000). The purpose of this measure is to screen children ages 4-12 years for possible sleep problems. The 35-item abbreviated version focuses on three main domains: dysomnias, parasomnias, and sleep-disordered breathing.

Although sometimes used in the ASD population, it was not developed for use among children with an ASD.

A second informant report is the Behavioral Evaluation of Disorders of Sleep (BEDS) (Schreck, 1997/1998). This measure is valid for children 5-12 years of age. It uses 5-point Likert-type questions that were developed based on criteria for sleep disorders from the ICSD-R. The five categories of sleep disorders described are: expressive sleep disturbances, sensitivity to the environment, disoriented awakenings, sleep facilitations, and apnea/bruxism. Though this measure has been used in studies focusing on children with ASD, during the development of the assessment, children with ASD were excluded from the sample (Schreck, Mulick, & Rojahn, 2003).

One sleep assessment measure that focuses on adults with ID is the Sleep Problems Inventory (SLEEPY) (Dixon, 2007). This measure is a 24-item questionnaire that is administered by a clinician to the caregiver. The SLEEPY has four main factors which aid in identifying sleep problems: daytime somnolence, sleep maintenance, breathing-related sleep disorders, and hypersomnia. Though this assessment has the benefit of being designed specifically for adults with ID, the author notes that the reliability for this instrument is not excellent and the assessment has a low sensitivity for detecting some sleep behaviors; therefore, it may not be ideal (Dixon, 2007).

The Diagnostic Assessment for the Severely Handicapped-II (DASH-II; Matson, 1993) is an informant based assessment examining psychopathology in adults with severe to profound ID. Eighty-four items make up the 13 subscales of the DASH-II: Impulse, Organic, Anxiety, Mood, ASD/Autism, Schizophrenia, Stereotypies, Impulse, Self-Injurious Behavior, Elimination, Eating, Sleep, and Sexual. Caregivers provide information on frequency, duration, and severity



of each behavior based on the previous two weeks. An elevation on any of the subscales represents clinical significance. Within the sleep subscale, five items probe for the presence of sleep problems. The DASH-II is a relatively short assessment but the sleep subscale has good validity (Matson & Malone, 2006) and reliability (Sevin, Matson, Williams, & Kirkpatrick-Sanchez, 1995). In addition, one of the strengths of the DASH-II is that it was designed for those with severe to profound ID which is sometimes seen in those with ASD and those with ASD were included in the development of the assessment.

## **SLEEP DISORDERS WITHIN ASD**

Sleep disorders are common in those with ASD. However, in this population, problems with sleep are often classified as behavioral problems as opposed to actual diagnoses (Matson & Malone, 2006). In addition, research examining sleep and ASD is limited, and the majority of research that exists assesses children and adolescents as opposed to adults. Within the literature on children, Richdale and Schreck (2009) found that, according to parental report, overall 80% of children with either ID or a developmental disability experienced some sort of sleep problem. When examining children with AD, sleep disorder prevalence rates were 44-83% (Miano, et al., 2007; Richdale & Schreck, 2009). The discrepancy in prevalence estimates is a result of differing methods across studies such as diagnostic criteria used, sample size, method of assessment, and age (Richdale & Schreck, 2009). In regards to sleep patterns in adults with low-functioning ASD, sleep patterns are currently estimated to be comparable to those with ID (Hare, Jones, & Evershed, 2006). The study by Hare and colleagues was unique in that it used objective measure of sleep as opposed to third-party informants which may have eliminated some reporting bias. However, the presence of actual sleep disorders was not examined in this study. Brylewski and Wiggs (1998) examined sleep problems in adults with ID living in a community based supported living. Their findings showed that approximately half of individuals have insomnia while fourteen percent exhibited a parasomnia. It should be noted that in general prevalence rates for specific sleep disorders in those with ASD are not known as studies which examine this area do not examine prevalence rates for individual disorders, but look at aggregate prevalence rates across all sleep disorders.

In regard to specific sleep disorders, insomnia is the most commonly reported sleep problem in those with ASD (Malow, et al., 2006). Insomnia is most likely to occur during onset

of sleep, followed by awakenings in the middle of the night and then awakenings in the early morning (Taira, Takase, & Sasaki, 1998). Insomnia, which often results in daytime sleepiness, is frequently due to inconsistent bedtime routines which then develop into behavior problems (Richdale & Wiggs, 2005; Wiggs & Stores, 2004).

Compared to insomnia, parasomnias are not reported as frequently (Richdale & Schreck, 2009). Matson and Malone (2006) observed no instances of sleepwalking in adults with ASD. In addition, diagnosing some parasomnias, such as Nightmare Disorder, in those with ASD may be more difficult due to impairments in communication. Due to nightmares being internal, non-observable events, caregivers may not be able to know a person had a nightmare (Polimeni, Richdale, & Francis, 2005). This impaired ability to communicate likely effects accurate reporting and tracking of most parasomnias.

Several other sleep problems occur in those with ASD, though at lower frequencies. Circadian rhythm disorders (Richdale & Schreck, 2009), enuresis, and repetitive behaviors during sleep (Miano, et al., 2007) have all been reported. Johnson and Malow (2008) also found that people with an ASD are not at a higher risk to develop breathing-related sleep disorders than those who are typically developing.

While sleep problems have been shown to be a fairly common occurrence in those with ASD, they also appear to be relatively stable over time. Longitudinal studies in children with ID show that 50-75% of sleep problems present at the start of the studies were still present two or more years later (Didden & Sigafoos, 2001; Richdale, Francis, Gavidia-Payne, & Cotton, 2000). However, it seems that some sleep problems may resolve themselves. Taira and colleagues (1998) found that over a third of the sleep problems in their sample ceased without treatment. More research is needed to determine the stability of sleep problems in this population.

## **Group Differences within ASD**

Several studies have examined the group differences in sleep problems between those people with ASD, other diagnoses, and typical development. Many of these studies group all ASD together during analysis. When comparing typically developing children to those with any of the ASDs, insomnia is the most frequently reported problem for both groups (Didden & Sigafoos, 2001; Malow, et al., 2006). Though typically developing children and children with ASD may have similar types of sleep problems, research shows that children with ASD exhibit more severe problems at higher frequencies. For example, Richdale and Schreck (2009) found that children with ASD may have nighttime awakenings that may last for several hours and may include laughing, screaming, talking, or playing with objects whereas typically developing children's awakenings last for a shorter duration and cause less disruption. Additionally, when comparing those with ASD to those with other developmental delays or disorders (e.g., Prader-Willi Syndrome and Down Syndrome), the ASD group obtains the most frequent endorsements of sleep problems from caregivers (Cotton & Richdale, 2006; Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hansen, 2008).

In general, limited research has examined group differences in adults. Hare, Jones, and Evershed (2006) used sleep logs and actigraphy, the non-invasive method of detecting sleep and activity to observe sleep patterns, to examine sleep-wake cycles in those with an ASD and those with only ID. Their finding showed that the sleep-wake cycles of those with low-functioning autism were similar to those with ID only. Conversely, Matson, Ancona, & Wilkins (2008) compared adults with ASD and ID to adults with ID only using third party informant reports and found significantly more sleep problems in the ASD and ID group. Based on the current

literature for adults, it remains unclear if those with ASD and ID differ from those with ID only in regard to sleep problems.

While the aforementioned studies grouped ASD together, several studies have attempted to look for differences in sleep problems within the ASD by comparing AD, Asperger's Syndrome, and PDD-NOS. Schreck and Mulick (2000) examined differences in sleep problems among five different groups of children: AD, PDD-NOS, ID only, those special education classes with no ASD or ID, and typically developing controls. During analysis, the AD group and PDD-NOS group were combined due to a lack of differences in the parental reports. The overall findings showed that the ASD group had higher scores on dysomnias and parasomnias than the other three groups. Specifically, the ASD group had more frequent endorsements of repeated awakenings, nightmare behavior, and breathing related problems. A similar study by Polimeni, Richdale and Francis (2005) examined sleep problems in children with AD, Asperger's Syndrome, and those who were typically developing. This study also utilized parent reports to obtain data. Parents of children with AD and Asperger's syndrome reported significantly more frequent sleep problems than those parents of typically developing children; however no difference was found in the severity or type of sleep problems between groups. Overall sleep problems were lowest in the typically developing group followed by the AD group and then the Asperger's group in which sleep problems were greatest.

Once again limited studies have been conducted which examine sleep problems in regard to specific ASD in adults. One study by Tani et al. (2003) examined rates of insomnia in those with Asperger syndrome in comparison to healthy typically developing adults. Using questionnaires and sleep diaries, results indicated that those with Asperger syndrome had higher rates of insomnia and had sleep problems that were more likely to interfere with daytime

functioning. At this time, no other studies using an adult samples which separated ASD diagnoses in comparing rates of sleep problems were found.

### **Factors Contributing to Sleep Problems**

In those with ASD, as well in typical populations, a variety of factors have been shown to increase the likelihood of sleep problems. Factors include age, level of cognitive impairment, the presence of psychopathology, and the presence of psychotropic medications. While in general the relation between sleep problems and these factors is supported, some findings have been contradictory.

In regards to age, findings show that as people age, sleep problems increase (Smallwood & Stern, 2004). In a community sample of adults with ID, Brylewski and Wiggs (1999) found that the average age of those with sleep disorders was significantly older than those without sleep disorders. While findings generally support that sleep problems increase with age, some findings bring into question this relationship. Schreck and Mulick (2000) did not find difference in sleep quality or quantity in those with ID and ASD based on age. Conflicting findings indicate further research is needed in this area.

Another contributing factor to sleep problems in those with ASD is the presence of a comorbid condition. The first comorbid condition to consider is ID since, as mentioned above, ASD and ID are highly comorbid (Fombonne, 2003; Lang, Bouma, Sytéma, Kraijer, & Minderaa, 2006). It has been well established that those with ID exhibit more sleep problems than those with typical cognitive functioning (Matson, Ancona, & Wilkins, 2008), but more specifically, it is important to consider if and to what extent the degree of cognitive impairment affects the severity of sleep problems. While some researchers have found no association between levels of ID and sleep problems (Clements, Wing, & Dunn, 1986), others have shown

that level of ID may be an important factor in sleep problems. Espie and Tweedie (1991) found significant differences in sleep problems when comparing adults with severe and profound ID to adults with mild and moderate ID. Likewise, Piazza, Fisher, & Kahng (1996) found a positive correlation between IQ scores and quality and amount of sleep indicating that those with lower IQs have more sleep disturbances.

In addition to comorbid ID, other comorbid psychopathology and sleep disorders co-occur fairly commonly. In a typical developing population, comorbid anxiety, depression, and other psychological disorders increase the occurrence of sleep problems (Roth, et al, 2006). In those with ASD, comorbid conditions (e.g., specific phobia, attention deficit/hyperactivity disorder, obsessive compulsive disorder) are exhibited at a relatively high rate (Leyfer, et al., 2006). This increased risk of comorbidity increases the likelihood that someone with ASD would develop sleep problems. It should be noted that the reason for the co-occurrence of psychopathology and sleep problems is not completely understood. In some cases, sleep problems can be thought of as a symptom of the disorder (e.g., as is seen in depression), while in other cases it is not clear what the direction of the relationship is, or if both psychopathology and sleep problems may be caused by some other common factor (Staner, 2010).

A final factor to consider in sleep problems in those with ASD is the presence of psychotropic medications. In both those with ASD and ID, pharmacotherapy is often used to control psychiatric conditions and challenging behaviors, such as aggression (Matson & Wilkins, 2008). This becomes problematic as the use of the medications often leads to a variety of side effects which sometimes become permanent, such as with tardive dyskinesia (Advokat, Mayville, & Matson, 2000). Estimates suggest that approximately 31-55 % of those with ASD are on some form of psychotropic medication (Aman, Sarpahre, & Burrow, 1995; Martin, Scahill, Klin, &

Volkmar, 1999). The types of psychotropic medications prescribed vary, with the most common types being antidepressants, antipsychotics, and stimulants (Lecalavie & Gadow, 2008). In some cases, psychotropic medications may be used to treat sleep disturbances while in other cases the use of these medications results in sedation and actually create sleep disturbances (Valdovinos, Schroeder, & Kim, 2003). It is important to note that sleep problems are not only caused by medications with sedative effects. Hogg (1992) found that a minority of the participants in his study were actually taking medications with sedative side effects indicating that other factors of the medications are contributing to sleep problems. At this time further research is needed in this area.

### **Effects of Sleep Problems**

It is important to study sleep disorders because problems with sleep often cause other challenges and difficulties in the individual's life. In the typically developing population, sleep problems interfere with many areas of living, such as general physical health, memory, metabolism, safety of others (Tibbitts, 2008), concentration, and ability to complete daily tasks (Roth & Ancoli-Israel, 1999). In order for impairment to result from sleep disturbances, awakenings from sleep do not have to be long in duration but could be frequent awakenings for short durations (Ohayon, 2009). Also, the more severe the sleep problem, the greater impairment and difficulty the person will have during the day (Roth & Ancoli-Israel, 1999). The degree of impairment from sleep problems has also been connected to level of daytime activity with lower levels of activity resulting in greater impairments from sleep problems (Lopes, Esteves, Bittencourt, Tufik, & Mello, 2008).

There are additional effects of sleep problems, other than the aforementioned effects, when examining sleep problems in people with ASD and ID. In both typically developing



children and children with ID, parents report that presence of a sleep problem leads to an increase in behavior problems during the day. However, for parents of children with ID, the disturbances during the day are reported as being more intense and frequent than is seen in typically developing children with sleep problems (Richdale, et al., 2000; Richdale & Prior, 1995). More specifically, increases in disruptive behaviors (e.g., impatience, tantruming), self-absorbed behaviors (e.g., humming, biting, screaming) (Richdale, et al., 2000), hyperactivity, and irritability (Brylewski & Wiggs, 1998) have been observed among people with ID and sleep disturbances. Schroeder (2001) found that a lack of sleep can lead to an increase in SIB during the day in those with developmental disorders. Additionally, some debate exists regarding whether more sleep problems lead to greater autistic symptoms or if greater autistic symptoms lead to more sleep problems. Schreck, Mulick and Smith (2004) found that sleep problems exacerbate a child's autistic-like symptoms, such as stereotypies. However, Mayes and Calhoun (2009) found that the severity of autistic symptoms were predictive of increased sleep problems. Therefore, as to whether sleep problems increase autistic symptoms or vice versa is not clear.

It is important to point out that sleep problems not only affect the individual, but also parents, caregivers, and spouses (Richdale, et al., 2000). This is especially true for those individuals with ASD and ID. Compared to typically developing individuals, parents and caregivers of people with ID describe that sleep problems produce higher levels of stress on the caregiver (Richdale et al., 2000). Schreck and colleagues (2004) found that this increase in stress is due to people with ASD and ID being more highly dependent on caregivers. As a result, the quality of care may not be optimal when sleep problems are present because demands are higher (Wiggs & Stores, 2001). For example, some caregivers in in-patient facilities may put the individual to bed early in an attempt to reduce their stress but by doing so may actually

exacerbate sleep problems and decrease day-time functioning. For all of these reasons, it is important to continue to examine sleep in those with developmental disabilities.

## **PURPOSE**

Overall, sleep disorders are under diagnosed in the developmentally disabled population (Richdale & Schreck, 2009). This may be due at least in part to a limited number of studies have examined sleep problems in adults with ASD (Espie, et al., 1999; Richdale, et al., 2000; Richdale & Prior, 1995; Schroeder, 2001). Studies conducted on ASD and sleep primarily focus on children (Didden & Sigafos, 2001; Malow, et al., 2009; Polimeni, et al., 2005), and while some studies examine group differences between those with ASD, ID, and typically developing people, results are not consistent (Hare, et al., 2006). The purpose of the current study was to determine if sleep problems occur at different rates when comparing adults with AD and ID to adults with PDD-NOS and ID to adults with ID alone. It is important to note that none of these groups are a pure control, but that is due to the dependent measure being designed for those with severe to profound ID, and in addition, the sample was obtained from inpatient residential facilities. Thus, it would be inappropriate to use a pure control group from the community with no ID. Comparing these groups' sleep data is important to make better determinations about the nosology of these disorders. Furthermore, information regarding sleep impairments and how they differ between diagnostic groups and on other demographic characteristics such as psychotropic drug use, has implications for assessment, diagnosis, and treatment. The DASH-II sleep subscale will be used to obtain information about sleep as this measure has been specifically designed for those with intellectual disabilities in the severe to profound range. Furthermore, the sleep disorders subscale is valid (Matson & Malone, 2006) and reliable (Sevin, et al., 1995) for adults with ID. On this measure, the sleep subscale was used to compare groups on their total sleep subscale score as well as to compare groups on individual items.

## **HYPOTHESES**

Based on the current literature, it is predicted that the AD with ID group will exhibit the greatest amount of sleep problems, followed by the PDD-NOS group with ID, and then the ID only group. This prediction is based on findings which support that those with AD show the greatest and most severe impairments in a variety of areas such as socialization, cognitive function, and repetitive behaviors (Mayes, et al., 1993; Walker, et al., 2004). The analysis to compare groups on specific items within the sleep subscale is exploratory in nature to determine if certain items are more likely to be endorsed by one group than the other groups.

## METHOD

### Participants

Each comparison group, AD and ID, PDD-NOS and ID, and ID only was composed of 71 participants. Data had previously been collected at two state-run developmental centers in the Southeastern United States. Residents at the facilities vary in their level of intellectual ability, age, race, and gender. One hundred and seventy-one adults met the inclusion criteria of having severe or profound ID. In the current sample, 85.4% were diagnosed as having profound mental retardation based on results of previously administered standardized intelligence testing. The age of the sample ranged from 16 to 87 years of age ( $M = 50.59$ ,  $SD = 13.35$ ). The sample was composed of 56.1% male, and the ethnicity of the sample was 77.2% Caucasian, 22.2% African American, and 0.6% Hispanic. Equal sample sizes were obtained to protect against violations of the assumptions for some of the statistical analyses conducted (Leech, Barrett, & Morgan, 2008). In addition to these demographics, the presence of psychotropic medications, and the presence of an Axis I diagnosis (other than ASD) were also examined. In the overall sample, 22.8% were taking a psychotropic medication and 26.9% had an Axis I diagnosis other than ASD. Table 1 shows the demographics for each group.

Table 1  
Demographic Information

	Group 1	Group 2	Group 3
Age			
Mean (SD)	47.28 (10.15)	49.18 (13.85)	55.02 (14.59)
Range	18-65	16-75	28-87
Gender			
Male	59.6%	49.1%	59.6%
Female	40.4%	50.9%	40.4%
Ethnicity			
Caucasian	84.2%	73.7%	73.3%
African-American	15.8%	24.6%	26.3%

Table 1 continued

Hispanic	0.0%	1.8%	0.0%
ID level			
Severe	3.5%	10.5%	29.8%
Profound	96.5%	89.5%	70.2%
Psychotropic med			
Yes	43.9%	21.1%	3.5%
No	56.1%	78.9%	96.5%
Axis I Dx			
Yes	50.9%	21.1%	8.8%
No	49.1%	78.9%	91.2%

*Note.* Group membership is as follows: Group 1- AD and ID; Group 2- PDD-NOS and ID; Group 3- ID only.

## Measures

**DSM-IV-TR/ICD-10 Checklist.** This informant based measure consists of 19 items based on the criteria for AD from the *DSM-IV-TR* (APA, 2000) and *International Classification of Diseases- 10<sup>th</sup> revision (ICD-10; World Health Organization, 1992)*. Inter-rater reliability ( $r = .89$ ), test-retest reliability ( $r = .96$ ), and internal consistency ( $\alpha = .99$ ) all proved to be strong (Matson, Gonzalez, Wilkins, & Rivet, 2008). In order to be given the diagnosis of AD, the individual needed to have one item endorsed on both the socialization and restrictive/repetitive domains and two items endorsed within the communication domain. If these requirements were not met, but three items were endorsed, the individual was given a diagnosis of PDD-NOS.

**DASH-II.** The DASH-II is an informant based assessment examining psychopathology in adults with severe to profound intellectual disabilities. The assessment is composed of 13 subscales, one of which is specific to sleep problems. The entire instrument is composed of 84-items and for each item, data is collected regarding the frequency, severity and duration of the behavior. Frequency is determined based on how often the behavior has occurred in the past two weeks. A score of 0 indicates that the behavior has not been observed in the past two weeks, a score of 1 indicates a frequency of 1 to 10 times, and a score of 2 indicates the behavior as

occurred more than ten times. Severity is also rated with scores of 0, 1 or 2 (i.e., caused no disruptions or damages = 0, caused no damages but interrupted the activities of peers, family, or staff members at least once = 1, caused injury or property damage at least once = 2). The length of time that the behavior has occurred is measured as follows: less than one month = 0, between 1 and 12 months = 1, over 12 months = 2.

The sleep subscale is the focus of this study. This subscale consists of five items. The items are (1) has difficulty staying awake during the day, (2) wakes up frequently during the night, (3) has difficulty getting to sleep, (4) sleepwalks, and (5) wakes up crying or screaming. The sleep subscale of the DASH-II has been found to be a valid measure of sleep problems. Using sleep logs to investigate validity, Matson and Malone (2006) found all items except sleepwalking, which could not be assessed because it did not occur during the data collection period, were significantly correlated with corresponding data on the sleep logs. For example, DASH-II item of “wakes up frequently during the night” was significantly correlated with number of disruptions per night interval. The reliability of the DASH-II has also been well established with .86 mean percentage agreement between respondents and .84 test-retest reliability (Sevin, et al., 1995).

## **Procedure**

The level of ID for each participant was previously assessed by a master’s-level clinician using a standardized intelligence test. The diagnosis for each participant, AD and ID, PDD-NOS and ID, or ID only was then determined. AD and PDD-NOS diagnoses were agreed upon by two doctoral level students who served as raters using checklists based on the diagnostic criteria presented in the *DSM-IV-TR* (APA, 2000) and *ICD-10* (World Health Organization, 1992). Raters separately interviewed two direct-care staff who were familiar with the individual. The

individual needed to meet criteria on both assessments in order to obtain the diagnosis of AD. For the current study, AD and ID, PDD-NOS and ID, and ID only are being compared with respect to sleep problems. To complete the DASH-II, a trained health-care specialist, typically a psychologist, Qualified Mental Retardation Professional, or PhD student interviewed a direct care staff member who had worked with the individual for at least a year.



## STATISTICAL PROCEDURES

In determining the sample, those from the original database with mild or moderate ID were excluded as the current study focused on those with severe and profound ID. In addition, only participants with no missing assessment data were included (i.e., demographic information, an administration of the DASH-II, etc.). As there were more individuals in the PDD-NOS group and ID only groups, a random numbers table was used to obtain a random sample for each group. Equal sample sizes were used in order to protect against any violations of the assumption of homogeneity of variance for the statistics used (Leech, Barrett, & Morgan, 2008).

Next, in order to assess differences between groups on categorical variables of gender, ethnicity, level of ID, presence of a psychotropic medication, and presence of a non ASD diagnosis, *a priori* Chi-square analyses were conducted. For the continuous variable of age, differences between groups were analyzed with using analysis of variance (ANOVA). The three groups did not differ on gender,  $\chi^2(2) = 1.71, p = .425$  or ethnicity,  $\chi^2(4) = 4.18, p = .383$ . Significant differences were found among groups for level of ID,  $\chi^2(2) = 16.96, p = .000$ , presence of psychotropic medications,  $\chi^2(2) = 26.51, p = .000$ , and the presence of a non ASD Axis 1 diagnosis,  $\chi^2(2) = 27.18, p = .000$ . The ANOVA for age was also significant,  $F(2, 168) = 5.48, p = .005$ .

Due to the groups varying significantly on level of ID, presence of psychotropic medications, presence of an Axis I diagnosis, and age, a correlation table was created to examine the relation between these variables (Table 2). Significant correlations were found between age and presence of psychotropic medications, presence of a non-ASD Axis I diagnosis and presence of psychotropic medications, and presence of a non-ASD Axis I diagnosis and level of ID. Though these correlations were significant, the percent of shared variance for each of these

correlations was relatively low, 5.0%, 20.8%, and 3.1% respectively; therefore, all variables were still entered as covariates in the ANCOVA since there is still a large amount of unexplained variance between all of these variables (Field, 2005).

Table 2  
Correlations Between Dependent Variables

	Age	Psychotropic Meds	Axis I (not ASD)	Level of ID
Age	1.00			
Psychotropic Meds	-.223**	.1.00		
Axis I (not ASD)	-.042	.456**	1.00	
Level of ID	-.014	-.067	-.176*	1.00

\*. Correlation is significant at the .05 level.

\*\* . Correlation significant at the .01 level.

From here, an ANCOVA was conducted to compare the groups on total sleep subscale scores with presence of psychotropic medication, age, presence of a non-ASD Axis I diagnosis and level of ID being entered as covariates. In order to determine the appropriate sample size needed, GPOWER, a power analysis computer program, was used *a priori* (Faul, Erdfelder, Buchner, & Lang, in press). For this analysis, a medium effect size of .25 was used, as well as a power of .80 and an alpha of .05. Cohen (1992) states that these are conventional and accepted levels within psychological research for power and alpha. Based on similar studies which have compared sleep problems in those with an ASD, it was expected that differences between groups will not be large, which justified using a medium effect size of .25 (Hare, et al., 2006; Schreck & Mulick, 2000). According to the results of the power analysis, a total sample of 158 was required; however, as the dataset is relatively large and allowed for a larger sample, the maximum number of participants was used.

As pointed out by Field (2005), one of the assumptions necessary for conducting an ANCOVA is that the data be normally distributed. In order to test this assumption, the

Kolmogorov-Smirnov test was conducted to ensure that this assumption had not been violated. The results of the Kolmogorov-Smirnov test were significant indicating that the data was not normally distributed,  $D(171) = .34$ ,  $p = .00$ . Field pointed out that these tests can sometimes come out significant when little variation from a normal distribution has occurred due to a large sample size; therefore, the histogram of the dependent variable (i.e., total sleep scale score) was also examined to determine if the data appear to violate the assumption of normality. Based on examination of the histograms for the total sleep score of the overall sample and the histograms for each individual group, the K-S test results appeared accurate. While these results would indicate the use of nonparametric statistics, for the purposes of this paper, parametric statistics were reported as both nonparametric and parametric statistics were run and no differences in results were found.

The next set of analyses compared the endorsements of the five items on the sleep subscale among the three groups using a multivariate analysis of covariance (MANCOVA). Field (2005) pointed out that performing a MANCOVA before conducting the follow-up ANCOVAs helps to protect against inflating alpha and the chance of finding a false positive. In addition, running a MANCOVA allowed us to examine the relationship between items and trends in how they may be related. Again, GPOWER was used to determine the appropriate number of participants in the sample. Based on a power of .8, alpha of .05, a medium effect size of .25, and 5 response variables, it was determined that a total of 39 participants would be needed. As in the first set of analyses using ANCOVAs, the MANCOVA also assumes that the data is normally distributed for each of the dependent variables (i.e., each of the DASH-II sleep subscale items). The data for each of the items was not distributed normally as the K-S test resulted in the following in regard to each item: item 14,  $D(171) = .47$ ,  $p = .00$ ; item 19,  $D(171)$

= .44,  $p = .00$ ; item 39,  $D(171) = .52$ ,  $p = .00$ ; item 40,  $D(171) = .53$ ,  $p = .00$ ; and item 79,  $D(171) = .53$ ,  $p = .00$ . These results indicate that a series of Kruskal-Wallis should be used; however, in this case, due to a lack of different outcomes with parametric versus nonparametric statistics, only parametric statistics were reported.

Lastly, based on the outcomes of the preliminary analyses, it was decided that a multiple regression be conducted to examine which factors predicted sleep problems in our sample. The factors included in the analysis were age, presence of psychotropic medications, level of ID, Axis I diagnosis (not ASD), and ASD group (Autism, PDD-NOS, or ID only). The variable of ASD group was dummy coded since it used three categorical variables, with the ID only group being the anchor. In this case, the enter method was chosen for the regression. The enter method is appropriate in the case when no *a priori* hypotheses exist regarding the amount each variable will contribute to the model (Field, 2005).

## RESULTS

An ANCOVA was conducted to examine the differences among the three groups, Autistic Disorder and ID, PDD-NOS and ID, and ID, only on total sleep subscale scores from the DASH-II. Of the four covariates entered (i.e., age, level of ID, presence of a non-ASD Axis I diagnosis, and presence of psychotropic medication), the presence of psychotropic medications was the only covariate significantly related to total sleep score,  $F(1, 164) = 18.10, p = .00$ . The ANCOVA showed no significant relationship between total scores on the sleep scale and group membership,  $F(2, 164) = 1.36, p = .26$ .

Next the MANCOVA was conducted in order to examine differences among the three groups on individual items of the sleep subscale. Once again, age, level of ID, presence of a non-ASD Axis I diagnosis, and presence of psychotropic medication were entered as covariates in the analysis. Using the Pillai's trace statistic, which is robust in cases of equal sample size (Field, 2005), there were no significant differences between the three groups in regard to individual items of the DASH-II,  $F(5, 160) = .91, p = .48$ .

Finally, using a multiple regression, factors which may predict sleep problems were examined. In regard to correlations between variables entered as predictors, there were no correlations greater than .90. According to Field (2005), this indicates that the assumption of multicollinearity was not violated and that the predictors are measuring different constructs. Using the enter method, a significant model was found,  $R^2 = .11, F(6, 164) = 3.48, p = .00$ . In this model, based on the correlations between the predictors and the outcome variable of total sleep score, and based on beta values, the only significant predictor is the presence of psychotropic medications (Table 3).

Table 3  
 Predictor Correlations with Sleep Score and Beta Values

	Correlation with sleep score	Standardized Beta
ASD 1	-.02	-.15
ASD 2	.05	-.01
Age	.00	.058
Level of ID	-.21	-.03
Presence of Meds	.30*	.37*
Axis 1 diagnosis, not ASD	.10	0.02

*Note:* \* indicates significance at the .05 level or lower.

## DISCUSSION

The current study aimed to determine if those with AD, PDD-NOS, and severe to profound ID only differed in sleep problems; however, the most important finding was discovered during follow up analyses which examined other predictors of sleep problems. These findings suggest that largest predictor of sleep problems in our sample was the use of psychotropic medications. Due to the many factors affecting sleep, a multiple regression which was completed to explore what variables predicted the presence of sleep problems in our sample. In this analysis, the variables of age, level of ID, presence of psychotropic medication, presence of a non-ASD Axis I diagnosis, and ASD group membership were entered into the regression. Based on the model produced, the only factor found to significantly be related to total sleep score was the presence of psychotropic medications. This finding is somewhat inconsistent with previous research which has lent some support to the effect of each of these variables on sleep. The current study found that increases in age did not result in significantly more sleep problems. As mentioned earlier, previous research has found that sleep problems increase with age (Brylewski & Wiggs, 1999; Smallwood & Stern, 2004) while other research has not found this relationship (Schreck & Mulick, 2000). It is suggested that this relationship be examined more closely in a more controlled study to determine the effect of age on sleep. Similarly, while previous research has found that more severe ID results in greater sleep problems, the current study found no such relation. The reason for this difference may be due to the fact that the current sample only included those with severe and profound ID. When Espie and Tweedie (1991) found differences in sleep based on ID level, they found differences between severe/profound and mild/moderate. Therefore, at the more severe range of ID, sleep differences

may not be as pronounced. Overall, the relationship between many of these variables and sleep in this population remains to be unclear and complex.

Based on the findings of the current study, there are clear implications for the use of psychotropic medications. Psychotropic medications were the only variable which significantly predicted sleep problems in our sample. Previous research has shown that these medications may be overprescribed in this population. More specifically, psychotropic medications may be used to control behavior problems in individuals with no psychiatric diagnoses (Matson and Wilkins, 2008). In our sample, 16% of participants that were taking psychotropic medications had no Axis 1 diagnoses. Due to these medications contributing to sleep problems as well as causing other side effects, it is important for prescribers to ensure that medications are necessary so that unnecessary side effects are not experienced. Future research should examine the effect of specific psychotropic medications and how they relate to sleep disorders more closely.

In regard to the other findings of this study, it was predicted that those with AD and ID would have the most sleep problems, followed by those with PDD-NOS and ID, and finally those with ID only exhibit the least amount of sleep problems due to greater impairments in functioning in the first two groups. The three groups were compared based on the total of the sleep subscale while controlling for age, level of ID, presence of a non ASD Axis 1 diagnosis, and presence of psychotropic medications, as these variables have been shown to effect sleep in previous literature (Brylewski & Wiggs, 1999; Fombonne, 2003; Lang, Bouma, Syttema, Kraijer, & Minderaa, 2006; Roth, et al, 2006; Smallwood & Stern, 2004). No significant differences were found among these three groups in regard to sleep problems.

Other analyses conducted aimed to examine if the three groups differed on individual items of the DASH-II sleep subscale. This portion of the study was exploratory in nature. When



examining the five sleep subscale items, while controlling for factors that have previously been found to affect sleep, no differences were found among those with AD and ID, those with PDD-NOS and ID, and those with ID only.

When examining the results of the current study, several other considerations and possible limitations need to be recognized. First, many other studies examining sleep in those with ASD and ID have used children. It is possible that the factors affecting sleep problems change throughout the life time. In addition, the current study used a sample from inpatient residential facilities as opposed to the home setting in which many studies with children and parents would be conducted. Residential facilities introduce several other factors to consider. For example, Wiggs and Stores (2001) pointed out that sleeping routines may differ for those in residential facilities. Some caregivers may put clients to bed earlier in order to attempt to reduce caregiver stress. Whether bedtime routines are more consistent in inpatient or home settings remains unknown.

Another consideration for the current study is the type of measure used. The DASH-II uses information from third party informants (i.e., direct care staff or other caregivers). Because these are not objective measurements, like actigraphy or polysomnography, it is possible that these informants introduce some sort of bias in their responding. In addition, there may be qualitative differences in how parents perceive problems in the home compared to direct care staff in residential facilities. This may be due to increased stress that caregivers in residential facilities experience since they are likely caring for multiple residents. Differences in the perception of sleep problems may explain why some factors previously found to contribute to sleep problems were not replicated here.

In terms of the statistical analyses used, the multiple regression has a limitation due to the types of variables entered. Several of the variables (i.e., presence of psychotropic medications, presence of a non ASD Axis 1 diagnosis, and level of ID) were dichotomous variables. As stated by Wolfle and Ethington (1985), using dichotomous variables in a multiple regression can underestimate the true effects of the variable. It is possible that this limitation could have effected to outcome of the results.

In sum, more controlled research is still needed to examine sleep problems in adults with ASD and ID. In sorting through the many factors which may affect the sleep of this population, it would be also be helpful to use both informant reports and objective measures to gain information. In continuing this research, the negative effects of sleep problems in this population will be able to be decreased.

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## **VITA**

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