## Louisiana State University LSU Digital Commons

LSU Doctoral Dissertations

Graduate School

2016

# Adaptive Behavior Deficits in Adults with Autism Spectrum Disorder- Cutoff Scores for the ASD-D-A according to DSM-5 Criteria.

Nicole Cassandra Turygin Louisiana State University and Agricultural and Mechanical College

Follow this and additional works at: https://digitalcommons.lsu.edu/gradschool\_dissertations Part of the <u>Psychology Commons</u>

#### **Recommended** Citation

Turygin, Nicole Cassandra, "Adaptive Behavior Deficits in Adults with Autism Spectrum Disorder- Cutoff Scores for the ASD-D-A according to DSM-5 Criteria." (2016). *LSU Doctoral Dissertations*. 883. https://digitalcommons.lsu.edu/gradschool\_dissertations/883

This Dissertation is brought to you for free and open access by the Graduate School at LSU Digital Commons. It has been accepted for inclusion in LSU Doctoral Dissertations by an authorized graduate school editor of LSU Digital Commons. For more information, please contactgradetd@lsu.edu.

### ADAPTIVE BEHAVIOR DEFICITS IN ADULTS WITH AUTISM SPECTRUM DISORDER- CUTOFF SCORES FOR THE ASD-D-A ACCORDING TO DSM-5 CRITERIA.

A Dissertation

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

in

The Department of Psychology

by Nicole Turygin B.S., University of Florida, 2006 M.A., George Mason University, 2010 May 2016

TABLE OF CONTENTS	TA	BLE	E OF	CON	TENTS
-------------------	----	-----	------	-----	-------

LIST OF TABLES	iii
LIST OF FIGURES	iv
ABSTRACT	V
INTRODUCTION	1
PURPOSE	40
METHOD	41
STUDY 1: RESULTS	50
STUDY 1: DISCUSSION	54
STUDY 2: RESULTS	56
STUDY 2: DISCUSSION	61
GENERAL DISCUSSION	63
REFERENCES	
APPENDIX A: DIAGNOSTIC GROUP ALGORITHM	84
APPENDIX B: IRB APPROVAL	85
VITA	86

## LIST OF TABLES

1. Demographic Information	43
2. Sex and Diagnostic Group	44
3. Measures of Central Tendency for DSM-5 ASD and Non-DSM-5 ASD Groups and DSM-4/DSM-5 ASD Groups Compared to Non-ASD Groups	51
4. Measures of Central Tendency- Log Transform Scores and Corresponding ASD-A Scores	52
5. Sensitivity, Specificity, and Predictive Value Scores Based on ROC analysis	53

## LIST OF FIGURES

1. ASD-D-A Scores for the Control and DSM-5 Groups	53
--	----

#### ABSTRACT

With the release of the DSM-5, measures of ASD must be adjusted to take into account the new diagnostic criteria. In the present study, 337 adults with varying levels of intellectual disability who reside in a residential facility were studied. The present study identified potential cutoff scores on an established measure of ASD symptoms in adults, the ASD-A, and compared adaptive behavior levels between groups identified by DSM-IV-TR and DSM-5. The sample was divided into three groups: non-ASD, ASD according to DSM-5 and ASD according to DSM-IV-TR ASD along DSM-IV-TR was determined based on previous diagnoses, while DSM-5 diagnoses were determined based on an algorithm based on previous criteria and new criteria. The optimal cutoff scores for the ASD-D-A were computed using measures of central tendency and ROC analysis, and presented with corresponding considerations. A MANOVA was conducted to examine differences in adaptive behavior between the new ASD vs. non-ASD group, and the DSM-5, DSM-IV, and non-ASD groups using the VABS-II. Differences in adaptive behavior were not found to be significant between the new DSM-5 and non-autistic groups using the new criteria. Results were discussed in light of the sample and potential implications of the DSM-IV. The changes in diagnostic criteria pose concerns, particularly for adults with intellectual and developmental disabilities (IDD). Changes with respect to new diagnostic criteria and their potential effects on individuals with ASD diagnoses were discussed.

#### INTRODUCTION

Autism Spectrum Disorders (ASDs) refer to a group of neurodevelopmental disorders which first occur in early childhood, and whose symptoms are currently believed to persist for the lifetime of the individual. ASDs are characterized by deficits in socialization and communication, and the presence of repetitive behaviors or restricted interests. Until the release of the 5<sup>th</sup> edition of the *Diagnostic and Statistical Manual* (American Psychiatric Association, 2014), ASDs were defined as comprising five separate diagnoses; Autistic disorder, Asperger disorder (AD), Childhood Disintegrative Disorder, Rett's Disorder, and Pervasive Developmental Disorder- Not Otherwise Specified (PDD-NOS). With the release of the *DSM-5* in 2013, these disorders have been collapsed into a single diagnostic category, which is referred to as Autism Spectrum Disorder (ASD).

Autism spectrum disorder as a distinct disorder has only been widely-recognized as such over the past several decades. In 2013, the American Psychiatric Association changed the manner in which autism spectrum disorders are categorized, described, and diagnosed. This has resulted in a great deal of controversy in the scientific community, as well as among public health officials, individuals with ASD, and their families. The concept of autism as a disorder has been relatively stable over the past few decades, but has shifted since its original inception.

#### History of Autism Spectrum Disorders

The meaning of the word "autism" has shifted from its earliest uses to its current usage denoting the disorder(s) that are referred to as ASD. The term "autism" is derived from the Latin term *autismus* and is further derived from the Greek root *autos*,

referring to excessive self-admiration. The word "autism" refers to the preference for "aloneness" that represented a symptom of the individuals we currently characterize as "autistic" as well as a core symptom of schizophrenia.

Eugen Bleuler, a Swiss psychiatrist, used the term "autism" to refer to a detachment from reality, and the "predominance of inner life" or focus on internal stimuli that he observed in individuals with schizophrenia (Bleuler, 1950). Autism, according to Bleuler, was a secondary symptom of schizophrenia. This symptom manifested when individuals engaged in behavior that ran contrary to society's norms, while those individuals remained oblivious and unconcerned. This lack of concern was due to detachment from reality, either absolute or partial. Examples of such behavior took a variety of forms. They included socially offensive behavior such as emptying bowels at social gatherings. Some examples included a repetitive quality, such as providing repetitively written phrases to one's psychiatrist or writing letters successively without waiting for responses. Another example included requesting items such as keys for a particular purpose but then exhibiting a lack of understanding when the desired item was attained. Indifference to everyday affairs, or hiding beneath blankets as a means to remove themselves from the environment also comprised behavior Bleuler described as "autistic".

Perhaps the first clinician to use the word "autism" to refer to the constellation of symptoms similar to current conceptions of ASD was Bruno Bettelheim. Bruno Bettelheim was a child psychologist who directed the Ortho-genic School for Children with Emotional Problems in Chicago. Bettelheim, in 1943 he posited that symptoms of social withdrawal, language delay, and repetitive behaviors in children resulted from

emotional or psychic stressors caused by environments lacking in stimulation and affection during the earliest years of life. In this hypothesis Bettelheim encouraged others to further investigate his hypothesis that poor parenting, particularly by cold "refrigerator" mothers resulted in developmental problems. Bettelheim continued to support and promote this hypothesis into the 1960s.

The explicit use of the word "autism" was first used to describe to the symptoms that approximate current conceptions of ASD occurred with the release of two seminal articles. ASD as a diagnosis can be attributed simultaneously to two groundbreaking researchers in Germany and the United States. One of these was the Austrian-American physician and father of child psychiatry, Leo Kanner. In his work in the first pediatric psychiatry service at Johns Hopkins University, he observed that certain children seemed to exhibit a particular syndrome of behavior. In his 1943 study, "Autistic disturbances of affective contact" he described 11 narrative accounts of these children with "Kanner syndrome", who exhibited "extreme autistic aloneness". He observed that these children also originated from well-to-do families of Jewish or European origin, and, that there was little warmth between the parents and children.

In Kanner's 1943 article "Autistic Disturbances of Affective Contact", he described a narrative account of 11 children between 2 and 8 years of age with language and social skills impairments who also exhibited behavioral rigidity and odd behaviors. These children were described by their parents as "self-sufficient", and "in a shell". Of the twenty, sixteen of Kanner's children were boys, and four were girls. Thirteen acquired language, and the other seven were described by Kanner as "mute" at the time of his examinations. Those children who were able to speak had "good rote"

memory, but the words were not used to convey meaning to others. The children described as "mute" were often able to emit single words or phrases, in a manner described by Kanner as "delayed echolalia". Kanner described these children as having a "good relation" to objects, and indicated that they appeared able to play happily for hours on their own. In contrast, these children often ignored others around them and preferred instead to continue to play with preferred objects.

In his "Early Infantile Autism," Kanner described the majority of these children as "feeble-minded". However, he described these children as having "strikingly intelligent physiognomies". Kanner stated that he was not able to assess or estimate the intellectual abilities of these children. For example, he did not administer the Binet or other formal measure of intellectual functioning due to "limited accessibility." He did report that these children "did well" with the Seguin form board, a measure of speed and memory.

Both Kanner and Asperger described the syndrome as present at birth. In 1944, Austrian graduate student Hans Asperger published "Die 'Autistischen Psychopathen im Kindesalter". "Autistic Psychopathology in Childhood" remained largely undiscovered in the USA until it was translated by Uta Frith in 1991(Asperger, 1991). The four children described by Asperger presented with impairments in communication and socialization, but in contrast to Kanner's "mute" children, the children observed by Hans Asperger demonstrated typical verbal abilities.

Kanner and Asperger both described as "autistic", those children presenting with deficits in socialization and communication who displayed repetitive or restricted interests. These children had been given a variety of diagnoses. Children with autism

were classified by Kanner as "pseudo-defective". "Defectiveness" and "Feeblemindedness," represented accepted diagnostic nomenclature for the classification of individuals presently diagnosed with "Intellectual Disability". Feeble-mindedness was categorized by Kanner into three categories: absolute, relative, and apparent/pseudodefective. The "absolute feeble-minded" individuals were believed to have irreversible pathology. The "relative feeble-minded" could be habilitated by "cultural training", and the "apparent or pseudo-feeble minded" included autistic and schizophrenic children. A colleague of Kanner, Leon Eisenburg (1957) followed 63 of Kanner's autistic children for four or more years. Three were classified as having resulted in a "good" outcome as they developed, and were able to function well academically and socially. Fourteen had a "fair" outcome and were able to attend school and perform at grade level, but were described as "deviant in personality". The majority had a "poor" outcome which was defined as "feeble-mindedness" and/or psychotic behavior.

A distinction between those deemed "feeble-minded", "schizophrenic", and "autistic" was not evident in the public policy of the time. Clemens Benda (1952) noted that the word "idiocy" shares the same derivation from Greek that "autism" shares from Latin. The term "idiot" referred to "one who lives in his own world, a private person or recluse" and was originally a generic term to refer to persons with impairing social and communication deficits who today may have been classified as having either an autistic disorder or an intellectual disability. Initially, a variety of terms were used interchangeably to describe individuals with the constellation of symptoms we could today classify as an ASD, including; dementia praecocissima, dementia infantilis, infantile autism, symbiotic psychosis, schizophrenic syndrome of childhood,

pseudopsychopathic schizophrenia, and latent schizophrenia" (Rutter, 1972). As an interesting foreshadow of current research findings, Kanner identified autism as a "unique syndrome recognizable as early as the first or second year of life."

The conceptions of autism continued to change throughout its early history. Bernard Rimland, an experimental psychologist who had a son diagnosed with autism, began to investigate the disorder. The resulting book, *Infantile Autism: The syndrome and its Implications for a Neural Theory of Behavior* reviewed the literature, controversies, and findings of the disorder (Rimland, 1964). Rimland was perhaps among the first to question that the disorder was psychogenic in nature, and therefore environmentally-based. Rimland continued to advocate for parents and families of individuals with the disorder, started the National Society for Autistic Children, and the Institute for Child Behavioral Research; one of the first institutes dedicated to determining the cause and finding treatment for the disorder.

#### The Changing Conceptions of ASD

The constellation of symptoms we currently characterize as autistic were captured within the context of a schizophrenia-spectrum disorder in the mid-20<sup>th</sup> century. According to the *DSM-I* (1968), "autism" was deemed a symptom of "Schizophrenic reaction, childhood type". This diagnosis was differentiated from other manifestations of schizophrenia by 'immaturity and plasticity of the patient at the time of onset of the reaction". In the *DSM-II* (1968), "autism" continued to be subsumed under the category "Schizophrenia, childhood type". Childhood type schizophrenia was characterized by "autistic, atypical, and withdrawn behavior; failure to develop identity separate from the mother's; and general unevenness, gross immaturity, and inadequacy

of development. These developmental defects were said to possibly "result in mental retardation, which should also be diagnosed". It was with this revision of the DSM that ASD came to explicitly resemble the construct similar to that, which is used in the recent versions of the diagnostic manual. Withdrawn behavior, coupled with developmental deficits differentiated the syndrome from developmental delay or ID.

**DSM-III.** The *DSM-III* was also notable in that it divided "autism" into five separate disorders; Infantile Autism, Residual Infantile Autism, Child Onset Pervasive Developmental Disorder (COPDD), Residual Childhood Onset Pervasive Developmental Disorder, and Atypical Pervasive Developmental Disorder. "Childhood type schizophrenia" became "infantile autism" and required onset before 30 months of age. Diagnostic criteria for infantile autism included "pervasive lack of responsiveness to other people (autism)", gross deficits in language development, if speech is present, peculiar speech patterns such as immediate and delayed echolalia, metaphorical language, pronominal reversal, bizarre responses to various aspects of the environment (e.g. resistance to change, peculiar interest in or attachments to animate or inanimate objects, and absence of delusions, hallucinations, loosening of associations, and incoherence as in schizophrenia; American Psychiatric Association, 1980).

Childhood onset pervasive developmental disorder required impairments in social relationships, constricted/inappropriate affect, resistance to changes in the environment, oddities in motor movement, speech abnormalities, hyper- or hypo-sensitivities to sensory stimuli, and self-mutilation, first occurring between 30 months and 12 years of age, with the absence of delusions or hallucinations. Residual forms of infantile autism and childhood onset pervasive developmental disorder were diagnosed

the criteria had been met previously but the individual no longer met criteria for the disorder. Atypical Pervasive Developmental Disorder included those children who did not meet full criteria and corresponds to Pervasive Developmental Disorder-Not Otherwise Specified in the DSM-III-TR (Spitzer & Siegel, 1990).

**DSM-III-R**. The revision of the DSM-III with the DSM-III-R further refined the criteria and introduced the diagnostic label of "Autistic Disorder" to encompass infantile autism and COPDD. This change was undertaken due to the recognition that autism was not a syndrome of infancy. Eight of 16 diagnostic criteria were now required to meet criteria for Autistic Disorder. For this disorder, additional criteria were added in order to include a broader range of deficit across developmental and chronological ages (Volkmar, Bregman, Cohen, & Cicchetti, 1988). Pervasive Developmental Disorder encompassed those who did not meet full criteria for the disorder.

**DSM-IV and DSM-IV-TR.** The *DSM-IV* introduced the five disorders comprising the autism spectrum; Autistic Disorder, Asperger syndrome, Rett's Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder- Not Otherwise Specified. Asperger Disorder omitted the criteria for language delay. For a diagnosis of autistic disorder, two symptoms from criteria A corresponding to qualitative impairments in social interaction must be present; as well as one from criteria B which involved impairments in verbal and nonverbal communication and imaginative activity, and one from C, which corresponded to a "markedly restricted repertoire of activities and interests". Autistic disorder required onset during infancy or early childhood, with "childhood onset" being used as a specifier for those who developed symptoms of the disorder after 36 months of age.

Autistic Disorder in the DSM-IV-TR retained separate criteria for communication, social interaction, and restricted and repetitive behavior, with a specifier that one of these criteria manifested prior to age 3. With the DSM-IV and DSM-IV-TR, researchers defined the "autism spectrum" to include individuals with diagnoses of Autistic Disorder, Rett's Disorder, Asperger Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder- Not Otherwise Specified.

During the time of DSM-IV and DSM-IV-TR, "Autism Spectrum Disorders" or "Pervasive Developmental Disorders" became the designated diagnostic terminology. Consistent with the DSM-IV and DSM-IV-TR, the disorders included Childhood Disintegrative Disorder, Asperger's Disorder, and Pervasive Developmental Disorder-Not Otherwise Specified (Lord, Cook, Leventhal, and Ameral, 2000). Rett's disorder has also historically been classified as an ASD/PDD due to the similarity of symptoms. However, recently, researchers have treated Rett's disorder as separate due to its discrete genetic cause and distinct course of the disorder. A clear genetic basis for the disorder has been identified; mutations on the methyl-CpG-binding protein 2 (MECP2) gene (Kilstrup-Nielsen & Landsberger, 2015; Percy, 2014). In contrast, no discrete genetic bases have been identified for Autistic Disorder, Asperger disorder, Childhood Disintegrative Disorder, or PDD-NOS.

#### DSM-5 Diagnostic Criteria

In 2013, with the release of the *DSM-5*, came a controversial and widelydiscussed change to the diagnostic criteria for ASD. In the new criteria, Rett's disorder was removed, and Asperger Disorder, Autistic Disorder, and Childhood Disintegrative Disorder were refined and collapsed to one overarching "Autism Spectrum Disorder". In addition, Pervasive Developmental Disorder- Not Otherwise Specified (PDD-NOS) was

eliminated. PDD-NOS was an ASD that was diagnosed in children with significant impairments in socialization, communication, and exhibited restricted/repetitive behavior who did not meet full criteria for one of the other disorders. The elimination of PDD-NOS resulted in the inability for a clinician to provide an individual with a sub-threshold diagnosis of autism for those cases in which symptoms were present and impairing, despite those children not meeting all of the criteria for the disorder. Recently, researchers have identified that the new criteria have been excluding significant numbers of individuals who previously were diagnosed with PDD-NOS (Mayes, Black, & Tierney, 2013).

The symptoms of communication and socialization deficits were became a single symptom category, social communication. In the DSM-IV-TR, symptoms for socialization and communication each received their own specific diagnostic criteria, which were collapsed to form the DSM-V criteria. Some researchers considered this change to be "more parsimonious" as compared to previous diagnostic criteria (Frazier et al, 2012). Others, including authors who provided some of the theoretical bases for the original criteria, argue that much has been lost with this change, as the current diagnostic criteria represent a movement away from some of the foundational theories related to the disorder, such as the Wing and Gould Triad.

Wing and Gould coined the "Wing and Gould Triad" of ASD symptoms in the late '70s and early '80s (Gould, 1982; Wing, 1981b; Wing & Gould, 1979; Wing, Gould, & Gillberg, 2011). This triad consists of impairments in social interaction, social communication, and social interaction. Social interaction impairments related to difficulties with nonverbal signs related to interest between individuals and included

affectionate contact and responses to smiling and physical affection. Impairments in social communication related to the ability to engage in verbal and nonverbal communication with others, including joint referencing. Social imagination impairments related to the abilities related to theory of mind, and the ability to understand and predict the consequences of ones' actions. This criterion was not included in any of the original criteria. However, the triad is still considered relevant to researchers who continue to consider and explain these constructs. Happe and Ronald (2009) relate the different impairments to specific genetic influences, and regard each portion of the triad as distinctly related to particular genetic influences. Other researchers have been examining potential cognitive underpinnings to these individual deficit areas (Ecker, Marquend, Mourcio-Miranda, Johnston, Daly, & Brammer, 2010; Yoshida, Dziobek, Kliemann, Heekeren, Friston, & Dolan, 2010).

The addition of sensory symptoms has been a less-addressed addition to the criteria, and is included under the restricted and repetitive behaviors category. However, sensory differences have been widely observed to occur at high rates in individuals with ASD, but this symptom has not been the focus of many studies. This addition is less controversial. For example, researchers have observed a positive relationship between social-communication impairment severity and hyporesponsivness to stimuli and sensory seeking behavior in individuals with ASD (Watson, Patten, Baranek, Poe, Boyd, Freuler, & Lorenzi, 2011). Sensory symptoms, though not included in previous diagnostic criteria, were widely included in measures of ASD.

In contrast to the specification that symptoms of the disorder became evident prior to age 3, the DSM-5 specifies only that symptoms were present in the early

developmental period. It further elaborates that the symptoms may not have become evident until the individual's social demands exceeded their capacities. This criterion also allows for the deficits to have been masked by compensatory strategies later in life. The change in this criterion allows for presentations for which symptoms of the disorder were not expressly evident in early childhood.

In addition, the disturbances must not be better explained by intellectual disability or global developmental delay. It further specifies that diagnoses of cooccurring ASD and ID must be supported with lower social communication scores compared to the individual's developmental level. This is, in practice, similar to how many clinicians diagnosed ASD previous to these explicit criteria. However, the degree to which social communication must be lower, coupled with a heterogeneous presentation of skill and deficit profiles among individuals with ID will likely pose challenges for the diagnosing clinician.

The differences between disorders were somewhat captured by the addition of specifiers for the presence of ID, language impairment, association with a known medical, genetic, or environmental factor, association with another neurodevelopmental, mental, or behavioral disorder, or with catatonia. The DSM-5 also added specifiers for deficits in social communication and restricted, repetitive behaviors. The specifiers are "Level 1, requiring support", "Level 2, requiring substantial support", and "Level 3, requiring very substantial support". These specifier levels include narratives to assist the clinician in determining the level of impairment observed within these two symptom categories. Overall changes made by the DSM-5 have been highly contested by autism researchers due to narrowed criteria and decreased specificity.

Some researchers have observed the DSM-5 criteria for autism to be overly restrictive. McPartland, Reichow, and Volkmar (2012) determined that 60.6% of individuals diagnosed with ASD in a field trial would continue to meet diagnostic criteria using DSM-5 criteria and DSM-IV, and 39.4% would not continue to meet criteria. The new diagnostic criteria were highly specific, and excluded 94.9% of individuals who did not meet criteria for the disorder.

#### Social Communication Disorder

Social communication disorder is a new disorder first introduced with the introduction of the DSM-5. This disorder occurs when an individual exhibits persistent difficulties in the social use of nonverbal and/or verbal communication. To be diagnosed with social communication disorder, an individual must exhibit all of the following: deficits in using communication for social purposes, impaired ability to change communication according to context, difficulties with following rules for conversation, including the use of nonverbal communication, and difficulties with non-explicit language including humor. These individuals should not exhibit restricted and repetitive behavior. In addition, these deficits must correspond to functional limitations with respect to one's social life, occur early in the developmental period (in a manner similar to the criteria specified for ASD), and not be attributed to another medical or neurological disorder or learning or intellectual disability.

Some researchers have raised the question as to whether social communication disorder is related to ASD, as it relates to difficulties with pragmatics and social uses of communication, but excludes the presence of restricted and repetitive behavior. Some researchers suspect that this disorder was included to capture more mild forms of ASD (Tanguay, 2011). This diagnosis, prior to its inclusion in the DSM-5 was considered

only by speech pathologists as a disorder. Currently, researchers have focused on the degree to which pragmatic language and social communication are overlapping constructs. This has not been well established, but there is some evidence that the two concepts are distinct, and as a result social communication disorder may be best described as a language disorder. However, the results thus far are mixed. Norbury (2013) outlines some of the relationships between these two constructs: lack of social instinct can result in delayed developmental language and vice versa. Some individuals who previously carried a diagnosis of ASD may receive this diagnostic criteria; in a study of individuals with social communication disorder, those with the disorder were likely to have met DSM-5 criteria for an ASD, largely as a result of sensory interests and repetitive speech, and that language capacities were observed to change greatly with age (Bishop & Norbury, 2002).

#### The Controversy over DSM-5 Criteria

The changes in diagnostic criteria have proven controversial among clinicians and researchers. Some researchers have commended the new criteria for its emphasis on additional diagnostic specificity with the addition of severity levels and numerous specifiers. Specifiers include: level of intellectual and language impairment, specific medical, genetic, or environmental factors, and neurodevelopmental, mental, or behavioral disorders that often co-occur with ASD. The new diagnostic guidelines result in diagnoses that are intended to be highly specific and detailed, and provide greater information compared to that which was incorporated in the DSM-IV-TR (Lai, Lombardo, Chakrabarti, & Baron-Cohen, 2013). Other researchers expressed concern that the

new diagnostic criteria are restrictive, exclude a number of individuals with impairments in the core symptom areas of ASD, will result in decreased rates in diagnosis, and limit access to treatment particularly among individuals with less severe presentations (Mayes, Calhoun, Murray, Pearl, Black, & Tierney, 2014).

The subsuming of the autism spectrum disorders into one overarching disorder has been controversial within the field of psychology, and among families of individuals with ASD as well as individuals with ASD themselves. The elimination of the individual disorders and shifts in criteria pose difficulties for researchers who study individuals with ASD, and shifting in diagnostic criteria may damage the ecological validity of these studies.

Researchers who supported the change cited a lack of diagnostic stability of the specific ASD subtypes. In particular, the distinction between AS and HFA resulted was determined by whether an individual had a history of language delays or deficits. Findings from recent research seem to indicate that the separate disorders as outlined in the DSM-IV-TR were somewhat nebulous, and that few individuals fit neatly into one particular disorder. A downside to the elimination of these disorders is that specific abilities and impairments that occur in individuals with ASD vary widely. Recently, based on genetic findings, as well as specific behavioral presentations, researchers now hypothesize that there may be several "autism spectra" or "autisms" rather than a single ASD disorder as described in the present criteria (Wing, Gould, & Gillberg, 2011). However, other than the disorders that have been long known to be associated with ASD and ASD-like symptoms (e.g. Rett's disorder, Fragile X syndrome, Angelman

syndrome) researchers have not yet begun to delineate the specific autisms in a manner that can be translated into clinical practice.

#### **Responses from Organizations**

Controversy over the new diagnostic criteria was highly publicized. Discussions over the merits and limitations of the new criteria occurred within the scientific community and were reported by conventional media. Notably, one of the most well-known national organizations for ASD, Autism Speaks (2015), maintains a website which provides news and updates on the criteria, and the surrounding controversy. Autism Speaks expressed concerns that the narrowed criteria may result in a reduction of services for individuals on the spectrum. The U.S. Department's Health and Human Services Interagency Autism Coordinating Committee (IACC, 2014) echoed these concerns and underscored the issues related to the effect of the new diagnostic criteria on research, and that researchers should be mindful of these changes when choosing to exclude or include individuals in further studies. In addition, the IACC provided questions related to who will now be identified as having ASD, how they will be identified, and identified implications for both practice and policy.

Research questions related to who will be identified under the new criteria highlighted the potential for the new criteria to exclude individuals with the disorder, the effect of the new disorder on the diagnosis of specific groups (minorities, females, adolescents, and adults), symptom profiles, and severity ratings. They also questioned the removal of the requirement of symptom onset to occur prior to age 3, and the effect this may have on the identification of young children with the disorder. Finally, they

questioned the reliability and validity of Social Communication disorder and the degree to which this disorder is distinct from ASD.

Concerns related to the way in which individuals with ASD will be identified raised concerns about the changes of the criteria on the existing diagnostic and screening instruments, and instruments related to Asperger Disorder in particular. In addition, questions related to how individualized diagnoses, presumably related to the specifiers, will be given. They mention the need for researchers to identify not only functioning with respect to core symptoms but overall strengths and weaknesses, as well as co-occurring disorders. Additionally, they raised concerns related to how these changes will affect how clinicians, other professionals, the community, and researchers identify ASD and implications related to education, service systems, and how the changes will be aligned with school policies.

Finally, the meaning of a diagnosis of ASD was questioned on several levels. First, they questioned the effect of these changing criteria on the culture of individuals with ASD, especially those with diagnoses of Asperger disorder. It was also questioned how the severity levels will be used with respect to acquisition of services. In addition, the degree to which the severity levels will affect the understanding of etiology and clinical management of the disorder has yet to be determined. They questioned how research on DSM-IV-TR diagnosed individuals will apply to the new population, and how datasets of DSM-IV-TR diagnosed individuals will be integrated with future data sets in which individuals will presumably be diagnosed along DSM-5 criteria. Finally, they voiced concerns with respect to how diagnostic changes will impact those with deficits in the core symptom areas who would have previously been diagnosed with

ASD, and how our current knowledge of treatment will relate to the new diagnosis of social communication disorder.

#### **Responses from the Research Community**

The research community also engaged in dialogue related to implications of the new criteria. Edward Ritvo (2012) published a letter to the editor in the Journal of Autism and Developmental Disorders outlining the major concerns of the new criteria. He and cosigning authors stated that the diagnostic changes were not based on empirical evidence and that 20-40% of individuals with the disorder will no longer meet criteria. In addition, he raised some practical concerns related to difficulties that the new criteria will present, as previous studies may become incompatible with current diagnostic criteria, and that these new criteria will cause difficulties for those who are engaging in longitudinal studies. Other concerns related to challenges retraining clinicians and potential loss of services as resulting from diagnostic changes were addressed as well.

The controversy resulting from proposed changes in criteria was observed in scientific journals as well as in the media. Joseph Buxbaum and Simon Baron-Cohen (2013) invited two key experts on autism to address their respective concerns in the journal *Molecular Autism*. Fred Volkmar, the primary author of the DSM-IV autism and Pervasive Developmental Disorders criteria, and Catherine Lord, a key researcher on the Neurodevelopmental Disorders workgroup who was instrumental in helping to develop criteria for the DSM-5, were each invited to discuss their perspectives on the changing criteria.

Volkmar and Reichow (2013) posed concerns related to the effect of the changing criteria on longitudinal studies of ASD. They also expressed concerns that

individuals, particularly those with less severe presentations of the disorder, will no longer meet criteria and that this may result in a loss of services for those individuals, as well as concerns related to the removal of AS. They raised a question of whether the removal of PDD-NOS may result in a disorder similar to "Kanner's disorder" in presentation compared to the population that now receives diagnoses of ASD. In contrast, however, they lauded the change in terminology from Pervasive Developmental Disorder to Autism Spectrum Disorder and the concept of creating dimensions of impairment within the disorder. They stated that the core symptom areas of socialization and communication deficits were not previously collapsed, as factor analyses of symptoms were not straightforward, yielding two, three, or five factor solutions and that the three factor model was chosen for reasons of historical continuity (Norris, Lecavalier, & Edwards, 2012).

Grzadzinski, Huerta, and Lord (2013), in a published response, stated that the changes are positive; the change of the disorder into a single diagnosis removes the issue related to low reliability of the individual ASD diagnoses. They posited that the inclusion of sensory abnormalities reflects a common symptom observed by many researchers. According to Huerta, Bishop, Duncan, Hus, and Lord (2012), 90% of those who met criteria along DSM-IV criteria also met DSM-5 criteria, which were also observed to demonstrate higher specificity. They also emphasized that the new criteria are more inclusive of the wide range of language and cognitive abilities of individuals with ASD. Moreover, specifiers are now used to indicate when regression occurs (e.g. in language, social skills, and motor and/or adaptive functioning) and provide for additional detail. Grzadzinski and colleagues (2013) emphasize that future researchers

should use a dimensional approach and account for many variables that are often affected in ASD (age of onset, and cognitive, language, social, and adaptive skills).

#### **Etiology and Prevalence**

Autism is a worldwide public health concern, particularly as researchers have been unable to identify a discrete cause, and because the prevalence of the disorder continues to increase. Autism is occurring at high prevalence rates all over the world. Observed prevalence estimates have been as high as 1 in 68 (Centers for Disease Control, 2014). Variation in observed rates may be due to whether the figures include more restrictive criteria (i.e., autistic disorder) only, or include all of the disorders including PDD-NOS. In an Icelandic study of ASD prevalence in children born between 1974 and 1983, a prevalence of 3.8 per 10,000 was observed, and increased to 8.6 per 10,000 for children born between 1984 and 1993 (Magnusson & Saemundsen, 2001). In a Taiwanese study of the disability registers between 2000 and 2007, researchers observed an increase of 249.5% of people with the disorder, with the most dramatic increases observed among school-aged children (Lin, Lin, & Wu, 2009). Current estimates of ASD prevalence range from as high as 1 in 68 to a more conservative 1 In 150 (CDC, 2014b; Lord & Bishop, 2010; Matson & Shoemaker, 2009).

Public interest in ASD has increased in recent decades, and it is currently considered to be among the top public health concerns worldwide. Both prevalence and awareness of the disorder have increased. Increased awareness of ASD in much of the world has resulted in an increase in services available to individuals diagnosed with the disorder. Early intervention has become a focus of services provided for children with ASD. In 2006, the American Academy of Pediatrics recommended screening for all children beginning at 9 months of age, and again at 18- and 24-month

checkups. In contrast to its previous guidelines released in 2001, in which general surveillance for developmental disorders was recommended, the 2006 guidelines were the first to specifically recommend screening of children and education for parents on the development of communication skills (American Academy of Pediatrics, 2001; American Academy of Pediatrics, 2006).

One major concern of the rising rates of ASD relates to questions resulting from its multifactorial nature and heterogeneous presentation. There are few variables that have been found that relate to the prevalence of ASD. No significant differences have been found with respect to race or ethnicity (Fombonne, 1999; Horovitz, Matson, Rieske, Kozlowski, & Sipes, 2011). Despite the many methodological challenges involved in comparing ASD prevalence with respect to geography, estimates of the disorder do not vary significantly between North America, Europe, and the Western Pacific (Elsabbagh et al, 2012). However, few prevalence estimates from developing countries have been conducted. Previous researchers have observed that the most severe and disabling cases of ASD tend to be identified in developing countries (Daley, 2004; Juneja, Mukherjee, & Sharma, 2004).

In all populations, however, males are observed to have a higher prevalence of the disorder, with an approximate ratio of 4:1 (Wing, 1981c). Consistent with Hans Asperger's observations that autism represented an excess of male intelligence, Baron-Cohen (2002) believes that autism represents an "extreme male brain" in which the brain is extremely predisposed to "systemizing" or correlating events with outcomes (Baron-Cohen, 2002). In contrast, this brain is less adept at "empathizing" which involves attributing mental states to others and being able to provide appropriate

affective responses. However, findings linking "maleness" to the deficits in ASD have been mixed (Falter, Plaisted, & Davis, 2007; Teatero & Netley, 2013).

**Genetic Bases for ASD.** Much research has been devoted to identifying the cause of ASD/PDD. Currently, no one discrete cause has been identified, despite genome-wide association studies and whole-exome sequencing. Geneticists have identified syndromic, mitochondrial, point mutations, and polygenetic associations between the genotype and the behavioral phenotype that is currently labeled "autism" (Persico & Napolioni, 2013). A variety of genetic/genomic disorders in which ASD is often observed include fragile X syndrome, tuberous sclerosis, neurofibromatosis, phenylketonuria, Smith-Lemli-Opitz syndrome, Cohen syndrome, Sotos Syndrome, Angelman Syndrome, Cornelia de Lange syndrome, and Down Syndrome (Persico & Napoliioni, 2013). Researchers have also identified other, rare monogenetic forms of autism, such as Rett's disorder which comprise approximately 1% of individuals with a diagnosis (Persico & Napolioni, 2013).

ASDs are also linked to copy number variants, which are segments of DNA consisting from 50 to thousands of base pairs with deletions, insertions, inversions, duplications, or re-combinations. These are present in typical controls (1-3%) but are more prevalent in individuals with ASD (6-10; Persico & Napolioni, 2013). Neroligins, notably SHANK and neurexin are also frequent locations for mutations linked to behavioral phenotypes. Morphogenetic, growth-regulating genes, and calcium-related genes are also linked to the autism behavioral phenotype.

Despite an observed genetic link in some cases, approximately 90-95% of cases of ASD occur without a known cause (Caronna, Milunsky, & Tager-Flusberg,

2008). Twin studies have observed concordance between 69-95% of monozygotic twins and 3-8% of dizygotic twins (Dawson, 2008) and will occur in a younger non-twin sibling at rates between 2-35% (Zwaigenbaum et al, 2007). Autism spectrum disorder occurs at a high rate with certain other disorders. For example, 15-25% of individuals with fragile X syndrome also meet criteria for ASD (Rogers, Wehner, & Hagerman, 2001).

Much research has focused on a potential link between vaccines and the development of ASD. According to a now-discredited study by the gastroenterologist Andrew Wakefield and colleagues (1998), intestinal inflammation observed in children with ASD was claimed to be linked to an exogenous influence on brain functioning, and suggested that the measles, mumps, rubella (MMR) vaccine may be this exogenous influence. Compelling findings from this study resulted in a wave of studies attempting to replicate his results. Most researchers did not observe any link between vaccines and the development of ASD; in those studies in which relationships were observed, the results were equivocal. Some researchers have argued strongly against a causal relationship between vaccination and ASD (Madsen, Hviid, Vestergaard, Schendel, Wohlfahrt, Thorsen, Olsen, & Melbye, 2002).

#### Neurobiological bases for ASD.

Much research has been conducted to determine the biological source of ASD. Definitive results have been elusive. Early studies in the neurobiology of ASD largely implicated dysfunctions within the amygdala (Amaral & Corbett, 2002). Bauman and Kemper (1984) observed structural differences (cell clusters) in the amygdalae of autistic, compared to control brains. In addition, they observed structural changes

throughout the brain, including within the hippocampus, septum, and cerebellum. Other researchers, however, have not produced consistent comparable results. The amygdala, as a center key social center of the brain, has been implicated. Animal research on the effects of lesions on the amygdala resulted in inexpression, lack of eye contact, and stereotypies (Bachevalier, 1994). Interestingly, amygdala volume has been found to correlate positively with anxiety/depression levels in individuals with ASD (Juranek, Filipek, Berenji, Modahl, Osann, & Spence, 2006).

However findings from animal studies have not translated neatly to humans. Results of neuroimaging studies on the amygdala have been complex and often contradictory. Gross volume of the amygdala in individuals with ASD has been observed to be both increased (Abell et al, 1999) and decreased (Aylward, Minshew, Goldstein, Honeycutt, Augustine, Yates, Barta, & Pearlson, 1999; Howard, Cowell, Boucher, Broks, Mayes, Farrant, & Roberts, 2000). Additionally, individuals with ASD have been observed to have less activation in the amygdala when engaging in social tasks compared to controls (Ashwin, Baron-Cohen, Fletcher, Bullmore, & Wheelwright, 2001; Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin, & Williams, 1999).

The cerebellum has also been largely implicated in the disorder. For example, a loss of Purkinje cells within the cerebellum has been a repeated finding, but is also observed in individuals with seizure disorders (Ritvo, Freeman, Scheibel, Duong, Robinson, Guthrie, & Ritvo, 1986; Bailey, Luthert, Dean, Harding, Janota, Montgomery, Rutter, & Lantos, 1998).

Widespread neurological differences in autism have been observed inconsistently across many of the neurological systems. For example, cortical thinning

has been observed in adolescents and adults with the disorder (Wallace, Eisenberg, Robustelli, Dankner, Kenworthy, Giedd, & Martin, 2015). As a result, one recent hypothesis that has been somewhat supported involves changes in neuroplasticity in individuals with ASD. Neuroplasticity refers to the ability of neurons to organize, reorganize, and alter their anatomical and functional connections according to environmental input (Desarkar, Raijji, Ameis, & Daskalakis, 2015).

#### ASD and Co-occurring Conditions

The impact of ASD upon individuals and populations is significant. The deficits observed in ASD persist across the lifespan, and often co-occur with other conditions that are associated with lifelong impairments. The most prevalent and disabling condition that frequently co-occurs with ASD is intellectual disability (ID). ID co-occurs with ASD at a rate of between 50 and 70 percent (Chakrabarti & Fombonne, 2001; Fombonne, 1999; LaMalfa, Lassi, Bertelli, Salvini, & Placidi, 2004; Volkmar, Lord, Bailey, Schultz, & Klin, 2004). Independently of other conditions, ID results in significant impairment and is linked to a wide array of long-term disadvantages including the need for lifelong supports with respect to daily living, maintaining and occupation and social relationships, and recreation.

The co-occurrence of ID and ASD in individuals results in decreases in social functioning, and adaptive behavior skills than by either disorder alone (Matson, Mayville, Lott, Bielecki, & Logan, 2003). Adults with autistic disorder score significantly lower on the social subscale of the Vineland Adaptive Behavior Scales (VABS) compared to adults with PDD-NOS or ID (Njardvick, Matson, & Cherry, 1999). Autism, compared to ID, tends to be associated with higher rates of co-occurring psychiatric disorders (Brereton, Tonge, & Einfeld, 2006). Moreover, individuals with diagnoses of ID and

ASD are at an even more elevated risk (Tsakanikos, Costello, Holt, Bouras, Sturmey, & Newton, 2006). Individuals with ASD and ID have been observed to experience the poorest outcomes with respect to independent living, education, and occupational attainment (Marriage, Wolverton, & Marriage, 2009).

**Anxiety**. Anxiety disorders co-occur with ASDs at high rates (between 40-84%), regardless of level of intellectual functioning (Mayes, Calhoun, Murray, Ahuja, & Smith, 2011). However, prevalence is higher among individuals with higher verbal abilities (Mayes, Calhoun, Murray, & Zahid, 2011). In a study by Maddox and White (2015), approximately 50% of adults with high-functioning ASD had co-occurring social anxiety disorder. Individuals with high-functioning AS have been observed to receive elevated scores compared to typically-developing children with respect to anxiety, social worries, separation anxiety, and obsessive-compulsive disorder (Gillott, Furniss, & Walter, 2001).

Presentations of anxiety seem to vary also with autism presentation. Wing and Gould (1979) separated individuals with ASD into specific behavioral subtypes. The "active but odd" subtype evinced higher levels of anxiety compared to the "aloof" subtype, which appeared to experience less anxiety. High-functioning adults with ASD were at heightened risk for social anxiety disorder. This information is particularly interesting, as many assume that individuals with ASD have decreased interest in social interaction. In addition, anxiety disorders are frequently observed in family members of individuals with ASD (Matson, Hess, & Boisjoli, 2010).

Attention-Deficit/Hyperactivity Disorder (ADHD). ADHD also co-occurs at high rates in individuals with ASD, at rates between 20-70% (Matson, Rieske, &

Williams, 2013; Rommelse, Franke, Geurts, Hartman, & Buitelar, 2010). According to DSM-IV-TR, ADHD could not be diagnosed in the presence of an ASD. However, with the release of the DSM-5, this was changed. This change was consistent with research in the area, as researchers have observed neurobiological links between the two disorders (Van Der Meer et al, 2012). In addition, other researchers have observed phenotypic overlaps between the two disorders. One such example, by Sinzig, Walter, and Doepfner (2009) identified "inattentive-stereotyped" and "hyperactive-communication impaired" as ADHD presentations within an ASD sample.

**Depression.** Depression is a disorder in which an individual experiences a sad, depressed mood, and/or a loss of interest or pleasure in things that were previously enjoyed. Individuals with ASD are at higher risk for depression compared to the general population (Ghaziuddin, Ghaziuddin, & Greden, 2002; Gillberg & Billstedt, 2000; Bradley, Summers, Wood, & Bryson, 2004). Matson and Williams (2014) found that depression co-occurs in approximately 50% of individuals with ASD. In an early study of adults with Asperger syndrome, 30% were observed to have depression (Wing, 1981a). However, depression is less diagnosed in individuals with more severe intellectual disability, or more severe social and communication difficulties. This may relate to the difficulties that some individuals with ASD have with matching facial expressions to affective experiences. The expression of depression in individuals with ASD may be idiosyncratic and less apparent to those who are not familiar with the individual. An increase in depressive symptoms in individuals with ASD may be positively associated with intellectual and adaptive behavior functioning; due to their ability to understand others and to be aware of their deficits (Sigman, Dissanayake,

Arbelle, & Ruskin, 1997). However, this difference may be due to the fact that individuals with higher-functioning ASD may be better able to express their symptoms to family, caregivers, and professionals.

**Obsessive-Compulsive Disorder (OCD).** OCD consists of recurring obsessions and compulsions. OCD and ASD have been found to have phenotypic and genotypic overlap. Bejerot (2007) suggests that higher-functioning forms of ASD may be obscured by OCD, and that HFA and OCD in combination is extremely common. Genetic studies of OCD and Asperger's disorder have shown a shared genetic bases (Ozaki et al, 2003) In a study by McDougle, Kresch, Goodman, Naylor, Volkmar, Cohen, and Price (1995), individuals with ASD were observed to report obsessional thoughts, cleaning, checking, and counting symptoms less frequently, and more likely to experience repeating, hoarding, touching, tapping, and self-injury compared to a matched sample with OCD. In a more recent study by Russell, Mataix-Cols, Anson, and Murphy (2005) individuals with ASD experienced similar symptoms to individuals diagnosed with OCD, with somatic obsessions, repeating, and checking being more common in those with OCD.

**Challenging Behavior**. Challenging behaviors, otherwise known as maladaptive, problem, or aberrant behaviors are behaviors which are considered inappropriate with respect to frequency, intensity, and/or duration. Challenging behaviors also may cause harm, or result in risk, hardship, or other negative consequences towards the individual, the environment, or those around the individual. Challenging Behaviors are often characterized into three types: self-injurious behavior, aggressive/destructive behavior, and stereotyped behavior. Self-injurious behaviors

(SIB) are those which threaten the safety of the individual emitting the behavior (Schroeder, Mulick, & Rojahn, 1980; Tate & Baroff, 1966). Aggressive behaviors are those with the potential to result in physical harm to those around the individual. Destructive behaviors involve the risk of harm to objects or features of the environment. Stereotyped behaviors are movements, gestures, postures, or utterances which are conspicuous and undesirable behavior, often with a repetitive quality (Berkson, 1983; Rojahn & Sisson, 1990). They are often disruptive and harmful, and may result in interrupted learning, social stigmatization, and exclusion from social and learning environments.

Because ASDs have become a public health concern, and autism awareness is focused mainly on children, there is considerable attention given to ensuring that children with the disorder receive appropriate education, which often addresses core symptoms, adaptive skills training, and access to a variety of treatments for the disorder. Increased awareness has resulted in insurance coverage for services for children with ASD, as well as research funding and the provision of public resources intended for these individuals. Much of the current research focuses on early identification and intervention intended to ameliorate the symptoms of the disorder in the hopes of improving lifelong functioning. However, as ASD is a lifelong disorder, a majority of children with the disorder, regardless of treatment, will require lifelong supports and services.

#### Current Lifespan Research in Autism Spectrum Disorder

ASD is conceptualized as a neurodevelopmental disorder characterized by deficits in social interaction, language, and the presence of restricted and repetitive

behavior. For many children, the disorder appears in infancy and can currently be diagnosed as early as 17 months of age. However, for children with milder forms of the disorder, it may not be apparent until later in childhood. For example, the majority of children with Asperger disorder were diagnosed by age 8. Approximately 60 percent of individuals with ASD disorder have co-occurring ID. Currently, there is a need for research that informs the continuity of care for individuals with ASD across the lifespan.

There is currently very little research that has examined the development or change in prevalence of specific symptoms of autism across the lifespan. However, researchers have identified specific symptoms as characteristic or typical of children with autism at various points in development. The developmental trajectory of ASD is currently not well-understood. For example, a subset of apparently typically-developing infants with PDD exhibit regression (Matson, Wilkins, & Fodstad, 2010). This presentation, when it occurred after several years of seemingly typical development, was classified as Childhood Disintegrative Disorder according to the DSM-IV-TR.

Childhood Disintegrative Disorder, which was eliminated from the *DSM-5* diagnostic criteria, is an ASD in which children appear to develop typically until after 2 years of age, after which they experience regression in language, social behavior, and other skills (Matson & Mahan, 2009). Regression is believed to be a common occurrence in individuals with ASD, but this topic has been less-studied. Goin-Kochel, Ensler, Kann, and Hus (2014) observed that 36.9% of children in an ASD sample exhibited skill regression. In this sample, those children who experienced early language losses exhibited more severe presentations of the disorder in later childhood. Regression of symptoms has also been observed to correlate with an increased risk of

co-occurring psychiatric disorders (Matson, Wilkins, & Fodstad, 2010); sleep problems (Gianotti et al, 2008). Werner, Dawson, Munson, & Osterling (2005) observed that the children who experienced skill regression did not differ from children without regression in developmental or adaptive behavior, but exhibited lower functioning in social reciprocity.

# Symptom Trajectory

Research on the symptoms of autism has focused mainly on children and adolescents with the disorder, and has focused on those symptoms that are presumed to be present across the lifespan. The developmental trajectory of ASD symptoms is less widely known. Puberty is associated with the exacerbation of challenging behavior in between 10 and 33 percent of individuals with ASD, and is observed at higher frequencies in females and those with an ID (Gillberg, 1984, Knickmeyer, Wheelwright, Hoekstra, & Baron-Cohen, 2006). Menarche has also been observed to occur later in females with ASD (Knickmeyer et al, 2006), and is hypothesized to relate to the exposure to higher levels of prenatal androgens in cases where menarche is greatly delayed. In a case study of adolescents diagnosed with ASD and Premenstrual Dysphoric Disorder, a cyclical exacerbation of ASD symptoms was observed in the 4 to 5 days prior to the onset of menses.

The lifespan development of ASD symptoms is heterogeneous. Some individuals exhibit a gradual loss of skills over time, whereas in others the core symptoms become less severe and overall functioning improves. Some researchers have observed that individuals without ID experience improvement in the core symptoms and co-occurring challenging behaviors (Piven, Harper, Palmer & Arndt, 1996; Seltzer, Shattuck, Abbeduto, & Greenburg, 2004; Shattuck, 2007). Seltzer et al

(2003) observed that approximately 55 of his sample of individuals with ASD who met criteria during adolescence continued to exhibit symptoms into adulthood. Autism subtypes have been associated with differential symptom trajectories. Starr, Szatmari, Bryson, and Zwaigenbaum (2003) observed that individuals with high functioning autism increased in their communication skills, and simultaneously became more impaired in socialization, with no change in the presence of restricted and repetitive behavior over the course of two years. Conversely, McGovern and Sigman (2005) observed overall improvement in communication, socialization, and restricted and repetitive behavior symptoms as children progressed into adolescence. In a literature review, Seltzer, Shattuck, Abbetduto, and Greenburg (2004) found that most studies observed overall improvement in communication from childhood, adolescence, into adulthood, with continued social impairment.

In a correlational study between service acquisition and social-communication impairments, the greatest overall improvements occurred in children and adolescents who received a therapy (speech, occupational, or behavior), compared to those children who received no therapy. Interestingly, the greatest response to treatment was observed in those with a higher nonverbal IQ, regardless of type of treatment (Mazurek, Kanne, & Miles, 2012). Individuals with Asperger disorder have been observed to experience deficits in communication into adulthood. Action fluency and category fluency were found to be lower than non-autistic individuals into adulthood (Inokuchi & Kamio, 2013).

Some researchers have observed that during adolescence, challenging behavior occurs in cycles of 1-2 years in which the challenging behavior increases. In

this sample, 22 percent continued to experience an increase in core symptoms and 50 percent exhibited stable impairments in nonverbal communication, social reciprocity, and presence and severity of maladaptive behavior.

### **Comorbidities in Adolescence and Adulthood**

Adults with ASD are also at risk for co-occurring psychiatric disorders. The association between increased levels of anxiety in individuals with ASD is wellestablished. Adults with Asperger disorder have been observed to have higher levels of social anxiety symptoms (Bejerot, Eriksson, & Mortberg, 2014). Farrugia and Hudson (2006) observed that baseline levels of anxiety for adolescents with Asperger Disorder were determined to be commensurate with adolescents diagnosed with anxiety disorders, along with dysfunctional automatic thoughts, behavior problems, and impairments associated with anxiety disorders. This trend has also been observed when comparing adults with ASD to typically-developing adults without ASD, as well as a sample matched along age, gender, and intellectual level (Gillot & Standen, 2007)

Other co-occurring disorders are also observed at high rates in adults and adolescents with ASDs. Challenging behaviors generally first occur during childhood. However, when left untreated, these behaviors frequently result in the disruption of the educational trajectory and increase the likelihood of placement into residential facilities (Howlin, 1997; King, State, Shah, Davanzo, & Dykens, 1995)

# **Current Trends in ASD Research and Intervention**

As a result of the focus on early intervention, there has been an increased focus on the development of diagnostic tools and treatment modalities for very young children who are at risk for developmental delay. Early diagnosis and treatment of ASD and its related symptoms is associated with the most desirable outcomes; namely the decrease

in severity of the symptoms of the disorder, including improved social and academic functioning. As of 2009, the mean age of children with autistic disorder was approximately 4 years of age (Zwaigenbaum, Bryson, Lord, Rogers, Carder, & Carver, 2009). However, the disorder can now be reliably diagnosed in much younger children, as young as 14-24 months of age (Lord, Risi, DiLavore, Shulman, Thurm, & Pickles, 2006; Stone, Lee, Ashford, Brissie, Hepburn, Coonrod, & Weiss, 1999; Stone, McMahon, & Henderson, 2008).

### Assessment of Adolescent and Adult ASD

Current research has focused on the early diagnosis of ASD. Early diagnosis in ASD is emphasized, as it provides children with the disorder access to early interventions intended to interrupt the development of ASD symptoms and provide for the most favorable outcomes in affected individuals. Early intervention treatments aim to improve outcomes with respect to the core deficits of social skills and language, and to ameliorate the presentation of restricted and repetitive interests. Early diagnosis and treatment is vital to the treatment of individuals with ASD and represents current conceptions of best practice.

There are also measures available that target the symptoms of ASD in young children, most notably the *Baby and Infant Screen for Infants with aUtIsm Traits* (*BISCUIT*; Matson, Boisjoli, & Wilkins, 2007), *Checklist for Autism in Toddlers* (*CHAT*; Baron-Cohen, Allen, & Gillberg, 1992), *Modified Checklist for Autism in Toddlers* (*M-CHAT* Robins, Fein, Barton, & Green, 2001), Early Screening of Autism Traits Questionnaire (ESAT; Dietz, Swinkels, van Daalen, van Engeland, & Buitelaar, 2006), First Year Inventory (FYI; Reznick, Baranek, Reavis, Watson, & Crais, 2007), the

Quantitative Checklist for Autism in Toddlers (Q-CHAT; Baird, Charman, Baron-Cohen, Cox, Swettenham, Wheelwright, & Drew, 2000), and the Autism Diagnostic Observation Schedule – 2 Toddler Module (ADOS-2; Lord, Luyster, Gotham, & Guthrie, 2012).

Measures developed to diagnose and monitor symptoms of ASD in school-age children are more numerous. The most widely-used and recognized include the Autism Spectrum Disorders- Diagnostic for Children (ASD-D-C), the Autism Diagnostic Observation Schedule Modules 1-4 (ADOS-2; Lord, Rutter, DiLavore, Risi, Gotham, & Bishop, 2012), Autism Diagnostic Interview-Revised (ADIR; Le Couteur, Lord, & Rutter, 2003), and the Childhood Autism Rating Scale- 2<sup>nd</sup> Edition (CARS-II; Schopler & Van Bourgondien, Wellman, & Love, 2010).

# Treatment of Adolescent and Adult ASD

Treatment for ASD in children continues to be widely-researched and is increasingly becoming more accessible. Much of this is due to greater awareness of the disorder among professionals, as well as the public. Moreover, laws have been enacted that have increased access to services for children with ASD on healthcare plans. However, it has been less-researched in adults with the disorder.

ASD is conceptualized as a lifelong disorder. Some researchers have identified that a subset of individuals on the spectrum, and particularly those with Asperger disorder, may no longer meet criteria for the disorder in adulthood (Magiati, Wei Tay, & Howlin, 2014). However, the majority of children diagnosed with the disorder will continue to require services and supports into adulthood. Research on treatment, diagnosis, and symptom monitoring of individuals with ASD has been a lesser focus of research, although this population will continue to require supports. In a longitudinal

study, Howlin, Moss, Savage, and Rutter (2013) observed that a population of individuals who were diagnosed with ASD as children who had average nonverbal IQs all continued to meet criteria for the disorder in adulthood and mostly achieved poor outcomes with respect to occupation and socialization, even though the severity of their symptoms decreased over time. Individuals with autism will require continued assessment and intervention throughout the lifespan.

Studies of adults with ASD often have focused either on "lower functioning" individuals with ID, or individuals with Asperger disorder. Even individuals with Asperger disorder are likely to exhibit poor educational and occupational outcomes, and are less likely to live independently in adulthood. In a study by Ventner, Lord, & Schopler (1992), only 27.3 percent of individuals were "competitively" employed, while 59.1 percent were employed in a supervised setting, such as a sheltered workshop, supervised work setting or school-based setting, and 13.6 percent were unemployed. In this sample, there were no individuals who were married, and only 9% lived independently. Similar studies have observed a range of 7-50 percent of individuals with Asperger disorder or "high functioning" autism pursuing higher education, 16-50 percent in a semi-independent living setting, and 5-44 employment (Larsen & Mouridsen, 1997; Mawhood & Howlin, 1999).

Treatments for adults with autism spectrum disorder have been less-studied compared to the child and adolescent ASD population. Treatment modalities for children with the disorder have been informed by both early intervention and educational models, and are often administered in highly-structured settings such as schools and/or specialized after-school programs.

Evidence-based practices for treatment of ASD in adults are generally behavioral in nature. However, because of the heterogeneous presentation of ASD in adults, adults with ASD may be found in a variety of settings. Some adults with ASD may require little to no support, having self-selected appropriate professions, partners, and environmental conditions under which they can flourish. For example, an adult with a restricted interest may choose a corresponding profession, and experience success and acclaim as a result. On the opposite end of this spectrum are those adults who require extensive supports in most domains of life, who experience severe deficits in the ability to communicate, interact with others, or may be severely impaired by repetitive, aggressive, or self-injurious behavior.

As a result, interventions for adults with ASD must be highly specialized to the individual's level of functioning and any co-occurring disorder or challenge he or she may be experiencing. For example, cognitive behavior therapy (CBT) has been found to be effective in adults with co-occurring depression and anxiety (Binnie & Blainey, 2013). In a study of group CBT in adults with Asperger Disorder, predictably, the scientific basis of the model, and the focus on concrete evidence were all appreciated as benefits by the participants (Weiss & Lunsky, 2010). It is notable, however, that CBT has been found to improve the symptoms of many co-occurring disorders, but has not been found to be effective for treating the core symptoms of the disorder.

For individuals who are lower-functioning or who seek treatment for core symptoms of the disorder, treatments based on the theories of Applied Behavior Analysis have shown increased success. This approach involves task analysis, time delay procedures, prompting, modeling, extinction, response interruption and

redirection, functional communication training, and differential reinforcement of behavior (alternate, other, incompatible, or appropriate behavior). Positive behavior supports (PBS) is one such modality which uses ABA principles to support individuals in specific targeted environments, such as educational or occupational settings (Schall, 2010).

# Adaptive Behavior and Autism Spectrum Disorder

Adaptive behavior is a broad construct which refers to those behaviors which are required for independence and self-sufficiency in one's life (Sparrow, Balla, Cicchetti, 1984). Adaptive behavior includes communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and occupation. These adaptive behavior domains have sometimes been divided into conceptual, practical, and social domains (American Association on Mental Retardation, 1992). Deficits in adaptive behavior comprise a core symptom of ID and also include two core symptom areas related to ASD; communication and social skills.

Compared with non-autistic individuals with ID, individuals with ID and ASD experience more severe deficits in social skill and communication on measures of adaptive functioning with respect to age-norms, but not with respect to standard scores (Carter, Volkmar, Sparrow, Wang, Lord, Dawson, Fombonne, Loveland, Mesibov, & Schopler, 1998). Adaptive functioning score profiles were found to differ with respect to age and verbal/nonverbal status (Carter et al, 1998). Researchers observed greatest impairments in communication in nonverbal individuals older than 10.

# Intellectual Disability, Autism, and Adaptive Behavior Deficits in Adults

Adults with ASD represent a heterogeneous population which comprises a wide spectrum of ability. These individuals are likely to experience co-occurring intellectual disability, challenging behavior, and psychiatric disorders. The change in diagnostic

criteria, in addition to posing specific challenges to researchers, clinicians, policymakers, and individuals with ASD and their families, may pose specific concerns for adults with ASD. Specifically, the relationship of the new criteria to the adult population of individuals with ID should be carefully considered.

As the majority of ASD research is conducted in young children through adolescents, it is less-known how the changes in diagnostic criteria are likely to affect the adult population. The combination between intellectual disabilities, differential abilities with respect to communication and socialization abilities within an ASD population, and the high frequency of co-occurring disorders poses challenges for research in this area. The presence of these risks underscores the need for additional research and consideration for these individuals. The increasing prevalence of ASD in children will lead to an increasing prevalence of the disorder in the adult population in the coming years, and a corresponding need for treatments, psychological services, and access to lifelong supports.

### PURPOSE

The changes in diagnostic criteria for ASD have wide-reaching consequences. With changes in diagnostic criteria come the potential for changes in rates of diagnosis of the disorder and the corresponding changes in service availability. Two related studies were conducted. The first determines the new cutoff scores that correspond to diagnoses of ASD using the ASD-D-A. The second examines changes in corresponding adaptive behavior skills of the population likely to receive a diagnosis of ASD in the future.

Together, potential cutoff scores according to the changes in diagnostic criteria and classification are calculated, and the degrees to which these changes may affect future rates of diagnosis of the disorder are examined. The present study also determines whether the changing criteria will result in degrees of impairment in those who will no longer qualify for a diagnosis of the disorder. The present study analyzes the adaptive functioning of individuals who meet ASD criteria, individuals who have been diagnosed with ASD but who will no longer meet criteria, and individuals without ASD. This determines both the changes likely to occur resulting from the changes in diagnostic criteria, and whether individuals with significant impairments in the core symptoms of ASD are likely to be excluded.

### METHOD

# **Participants**

The participants for study 1 were selected from a pre-existing database of adults who resided at a facility for individuals with intellectual and developmental disabilities at the time of assessment. The sample consists of 337 adults with varying levels of intellectual disability. The participants in this sample were administered the ASD-D-A as a part of a test battery which included measures of adaptive behavior, behavior problems, and symptoms of autism and co-occurring disorders. The data set also includes DSM-4-TR Axis I and Axis 2 diagnoses from the participants' chart. Diagnoses were given based on clinical judgment of a licensed psychologist from the individual's treatment team following an evaluation. The evaluation included a personal history and review of records, behavioral observations, and assessment measures chosen by the psychologist. Participants with less than 90% of target data points completed will have those points replaced with the mean for that item (Field, 2005).

Level of intellectual ability was determined by a licensed clinical psychologist employed by the facility. These diagnoses were based on standardized assessments of intellectual functioning (i.e. *Stanford-Binet Intelligence Scales, WISC-IV, Leiter-R*) and adaptive behavior (ie. *Vineland Adaptive Behavior Scales, 2<sup>nd</sup> Edition*), in addition to clinical judgment, expertise, and a review of the individual's history and previous records, as provided to the facility. Diagnoses of intellectual and/or developmental disability were conducted as a matter of course upon intake, and periodically thereafter, as the presence of an intellectual and/or developmental disability was a requirement for

residence at the facility. A total of 337 individuals were eligible to participate in the study.

#### Measures

### Autism Spectrum Disorder- Diagnostic- Adult (ASD-D-A).

The *ASD-D-A* is a 31-item measure assessing for autistic symptomatology in adults with intellectual and developmental disabilities. All items are scored on a dichotomous scale according to whether they were found to be "0 = not different; not a problem", or "1= different; a problem". All items were developed by a clinical psychologist with more than 30 years of experience with IDs through a review of the literature, other ASD assessment measures (e.g., *ADOS*, *CARS*), and the diagnostic criteria (*DSM-IV*, *ICD-10*). This instrument includes three subscale scores- social, communication, and repetitive behaviors/restricted interests. The *ASD-D-A* has high internal consistency (Cronbach's alpha = .94; Matson, Wilkins, & Gonzalez, 2007).

Advanced doctoral-level graduate students administered the *ASD-D-A*; the students interviewed staff members who had worked with the participant for at least 6 months. Prior to the administration of the instrument, the participant was given a brief explanation as to the purpose of the study, and provided verbal assent in the presence of a staff member. The questions related to the core symptoms of autism and included the following items: communication skills, verbal communication, ability to recognize the emotions of others, reaction to normal everyday lights, use of language in conversations with others, and limited number of interests. Items were rated on a dichotomous scale to the extent that the behavior was a problem during the observation: "0=not different, no impairment", or "1 =different, some impairment".

	400			TOTAL
	ASD DSM-5	ASD DSM-4	Non-ASD	TOTAL
		( <i>n</i> = 83)	( <i>n</i> =115)	(N = 337 )
	( <i>n</i> = 83)			
Age in Years	50.12	48.67	55.22	51.52
(SD)	(11.6)	(12.95)	(14.2)	(13.2)
Gender				
Male	46(52.6)	71(55.4)	64 (54.8)	180 (54.1)
Female	37 (47.4)	64 (44.6)	55 (45.2)	153 (45.9)
Race				
Caucasian	66 (76.3)	103(20.5)	91 (76.5)	260 (77.2)
African-Am.	17 (23.0)	31(20.5)	28 (23.5)	76 (22.5)
Hispanic	0	1	0	1
Verbal	24 (40.0)	54 (28.9)	56 (48.7)	134 (40.2)
Nonverbal	59 (60.0)	81	63(51.3)	203 (59.8)
Intellectual Disability				
Mild	0	1	1	2
Moderate	3	6	10	19
Severe	9	14	24	47
Profound	68	107	81	256
Unspecified	3	7	3	13

Table 1. Demographic Information

Note: SD = Standard Deviation.

Table 2. Sex and Diagnostic Groups

	DSM-5	DSM-IV-TR	Non-ASD	Total
Males	71	46	64	181
Females	64	37	55	156
Total	135	83	119	337
				38.1% loss

The *ASD-D-A* has three factors which corresponded to the three autism symptom areas specified in the DSM-IV-TR; social, communication, and repetitive and restricted interests. Factor 1 includes items related to socialization, factor II includes those items that were mostly related to communication, and factor III consisted of those items related to restricted or repetitive behaviors (Matson, Boisjoli, Gonzalez, Smith, & Wilkins, 2007).

The *ASD-D-A* was found to be highly correlated with a measure of autism symptoms according to the previous version of the DSM (*DSM-IV-TR Checklist* (r =.60, p < .01) and highly negatively correlated with a measure of social skills in individuals with ID (*MESSIER*; r = -.67, p < .01) as well as the socialization domain of the Vineland Adaptive Behavior Scales (r =-.42, p < .01). Discriminant validity of the *ASD-D-A* was determined through a non-significant result (r - .12, p > .05) when correlated with scores on a measure of symptoms of co-occurring disorders in individuals with ID (*DASH-II*; Matson, Wilkins, Boisjoli, & Smith, 2007). The *ASD-D-A* yielded an excellent inter-rater reliability of .99 and a good internal consistency of .77 (Matson, Gonzalez, Wilkins, & Rivet, 2006).

# **DSM-IV-TR Checklist**

The *DSM-IV-TR/ICD-10 checklist* is a 19-item diagnostic checklist which corresponds to the diagnostic criteria outlined in the DSM-IV-TR and ICD-10 along the core symptom areas of socialization, communication, and restricted and repetitive interests. In this measure respondents indicate "yes" if the symptom applies to the participant. Internal consistency ( $\alpha$  = .95), test-retest reliability (r=.97), and inter-rater reliability (r=.89) for this measure were all found to be high (Matson, Gonzalez, Wilkins, & Rivet, 2008). The criteria by which the participants were selected into the DSM-5 group are provided in the appendix. All participants were coded as non-autistic, ASD according to DSM-IV only, and ASD according to DSM-5 based upon responses on the *DSM-IV-TR checklist* and the corresponding items. Results of this measure, and not clinical diagnoses from the professionals at the center, were used as the basis for DSM-IV-TR diagnoses for the present study, as an analogue to the DSM-5 diagnostic algorithm to determine DSM-5 diagnosis.

### **DSM-5** Diagnostic Algorithm

ASD Diagnostic group membership was determined by an algorithm that utilized information from the DSM-IV-TR checklist supplemented with items from other measures as necessary. The DSM-5 diagnostic criteria were recorded and mapped onto DSM-IV-TR criteria. Notably, DASH-II items related to under-reactivity to light and sound were used to map onto the sensory reactivity criterion. The DSM-5 Diagnostic algorithm used for the present study is presented in the Appendix. Per the DSM-5, participants must have met criteria for each of the criteria in criterion A, and at least two from criterion B.

# Vineland Adaptive Behavior Scale- 2<sup>nd</sup> edition (VABS-II)

The VABS-II is an informant-based, semi-structured interview that assesses adaptive behavior skills, and is widely used to evaluate children and adults with intellectual disabilities. Adaptive behavior skill domains include: communication, daily living skills, socialization, and motor skills. This measure also provides a composite score of overall adaptive functioning in individuals from birth through 90 years of age (Sparrow, Balla, & Ciccheti, 2005). The original version of the VABS correlated highly with IQ scores at .72, although this relationship has not been evaluated using the current version (Freeman, Ritvo, Yokota, Childs, & Pollard, 1988). The communication subscale is divided into receptive, expressive, and written communication domains. The daily living skills subscale includes skills related to hygiene, self-care, household maintenance, and use of time, money, and some vocational skills. Leisure skills are defined within the socialization domain, which includes a subdomain for Play and Leisure skills, Interpersonal Relationship Skills, and Coping skills. The VABS-II is a highly reliable measure, with high validity for all of the behavioral domains and the composite scores (Sparrow et al, 2005). Internal consistency was good to excellent for all domains (.80-.96). Inter-rater reliability was high, with a range from .75-.85 for domains and .67-.8 for subdomains. The VABS-II domains have been validated by a confirmatory factor analysis, and population norms have included individuals with autism, intellectual disabilities, learning disabilities, and hearing and visual impairments.

The *VABS-II* has been compared to other adaptive behavior skills measures in individuals with high-functioning ASD. In these individuals, socialization scores were measured in the "moderately low" range. This pattern was not observed with respect to the communication subscale, although the target sample included individuals with an

average IQ, and required relatively high communication skills for inclusion in the study (Lopata, Smith, Volker, Thomeer, Lee, & McDonald, 2013).

### Procedure

The initial participant pool included all individuals with ID who resided at a residential treatment facility in Louisiana and provided assent for the study. Chart reviews were conducted for each participant to obtain information on diagnoses, levels of intellectual functioning, and demographic information. Doctoral level graduate students administered the battery to a staff member who was familiar with the participant, and had worked with them for at least six months. The ASD-D-A was administered as a portion of a test battery that measured symptoms of ASD, co-occurring conditions, and other conditions for research purposes.

**Diagnostic Group Assignment**. Participants were divided into non-autistic, DSM-5 autism, and DSM-IV-TR autism groups according to the criteria specified in the DSM-IV-TR checklist. Criteria for inclusion in the DSM-5 group were based on a symptom algorithm as outlined in Appendix 1 based on the final published criteria for ASD (American Psychiatric Association, 2013). Rubrics for inclusion into diagnostic categories based on items from other scales have been conducted previously (Frazier et al., 2012; Matson et al, 2007). It is notable that all participants who met criteria for DSM-5 also met criteria for ASD according to DSM-IV-TR.

After applying the diagnostic algorithms to the data set, 135 participants met criteria for ASD according to DSM-5, 83 met criteria for DSM-IV-TR only, and 119 did not meet criteria for an ASD. An a priori profile analysis was conducted based on the

diagnostic categories determined by the DSM-5 algorithm (i.e., DSM-5 ASD vs. non-ASD along DSM-5).

Parents and/or legal guardians of each participant provided informed consent. The purpose of the study and the manner in which the data would be used was discussed with each parent and participant, and the participant provided informed assent when developmentally appropriate. The present studies have received prior approval from institutional review boards. Use of the data has been approved by the Louisiana State University Institutional review board and the Louisiana State Human Research Committee.

# Study 1. Preliminary Data Exploration, Parametric Assumptions, and Transformations

For the purposes of measures of central tendency and the ROC curve, data were analyzed without transformation. ASD-D-A data were inspected for parametric assumptions and outliers. Within the ASD-A scores, there were 335 valid data points, with a mean score was 20.10 (8.74), with a median of 23. Skewness was -.773 and kurtosis was -.53. Visual exploration of data revealed a strong negative skew. Using Kolmogorov-Smirnov tests, it was found that the distribution of scores on the ASD-D-A, were non-normal within the DSM-5 group D(119) = .13, p = .00, DSM-IV group D(83) = .16, p = .00, and the non-ASD group D(135), p = .00. Variances between the three groups were also significantly different F(2, 334) = 25.89, p = .00.

Scores for the ASD-D-A were reverse score transformed, due to negative skew and log, square root, and reciprocal transformed, and inspected for normality and homogeneity of variance. Visual inspection revealed an approximately normal distribution after the log transform. Statistical tests of normality revealed that the tests were normal within each groups, the DSM-5 group D(135) = .08, p = .02, and the nonautistic group D(119) = .00, and non-significant for the DSM-4 group, D(83) = .09, p = .07. The log transformed scores met the assumption for homogeneity of variance F(2, 334) = .82, p = .44. Because the ROC curves are non-parametric, non-transformed scores were used to compute the data using this method. Transformed data were used for parametric analyses.

**Hypotheses.** Based on previous studies on the relationship between the new diagnostic criteria and levels of adaptive behavior (Turygin, Matson, Beighley, & Adams, 2013) that determined that individuals with established diagnoses of ASD who will no longer meet criteria exhibited comparable levels of social and communication deficit. It is hypothesized that individuals with ASD according to DSM-5 and ASD according to the DSM-IV criteria only will not differ significantly from one another on levels of adaptive behavior skills as measured by the VABS-II, including ASD core symptoms of communication and socialization skills. Additionally, levels of non-ASD related adaptive skills areas will not significantly differ between the non-ASD, DSM-5 ASD, and DSM-IV ASD groups.

### **STUDY 1: RESULTS**

# Analysis 1. Method- Central Tendency

The present analysis sought to determine cut-off scores in the present sample of adults in a residential facility for individuals with intellectual and developmental disabilities.

Following Matson et al (2007), a standard deviation model was conducted to determine the clinical significance according to the two standard deviations between the non-ASD and DSM-5 ASD populations. Jacobson and Truax (1991) suggested that a cutoff point two standard deviations from the mean of the functional population. This approach ensures that as one crosses a particular cutoff in the direction of the mean of the "functioning" population; one can be considered to be "entering the functional population". This approach is preferable when functional and non-functional populations are non-overlapping. In overlapping populations, determining the point at which one is closer to the functional mean compared to the non-functional mean can also be used as a cut-off point (Jacobson & Truax, 1991). One limitation to this approach, however, is that the severity of each dysfunctional population will result in differential cutoff scores. It is for this reason that the present study will utilize the former method.

# Analysis 1. Results

An a priori profile analysis was conducted comparing cutoff scores along DSM4/DSM5 criteria vs. the non-ASD group, and cutoff scores comparing DSM-5 and non-ASD group. Measures of central tendency were calculated. To determine potential cutoff points, the mean scores were examined, to determine which resulted in the

largest spread between those in the ASD and non-ASD groups. Based on cutoff point guidelines by Jacobson and Truax (1991), scores of 23, 24, 25, were identified as potential cutoff points. The method of using 1 standard deviation resulted in potential cutoff scores of 27 and 28, and the method of using 1.5 standard deviations above the control mean was not appropriate, due to low variance of scores between the ASD

DSM-5 and control groups.

Table 3. DSM-5 ASD and Non-DSM-5 ASD Groups and DSM-4/DSM-5 ASD Groups compared to Non-ASD Groups

	Mean (SD)	Median	Mode
ASD-DSM-5	22.6 (6.84)	24.00	27.00
Non-ASD (DSM- 5)	18.43 (9.46)	21.00	27.00
ASD-DSM-4	22.28 (6.95)	24.00	27.00
Non-ASD (DSM- 4)	16.17 (10.22)	18.0	26.00

# Analysis 2. ROC Analysis

ROC analysis is a flexible and useful method for developing cut-off scores in psychological assessments (Schisterman, Perkins, Liu, & Bondell, 2005). This method provides sensitivity and specificity levels for each potential cutoff point, which allows the appropriate cut-off score to be chosen based on the intended function of the assessment (Metz, 1978; Hanley & McNeil, 1983; Wuench, 2015).

	Mean (SD)	Mean Score	Median	Mode
ASD-DSM-5	.84 (.37)	25	.90 (24)	.60 (28)
Non-ASD (DSM-5)	1.00 (.36)	22	1.04 (21)	.70 (27)

 Table 4. Measures of Central Tendency- Log Transform Scores and Corresponding

 ASD-A scores

ROC curves are used extensively in medical literature to distinguish "diseased" from "non-diseased" subjects. The probability of correctly ranking a "diseased" and "non-diseased" pair is related conceptually to the Wilcoxon/Mann-Whitney nonparametric tests. As the Wilcoxon test measures the probability  $\theta$  that when "diseased" and "non-diseased" scores are chosen randomly, they will be correctly ranked. Correspondingly, the area under the ROC curve denotes the probability  $\theta$  that paired scores will be correctly identified. AUC values range between .50 and 1; values of .5 represent values that are no better than chance, and 1 represents a perfectly accurate model (Park, Goo, & Jo; 2004). This procedure is contrasted from the Standard Deviation from Central Tendency method as it is nonparametric. Moreover, this procedure allows the desired sensitivity and specificity to be selected.

# Analysis 2. ROC Analysis Results

A ROC analysis was conducted to determine the cutoff point that best differentiated the DSM-5 from the control population. For the analysis, SPSS Roc Curve Analysis was utilized to determine potential sensitivity and specificity scores for each potential cutoff score. The area under the curve (AUC) for the analysis was .627, CI[.57-.69], S.E. .30, p = .00. This represents that the model has some limited value in optimizing sensitivity and specificity with respect to ASD diagnoses. ROC analysis of

the cutoff point revealed an optimal cutoff score of 17, which maximized the sensitivity at .78 and specificity at .38.

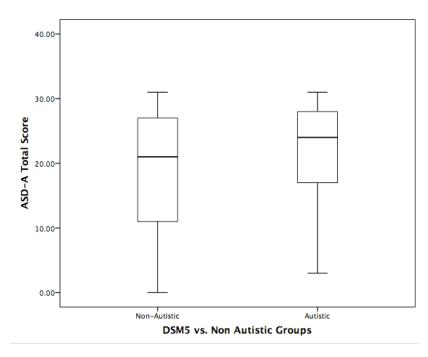


Figure 1. ASD-D-A Scores for the Control and DSM-5 groups

Table 5.	Sensitivity,	Specificity,	and Predictive	Value Sco	ores Based o	n ROC Analysis
----------	--------------	--------------	----------------	-----------	--------------	----------------

Score	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
17	.78	.38	45%	72%
23	.59	.54	45%	66%
24	.56	.60	48%	67%
25	.50	.62	46%	65%
27	.41	.74	51%	65%
28	.31	.82	54%	64%

### **STUDY 1: DISCUSSION**

Potential cutoff scores for the ASD-D-A ranged from 17 based on the ROC analysis, but then ranged as high as 23, 24, 25, 27, and 28 for the measures of central tendency methods. The relatively low value calculated by the ROC curve may be the result of a relatively low AUC, which suggests that this measure has a limited value for distinguishing affected vs. unaffected individuals. This is unsurprising, given that the new diagnostic criteria result in an overall "loss" of individuals who were previously identified as having the disorder. This suggests that the present algorithm for the ASD-*D-A* is of limited value for diagnosing ASD. Those individuals who no longer meet criteria for the disorder may now be considered "false positives" according to DSM-5 criteria. Alternately, the issue may be that due to the new diagnostic criteria, individuals who are known to be affected with the disorder are now being considered "false negatives". The follow-up analyses in Study 2 examine whether this hypothesis can be supported. Should individuals who no longer meet criteria be considered "false negatives" if they are excluded but still exhibit comparable levels of impairment relative to those with the disorder?

One challenge and limitation to this approach, however, is that it does not consider the presence of restricted interests and repetitive behavior a core symptom of the disorder. The presence of this symptom is necessary to diagnose ASD, and in particular to differentiate it from the new DSM-5 diagnosis of social communication disorder. The present sample does not contain a measure of restricted and repetitive behavior that would provide the data necessary to make such a comparison. However,

future studies may wish to consider this when determining whether individuals will meet criteria for ASD.

### **STUDY 2: RESULTS**

# Study 2. Preliminary Analyses

For the multivariate analysis of variance (MANOVA), VABS-II scores were visually examined for parametric assumptions and outliers. There were 337 valid data points for all subscale scores. As expected, due to the severity and the nature of a residentially-based sample of adults with intellectual and developmental disabilities, a significant positive skew for all three subscales was observed through visual inspection of the data. One outlier was observed within the communication domain only, which was within the range of the subscale, and corresponded to relatively high scores on the other subscales. Within the communication scores there was a mean score of 22.73 with a median of 16, skewness of 2.18, and kurtosis of 6.50. With respect to tests of normality with respect to scores on the ASD-D-A, Kolmogorov-Smirnov test revealed that scores were significantly non-normal within the DSM-5 group D(83) = .16, p = .00, DSM-IV group D(130) = .23, p = .00, and the non-ASD group D(117)= .18, p = .00. Mean variances between the three groups were not significantly different between groups *F*(2, 327) = 1.92, *p* = .00 in the communication group.

With respect to daily living skills scores, there was a mean score of 35.61 (SD= 36.70), with a median of 22. Skewness was 1.33 and kurtosis was 1.11. With respect to tests of normality with respect to daily living skills scores, a Kolmogorov-Smirnov test revealed that scores were significantly non-normal within the DSM-5 group D(83) = .16, p = .00, DSM-IV group D(130) = .18, p = .00, and the non-ASD group D(117)=.19, p = .00. Mean variances between the three groups were significantly different between groups F(2, 327) = 12.50, p = .00 in the daily living skills group.

With respect to socialization scores, there was a mean score of 23.89 (SD= 36.70), with a median of 17. Skewness was 1.55 and kurtosis was 2.55. With respect to tests of normality with respect to daily living skills scores, a Kolmogorov-Smirnov test revealed that scores were significantly non-normal within the DSM-5 group D(83) = .16, p = .00, DSM-IV group D(130) = .18, p = .00, and the non-ASD group D(117) = .18, p = .00. Mean variances between the three groups were significantly different between groups F(2, 327) = 7.47, p = .00 in the socialization group.

Therefore, transformations were considered in order to determine whether the assumption of normality could be met for these data, in order to be able to use parametric statistics. After running tests of normality using log, square root, and reciprocal transformations, it was determined that a log transformation resulted in the greatest improvement on the normality of data. This transformation also improved the visual distribution of the data, reduced the positive skew. When data were log transformed, data based on the mean all satisfied the Levene's test, (communication, p = .36; daily living skills, p = .09, and socialization, p = .15).

Post transformation, Kolmogorov-Smirnov tests continued to demonstrate nonnormality of the data for most of the subscales within diagnostic groups. However, large sample sizes frequently result in significant findings with this test when large sample sizes are used. Skewness and kurtosis were improved for all variables. Within all of the subscale scores, normality was improved visually and when examining skewness and kurtosis scores. Within the communication scores, skewness was -.53 and kurtosis was .16. Within daily living skills scores, skewness was -.56 and kurtosis was .46, and within socialization scores, skewness was .59 and kurtosis was -.31. In addition, the

assumption of homogeneity of variance was met for all three subscales for the mean; the communication subscale D(2, 327) = 1.03, p = .36, daily living skills D(2, 327) = 2.47, p = .09, and socialization subscale D(2, 327) = 1.94, p = .15.

# Adaptive Behavior Scores Between Diagnostic Groups

# Analysis 1. Adaptive Behavior Score Differences between DSM-5 and non-ASD diagnostic groups

To determine the effect of potential covariates, a variety of tests were conducted. Chi-square tests were conducted to determine whether gender, race or intellectual ability had an effect on adaptive behavior scores. No significant effect of race  $\chi^2(2) =$ 1.53, p = .46, gender,  $\chi^2(1) = .113$ , p = .74, or intellectual disability were observed  $\chi^2(4)$ = 4.07, p = .40.

A follow-up multivariate analysis of variance (MANOVA) was conducted to determine differences between adaptive behavior subscores and the diagnostic groups. Preliminary analyses were conducted to ensure that the assumptions for the analyses were satisfied. Prior analyses of normality ensure that this assumption was met. Box's test was run to ensure that the homogeneity of covariance matrices was met, as Box's test was not significant, Box's M = 8.87, p = .1.9.

In order to interpret the results of the MANOVA, Roy's largest root was selected as the test statistic due to its robustness when dependent variables are related to one construct, in this case, adaptive behavior (Field, 2009, Warne, 2014). There was no significant effect of diagnostic group on adaptive behavior scores  $\Theta$ = .16, F(3, 326) = 1.59, p = .19. As a result, the null hypothesis that adaptive behavior scores differ between diagnostic groups cannot be rejected. As a result, follow-up univariate ANOVAs and discriminant analyses cannot be conducted.

# Analysis 2. Adaptive Behavior Scores Differences between DSM-5, DSM-IV-TR, and Non-ASD groups.

Due to the non-significant findings of the original analysis, a subsequent analysis was run to determine whether the DSM-5, DSM-IV-TR, and non-ASD groups differed significantly with respect to adaptive behavior scores. To determine the effect of potential covariates, a variety of tests were conducted. Chi-square tests were conducted to determine whether gender, race or intellectual ability had an effect on adaptive behavior scores. No significant effect of race  $\chi^2(4) = 1.80$ , p = .77. gender,  $\chi^2(2) = .167$ , p = .92, or intellectual disability were observed  $\chi^2(8) = 11.08$ , p = 20.

A follow-up multivariate analysis of variance (MANOVA) was conducted to determine differences between adaptive behavior subscores and the diagnostic groups. Preliminary analyses were conducted to ensure that the assumptions for the analyses were satisfied. Prior analyses of normality ensure that this assumption was met. Box's test was run to ensure that the homogeneity of covariance matrices was met, Box's *M* = 17.56, *p* = .14.

In order to interpret the results of the MANOVA, Roy's largest root was selected as the test statistic due to its robustness when dependent variables are related to one construct, in this case, adaptive behavior (Field, 2009, Warne, 2014). There was a significant effect of diagnostic group on adaptive behavior scores  $\Theta$ = .023, *F*(3, 326) = 2.49, p = .05. Follow-up analyses revealed that significant differences were observed for the communication scores *F*(2) = 3.41, p = .04. Differences in adaptive behavior scores were not observed for the daily living skills scores *F*(2) = 1.08, p = .34, and were significantly different within socialization scores *F*(2) = 3.20, p = .04.

Scheffe post-hoc statistics were used due to sample size differences. Within the communication scores, only the control group (M = 25.95) was found to differ significantly from the DSM-5 group (M = 21.34) p = .04. Within the socialization scores, only the DSM-5 group (M = 21.16) differed from the control group (M = 29.10), p = .04

### **STUDY 2: DISCUSSION**

No significant differences were observed between the DSM-5 group and the control group with respect to adaptive behavior scores. It is notable that the adaptive behavior scores, particularly those that relate to the core symptoms of ASD, were not significantly impaired in the DSM-5 group compared to the control group. Adaptive behavior includes two domains that represent the core symptoms of ASD, and should necessarily result in differential deficits between the groups. This finding suggests that individuals in the new "non-ASD group" were comparably impaired. The second analysis compared the control (without those who only met criteria for ASD according to DSM-IV-TR only), the DSM-IV-TR group (representing those who will likely be excluded from ASD diagnoses in the future), and those who met criteria for ASD according to DSM-5. This analysis represented the effects that are likely to reflect what will occur when the DSM-5 comes into widespread use: individuals with significant impairments related to the core symptoms of ASD will be considered among the "controls." Alternately, they may meet criteria for social communication disorder, as suspected by Tanguay (2011). Future studies should examine the degree to which this may occur. However, due to the differing criteria for the disorder, data were not adequate to categorize the present sample with respect to social communication disorder. Additional research on social communication disorder and its relationship to ASD should also be a focus of study.

The second analysis was conducted to determine whether there was a differential effect of adaptive skills impairment across the groups. It was notable that only the DSM-5 group differed from the control group with respect to communication

and socialization scores. To an extent, this difference is unsurprising, given that the most severely affected individuals are likely to continue to meet criteria for ASD based on DSM-5 criteria, and the DSM-5 group is likely to include the most impaired individuals from the present sample, with respect to the core symptoms of ASD. Moreover, the sample used in the present study was comprised of individuals who resided at a long-term facility for individuals with IDD. As a result, many of the individuals in the DSM-5 group likely represent some of the most severely impaired individuals on the autism spectrum, and also included individuals with severe global delays. It was evident that the present sample was comprised of individuals with adaptive skills deficits that were quite severe. Additional research on the effect of these changes as they relate to adults, and individuals with IDD is needed.

On the other hand, these findings were also surprising, given that the DSM-IV-TR group did not significantly differ from the DSM-5 group. This indicates that individuals in the DSM-IV-TR group are not significantly less impaired than individuals who met criteria along DSM-5 criteria. Given that the sample likely includes individuals with ASD who represent the most severe presentations, given that they require the supports of a residential treatment facility. Regardless of this distinction, the DSM-IV-TR group was not significantly less impaired, which suggests that the new criteria does not adequately capture impairment in individuals with ASD.

### **GENERAL DISCUSSION**

As autism spectrum disorders currently occur at a relatively high rate, are considered a public health concern worldwide, and persist across the lifespan, ASD should continue to be a major focus of research. The changes in diagnostic criteria pose difficulties for researchers in ASD, as longitudinal research is affected, and the change in diagnostic criteria result in a difference in which individuals may or may not qualify for a diagnosis of the disorder. In the present study, 38.1 percent of individuals with an existing diagnosis of ASD based on DSM-IV-TR criteria no longer qualified based on the change in criteria. This is consistent with previous studies, in which individuals who qualified for diagnoses of the disorder were be decreased from between 25-68% (Huerta, Bishop, Duncan, Hus, & Lord, 2012; Kulage, Smaldone, & Cohn, 2014; Matson, Belva, Horovitz, Kozlowsky, & Bamberg, 2012; Matson, Kozlowski, Hattier, Horovitz, & Sipes, 2012; Young & Rodi, 2014).

As ASD is a lifelong disorder, children with the disorder will require supports and services across the lifespan. The sharp increase in diagnoses of children with ASD will result in a sharp increase in adults who require diagnostic, treatment, and supportive services. Little research has been conducted in symptom presentation, stability, and diagnostic procedures in adults with the disorder. There is a paucity of research, and a corresponding paucity of diagnostic measures that may be used to diagnose ASD in adults. Thus, it is imperative that further measures be developed that diagnose and monitor symptoms of ASD in adults.

In the present study, several potential cutoff scores and their corresponding sensitivities and specificities were calculated. However, sensitivity and specificity for

the ASD-D-A cutoffs was significantly lower than observed with the DSM-IV-TR criteria. This is unsurprising, given that the measure was developed along those criteria. It is also unsurprising that the sensitivity and specificity of the ASD-D-A changed significantly, given that a large number, 38.1 percent of individuals in the present study no longer qualified for a diagnosis when measured along DSM-5 criteria. In effect, these individuals, who represented "true positives" when DSM-IV-TR criteria were used became negatives while still experiencing the core deficits of the disorder.

Analyses for cutoff scores on the ASD-A result in a cutoff score which is higher than that calculated for distinguishing individuals with and without ASD along DSM-IV-TR criteria. This was an expected finding given that the diagnostic criteria along DSM-5 criteria are more restrictive. This was observed even with the incorporation of the additional DSM diagnostic criteria of "sensory issues" incorporated into the second symptom category, restricted and repetitive behavior. The diagnostic algorithm incorporated two items from the Diagnostic Assessment for the Severely Handicapped, 2<sup>nd</sup> edition (DASH-II) which related to responsiveness to visual and auditory sensory stimuli. Consistent with findings from previous studies, the criteria for ASD along the new criteria resulted in a loss of ASD diagnosis for 52 individuals (38.5%) with a previous diagnosis of ASD. This observed change is consistent with the present range observed in a literature by Tsai (2014), which observed a range of 9-54% (median of 30%) loss of ASD diagnosis in individuals diagnosed with DSM-5 criteria.

In the present study, no significant differences were observed between those with DSM-5, and those without ASD (including those who no longer qualified for the diagnosis) with respect to socialization, communication, and daily living skills scores.

First, this finding was surprising because ASD is a disorder for which the core symptoms include core deficits in socialization and communication abilities. Communication and socialization scores, therefore, were hypothesized to have been significantly lower in the ASD sample. However, this was not observed. In the follow-up study which compared adaptive behavior scores between the three diagnostic groups (DSM-5, DSM-IV-TR, and non-ASD) only daily living skills and socialization scores were significantly lower in the DSM-5 group when compared to the non-ASD group only. This suggests that the DSM-5 group was more impaired with respect to daily living skills and socialization than those without ASD. The DSM-IV-TR group was not significantly different than either the non-ASD or DSM-IV-TR groups in the present study.

The present studies on the cutoff scores for an established measure of ASD symptoms and the corresponding social deficits highlights a question related to the changes in criteria that has been posed by several researchers. Namely, how will the changes in diagnostic criteria affect individuals with ASD with respect to their ability to receive services, especially when insurance coverage or educational accommodations are dependent on the individual receiving a diagnosis. Although individuals with co-occurring ID may continue to qualify, individuals on the autism spectrum who are less severely affected may be denied services, although they are the individuals who are most likely to exhibit more significant improvements with treatment. These consequences are likely to occur across the lifespan as well; early intervention services may not be provided to children who do not have global developmental delays but impairments in social and communication skills. In older individuals, social skills

training, occupational supports, and access to professional therapeutic services may be limited.

Due to increased nuance in research, as well as the focus on specific deficits observed in ASD, it will be increasingly necessary for clinicians to be able to measure symptom presentation and changes in symptom severity over time. In addition, the fact that intellectual disability and autism spectrum disorders co-occur at a relatively high rate necessitates further investigation in the extent to how social skills deficits differ between diagnostic groups and whether there are specific symptoms or presentations that distinguish deficits observed in ASD, deficits observed in ID, and deficits observed when an individual has co-occurring ASD/ID.

Due to the controversy surrounding the new diagnostic criteria, DSM-5 criteria have not been universally adopted. Some clinicians and hospital systems, as an alternative, continue to adhere to ICD-10 criteria, which correspond to those of the DSM-IV-TR. For the *ASD-D-A* based on DSM-IV-TR criteria, a cutoff score of 19 was considered indicative of ASD, along DSM-IV-TR criteria (Matson, Boisjoli, Gonzalez, Smith, & Wilkins, 2007), and the previous cutoff should continue to be used if the *ASD-D-A* is to be used for these purposes.

Future researchers should continue to examine the effect of the new diagnostic criteria. The present study did not observe significant differences in adaptive behavior scores between the typically-developing, DSM-IV-TR, and DSM-5 diagnostic groups. This finding was unexpected, as the core deficits of ASD should correspond to decreases in at least communication and socialization scores in the diagnostic groups. In other studies which have contrasted adaptive behavior scores between DSM-IV,

DSM-5, and typically-developing groups, researchers have observed that socialization and communication scores were significantly lower in the diagnostic groups, but not significantly different between the ASD groups, suggesting that those who no longer met criteria for diagnoses represented a population that was similarly impaired in the core symptoms as those who retained their diagnosis.

One potential reason for the present findings may be due to the target population. Participants in the present study comprised mainly individuals with profound and severe ID, and comprise a sample who resides in a state-run residential facility. As a result, individuals in all of the diagnostic categories likely represent those with severe challenges which may correspond to greater overall impairment. Increases in other disabling conditions may create additional difficulties with adaptive behavior. Notably, for the purposes of the present study, raw scores for VABS-II data were used due to the overall severity of impairment. Consistent with the level of intellectual disability, the majority of participants in the sample had adaptive behavior subscale scores in the lowest range.

Despite the challenges inherent in detecting differences in adaptive behavior in the present population, this area should continue to be studied. Adults, and individuals with severe presentations of ASD represent a relatively underserved population in the area of IDD, and ASD. In addition, the myriad of impairments experienced by these individuals result in a decreased quality of life, and increased medical and supportive expenses.

67

## REFERENCES

- Abell, F., Krams, M., Ashrubner, J., Passingham, R., Friston, K., Frackowiak, R.,...& Frith, U. (1999). The neuroanatomy of autism: A voxel-based whole brain analysis of structural scans. *Neuroreport*, *10*, 1647-1651.
- Amaral, D. G. and Corbett, B. A. (2003) The Amygdala, Autism and Anxiety, in Autism: Neural Basis and Treatment Possibilities: Novartis Foundation Symposium 251 (eds G. Bock and J. Goode), John Wiley & Sons, Ltd, Chichester, UK.
- American Academy of Pediatrics, Committee on Children With Disabilities (2001). Developmental surveillance and screening of infants and young children. *Pediatrics, 108*, 192–195.
- American Academy of Pediatrics, Council on Children With Disabilities (2006). Identifying Infants and Young Children With Developmental Disorders in the Medical Home: An Algorithm for Developmental Surveillance and Screening. *Pediatrics, 118*, 405–420.
- American Association on Mental Retardation (1992). *Mental Retardation: Definition, Classification, and Systems of Supports, (9<sup>th</sup> ed).* Washington, DC: AAMR.
- American Psychiatric Association. (1952). Diagnostic and Statistical Manual, Mental Disorders, (1<sup>st</sup> ed). Washington, DC: American Psychiatric Association.
- American Psychiatric Association. Committee on Nomenclature. (1965). *Diagnostic and Statistical Manual, Mental Disorders: With Special Supplement on Plans for Revision*. American Psychiatric Association.
- American Psychiatric Association (1968). DSM-II: Diagnostic and statistical manual of the mental disorders (2<sup>nd</sup> ed.). *Washington, DC: Author*.
- American Psychiatric Association. (1980). DSM-III: Diagnostic and Statistical Manual of Mental Disorders (3<sup>rd</sup> ed.). *Washington, DC: Author*.
- American Psychiatric Association. (1987). DSM-III-R: Diagnostic and Statistical Manual of Mental Disorders, -revised (3<sup>rd</sup> ed., revised) *Washington, DC: Author.*
- American Psychiatric Association. (1994). DSM-IV: Diagnostic and statistical manual of mental disorders (4<sup>th</sup> ed.) *Washington, DC: Author.*
- American Psychiatric Association (2000). DSM-IV-TR: Diagnostic and statistical manual of mental disorders (4th ed., Text Revision). *Washington, DC: Author*.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.).Washington, DC: Author.

- Ashwin, C., Baron-Cohen, S., Fletcher, P., Bullmore, E., Wheelwright, S. (2001). fMRI study of social cognition in people with and without autism. International meeting for Autism Research.
- Autism Speaks. (2015). DSM-5 News and Updates. Retrieved from https://www.autismspeaks.org/what-autism/dsm-5.
- Aylward, E. H., Minshew, N. J., Goldstein, G., Honeycutt, N. A., Augustine, A. M., Yates, K. O., ...,& Pearlson, G. D. (1999). MRI volumes of amygdala and hippocampus in non-mentally retarded autistic adolescents and adults. *Neurology*, *53*, 2145-2150.
- Bachevalier, J. (1994) Medial temporal lobe structures and autism: A review of clinical and experimental findings. *Neuropsychologia*, *32*, 627-648.
- Bailey, A., Luthert, P., Dean, A., Harding, B., Janota, I., Montgomery, M., Rutter, M., & Lantos, P. A full genome screen for autism with evidence for linkage to a region on chromosome 7q. *Brain*, *121*, 889-905.
- Baird, G., Charman, T., Baron-Cohen, S., Cox, A., Swettenham, J., Wheelwright, S., & Drew, A. (2000). A screening instrument for autism at 18 months of age: a 6– year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 694-702.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences, 6,* 248-254.
- Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *British Journal of Psychiatry*, *161*, 839-843.
- Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, C., Williams, S. C. (2000). The amygdala theory of autism. *Neuroscience & Biobehavioral Reviews*, 24, 355-364.
- Bauman, M., & Kemper, T L. (1985). Histoanatomic observations of the brain in early infantile autism. *Neurology*, *35*, 866-874.
- Bejerot, S., Eriksson, J. M., & Mortberg, E. (2014). Social anxiety in adult autism spectrum disorder. *Psychiatry Research*, 220, 705-707.
- Benda, C. E. (1952). Developmental Disorders of Mentation and Cerebral Palsies. New York: Grune and Stratton.

- Berkson, G. (1983). Repetitive stereotyped behaviors. *American Journal of Mental Deficiency*, 88, 239–246.
- Binnie, J., & Blainey, S. (2013). The use of cognitive behavioural therapy for adults with autism spectrum disorders: a review of the evidence. *The Mental Health Review*, *18*, 93-104.
- Bishop, D. V. M., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorder and specific language impairment: A study using standardized diagnostic instruments. *Journal of Child Psychology and Psychiatry, 43*, 917-929.
- Bleuler, E. (1950). Dementia Praecox or the Group of Schizophrenias. Zinkin, J (ed.) New York, NY International Universities Press.
- Bradley, E. A., Summers, J. A., Wood, H. L., & Bryson, S. E. (2004). Comparing rates of psychiatric and behavior disorders in adolescents and young adults with severe intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, *34*, 151-161.
- Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and Adolescents with Autism Compared to Young People with Intellectual Disability, *Journal of Autism and Developmental Disorders, 36*, 863-870.
- Buxbaum, J. D., & Baron-Cohen, S. (2013). DSM-5: the debate continues. *Molecular Autism, 4*, 11.
- Caronna, E. B., Milunsky, J. M., & Tager-Flusberg, H. (2008). Autism spectrum disorders: Clinical and research frontiers. *Archives of Disease in Childhood, 93*, 518-523.
- Centers for Disease Control and Prevention (2014a). Community Report on Autism. Retrieved from http://www.cdc.gov/ncbddd/autism/states/comm\_report\_autism\_2014.pdf.
- Centers for Disease Control and Prevention. (2014b). Prevalence of autism spectrum disorder among children aged 8 years: Autism and developmental disabilities monitoring network, 11 sites, United States, 2010. *Morbidity and Mortality Weekly Report, Surveillance Summaries, 63,* 1-21.
- Chakrabarti, S. & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association, 285*, 3093-3099.
- Daley, T. C. (2004). From symptom recognition to diagnosis: children with autism in urban India. *Social Science and Medicine, 58*, 1323-1335.

- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology, 20*, 775-803.
- Desarkar, P., Rajiji, T. K., Ameis, S. H., & Daskalakis, Z. J. (2015). Assessing and Stabilizing Aberrant Neuroplasticity in Autism Spectrum Disorder: The potential role of transcranial magnetic stimulation. *Frontiers of Psychiatry, 6,* 124.
- Dietz, C., Swinkels, S., van Daalen, E., van Engeland,; H., & Buitelaar, J. K. (2006).
   Screening for autistic spectrum disorder in children aged 14-15 months. II: Population screening with the early Screening of Autistic Traits Questionnaire (ESAT). Design and general findings. *Journal of Autism and Developmental Disorders*, 36, 713-722.
- Ecker, C., Marquend, A., Mourcio-Miranda, J., Johnston, P., Daly, E. M., Brammer, M. J., et al (2010). Describing the brain in five dimensions- magnetic resource imaging assisted diagnosis of autism spectrum disorder using a multi-parameter classification approach. *Journal of Neuroscience*, *30*, 8815-8818.
- Eisenburg, L. (1957). The Course of Child-hood Schizophrenia". Archives of Neurology and Psychiatry, 78, 69-83.
- Elsabbagh, M., Divan, G., Koh, Y.-J., Shin Kim, Y., Kauchali, S., Marcin, C., Montiel-Nava, C., Patel, V., Paula, C. S., Wang, C., Yasamy, Y. T., & Fombonne, E. (2012). Global Prevalence of Autism and other Pervasive Developmental Disorders. *Autism Research*, *5*, 160-179.
- Falter, C. M., Plaisted, K. C., & Davis, G. (2007). Visuo-spatial Processing in Autism-Testing the predictions of Extreme Male Brain Theory. *Journal of Autism and Developmental Disorders, 38*, 507-515.
- Farrugia & Hudson, (2006). Anxiety in adolescents with Asperger Syndrome: Negative thoughts, behavioral problems, and life interference. *Focus on Autism and other Developmetnal Disabilities.* 21, 25-35.
- Field, A. (2009). *Discovering statistics using SPSS* (third edition). London: Sage Publications.
- Fombonne, E. (1999). The epidemiology of autism: a review. *Psychological Medicine*, 29, 769-786.
- Frazier, T. W., Youngstrom, E. A., Speer, L., Embacher, R., Law, P., Constantino, J., Findling, R. L., Hardan, A. Y., & Eng, C. (2012). Validation of Proposed DSM-5 Criteria for Autism Spectrum Disorder. *Journal of the American Academy of Child* & Adolescent Psychiatry, 51, 28-40.

- Freeman, B. J., Ritvo, E. R., Yokota, A., Childs, J., & Pollard, J. (1988). WISC-R and Vineland Adpative Behavior Scale Scores in Autistic Children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27, 428-429.
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, *32*, 299-306.
- Giannotti, F., Cortesi, F., Cerquiglini, A., Miraglia, D., Vagnoni, C., Sebastiani, T. et al (2003). Langauge and other regression: Assessment and timing. *Journal of Autism and Developmental Disorders, 38*, 1888-1897.
- Gillberg, C. (1994). Autistic children growing up: Problems during puberty and adolescence. *Developmental Medicine & Child Neurology, 26*, 125-129.
- Gillberg, C., & Billstedt, E. (2000). Autism and Asperger Syndrome: Co-existence with other clinical disorders. *Acta Psychiatrica Scandinavica*, *102*, 321-330.
- Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism*, *5*(3), 277-286.
- Gillot, A., & Standen, P. J. (2007). Levels of anxiety and sources of stress in adults with autism. *Journal of Intellectual Disabilities*, *11*, 4, 359-370.
- Goin-Kochel, R. P., Ensler, A. N., Kanne, S. M., & Hus, V. (2014). Developmental regression among children with autism spectrum disorder: Onset, duration, and effects on functional outcomes. *Research in Autism Spectrum Disorders, 8*, 890-898.
- Gould, J. (1982). Social communication and imagination in children with cognitive and language impairments. PhD thesis. University of London.
- Hanley, J. A., McNeil, B. J. (1982). The meaning and use of the area under the Reciever Operating Characteristic (ROC) curve. *Radiology*, *148*, 839-843.
- Hanley, J. A.,& McNeil, B. J. (1983). A method of comparing the areas under Receiver Operating Characteristic curves derived from the same cases. *Radiology*, *143*, 29-36.
- Happe, F., & Ronald, A. (2009). The 'fractionable autism triad' : A review of evidence from behavioural, genetic, cognitive, and neural research. *Neuropsychology Review, 18*, 287-304.
- Horovitz, M., Matson, J. L., Rieske, R., Kozlowski, A. M., & Sipes, M. (2011). The relationship between race and challenging behaviors in infants and toddlers with

autistic disorder and pervasive developmental disorder- not otherwise specified. *Developmental Neurorehabilitation, 14*, 208-214.

- Howard, M. A., Cowell, P. E., Boucher, J., Broks, P., Mayes, A., Farrant, A., & Roberts, N. (2000). Convergent neuroanatomical and behavioural evidence of an amygdala hypothesis of autism. *Neuroreport*, *11*, 2931-2935.
- Howlin, P. (1997). Prognosis in autism: do specialist treatments affect long-term outcome?. *European Child & Adolescent Psychiatry*, 6, 55-72.
- Howlin, P., Moss, P., Savage, S., & Rutter, M. (2013). Social Outcomes in Mid- to Later Adulthood Among Individuals Diagnosed with Autism and Average Nonverbal IQ as Children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*, 572- 581.
- Huerta, M., Bishop, S., Duncan, A., Hus, V., & Lord, C. (2012). Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *American Journal of Psychiatry*, 169, 1056-1064.
- Inokuchi, E., & Kamio, Y. (2013). Qualitative analyses of verbal fluency in adolescents and young adults with high-functioning autism spectrum diosrder. *Research in Autism Spectrum Disorders, 7*, 1403-1410.
- Interagency Autism Coordinating Committee (2014). Statement Regarding Scientific Practice, and Policy Implications of Changes in the Diagnostic Criteria for Autism Spectrum Disorder. Retrieved from https://iacc.hhs.gov/publications/2014/statement\_iacc\_dsm5\_changes\_criteria\_0 40214.pdf.
- Jacobson, N. S., & Truax, P. (1991). Clinical Significance: A Statistical Approach to Defining Meaningful Change in Psychotherapy Research. *Journal of Consulting and Clinical Psychology*, *5*9, 12-19.
- Juneja, M., Mukherjee, S. B., & Sharma, S. (2004). A descriptive hospital based study of children with autism. *Indian Pediatrics, 42*, 453-458.
- Juranek, J. Filipek, P. A., Berenji, G. R., Modahl, C., Osann, K., & Spence, M. A. (2006). Association Between Amygdala Volume and Anxiety Level: Magnetic Resonance Imaging (MRI) study in Autistic Children. *Journal of Child Neurology*, 21, 1051-1058.
- Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child, 2*, 217-250.

Kanner, L. (1944). Early infantile autism. Journal of Pediatrics, 25, 211-217.

- Kilstrup-Nielsen, C., & Landsberger, N. (2015). Rett Syndrome: From the involved Gene(s) to Treatment. *Neurobiology of Brain Disorders*, 98-119.
- King, B. H., State, M. W., Shah, B., Davanzo, P., & Dykens, E. (1997). Mental Retardation: A review of the past 10 years. Part 1. *Journal of the American Academy of Child & Adolescent Psychiatry.* 36, 1656-1663.
- Knickmeyer, R. C., Wheelwright, S., Hoekstra, R., & Baron-Cohen, S. (2006). Age of menarche in females with autism specrum conditions. *Developmental Medicine* & Child Neurology, 28, 1007-1008.
- Kulage, K. M., Smaldone, A. M., & Cohn, E. G. (2014). How will DSM-5 Affect Autism Diagnosis? A Systematic Literature Review and Meta-Analysis. *Journal of Autism and Developmental Disorders, 44*, 1918-1932.
- Lai, M. C., Lombardo, M. V., Chakrabarti, B., & Baron-Cohen, S. (2013). Subgrouping the Autism "Spectrum": Reflections on DSM-5.LaMalfa, G., Lassi, G., Bertelli, M., Salvini, R., & Placidi, G. F. (2004). Autism and intellectual disability: A study of prevalence on a sample of the Italian population. *Journal of Intellectual Disability Research*, 48, 262-267.
- Larsen, F. W., & Mouridsen, S. E. (1997). The outcome in children with childhood autism and Asperger syndrome originally diagnosed as psychotic. A 30-year follow-up study of subjects hosptialzied as children. *European Child and Adolescent Psychiatry*, *6*, 181-190.
- Le Couteur, A., Lord, C., & Rutter, M. (2003). Autism Diagnostic Interview-Revised (ADI-R), Los Angeles, CA: Western Psychological Services.
- Lin, J-D., Lin, L-P., & Wu, J-L. (2009). Administrative prevalene of autism spectrum disorders based on national disability registers in Taiwan. *Research in Autism Spectrum Disorders*, *3*, 269-274.
- Lopata, C., Smith, R. A., Volker, M. A., Thomeer, M. L., Lee, G. K., & McDonald, C. A. (2013). Comparison of adaptive behavior measures for children with HFASDs. *Autism research and treatment*, *2013*.
- Lord, C., Cook, E. H., Leventhal, B. L., & Ameral, D. G. (2000). Autism Spectrum Disorders, *Neuron, 28*, 355-363.
- Lord, C., & Bishop, S. L. (2010). Autism spectrum disorders: Diagnosis, prevalence, and services for children and families. *Social Policy Report, 24*.
- Lord, C., Luyster, R. J., Gotham, K., & Guthrie, W. (2012). Autism Diagnostic Observation Schedule, Second Edition Toddler Module. Western Psychological Services, Los Angeles, CA.

- Lord, C., Risi, S., Dilavore, P., Shulman, C., Thurm, A., & Pickles, A (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, *63*, 694-701.
- Lord, C. Rutter, M., DiLavore, P., Risi, S., & Gotham, K. (2012). Autism Diagnostic Observation Schedule, Modules 1-4. Western Psychological Services, Los Angeles, CA.
- Madsen, K. M., Hviid, A., Vestergaard, M., Schendel, D., Wohlfahrt, J., Thorsen, P., Olsen, J., & Melbye, M. (2002). A population-based study of measles, mumps, and rubella vaccination and autism. *New England Journal of Medicine*, *19*, 1477-1482.
- Magiati, I., Wei Tay, X., & Howlin, P. (2014). Cognitive, language, social and behavioural outcomes in adults with autism spectrum disorders: A systematic review of longitudinal follow-up studies in adulthood. *Clinical Psychology Review, 34*, 73-86.
- Magnusson, P., & Saemundsen, E. (2001). Prevalence of autism in Iceland. *Journal of Autism and Developmental Disorders, 31*, 153-163.
- Maddox, B., & White, S. (2015). Comorbid Social Anxiety Disorder in Adults with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders, 45*, 3949-3960.
- Marriage, S., Wolverton A., & Marriage, K. (2009). Autism Spectrum Disoder Grown Up: A chart review of Adult Functioning. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 18*, 322-328.
- Matson, J. L., Baglio, C. S., Smiroldo, B. B., Hamilton M., Packlowskyj, T., Williams, D., Kirkpatrick-Sanchez, S. (1996). Characteristics of Autism as assessed by the Diagnostic Assessment for the Severely Handicapped- II (DASH-II). *Research In Developmental Disabilities, 17,* 135-143.
- Matson, J. L., Belva, B. C., Horovitz, M., Kozlowski, A. M., & Bamberg, J. W. (2012). Comparing symptoms of autism spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR criteria and the proposed DSM-5 diagnostic criteria. *Journal of Developmental and Physical Disabilities*, 24, 403-414.
- Matson, J. L., Boisjoli, J. A., Mat& Dempsey, T. (2009). Factor Structure of the Autism Spectrum Disorders- Diagnostic for Children (ASD-DC). *Journal of Developmental and Physical Disabilities, 21*, 195-211.
- Matson, J. L., Boisjoli, J. A., Gonzalez, M. L., Smith, K. R., & Wilkins, J. (2007). Norms and cut off scores for the autism spectrum disorders diagnosis for adults (ASD-

DA) with intellectual disability. *Research in Autism Spectrum Disorders, 1*, 330-338.

- Matson, J. L., Boisjoli, J. A., & Wilkins, J. (2007). *Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT)*. Baton Rouge, LA. Disability Consultants, LLC.
- Matson, J.L., Hess, J. A., Mahan, S., & Fodstad, J. C. (2010). Convergent Validity of the Autism Spectrum Disorder- Diagostic for Children (ASD-DC) and Autism Diagnostic Interview- Revised (ADI-R). *Research in Autism Spectrum Disorders*, 4, 741-745.
- Matson, J. L., Gardner, W. I., Coe, D. A., & Sovner, R. (1991). A scale for evaluating emotional disorders in severely and profoundly mentally retarded persons. *British Journal of Psychiatry*, *159*, 404-409.
- Matson, J. L., Gonzalez, M., & Wilkins, J. (2009). Valditiy Study of the Autism Spectrum Disorders- Diagnostic for Children (ASD-DC). *Research in Autism Spectrum Disorders, 3*, 196-206.
- Matson, J. L., Gonzalez, M, Wilkins, J., & Rivet, T. T. (2006). Reliability of ht eAutism Spectrum Disorder- Diagnostic for Children (ASD-DC). Research in Autism Spectrum Disorders, 2, 533-545.
- Matson, J. L., González, M. L., Wilkins, J., & Rivet, T. T. (2008). Reliability of the Autism Spectrum Disorders-Diagnostic for Children (ASD-DC). *Research in Autism Spectrum Disorders, 2*, 696-706.
- Matson, J. L., Hess, J. A., & Boisjoli, J. A. (2010). Comorbid psychopathology in infants and toddlers with autism and Pervasive Developmental Disorders- Not otherwise specified. *Research in Autism Spectrum Disorders, 4*, 300-304.
- Matson, J. L., & Kozlowski, A. (2011). The increasing prevalence of autism spectrum disorders. *Research in Autism Spectrum Disorders, 5*, 418-425.
- Matson, J. L., Kozlowski, A. M., Hattier, M. A., Horovitz, M., & Sipes, M. (2012). DSM-IV vs DSM-5 diagnostic criteria for toddlers with autism. *Developmental Neurorehabilitation, 15*, 185-190.
- Matson, J. L., & Mahan, S. (2009). Current Status of research on childhood disintegrative disorder. *Research in Autism Spectrum Diosrders, 3*, 861-867.
- Matson, J. L., Mayville, E. A., Lott, J. D., Bielecki, J., & Logan, R. (2003). A comparison of social and adaptive functioning in persons with psychosis, autism, and severe or profound mental retardation. *Journal of Developmental and Physical Disabilities*, *15*, 57-85.

- Matson, J. L., Rieske, R., & Williams, L. W. (2013). The relationship between autism spectrum disorders and attention-deficit/hyperactivity disorder: An overview. *Research in Developmental Disabilities, 34*, 2475-2484.
- Matson, J. L., Rivet, T. T., Fodstad, J. C., Dempsey, T., Boisjoli, J. A. (2009). Examination of adaptive behavior differences in adults with autism spectrum disorders and intellectual disability. *Research in Developmental Disabilities, 30*, 1317-1325.
- Matson, J. L., & Shoemaker, M. (2009). Intellectual disability and its relationship to autism spectrum disorders. *Research in Developmental Disabilities, 30*, 1107-1114.
- Matson, J. L., Wilkins, J., & Fodstad, J. (2010). Children with autism spectrum disorders: A comparison of those who regress versus those who do not. *Developmental Neurorehabilitation*, *13*, 37-45.
- Matson, J. L., Wilkins, J., & Gonzalez, M. (2007). Reliability and factor structure of the Autsim Spectrum Disorders- Diagnosis scale for Intellectually Disabled Adults (ASD-A). *Journal of Developmental and Physical Disabiliies, 19*, 565-577.
- Matson, J. L., Wilkins, J., Sharp, B., Knight, C., Sevin, J. A., & Boisjoli, J. A. (2009). Sensitivity and Specificity of the Baby and Infant Screen for Children with aUtism Traits (BISCUIT): Validity and cut-off scores for autism and PDD-NOS in toddlers. *Research in Autism Spectrum Disorders*, *3*, 924-930.
- Mawhood, L., & Howlin, P. (1999). The outcome of a supported employment scheme for high-functioning adults with autism or Asperger syndrome. *Autism*, *3*, 229-254.
- Mayes, S. D., Black, A., & Tierney, C. D. (2013). DSM-5 under-identifies PDD-NOS: Diagnostic agreement between DSM-5, DSM-IV, and Checklist for Autism Spectrum Disorder. *Research in Autism Spectrum Disorders, 7*, 298-306.
- Mayes, S. D., Calhoun, S. L., Murray, M. J., Ahuja, M., & Smith, L. A. (2011). Anxiety, depression, and irritability in children with autism relative to other neuropsychiatric disorders and typical development. *Research in Autism Spectrum Disorders*, *5*, 474-485.
- Mayes, S.D., Calhoun, S. L., Murray, M. J., Pearl, A., Black, A., & Tierney, C. D. (2014). Final DSM-5 under-identifies mild Autism Spectrum Disorder: Agreement between the DSM-5, CARS, CASD, and clinical diagnoses. *Research in Autism Spectrum Disorder, 8,* 68-73.
- Mayes, S. D., Calhoun, S. L., Murray, M. J., & Zahid, J. (2011). Variables associated with anxiety and depression in children with autism. *Journal of Developmental and Physical Disabilities*, *23*, 325-337.

- Mazurek, M. O., Kanne, S. M., Miles, J. H. (2012). Predicting improvement in socialcommunication symptoms of autism spectrum disorders using retrospective treatment data. *Research in Autism Spectrum Disorders, 6*, 535-545.
- McDougle, C. J., Kresch, L. E., & Goodman, W. K., Naylor, S. T., Volkmar, F. R., Cohen, D. J., & Price, L. H. (1995). A case-controlled study of repetitive thoughts and behavior in adults with autistic disorder and obsessive-compulsive disorder. *American Journal of Psychiatry*, 152, 772-777.
- McGovern, C. W. & Sigman, M. (2005). Continuity and change from early childhood to adolescence in autism. *Journal of Child Psychology and Psychiatry, 46*, 401-408.
- Metz, C.E. (1978) Basic principles of ROC analysis. *Seminars in Nuclear Medicine 8*, 283-298.
- Norbury, C. F. (2013). Practitioner Review: Social (pragmatic) communication disorder conceptualization, evidence, and clinical implications. *The Journal of Child Psychology and Psychiatry, 55*, 204-216.
- Norris, M., Lecavalier, L., Edwards, M. C. (2012). The structure of autism symptoms as measured by the autism diagnostic observation schedule. *Journal of Autism and Developmental Disorders, 42*, 1075-1086.
- Njardvik, U., Matson, J. L., & Cherry, K. (1999). A comparison of social skills in adults with autistic disorder, pervasive developmental disorder not otherwise specified, and mental retardation. *Journal of Autism and Developmental Disorders, 29*, 287-295.
- Ozaki, N., Goldman, D., Kaye, W. H., Plotnikov, K., Greenberg, B. D., Lappalainen, J., Rudnick, G., & Murphy, D. L. (2003). Serotonin Transporter Missense Mutation Association with a Complex Neuropsychiatric Phenotype. *Molecular Psychiatry*, *8*, 933-936.
- Park, S. H., Goo, J. M., & Jo, C-H. (2004). Receiver Operating Characteristic (ROC) Curve: Practical Review for Radiologists. *Korean Journal of Radiology, 5*, 11-18.
- Percy, A. (2014). The American History of Rett Syndrome. Pediatric Neurology, 50, 1-3.
- Persico, A. M., & Napolioni, V. (2013). Autism Genetics, *Behavioral Brain Research*, *15*, 95-112.
- Piven, J., Harper, J., Palmer, Pl., & Arndt, S. (1996). Course of behavioral change in autism: A restosepctive study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 523-529.

- Reznick, J.S., Baranek, J. T., Reavis, S., Watson, L. R., & Crais, E. R (2007). A parent report instrument for identifying one-year-olds at risk for eventual diagnosis of autism: the first year inventory. *Journal of Autism and Developmental Disorders*, 37, 1691-1710.
- Rimland, B. (1964). Infantile Autism: The syndrome and its implications for a neural theory of behavior: Englewood Cliffs, NJ: Prentice Hall.
- Ritvo, E. R. (2012). Postponing the Proposed Changes in DSM 5 for Autistic Spectrum Disorder Until New Scientific Evidence Adequately Supports Them. *Journal of Autism and Developmental Disorders, 42*, 26-29.
- Ritvo, E. R., Freeman, B. J., Scheibel, A. B., Duong, T., Robinson, H., Guthrie, D., & Ritvo, A. (1986). Lower Purkinje cell counts in the cerebella of four autistic subjects: initial findings of the UCLA-NSAC Autopsy research report. *American Journal of Psychiatry*, *143*, 862-866.
- Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The Modified Checklist for Autism in Toddlers: an Initial study investigating the early detection of autism and pervsiave developmental disorders. *Journal of Autism and Developmental Disorders, 31,* 131-144.
- Rogers, S. J., Wehner, E., & Hagerman, R. (2001). The behavioral phenotype in Fragile X: Symptoms of autism in very young children with fragile-X syndrome, idiopathic autism, and other developmental disorders. *Journal of Developmental and Behavioral Pediatrics*, 22, 409-417.
- Rojahn, J., & Sisson, L. A. (1990). Stereotyped behavior. In *Handbook of behavior modification with the mentally retarded* (pp. 181-223). Springer US.
- Rommelse, N. N., Franke, B., Geurts, H. M., Hartman, C. A., & Buitelaar, J. K. (2010). Shared heritability of attention-deficit/hyperactivity diosrder and autism spectrum disorder. *European Child and Adolescent Psychiatry*, *19*, 281-295.
- Russell, A. J., Mataix-Cols, D., Anson, M., & Murphy, D. G. (2005). Obsessions and compulsions in Asperger syndrome and high-functioning autism. *The British Journal of Psychiatry*, *186*, 525-528.
- Schall, C. M. (2010). Positive behavior support: Supporting adults with autism spectrum disorders in the workplace. *Journal Of Vocational Rehabilitation*, *32*, 109-115.
- Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). Childhood Autism Rating Scale, 2<sup>nd</sup> Edition. Los Angeles, CA: Western Psychological Services.

- Schroeder, S., Mulick, J. A., & Rojahn, J. (1980). The definition, taxonomy, epidemiology, and ecology of self-injurious behavior. *Journal of Autism and Developmental Disorders, 10*, 417- 432.
- Seltzer, M. M., Shattuck, P, Abbetuto, L, & Greenburg, J S. (2004). Trajectory of development in adolescents and adults with autism. *Mental Retardation and Developmental Disabilities Research Reviews, 10*, 234-247.
- Shattuck, P. T., Seltzer, M. M., Greenburg, J. S., Orsmond, G. I., Bolt, D. Kring, S. Et al, (2007). Change in autism symptoms and maladaptive behaviorsi n adolescents and adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders, 37*, 1735-1747.
- Sigman, M., Dissanayake, C., Arbelle S., & Ruskin, E. (1997). Cognition and emotion in children and adolescents with autism. In D.Cohen and F. R. Volkmar (Eds.), *Handbook of Autism and Pervasive Developmental Disorders*, 2<sup>nd</sup> edition. New York: John Wiley and Sons.
- Sinzig, J., Walter, D., & Doepfner, M. (2009). Attention Deficit/Hyperactivity Disorder in Children and Adolescents with Autism Spectrum Disorder. *Journal of Attention Disorders, 13,* 117-126.
- Sparrow, S. S., Balla, D., & Cicchetti, D. (1984). *Vineland Adaptive Behavior Scales*. Circle Pines, MN: American Guidance Service.
- Sparrow, S. S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland-II: Vineland Adaptive Behavior Scales, Second Edition. Survey Forms Manual.* Minneapolis, MN: Pearson Assessments.
- Spitzer, R. L. & Siegel, B. (1990). The DSM-III-R Field Trial of Pervasive Developmental Disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 29, 855-862.
- Starr, E., Szatmari, P., Bryson, S., & Zwaigenbaum, L. (2003). Stability and change among high-functioning children with pervasive developmental disorders: A 2year outcome study. *Journal of Autism and Developmental Disorders*, 33, 15-22.
- Stone, W., Lee, E., Ashford, L., Brissie, J., Hepburn, S., Coonrod, E., & Weiss, B. H. (1999). Can autism be reliably diagnosed in children under 3 years? *Journal of Child Psychology and Psychiatry, 40*, 219-226.
- Stone, W.L., McMaohn, C. R., & Henderson, L. M. (2008). Use of the screening tool for autism in two-year-olds (STAT) for children under 24 months: An exploratory study. *Autism: The international Journal of Research and Practice, 12*, 557-573.

- Tanguay, P. E. (2011). Autism in DSM-5. *The American Journal of Psychiatry, 168*, 1142-1144.
- Tate, B., & Baroff, G. (1966) Aversive control of self-injuriou behavior in a psychotic boy. *Behaviour Research and Therapy, 4*, 281-287.
- Teatero, M. L., & Netley, C. (2013). A Critical Review of the Research on the Extreme Male brain Theory and Digit Ratio (2D:4D). *Journal of Autism and Developmental disorders, 43*, 2664-2676.
- Turygin, N., Matson, J. L., Beighley, J., & Adams, H. (2013). The effect of DSM-5 criteria on the developmental quotient in toddlers diagnosed with autism spectrum disorder. *Developmental Neurorehabilitation, 16*, 38-43.
- Tsai, L. (2014). Impact of DSM-5 on epidemiology of Autism Spectrum Disorder. *Research in Autism Spectrum Disorders, 8*, 1454-1470.
- Tsakanikos, E., Costello, H., Holt, G., Bouras, N., Sturmey, P., & Newton, T. (2006). Psychopathology in Adults with Autism and Intellectual Disability. *Journal of Autism and Developmental Disorders, 36*, 1123-1129.
- Van der Meer, J. M. J., Oerlemans, A. M., van Steijn, D. J., Lappenschaar, M. G. A., de Sonneville, L. M. J., Buitelaar, J. K., & Rommelse, N. N. (2012). Are autism spectrum disorder and attention/deficit hyperactivity disorder different manifestations of one overarching disorder? Cognitive and symptom evidence from a clinical and population based sample. *Journal of the American Academy* of child and Adolescent Psychiatry, 51, 1160-1172.
- Ventner, A., Lord, C., & Schopler, E. (1992). A follow-up study of high-fucntioning autistic children. *Journal of Child Psychology and Psychiatry, 3*, 489-507.
- Volkmar, F. R., Bregman, J., Cohen, D. J., & Cicchetti, D. V. (1988). DSM-III and DSM-III-R Diagnoses of Autism. *The American Journal of Psychiatry*, *145*, 1404-1408.
- Volkmar, F., Lord, C., Bailey, A., Schultz, R., & Klin, A. (2004). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, *45*, 135-170.
- Volkmar, F. R., & Reichow, B. (2013). Autism in DSM-5: progress and challenges. *Molecular Autism, 4*, 13.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., Berelowitz, M., Dhillon, A. P., Thomson, M. A., Harvey, P., Valentine, A., Davies, S. E., & Walker-Smith, J. A. (1998). Retracted: Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351.

- Wallace, G. L., Eisenburg, I. W., Robustelli, B., Dankner, N., Kenworthy, L., Giedd, J. N., & Martin, A. Longitudinal cortical development during adolescence and young adulthood in autism spectrum disorder: increased cortical thinning but comparable surface area changes. *Journal of American Child and Adolescent Psychiatry, 54*, 464-469.
- Warne, R. (2014). A Primer on Multivariate Analysis of Variance (MANOVA) for Behavioral Scientists. *Practical Assessment, Research & Evaluation*, 19, 1-10.
- Watson, L. R., Patten, E., Baranek, G. T., Poe, M., Boyd, B.A., Freuler, A., & Lorenzi, J. (2011). Differential associations between sensory response patterns and langauge, social, and communication measures in children with autism or other developmental disabilities. *Journal of Speech, Language, and Hearing Research, 54*, 1562-1576.
- Werner, E., Dawson, G., Munson, J., & Osterling, J. (2005). Variation in early developmental course in autism and its relation with behavioral outcome at 3-4 years of age. *Journal of Autism and Developmental Disorders, 35,* 337-350.
- Weiss, J. Y., & Lunsky, Y. (2010). Group cognitive behaviour therapy for adults with Asperger syndrome and anxiety or mood disorder: a case series. *Clinical Psychology & Psychotherapy*, *17*, 438-446.
- Wing, L. (1981a). Asperger's syndrome: a clinical account. *Psychological Medicine, 11*, 115-119.
- Wing, L. (1981b). Language, social, and cognitive impairments in autism and severe mental retardation. *Journal of Autism and Developmental Disorders, 10*, 31-44.
- Wing, L. (1981c) Sex ratios in early childhood autism and related conditions. *Psychiatry Research*, *5*, 129–137.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children. Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*, 11-29.
- Wing, L., Gould, J., & Gillberg, C. (2011). Autism spectrum disorders in the DSM-V: Better or worse than the DSM-IV? *Research in Developmental Disabilities, 32*, 768, 773.
- Wuench, K. (2015). Binary Logistic Regression with SPSS. http://core.ecu.edu/psyc/wuenschk/MV/multReg/Logistic-SPSS.pdf.

- Yoshida, W., Dziobek, I., Kliemann, D., Heekeren, H. R., Friston, K. J., & Dolan, R. J. (2010). Cooperation and heterogenity of the autistic mind. *Journal of Neuroscience*, *30*, 8815-8818.
- Young, R. L., & Rodi, M. L. (2014). Redefining Autism Spectrum Disorders using DSM-5: The Implications of the Proposed DSM-5 criteria for Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 44, 758-765.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carder, A., Carver, L. (2009). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Insights from studies of high-risk infants. *Pediatrics, 123*, 1383-1391.
- Zwaigenbaum, L., Thurm, A., Stone, W., Baranek, G., Bryson, S., Iverson, J., Kau, A., Klin, A., Lord, C., Landa, R., Rogers, S., & Sigman, M. (2007). Studying the emergence of autism spectrum disorders in high-risk infants: Methodological and practical issues. *Journal of Autism and Developmental Disorders*, *37*, 466-480.

# APPENDIX A: DIAGNOSTIC GROUP ALGORITHM

DSM-IV-TR	DSM-5
Social Interaction (need 1)	Social Communication/interaction- multiple contexts
<ul> <li>1d. Social/emotional reciprocity</li> <li>1e. Not seeking/using others for comfort</li> <li>2b. Impairment to initiate/sustain</li> <li>conversation</li> <li>2e. Lack of emotional response</li> </ul>	A1. Deficits in social emotional reciprocity
<ul><li>1a. Nonverbal behavior, eye contact</li><li>1c. Seeking to share enjoyment</li><li>g. Impaired use of gestures</li></ul>	A2. Deficits on nonverbal communicative behavior
1b. Relationships	A3. Deficits in developing, maintaining, understanding relationships
Need one from each of 2	Restricted/repetitive
2c. Stereotyped repetitive language	B1. Stereotyped/repetitive
3c. Stereotyped motor mannerisms	movements, objects, speech
3b. Inflexible routines/rituals 2d. Lack of varied, spontaneous make- believe play	B2. Insistence on sameness, inflexible adherence to rituals of verbal/nonverbal behavior
<ul> <li>3a. stereotyped/restricted patterns of interest</li> <li>3e. Interest in unusual objects</li> <li>3f. Distress over non-functional details</li> <li>3d. Persistent preoccupation w/ parts of objects</li> </ul>	B3. Highly restricted/fixated interests
DASH 11 Does not respond to nearby light/movement w/ vision present DASH 17 Does not respond to nearby sound w/ hearing present	B4. Hyper-or hypo-reactivity to sensory input
Delay or abnormal function age 3	C. Symptoms present in early dev. period
Delay or abnormal function age 3	D. Symptoms are impairing
Not Better explained by ID	E. Not better explained by ID or global developmental delay

## APPENDIX B: IRB APPROVAL

### ACTION ON PROTOCOL CONTINUATION REQUEST



Institutional Review Board Dr. Dennis Landin, Chair 130 David Boyd Hall

Baton Rouge, LA 70803 P: 225.578.8692

F: 225.578.5983 irb@lsu.edu | lsu.edu/irb

Johnny Matson Psychology Dennis Landin

DATE: October 14, 2015

RE: IRB# 3288

TO:

FROM:

TITLE: Norming and Development of Measures for Treatment and Diagnosis of Individuals with Intellectual and Developmental Disabilities

New Protocol/Modification/Continuation: Continuation

Chair, Institutional Review Board

Review type: Full \_\_\_ Expedited X Review date: 10/13/2015

Risk Factor: Minimal X Uncertain Greater Than Minimal

Approved X Disapproved

Approval Date: 10/13/2015 Approval Expiration Date: 10/12/2016

Re-review frequency: (annual unless otherwise stated)

Number of subjects approved: 500

LSU Proposal Number (if applicable):

Protocol Matches Scope of Work in Grant proposal: (if applicable)

By: Dennis Landin, Chairman

#### PRINCIPAL INVESTIGATOR: PLEASE READ THE FOLLOWING -Continuing approval is CONDITIONAL on:

- 1. Adherence to the approved protocol, familiarity with, and adherence to the ethical standards of the Belmont Report, and LSU's Assurance of Compliance with DHHS regulations for the protection of human subjects\*
- 2. Prior approval of a change in protocol, including revision of the consent documents or an increase in the number of subjects over that approved.
- 3. Obtaining renewed approval (or submittal of a termination report), prior to the approval expiration date, upon request by the IRB office (irrespective of when the project actually begins); notification of project termination.
- 4. Retention of documentation of informed consent and study records for at least 3 years after the study ends.
- 5. Continuing attention to the physical and psychological well-being and informed consent of the individual participants, including notification of new information that might affect consent.
- 6. A prompt report to the IRB of any adverse event affecting a participant potentially arising from the study.

undin

- 7. Notification of the IRB of a serious compliance failure.
- 8. SPECIAL NOTE:

\*All investigators and support staff have access to copies of the Belmont Report, LSU's Assurance with DHHS. DHHS (45 CFR 46) and FDA regulations governing use of human subjects, and other relevant documents in print in this office or on our World Wide Web site at http://www.lsu.edu/irb

## VITA

Nicole Turygin earned her Bachelor of Science degree in psychology from the University of Florida with honors in 2006. 2010, she completed her Master of Arts from George Mason University, where she completed her master's thesis entitled "Visual Scanning of Facially Displayed Affect by Individuals with Autism Spectrum Disorders." She enrolled in the clinical psychology graduate program at Louisiana State University in Fall 2010 under the supervision of Johnny L. Matson. She completed her predoctoral internship at Indiana University Medical Center with an autism emphasis.