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Factor Analysis and Cut-Off Scores for the Autism Spectrum Disorders-Observation for Children

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FACTOR ANALYSIS AND CUT-OFF SCORES FOR THE
AUTISM SPECTRUM DISORDERS-OBSERVATION FOR CHILDREN

A Dissertation

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

in

The Department of Psychology

by

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Commonly Used Abbreviations

AD	Autistic Disorder
ADDM	Autism and Developmental Disorders Monitoring Network
ADOS	Autism Diagnostic Observation Schedule
ADOS-2	Autism Diagnostic Observation Schedule, Second Edition
ADOS-G	Autism Diagnostic Observation Schedule – Generic
AGRE	Autism Genetic Resource Exchange
AOSI	Autism Observation Scale for Infants
APA	American Psychiatric Association
ASD	Autism Spectrum Disorder
ASD-OC	Autism Spectrum Disorders – Observation for Children
AtypDev	Atypically Developing
AUC	Area Under the Curve
BOS	Behavior Observation System for Autism
BRIAAC	Behavior Rating Instrument for Autistic and Atypical Children
CARS	Childhood Autism Rating Scale
CDC	Centers for Disease Control and Prevention
DD	Developmental Disability
DSM	Diagnostic and Statistical Manual of Mental Disorders
EFA	Exploratory Factor Analysis
ICD-10	International Statistical Classification of Diseases and Health Related Problems, 10 th Edition
ID	Intellectual Disability
MMR	Measles, Mumps, and Rubella
NPV	Negative Predictive Value
PAF	Principal Axis Factors
PDD	Pervasive Developmental Disorder
PDD-NOS	Pervasive Developmental Disorder-Not Otherwise Specified
PL-ADOS	Prelinguistic Autism Diagnostic Observation Schedule
PPV	Positive Predictive Value
ROC	Receiver Operating Characteristics
RRBIs	Restricted and Repetitive Behaviors and Interests
STAT	Screening Test for Autism in Two-year-olds
TypDev	Typically Developing

Abstract

Optimal prognoses for children with Autism Spectrum Disorders (ASDs) often rely upon early intervention; thus, there has been a call for reliable and valid assessment tools in order to ensure accurate diagnoses among youth at risk for developmental disabilities (DDs) such as autism. The target of this paper is to inspect the underlying factor structure of a recently developed observation tool for assessing autistic symptoms, the *Autism Spectrum Disorders – Observation for Children (ASD-OC)*. More importantly, cutoff scores were also developed for clinical use in order to distinguish between those with and without an ASD. Given that marked changes were made to ASD diagnostic criteria with the release of the most recent *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)*, two sets of cutoff scores were developed according to the *DSM-IV-TR* and *DSM-5*. Study 1 found that the underlying factor structure of the *ASD-OC* was best explained by two components (i.e., social/communicative behaviors and repetitive/restricted behaviors and interests). Studies 2 and 3 found the *ASD-OC* to have excellent discriminating ability when differentiating between those with and without ASD according to both the *DSM-IV-TR* and the *DSM-5*. Corresponding cutoff scores were developed based upon these analyses. Although there are a number of ASD observation tools already in existence, the ability of the *ASD-OC* to satisfy many of the shortcomings of these pre-existing measures appears to be promising.

Introduction

Autism Spectrum Disorders (ASDs), also known as Pervasive Developmental Disorders (PDDs), are a group of neurodevelopmental disorders which manifest in early childhood. ASDs include Autistic Disorder (more commonly known as autism), Asperger's Disorder, Childhood Disintegrative Disorder, Rett's Disorder, and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS). These disorders are characterized by pervasive impairments in socialization and communication, as well as the presence of repetitive or restricted behaviors or interests (Barbaro & Dissanayake, 2009; Bodfish, Symons, Parker, & Lewis, 2000; Cederlund, Hagberg, & Gillberg, 2010; Charman, 2008; Duffy & Healy, 2011; Fodstad, Matson, Hess, & Neal, 2009; Landa & Garrett-Mayer, 2006; Lord & Luyster, 2006; Matson, Dempsey, et al., 2012; Matson, Wilkins, & Gonzalez, 2008; Matson, 2008, 2009; Zwaigenbaum et al., 2007).

Over the past decade, ASDs have become popular both within the public media and the scientific community. Etiology, prevalence, treatment efficacy, and diagnostic criteria of ASDs have all been points of contention between researchers, clinicians, and parents, alike. Nonetheless, researchers generally agree that optimal performance outcomes of children with ASDs depend on early intervention, which relies on early assessment, identification, and diagnosis (Dawson, 2013; Elsabbagh & Johnson, 2007; Frazier et al., 2011; Manning-Courtney et al., 2003; Martinez-Pedraza & Carter, 2009; Matson, 2007a; Matson & Konst, 2013; Peters-Scheffer, Didden, Korzilius, & Sturmey, 2011; Reichow, Barton, Boyd, & Hume, 2012; Werner, Dawson, Osterling, & Dinno, 2000; Zwaigenbaum et al., 2009a). Therefore, the importance of research into the development of assessment tools to assist clinicians in the early diagnosis of ASDs cannot be underestimated.

The *Autism Spectrum Disorder-Observation for Children (ASD-OC)* is a recently developed clinician-rated direct observation scale which assesses autistic symptomatology in children (Neal, Matson, & Belva, 2013). Neal and colleagues have recently established strong reliability (Neal et al., 2013), validity (Neal, Matson, & Hattier, 2014), and discriminative ability (Neal, Matson, & Belva, 2012) for this measure, and the current study builds upon this previous research. The aim of this study is three fold. First, the factor structure of the *ASD-OC* was established. Second, total cutoff scores were determined in order to distinguish between individuals with and without an ASD according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)* formalized diagnostic criteria. Finally, total cutoff scores were also determined to distinguish between those with and without ASD according to the revised *DSM-5* criteria. This is one of the first ASD observation tools with two sets of cutoff scores matching each of the two sets of formalized diagnostic criteria for autism. Having dual cutoff scores will assist practitioners in the transition to the newly adopted criteria, while providing continuity across diagnoses. The history of autism, core features of ASDs, and comprehensive assessment of these disorders is discussed. Additionally, a detailed review of all currently available ASD observation measures is provided.

Autism Spectrum Disorders

History

Although many today understand the term “autism” as referring to a disorder manifesting in early childhood characterized by deficits in communication and socialization, as well as repetitive behaviors, this was not always the case. In 1908, a Swiss psychiatrist by the name of Eugen Bleuler coined the term “autism” to refer to a cluster of symptoms present in many individuals diagnosed with schizophrenia (Bleuler, 1913). This “autistic” symptom cluster consisted of a withdrawal from interactions not only with other individuals but from the world as a whole. Bleuler described an autistic schizophrenic individual as one who “ceases to care about the real world. He shows a lack of initiative, aimlessness, neglect of reality, distractedness, but also impulsive and bizarre behaviour. Many of his actions, as well as his whole attitude to life are insufficiently externally motivated” (Bleuler, 1919). During this time, children presenting with autistic-like symptoms were usually diagnosed with childhood schizophrenia. While Bleuler’s autism is, in some ways, similar to the present day definition, it was not until 1943 when the term “autism” would be used in a way synonymous with today’s interpretation of the disorder.

“Autistic Disturbances of Affective Contact” would be the revolutionary publication bringing heed to one of the most serious disorders of childhood (Kanner, 1943). Leo Kanner, an Austrian child psychiatrist practicing at Johns Hopkins University, was the first to publicly depict his observations of children with autistic features. In this seminal paper, Kanner described the behavioral presentation of 11 children between 2 and 8 years of age, each with symptoms not fully meeting the criteria of any existing diagnoses at the time. He observed eight male and three female children all with impairments in language, social skills, and an unrelenting adherence to

nonfunctional routines. Kanner labeled this constellation of deficits as “early infantile autism” (Kanner & Eisenberg, 1956).

With regard to communication deficits, Kanner described all 11 children as having some sort of language impairment. In fact, three of the 11 children never acquired verbal communication, while the other eight were able to speak but with unusual or idiosyncratic verbalizations. Immediate and delayed echolalia (i.e., the repetition of previously heard words or phrases) and pronoun reversal (e.g., confusing ‘you’ and ‘I’) were also observed in several of the children. Socially, the children preferred to isolate themselves from others and actively avoided attempts at interaction from others; Kanner described this as an “extreme autistic aloneness which, whenever possible, disregards, ignores, and shuts out anything that comes to the child from the outside” (Kanner, 1944, p. 211). He noted that this desire to isolate oneself was present even from birth. While some of the children would respond to simple instructions, none would actively engage in reciprocal conversation with others. Lastly, all 11 children displayed some sort of persistence to maintain sameness throughout their daily routines. For example, some of the children insisted on following the same path or playing with the same toys/objects each and every day. The children seemed to expend all of their time and attention on the toys/objects that were of interest to them and would rarely vary their play to include other toys. Additionally, any deviation from their routine would typically result in extreme agitation (Kanner, 1944).

Unknowingly, an Austrian graduate student by the name of Hans Asperger concurrently published a similar description of childhood symptoms. Asperger’s 1944 thesis entitled “Autistic Psychopathy in Childhood” did not receive much notoriety until 1991 when Uta Frith translated it into English (Asperger, 1991). He described in detail four children who each presented with communication and socialization impairments, as well as other unusual and

restricted behaviors. Asperger's descriptions, however, differed slightly from Kanner's observations. For example, Asperger described some of the children he observed as having overly sophisticated speech and some even "talking like an adult." He also highlighted that in many cases the children displayed severe conduct problems, which were often the reason for referral.

As confusion grew in many diagnosticians due to the conflicting descriptions of the term "autism," it was crucial that researchers set forth to clearly delineate infantile autism from other analogous conditions (e.g., childhood schizophrenia, intellectual disability [ID]; Hingtgen & Bryson, 1972; Kanner, 1965). Unfortunately, it was this confusion that led many clinicians to use several diagnostic terms interchangeably, including "infantile autism, the atypical child, symbiotic psychosis, dementia praecocissima, dementia infantilis, schizophrenic syndrome of childhood, pseudopsycopathic schizophrenia, and latent schizophrenia" (Rutter, 1972). The volume of research that would follow is what ultimately warranted autism to be established as its own distinct disorder, and Kanner, Asperger, and Rutter would be some of the key players in this pivotal exploration.

First and foremost, it was essential that autism and childhood schizophrenia be distinguished from one another (Kanner, 1965). Although autistic symptomatology closely resembled that of childhood schizophrenia, there were multiple elements that set the two apart including age of onset, manifestation of isolation from others, the trajectory of the disorder, gender ratio, and other ancillary symptoms (Kanner & Eisenberg, 1956). In those with autism, the age of onset is typically during infancy and even sometimes from birth; whereas, symptoms do not usually arise until early adolescence for those with childhood schizophrenia (Volkmar, 1996). The way in which children with autism and children with childhood schizophrenia tend

to relate to others or engage in social isolation from others also tends to differ. Children with schizophrenia are generally able to initially establish relationships with others but then later withdraw from this social interaction as they age. Conversely, children with autism fail to ever develop social relationships with others from the very beginning (Asperger, 1991; Rutter, 1968, 1978). The trajectory of these two disorders also differs, where autism has been found to be a relatively stable disorder but childhood schizophrenia tends to present in cyclical periods of remissions and relapses (Volkmar, 1996). In 1978, Rutter also found that autism tended to have a 4:1 gender ratio from males to females, which has been replicated by other researchers (Baron-Cohen et al., 2011), yet childhood schizophrenia is found to generally be evenly dispersed among both genders. Finally, those with childhood schizophrenia tend to experience delusions and hallucinations, which are not evident in those with autism (Rutter, 1968). With this evidence, researchers and clinicians began to recognize autism as a separate and distinct entity (Kanner, 1965; Rimland, 1964).

Second, researchers also strived to differentiate autism from ID. Originally, it was believed that ID did not occur in those with autism, as Kanner described that “even though most of these children were at one time or another looked upon as feeble-minded, they are unquestionably endowed with good cognitive potentialities. They all have strikingly intelligent physiognomies” (Kanner, 1944, p. 217). He attributed this “good intelligence” to their superior rote memory and normal physical appearance. At the time, many others also believed that most children with a diagnosis of autism had average intelligence (Bettelheim, 1967; Kanner & Lesser, 1958; Rimland, 1964). However, Rutter (1968) did not believe this to be true due to surmounting evidence that many children with autism “function at a mentally subnormal level” (p. 5). Just as in the typically developing population, Lockyer and Rutter (1968) found the IQ of

children with autism to remain relatively stable over time despite improvements in their autistic symptoms. Nonetheless, approximately 25% to 33% of children with autism were found to function in the average range of intellect (Rutter, 1968; Rutter & Lockyer, 1967). Ben-Izchak and Zachor (2007) noted that researchers from the 1970's generally found that the majority of children with autism have mean IQ scores ranging from 45 to 50. Today, researchers have found that about 50% to 75% of all children with an ASD have a comorbid diagnosis of ID (Matson & Shoemaker, 2009).

In sum, autism has evolved over time from a subset of symptoms of schizophrenia to its own separate entity. Through Rutter's work distinguishing autism from childhood schizophrenia, ID, other neuroses, and developmental language disorders, autism was confirmed to be an independent diagnosis. Researchers were now faced with the task of clearly delineating the diagnostic criteria one must meet to obtain a diagnosis of autism. Although some criteria have changed, ASDs, today, are still characterized with Kanner's original three core deficits (i.e., communication, socialization, and restricted/repetitive behaviors or interests). The progression of these changes in diagnostic criteria will now be discussed.

Autism Defined

Naturally, one of the first to establish solid criteria for diagnosing autism was Kanner along with his colleague, Eisenberg (1956). They defined early infantile autism as having two core features: "extreme aloneness and preoccupation with the preservation of sameness" (Kanner & Eisenberg, 1956, p.63). Additionally, Kanner and Eisenberg noted that these symptoms manifest prior to 2 years of age. Since Kanner's (1943) initial description of autism, several other researchers have offered their own definitions and explanations of the disorder (e.g., Creak, 1961; Ritvo, 1978; Rutter, 1968, 1972, 1978; Rutter & Bartak, 1971). Despite this, the three

core symptoms originally described by Kanner (1943) have remained the core diagnostic characteristics in formal diagnostic classification systems.

The American Psychiatric Association (APA) established a task force of medical professionals to devise a classification system of psychological disorders to which physicians could refer in order to make the process of diagnosing simpler and more accurate due to the surge of veterans experiencing psychiatric problems following World War II (Shorter, 1997). In 1952, this task force published the *Diagnostic and Statistical Manual of Mental Disorders, First Edition (DSM-I)*. Autism first appeared in the third edition of the *DSM* (APA, 1980) as “infantile autism,” which was subsumed within the category of Pervasive Developmental Disorders (also known as ASDs). Other disorders classified within the PDD category in the *DSM-III* included residual infantile autism, childhood onset PDD, residual childhood onset PDD, and atypical autism (Volkmar & Klin, 2005). In the revised version of the *DSM-III*, the PDD category was simplified to only two disorders: Autistic Disorder and PDD-NOS (APA, 1987; Waterhouse, Wing, Spitzer, & Siegel, 1989). Additionally, the *DSM-III-R* included an age of onset criterion requiring pervasive deficits to be evident prior to 30 months of age (APA, 1987).

Today, the *DSM-IV-TR* provides a multi-axial approach to the diagnosis of psychological disorders, which was initially introduced in the third edition of the *DSM* and is still in effect today (APA, 2000). In the *DSM-IV-TR*, ASDs include Autistic Disorder, PDD-NOS, Asperger’s Disorder, Childhood Disintegrative Disorder, and Rett’s Disorder. Another prominent classification system is the *International Statistical Classification of Diseases and Health Related Problems, 10th Edition (ICD-10)*; World Health Organization [WHO], 1992). The criteria for all ASDs significantly overlap as the diagnostic criteria for the *DSM-IV-TR* was based upon a field trial which used criteria from the *DSM-III*, *DSM-III-R*, and *ICD-10* (Volkmar et al., 1994).

As the *DSM-IV-TR* is more widely used throughout the United States, the remainder of the paper will refer to this classification system for diagnostic criteria purposes (Matson & Minshawi, 2006; Volkmar & Pauls, 2003). Kanner's three core characteristics are apparent in all five ASDs; however, each disorder has its own more specific qualifications distinguishing itself from the rest. A brief but comprehensive summary of the current diagnostic criteria for Autistic Disorder and PDD-NOS will follow, as these are the two diagnoses germane to this current study.

Autistic Disorder. In order to qualify for a diagnosis of Autistic Disorder, or autism, the child must exhibit deficits in each of the three core features. In total, at least six of the following criteria must be met and the child's symptomatology must not be better explained by another disorder (e.g., Rett's Disorder or CDD). Within the socialization domain, at least two of these four impairments must be evident: (1) impairment in multiple nonverbal behaviors; (2) failure to develop social relationships with peers; (3) failure to engage in spontaneous sharing of interests with others; or (4) a lack of social or emotional reciprocity. At least one of the total six impairments must stem from the communication domain: (1) impairment in, or lack of, verbal communication (without compensation through alternative forms of communication); (2) impairment in initiating and maintaining conversations; (3) stereotyped or idiosyncratic language characteristics; or (4) a lack of age-appropriate pretend play. Finally, at least one of the six total symptoms must derive from the repetitive/restricted behaviors and interests domain: (1) preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal in intensity or focus; (2) a fixation with adhering to nonfunctional routines or rituals; (3) stereotyped or repetitive motor movements; or (4) a persistent preoccupation with parts of

objects. Symptoms in at least one of these three areas (social interaction; language use; pretend play) must be evident prior to 3 years of age (APA, 2000).

PDD-NOS. A child may qualify for a diagnosis of PDD-NOS, also known as “atypical autism” (Inglese & Elder, 2009b), for a number of reasons. Although PDD-NOS is the most commonly diagnosed ASD (Chakrabarti & Fombonne, 2005; Matson & Boisjoli, 2007), there are no specific criteria or guidelines one must meet when diagnosing PDD-NOS (APA, 2000). As stated in the *DSM-IV-TR*, the individual must have “a severe and pervasive impairment in the development of reciprocal social interactions or with the presence of stereotyped behavior, interests, and activities” to receive this diagnosis (APA, 2000, p. 84). In addition, the symptoms must not be better explained by another mental disorder.

Due to this vague description, there are a number of potential reasons a child may be given a diagnosis of PDD-NOS. Many clinicians have described this diagnosis as a “catchall” diagnosis for those children who do not meet full criteria for any other ASD, yet still exhibit significant ASD-like characteristics (Matson & Boisjoli, 2007; Matson & Minshawi, 2006; Tidmarsh & Volkmar, 2003). Walker et al. (2004) describe PDD-NOS as a “midway between the autism and [Asperger’s Disorder] groups on IQ, measures of adaptive behavior, and language milestones” (p. 178). This lack of any clear definition has posed many problems for diagnosticians when attempting to reliably diagnose the disorder; therefore, some have attempted to establish clear-cut description for this disorder. For example, Buitelaar and Van der Gaag (1998) established four situations in which a diagnosis of PDD-NOS is warranted: (1) when the age of onset is after 3 years of age; (2) when there is a presence of atypical symptoms that do not map on exactly to the *DSM-IV-TR* criteria for another ASD; (3) when the child’s symptoms are subthreshold; or (4) when the child’s symptoms do not meet the requirements for a diagnosis of

autism. Walker and colleagues (2004) also established more specific diagnostic criteria for PDD-NOS: (1) when the child fails to meet the domain criteria of Autistic Disorder in one of two domains (communication deficits or the presence of repetitive, stereotyped behaviors), or (2) when the child has fewer than six symptoms total. While this gives some guidance to diagnosticians, there is still no clear definition of PDD-NOS.

DSM-5. With the recent release of the fifth edition of the *DSM* in May of 2013, the diagnostic criteria and conceptualizations for ASD were vastly changed. To begin, the term ‘Autism Spectrum Disorder’ now replaces ‘Pervasive Developmental Disorder,’ since this has been the trend amongst clinicians, researchers, and parents alike for quite some time (APA, 2011; Kim & Lord, 2013). Furthermore, ASD no longer subsumes five distinct disorders but functions as its own singular disorder with severity qualifiers. More importantly, the longstanding triad of core features were collapsed into a dyad of symptoms: (1) social communication and interaction and (2) restricted/repetitive behaviors or interests. To qualify for an ASD diagnosis, the child must first meet the following three criteria within the first domain of social communication and interaction: (a) impairment in social and emotional reciprocity; (b) impairment in nonverbal behaviors used for social interaction; and (c) deficits in the development and maintenance of peer relationships. The child must also have at least two of the following criteria within the second domain of restricted/repetitive behaviors: (a) stereotyped or repetitive speech, motor movements, or use of objects; (b) inflexible adherence to nonfunctional routines or excessive resistance to change; (c) highly restricted interests abnormal in intensity and focus; and (d) hyper- or hypo-reactivity to or an abnormal interest in sensory stimuli. Lastly, these symptoms must be present in early childhood and must cause significant impairments in daily functioning. Each ASD diagnosis can also be qualified with a severity indicator: Level 3 –

requires very substantial support; Level 2 – requires substantial support; Level 1 – requires support. Kim and Lord (2013) state that the *DSM-5* also provides examples of symptoms for different age ranges and level of linguistic ability.

These changes have been a source of contention among clinicians and researchers in the field of ASDs (Ghaziuddin, 2010; Matson, Belva, Horovitz, Kozlowski, & Bamburg, 2012; Matson, Hattier, & Williams, 2012; Matson, Kozlowski, Hattier, Horovitz, & Sipes, 2012; McPartland, Reichow, & Volkmar, 2012; Wing, Gould, & Gillberg, 2011; Worley & Matson, 2012). Given that autism is characterized by a variety of different impairments, Szatmari (2000) argued that specifying the severity of this disorder would be difficult, as one child may be more impaired in one area than another when compared other children. In fact, severity ratings were piloted for a number of disorders in the *DSM-III-R* but were not included in the *DSM-IV* due to a lack of validation (Frances, 2010). Frances (2010) foresees the severity ratings in the *DSM-5* to be complicated and impractical for clinical purposes. Similarly, autistic symptoms have been shown to change as the child ages. For example, lower-order repetitive motor movements (e.g., hand-flapping) are more evident in early childhood, while these develop into more higher-order ritualized routines as that child enters adulthood (Bishop, Richler, & Lord, 2006). Since there can be variability in symptoms over time, one's severity qualifier may need to be modified to accurately reflect their current behavioral presentation. Another area of concern for many is the removal of Asperger's syndrome and PDD-NOS, which will greatly affect the social stigma associated with a diagnosis of ASD and the availability of treatment services (Frances, 2010; Matson, Hattier, et al., 2012; Wing et al., 2011).

Most importantly, a number of researchers have estimated that the new *DSM-5* diagnostic criteria will have a large impact on prevalence rates. Specifically, McPartland et al. (2012),

Matson, Belva et al. (2012), Matson, Kozlowski et al. (2012), and Worley and Matson (2012) all found that approximately 30% to 45% of individuals currently diagnosed with ASDs according to *DSM-IV-TR* criteria will no longer qualify for an ASD diagnosis when the new *DSM-5* criteria is applied. This will likely result in a loss of treatment services, given that most insurance plans require a formal diagnosis to qualify for reimbursement of services (Worley & Matson, 2012). In addition to clinicians and clients, these changes may also affect researchers, as it will be difficult to extend longitudinal studies across both the *DSM-IV-TR* and *DSM-5*.

Etiology

As expected, since a definitive etiology for ASDs does not exist, theories of etiology continue to multiply. Michael Rutter (Rutter, 2002), a prominent researcher in the field of autism, once stated, “it’s a dull month that goes by without a new cause for autism.” Shortly after Kanner’s first description of autism, researchers began searching for the source or cause of this disorder. Initially, all hypotheses regarding the etiology of autism were related to the social environment. The first theory to gain popularity was the psychogenic theory of autism. This was the idea that the parents or other family-related factors (e.g., parental rejection, insufficient stimulation, faulty communication patterns, family stress) actually caused autism. One popular proponent of the psychogenic theory was Bruno Bettelheim (1967) who proposed that autistic symptomatology was, in fact, the child’s response to a distant or cold parent (i.e., “refrigerator mothers”). According to Bettelheim, “the precipitating factor in infantile autism is the parent’s wish that his child should not exist” (Bettelheim, 1967). The child then interprets their parent’s cold demeanor with hostility and responds by disconnecting from their surrounding environment. Herbert Elovoff (1960) also supported this theory of refrigerator mothers. Although this theory

is not supported by any empirical research, PBS recently aired the film *Refrigerator Mothers* in 2001 as a piece in their *Point of View* series (Schreibman, 2005).

Another environmentally-related theory offered was the learning theory of autism. In the 1960s, Charles Ferster reported that the parents of children with autism typically have a predisposition to depression and often do not attend to their child's behavior; thereby, they are less likely to reinforce their child's positive behavior and more likely to inadvertently only pay attention to the negative behaviors which often cannot be ignored (e.g., tantrums). Ferster suggested that this ultimately reinforces autistic-like behavior in the child (Ferster, 1961). Although researchers have since found that behaviors associated with ASDs can be changed by positive or negative reinforcement, the manifestation of autistic symptoms is not a reaction to depressed or inattentive parents (Lovaas & Smith, 1989).

Once thought to be a disorder caused entirely by environmental factors (Bettelheim, 1967; Inglese & Elder, 2009a), growing evidence now supports the idea that genetic factors also play a role in ASDs. It took many years for researchers to consider that there may be a genetic component to autism, because children with autism rarely have parents also diagnosed with the disorder (Pennington, 2009). Schreibman (2005) lists eight prominent arguments against these social/environmental theories of the cause of autism: (1) researchers have never studied controlled observations of the behavior or personality of parents of children with autism; (2) parents who may be classified as "refrigerator" parents typically have typically developing children; (3) most parents of children with autism do not fit the personalities described above; (4) most of the siblings of children with autism are typically developing; (5) autistic symptomatology has been reported to be present since birth, too early to be affected by parents' personalities; (6) autistic symptoms overlap with some specific types of brain damage; (7) the

3:1 male to female ratio is consistent with other organic disorders; and (8) there is a higher concordance rate for monozygotic twins.

Twin studies have given scientists the first glimpse into the genetic characteristics of ASDs. In 1977, Folstein and Rutter conducted the first twin study of autism and found the concordance rates in monozygotic twins (36%) to be significantly greater than dizygotic twins (0%). Since this original study, other researchers have replicated these findings; however, the concordance rates of autism in monozygotic twins have been found to range from 60-90% (Frith & Happe, 2005; Rosenberg et al., 2009).

In addition to twin studies, researchers have also examined the likelihood of siblings of children already diagnosed with autism to also develop the disorder. Researchers suggest that siblings have a 5-10% risk of developing the disorder themselves (Charman, 2008). In other words, the risk of autism is 20-60% higher in siblings compared to the general population (Geschwind & Konopka, 2009; Pennington, 2009). Another finding supporting the genetic argument for autism is the “broader autism phenotype.” The broader autism phenotype refers to milder forms of autistic symptoms, usually within the communication and socialization domains (e.g., shyness, aloofness, communication delays; Rutter, 2000). Many siblings of children with autism who do not also have an ASD themselves tend to exhibit these more faint forms autistic-like characteristics. About 10-20% of first-degree relatives will experience some sort of mild social and/or communicative delays (Charman, 2008).

Although no identifiable biological markers of ASDs currently exist (Barbaro & Dissanayake, 2009; Bryson et al., 2007; Charman, 2008; Manning-Courtney et al., 2003; Manning-Courtney et al., 2013; Zwaigenbaum et al., 2009b), there is evidence suggesting that chromosomes 2, 7, 16, and 17 house genes susceptible to the development of autism (Folstein &

Rosen-Sheidley, 2001). Some evidence even indicates that social impairments, communication impairments, and RRBI's are all linked to different genes (Happé, Ronald, & Plomin, 2006). This suggests that some genes may be more negatively affected than others in certain ASD children, accounting for the great variability seen across the ASD continuum.

As a supporter of genetic research, Rutter (2000) noted seven reasons why this line of study is beneficial: (1) genetic findings have already affected previously upheld theories of autism; (2) this research raises the need for genetic counseling for families; (3) genetic studies can help better identify the broader autism phenotype; (4) this research may help find the underlying neurological processes that lead to the development of autism; (5) genetic findings may also guide researchers to possible protective factors; (6) this research can inform effective drug treatments; and (7) genetic findings may also help identify environmental risk factors.

Despite the great amount of research supporting a genetic component, some researchers argue that the genetic causes of autism have been overestimated due to methodological flaws, misinterpretations, and exaggerations (Chamak, 2010).

In addition to genetic factors, researchers have attempted to identify many environmental causal factors as well. In 1998, Wakefield and colleagues published an article describing 12 children with gastrointestinal problems. He stated that the measles, mumps, and rubella (MMR) vaccine caused these certain bowel symptoms, which ultimately led to the specific behavioral symptoms indicative of autism (Wakefield et al., 1998). Despite the fact that Wakefield's claim was not founded on empirical evidence and could not be replicated by other researchers, his theory gained popularity with many parents through widespread media coverage (Charman, 2008). In fact, in one survey researchers found that 29% of parents cite immunizations as their cause of their child's autism diagnosis (Harrington, Patrick, Edwards, & Brand, 2006). This

belief among parents ultimately led to a 12% drop in the administration of this vaccine in the UK. In turn, the incidence of measles increased 24-fold over the decade following the release of Wakefield's article (Thomas, 2010).

A year after Wakefield's publication, Taylor and colleagues (1999) conducted a study examining any possible causal links between the MMR vaccine and the incidence of ASDs. The authors found no spike in the number of ASD diagnoses following the introduction of the MMR vaccine in the UK in 1988. This study was extended in 2001, and once again no association between the vaccine and ASDs was found (Farrington, Miller, & Taylor, 2001). It is now widely accepted that there is no causal link between the MMR vaccine and autism (Evans et al., 2001; Farrington et al., 2001; Hornig et al., 2008; Madsen et al., 2003; Offit & Coffin, 2003; Rutter, 2005; Thomas, 2010; Wing & Potter, 2002).

With the MMR vaccine being the most contentious factor claimed to cause autism (Charman, 2008; Wakefield et al., 1998), other suggested environmental influences include valproic acid, medications, prematurity, infections, anoxia at birth, high metal toxicity levels, gluten, and casein (Arndt, Stodgell, & Rodier, 2005; Frith & Happe, 2005; Inglese & Elder, 2009a). Despite the popularity of many of these factors in the media, many of these causes remain uncorroborated (Farrington et al., 2001; Kaye, del Melero-Montes, & Jick, 2001; WHO, 2001; Taylor et al., 1999). The study of environmental factors affecting ASDs has been largely controversial and has yet to result in any scientifically validated environmental factors. Most researchers continue to hold the belief that the cause of ASDs is attributable to a combination of both genetic and environmental factors (Frith & Happe, 2005; Inglese & Elder, 2009a).

Prevalence

Just as with etiology, the prevalence rates of ASDs is a largely debated topic amongst researchers and clinicians. Over time with every new publication of the *DSM*, there has been a substantial rise in the prevalence of autism, and the underlying cause for this increase is still unclear. A number of possible causes have been suggested including: changes in diagnostic tendencies, an increase in possible triggers (e.g., gluten, environmental pollutants, mercury, vaccinations), a broadening of diagnostic criteria and less stringent screeners and assessments, a greater amount of attention dedicated to this disorder and public awareness, or a genuine growth in the disorder (Bertoglio & Hendren, 2009; CDC, 2009b; Chakrabarti, 2001; Chakrabarti & Fombonne, 2005; Elsabbagh & Johnson, 2007; Hebert, Sharp, & Gaudiano, 2002; Inglese & Elder, 2009a; Leonard et al., 2010; Matson & Kozlowski, 2011; Matson, Kozlowski, et al., 2012; Rice et al., 2010; Wing & Potter, 2002).

Initially thought to be very rare, ASDs are one of the most common childhood developmental disorders (Kim & Lord, 2013). The Autism and Developmental Disorders Monitoring Network (ADDM) founded by the Centers for Disease Control and Prevention (CDC) regularly monitors the prevalence of ASDs. CDC estimates from 2009 reported that ASDs occur in approximately 1 in every 110 children (CDC, 2011). Just recently, the CDC updated this statistic to 1 in every 88 children (ADDMN, 2012; CDC, 2012). In a review of epidemiological studies, Campbell, Davarya, Elsabbagh, Madden, and Fombonne (2011) reported the prevalence rates of ASDs overall to be 1 in 143 individuals or 70/10,000. More specifically, Autistic Disorder is estimated to occur in 1 out of every 455 individuals (22/10,000). Current estimates indicate that PDD-NOS is the most prevalent ASD with

prevalence rates of 21 to 36.1 per 10,000 individuals (Chakrabarti, 2001; Chakrabarti & Fombonne, 2005; Fombonne, 2005; Howlin, 2006).

Assessment of ASDs

Due to the apparent rise in ASD prevalence, greater public attention has been brought upon this disorder in recent years (Boyd, Odom, Humphreys, & Sam, 2010; Evans et al., 2001; Inglese & Elder, 2009b; Lord & Luyster, 2006; C. Rice, 2007). While this added attention has led to many advances in this field, it has also brought about great controversy regarding the actual cause for this increase in prevalence (e.g., misdiagnosis, an increase in external triggers, more general diagnostic criteria, more public awareness, or a true growth in ASDs; (Bertoglio & Hendren, 2009; Boyd et al., 2010; Campbell et al., 2011; CDC, 2009a; Chakrabarti, 2001; Chakrabarti & Fombonne, 2005; Croen, Grether, Hoogstrate, & Selvin, 2002; Kogan, 2009; Lynn Waterhouse, 2013). For that reason, it is the utmost responsibility of today's clinicians and diagnosticians to remain well-informed on the most accurate and effective ways to differentiate children with ASDs from those who are typically developing and those with other various developmental disabilities (DDs). Because there are no identifiable biological markers for ASDs (Barbaro & Dissanayake, 2009; Bryson et al., 2007; Manning-Courtney et al., 2003), diagnosis relies heavily upon parent-report and astute behavioral observations (Zwaigenbaum et al., 2009a).

Next, the three main characteristics of ASDs (i.e., socialization impairments, communication deficits, and repetitive/restricted behaviors and interest) will be reviewed. Within each ASD domain, the developmental pathways of children with ASDs will be compared to the development of children with other DDs and children who are typically developing. The developmental trajectory is good to consider since ASDs are believed to be disorders of developmental origin. This developmental aspect of ASDs also suggests that the behavioral presentation may change as the child ages. Therefore, it is also important to examine ASD

symptomatology across various ages in childhood. Researchers have found that ASD symptoms vary substantially across each child and present themselves in different ways. However, nearly all children with an ASD exhibit both an excess of abnormal behaviors (e.g., repetitive hand-flapping) and a lack of appropriate typical behaviors (e.g., failure to initiate interactions with others, failure to respond to one's name; (Lord & Luyster, 2006; Matson & Wilkins, 2007). Therefore, positive and negative symptoms relating to each of the three core features of ASD is also important to consider.

Core Features

Socialization. Bedell & Lennox (1997) define social skills as “the abilities to (a) accurately select relevant and useful information from an interpersonal context, (b) use that information to determine appropriate goal-directed behavior, and (c) execute verbal and nonverbal behaviors that maximize the likelihood of goal attainment and the maintenance of good relations with others” (p. 9). Typically developing infants begin to exhibit social behavior from birth, including recognition of their mothers, a preference for direct eye contact, and social smiling at 2 months of age (Grossman & Johnson, 2007; Johnson, Grossman, & Farroni, 2010). Children with autism, however, exhibit a number of socialization impairments, including: limited eye contact, impaired joint attention, failing to respond to one’s name when called, inappropriate facial expressions and gestures, inability to share enjoyment and interests with others, preferring to be alone, impaired social smiling, impaired pretend play, and lack of social or emotional reciprocity (APA, 2000; Baranek, 1999; Kaland, Mortensen, & Smith, 2011; Smith & Matson, 2010). Impairments in socialization are generally considered to be the main feature of ASDs (Sevin, Knight, & Braud, 2007).

Approximately 30-50% of parents recognize abnormal development during the 1st year of their child's life (Bryson et al., 2007; Gillberg et al., 1990; Hoshino et al., 1987), and socialization impairments, in particular, are often initially mistaken for hearing impairments (e.g., when the child fails to respond to his name when spoken; Eveloff, 1960; Manning-Courtney et al., 2003; Zwaigenbaum et al., 2009a). In a retrospective study, Werner, Dawson, Osterling, and Dinno (2000) found significant differences in responding to one's name between children with autism and typically developing children at 12 months of age. Many note deficits from the time of birth as well, such as preferring to be alone as an infant and not assuming the anticipatory posture when being held (Kanner, 1943; Zwaigenbaum et al., 2005). These recollections from parents regarding their child's early development, however, are often from retrospective studies which are usually prone to biases (Tager-Flusberg, 2010). Through the use of prospective studies, researchers have been able to better examine when certain autistic symptoms arise and the trajectory of those symptoms (Bryson et al., 2007; Landa & Garrett-Mayer, 2006; Ozonoff, Ana-Maria, et al., 2010; Zwaigenbaum et al., 2007).

Longitudinal studies examining specific social deficits, however, are sparse. It is difficult to define and measure social behavior throughout childhood because social behavior relevant to a toddler is very different than what is expected of a 10 year old child. Most longitudinal studies examine more general parent-reported socialization scores rather than specific social behaviors (Thurm, Bishop, & Shumway, 2011). Pry, Peterson, and Baghdadli (2009), however, were of the first to examine the developmental trajectories of several specific social communicative abilities (i.e., expressive language, joint attention, imitation, and play competence) in young children (at least 5 years of age) with ASD over the course of 3 years. The authors found that the development of social skills varied depending on the child's linguistic abilities. For those whose

language improved, their social skills also showed the most improvement. Whereas, for the group who regressed with regards to their language, no significant gains in socialization were found.

As the child ages, some have reported that social withdrawal slightly diminishes (Charman, Taylor, & Drew, 2005; Rutter, 1968); nevertheless, these social impairments usually still have adverse implications for academics and vocational work (Matson, Dempsey, & LoVullo, 2009) and persist over the lifetime (Gallo, 2010). As the child ages, isolating oneself and impaired social and emotional reciprocity are probably two of the most recognizable and impairing social deficits in individuals with ASDs. Llaneza and colleagues (2010) suggest that varying degrees of impairment in these areas can lead to virtually three types of autistic personalities: (1) aloof, (2) passive, and (3) the socially extremely awkward person. A child with ASD with an “aloof” personality tends to avoid physical touch and eye contact with others. A child with a “passive” personality does not necessarily *avoid* contact with others but just does not initiate those interactions. The “socially extremely awkward person” is said to be the least common of the three personalities and is someone who initiates contact with others but is socially awkward in doing so.

Communication. In the *DSM-5* communication impairments are collapsed into one core feature of ASD with socialization impairments. This is due to the fact that some researchers find it difficult to differentiate between communicative and social deficits as these two features are often interrelated with one another (APA, 2011). Communication impairments are often the first concerns reported by parents of children with ASDs (Kozlowski, Matson, Horovitz, Worley, & Neal, 2011; Tager-Flusberg & Caronna, 2007). Problems in this area, however, can often pose the greatest diagnostic difficulties for clinicians as many other disorders present with

communicative impairments as well; thus, it is crucial for diagnosticians to carefully consider other possible diagnoses with similar features (Matson & Neal, 2010).

Typically developing children usually speak their first words around 12 months of age and short phrases around 24 months of age (Tager-Flusberg, 2002). Many researchers have outlined several signs and symptoms of ASDs throughout childhood. Some of the communicative related symptoms include: no babbling by 12 months, no gestural communication by 12 months (e.g., pointing, nodding for *yes* or *no*), no single words by 16 months, no pretend play by 18 months, no two-word phrases by 24 months, and any loss of language in the preschool years (Campbell, 2011; Charman, 2008; Howlin, 2006; Tager-Flusberg & Caronna, 2007). It should be noted, however, that there is great variability in communication and language development in children with ASDs (Tager-Flusberg & Caronna, 2007; Thurm et al., 2011). In fact, Lewis, Murdoch, and Woodyatt (2007) studied linguistic abilities in children with Asperger's Syndrome, high-functioning autism, and typical development and found no significant differences between groups with respect to their comprehensive linguistic assessments. Nevertheless, the majority of children with ASDs exhibit pervasive communication deficits early on, and these deficits persist into adulthood placing a negative impact on other areas of daily functioning.

It has been estimated that approximately 25-50% of children with ASDs never develop any functional speech (Dawson & Murias, 2009; Howlin, 2006; Tager-Flusberg, 2001). Usually those who are able to speak still have great difficulty expressing their wants and needs to others, which can lead to frustration and other problematic behaviors (Beitchman, 2006; Sigafos, 2000). A percentage of children with ASDs (15-50%) also experience a regression in linguistic abilities, typically between the ages of 15 to 24 months (Bertoglio & Hendren, 2009; Hansen et

al., 2008; Johnson & Myers, 2008; Matson, Wilkins, et al., 2008; Tager-Flusberg & Caronna, 2007). Possessing functional speech by the age of 5 is generally believed to be one of the best prognosticators for positive outcomes in children with ASDs (Thurm et al., 2011).

Other oddities seen in the communication of children with ASDs include echolalia (Bertoglio & Hendren, 2009; Eveloff, 1960). While a degree of echolalia is common in typical development, this repetitive use of language extends beyond what is typical (Dawson, Mottron, & Gernsbacher, 2008). This may present as either immediate or delayed echolalia in which the child repeats previously heard words or phrases from various sources (e.g., other people, movies, video games, or books). Their speech may also possess several inappropriate characteristics, such as problems with volume, pitch, intonation, stress, rate, or rhythm.

Young children often present with limited or a lack of imitative or pretend play (APA, 2000; Charman, 2008). For example, the child may only play with a toy or object in the manner in which it is intended and may be unable to pretend that a cardboard box is a house. As this child ages, this deficit in pretend play expands to a deficit in understanding abstract ideas. Older children with ASDs often show problems with comprehension (Llaneza et al., 2010) and understanding or integrating abstract concepts (Bertoglio & Hendren, 2009). This may result in a child with ASDs interpreting a joke or non-literal phrase in a very literal sense. Other communication deficits that are often seen in older children with ASDs include initiating and maintaining conversations unrelated to their restricted interests (APA, 2000; Bertoglio & Hendren, 2009).

Repetitive and Restricted Behaviors and Interests. The third core feature of ASDs is repetitive and restricted behaviors or interests, also known as RRBI or stereotypies. Depending on level of severity and intensity, these behaviors can be some of the most “socially

stigmatizing” (Cunningham & Schreibman, 2008, p. 471) and most difficult to treat using behavioral principles (Matson, Dempsey, & Fodstad, 2009), thereby often resulting in the use of psychotropic interventions (Memari, Ziaee, Beygi, Moshayedi, & Mirfazeli, 2012; Rapp & Vollmer, 2005). RRBI is an umbrella term which encompasses a wide variety of behaviors, which Chowdhury, Benson, and Hillier (2010) classify into four main groups: behavioral, communicative, cognitive, and sensory. The behavioral group includes motoric repetitive behaviors (e.g., hand flapping, body rocking, repetitive finger mannerisms). RRBI in the communicative group include the use of repetitive or idiosyncratic language. The cognitive group includes examples like obsessions, insistence on sameness, and adherence to nonfunctional rituals or routines. Lastly, the sensory group includes hyper- or hypo-sensitivities to various sensory stimuli (e.g., sensitivity to lights, sounds, textures).

In contrast to Chowdhury and colleagues’ groupings, Turner (1999) proposed a different classification system for RRBI: lower-order and higher-order behaviors. He defined lower-order repetitive behaviors as ones that involved stereotyped motor movements, self-injury, and repetitive manipulation of objects. Whereas, higher-order repetitive behaviors included restricted interests, obsessions, compulsions, rigid adherence to routines or rituals, insistence on sameness, and abnormal attachments to objects.

Regardless of the specific type of behavior, some researchers generally accept that RRBI are non-functional (Lewis & Baumeister, 1982; Rapp & Vollmer, 2005); although, more recently researchers have begun to recognize that RRBI may be maintained by a variety of functions (i.e., sensory, automatic/non-social, social or nonsocial positive and negative reinforcement; Cunningham & Schreibman, 2008). Although RRBI may be one of the most recognizable

characteristics of ASD, these behaviors are also present in other developmental disorders and even in children with typical development (Matson & Nebel-Schwalm, 2007; Thelen, 1979).

While stereotypies are common during typical development, these behaviors begin to significantly diminish around 12 months of age (Thelen, 1979). Consequently, it can be difficult to distinguish between ASD and typical development in infants and toddlers with regard to stereotypic behaviors. Some report that repetitive behaviors can distinguish between ASD and typical development around 24 months of age (Lord, 1995). However, MacDonald and colleagues (2007) found that children with autism and typically developing children did not substantially differentiate from one another until 4 years of age. Consistent with these findings, other have found RRBI to manifest at later ages when compared to social and communicative deficits, making this third core feature of ASDs a poorer indicator of autism (Charman, 2008; Gray & Tonge, 2001; Happe et al., 2006). Authors of a recent 2012 study reported that about 27% of parents of children with ASDs reported their first concern to be related to RRBI (Guinchat et al., 2012).

Observation Measures

There is a growing amount of literature providing evidence for the argument that ASDs are present from birth and can be distinguished from typical development and other DDs at very early ages (Barton, Orinstein, Troyb, & Fein, 2013; Matson, Wilkins, et al., 2008; Osterling & Dawson, 1994; Ozonoff, Iosif, et al., 2010). Therefore, researchers are continually attempting to develop and validate screening measures and diagnostic instruments for infants and toddlers (Maestro et al., 2002; Matson, 2007a).

Early detection of ASDs allows parents the early-on advantage of developing plans to help their child through their academic development, establishing a support system of specialists, seeking out early genetics testing, lessening parental stress, and, most importantly, finding opportunities and services for early intervention (Rogers, 2000). Early intervention has resulted in improved outcomes across several domains including: social skills, communication skills, adaptive behaviors, and even IQ (Boyd et al., 2010; Council, 2001; Maestro et al., 2002; Martinez-Pedraza & Carter, 2009; Matson, 2007b; Rogers, 2000). Because of continued research in this area, identifying and diagnosing autism at younger ages has become less challenging and more reliable (Boyd et al., 2010).

It is widely accepted that a comprehensive ASD assessment should gather information through multiple methods (e.g., parent-report measures, direct observation, diagnostic interviews) and multiple informants (e.g., mother, father, teacher, other caregivers; Charman, 2008; Gallo, 2010; Haynes & O'Brien, 2000; Manning-Courtney et al., 2003; Risi et al., 2006; Zwaigenbaum et al., 2009a). As the current study examines a recently developed ASD observation tool, direct observation instruments will be the focus of the following review.

Although direct observation can be susceptible to certain biases, many consider it to be the best

method of assessment (Gardner, 2000; Hartmann, Barrios, & Wood, 2004; Kazdin, 1982; Lipinski & Nelson, 1974; Romanczyk, Kent, Diament, & O'Leary, 1973). Over the years, there has been several observation measures published for the use of diagnosing and detecting ASDs.

Behavior Rating Instrument for Autistic and Atypical Children

One of the first ASD observation tools was the *Behavior Rating Instrument for Autistic and Atypical Children (BRIAC; Rutterberg, Dratman, Franknoi, & Wenar, 1966; Rutterberg, Kalish, Wenar, & Wolf, 1977; Wenar & Rutterberg, 1976)*. This test was developed in 1966 by Rutterberg and colleagues. It consisted of eight scales: (1) relationship to adults, (2) communication, (3) drive for mastery, (4) vocalization and expressive speech, (5) sound and speech reception, (6) social responsiveness, (7) body movement, and (8) psychobiological development. These items were empirically derived from behavioral observations of children in a psychoanalytic preschool classroom (Matson & Minshawi, 2006). The scales were revised in 1991 with the release of the 2nd edition of the *BRIAC*. The body movement scale was removed and two supplemental scales for nonverbal children were added: 1. expressive gesture and sign language and 2. receptive gesture and sign language (Rutterberg, Wolf-Schein, & Wenar, 1991). Examiners must first undergo extensive training to be considered qualified to administer the *BRIAC*. Training involves learning the correct observation procedures which usually last two hours and the complex coding and scoring system. Each scale is based on a 10-point scale and are scored for severity, duration, and frequency of the behavior (Rutterberg et al., 1991).

Although the *BRIAC* total score has been shown to have good correlation with clinical judgment and some of the scales, reliability studies concerning more sophisticated interrater and test-retest estimates have yet to be published (Lord & Corsello, 2005), as all psychometric studies have only inspected the original version of the *BRIAC* (Rutterberg et al., 1977;

Ruttenberg et al., 1991). Cohen and colleagues (1978) were unable to establish discriminant validity for the *BRIAAC* with other instruments or clinical judgment, and the *BRIAAC* was unable to discriminate between children with autism and other disorders (e.g., childhood psychosis, developmental aphasia).

Behavior Observation System for Autism

In 1984, the *Behavior Observation System for Autism (BOS)* was developed. This was the first measure to emphasize the importance of a controlled environment during behavioral observations (Lord & Corsello, 2005). This measure assesses 24 behaviors associated with Autistic Disorder, which are divided into four groups: (1) solitary, (2) relationship to objects, (3) relationship to people, and (4) language (Freeman, Ritvo, & Schroth, 1984). Additionally, repetitive and nonrepetitive behaviors are coded separately within each of these four groups. These items were developed according to Ritvo's definition of autism, literature review, and clinical judgment. The intended use of the *BOS* was to distinguish between different types of ASDs, ID, and other DDs, along with monitoring the developmental trajectory of ASD symptomatology over time (Freeman, Ritvo, & Schroth, 1984).

Prior to administration of the *BOS*, examiners first must undergo a training process. First, examiners learn memorize the complex coding system of the *BOS*. Then, they must familiarize themselves with the procedures used to record the behaviors. Finally, each trained examiner must then practice using this coding system by watching and rating pre-recorded videotaped sessions. Due to the complex coding system, training on the administration of the *BOS* can last up to 2 months (Freeman et al., 1984; Freeman & Schroth, 1984).

The *BOS* is administered by first allowing the child to engage in free play with developmentally appropriate toys in an observation room. The assessor videotapes this session,

which is comprised of nine 3-minute intervals, with two baseline intervals at the beginning and end of the session and one interval in which the assessor attempts to engage in interactive play with the child. The examiner then watches the videotaped session and records targeted behaviors as either 0 (*did not occur at all*), 1 (*occurred once*), 2 (*occurred twice*), or 3 (*occurred regularly*). Although there is a complex coding system, the *BOS* lacks a diagnostic cutoff score (Freeman et al., 1984).

Freeman et al. (1984) examined the psychometric properties of the *BOS*. The authors found interrater reliability to be greater than .70 for 16 of the 24 items (Freeman et al., 1984). Four of the 24 behaviors were found to differ significantly between the autism group and the control group, establishing discriminant validity for these items. Although the authors have found the *BOS* to differentiate between autism and ID, the *BOS* has not yet been found to reliably distinguish between various types of ASDs or other DDs (Freeman et al., 1979). Therefore, this measure lacks utility for differential diagnosis (Matson & Minshawi, 2006). Unfortunately, test-retest and internal consistency have not been studied as of to date, and the studies of the psychometric properties has not been updated since 1984.

Childhood Autism Rating Scale

With the intent of distinguishing children with autism from those with other developmental delays, the *Childhood Autism Rating Scale (CARS)* is a measure of direct observation in which the clinician engages in structured play with the child and subsequently provides ratings on 15 items based upon their observations. These 15 items include: Relation to people; Imitation; Emotional response; Body use; Object use; Adaptation to change; Visual response; Listening response; Taste, smell, and touch response and use; Fear or nervousness; Verbal communication; Nonverbal communication; Activity level; Level and consistency of

intellectual response; and General impressions (Schopler, Reichler, DeVellis, & Daly, 1980; Schopler, Reichler, & Renner, 1988). Assessors also have the option of supplementing their observations with parent-reported information and/or relevant medical records. Each item can be rated as: 1 (within normal limits); 1.5; 2 (mildly abnormal); 2.5; 3 (moderately abnormal); 3.5; or 4 (severely abnormal). The summation of all 15 item scores provide a total score of 15 to 60 which can then fall within three ranges on the total score scale: Non-autistic range (<30); Mild to moderate autistic range (30-36.5); Severe autistic range (37+). Reliable psychometric properties have been found for the *CARS* with an internal consistency of .94, an interrater reliability of .84, and a test-retest reliability of .88. The *CARS* is available in multiple languages and is easy and brief to administer and score.

In 2010, a second version of the *CARS* was published. With regard to scoring, the examiner is able to provide .5 points for a child falling between two item scores. Specifically, on the Level and consistency of intellectual response item, the *CARS-2* provides descriptors for each of the half points as well and relates the scoring to their IQ scores if available. For example, the scores for the Level and consistency of intellectual response item are as follows: 1 (*the child is as intelligent as typical children of the same age and does not have any unusual intellectual skills or problems*); 1.5 (*the child has low intelligence [IQ score between 71 and 85] and does not have unusual intellectual skills or problems*); 2 (*the child has very low intelligence [IQ score is 70 or lower] and his or her skills appear fairly evenly delayed across all areas*); 2.5 (*the child has very low intelligence [IQ score is 70 or lower] and skills appear to vary across areas, but none is at or above average*); 3 (*the child's overall intelligence is in the range from intellectually disabled to average [IQ score less than 115], and there is significant variability in skills. At least one skill is in average range.*); 3.5 (*the child's overall intelligence is in the range from*

intellectual disability to average [IQ score less than 115], and there is significant variability in skills. At least one skill is above average range. Extreme savant skills are not included here but are rated in category 4); 4 (a rating of 4 is given when extreme savant skills are present, regardless of overall level of intelligence).

The *CARS-2* also builds upon the original *CARS* by making it more responsive to higher functioning children by including a rating scale to identify those with high-functioning autism (*CARS2-HF*). The *CARS2-HF* can be used for individuals over 6 years of age with an IQ above 80 and fluent communication. The second addition also offers a separate parent rating scale, the Questionnaire for Parents or Caregivers (*CARS2-QPC*). The *CARS-2* is intended for helping to inform diagnosis and intervention planning but is not intended for use as a diagnostic tool (Vaughan, 2011).

The *CARS-2* has a robust internal consistency reliability with a coefficient of .93 for the *CARS-2* standard form and .96 for the *CARS2-HF*. The *CARS2-HF* has an interrater reliability estimate of .95. Concurrent validity of the *CARS-2* with the *ADOS* was established with a correlation of .79. The *CARS-2* has a sensitivity value of .88 and a specificity value of .86 when distinguishing between those with and without a diagnosis of autism (Vaughan, 2011).

Although the *CARS* is considered to be one of the most popular assessment tools for autism, there are a number of limitations this measure has yet to overcome. Some criticize the *CARS* for loosely corresponding to the *DSM-IV-TR* diagnostic criteria for autism which may lead to higher levels of sensitivity (Inglese & Elder, 2009b; Lord & Risi, 1998). For example, Lord (1995) found that the *CARS* consistently identified non-autistic children with ID as having autism. While the *CARS-2* can be used to help inform diagnosis and plan interventions, is not intended for use as a diagnostic tool (Vaughan, 2011). Additionally, interrater reliability

estimates for the *CARS-2* standard form has not yet been provided. Finally, although the items of the *CARS-2* are consistent with current diagnostic system criteria, the factor structure of the *CARS-2* is not consistent with these current criteria (APA, 2000).

Autism Diagnostic Observation Schedule

Unlike the previously reviewed measures, the *Autism Diagnostic Observation Schedule* (*ADOS*) is a measure of direct observation that provides a specific set of standard interactions that the examiner can use to assess autistic symptomatology in children and adults (Lord et al., 1989; Matson & Minshawi, 2006). The scale is composed of four modules. Modules 1-3 are intended for use with children over the age of 5, while Module 4 should be used with adolescents and adults with fluent speech. The *ADOS* is intended to supplement information gathered through caregiver interview and other developmental tests. This semi-structured, standardized assessment of communication, social interaction, play and imagination was designed to operationalize the *DSM* criteria (Molloy, Murray, Akers, Mitchell, & Manning-Courtney, 2011).

Prior to administration, examiners must undergo a two-day clinical training course or watch a number of training DVDs. Once trained, the examiner is then ready for clinical administration of the measure. There are a set of eight standardized interactions for the examiner to use as prompts with the child during interactive play and include: (1) a construction task, (2) unstructured presentation of toys, (3) a drawing game, (4) a demonstration task, (5) a poster task, (6) a book task, (7) conversation, and (8) socioemotional questions. These various activities assess a range of various social and communicative skills (e.g., symbolic play, reciprocal play, turn taking, gesturing, storytelling, reciprocal communication, language use, asking for help, giving help, imitation, describing skills). Each behavior is rated on a 3-point scale: 0 (*within normal limits*), 1 (*infrequent or possible abnormality*), or 2 (*definite abnormality*). In addition to

rating each task, the examiner provides overall scores for reciprocal social interaction, communication, nonspecific abnormal behaviors (e.g., anxiety, attention, hyperactivity), and repetitive/restricted behaviors or interests. Kim & Lord (2013) state that the *ADOS* can usually be administered in 30 to 45 minutes, while others have stated that this measure generally takes no more than 90 minutes to administer and score (Molloy et al., 2011).

Based upon one's score, an individual can fall within either the Autism range or the ASD range. Their score must meet separate cutoffs for the communication domain, the social domain, and the total score. To improve the sensitivity and specificity of the *ADOS* Modules 1 through 3, revised algorithms were introduced in 2007 to reflect that social and communication items are better represented on one factor as opposed to two distinct factors. Additionally, the repetitive and restricted item scores are now included into the individual's total *ADOS* score (Gotham, Risi, Pickles, & Lord, 2007; Molloy et al., 2011).

Interrater reliability for the individual tasks comprising the *ADOS* ranged from .61-.92, while the interrater reliability for the total ratings ranged from .58-.87. The *ADOS* also has good test-retest reliability with coefficients ranging from .57-.84 for the tasks and .58-.92 for the general ratings. Half of the general rating items on the *ADOS* were found to reliably differentiate between autism with mild ID, ID alone, autism with normal IQ, and a typically developing group. Five of the eight tasks were found to significantly differentiate between autistic and non-autistic children (Lord et al., 1989). With regard to the new algorithms, the new and old algorithms were found to have similar ratings of sensitivity in a sample of autistic versus non-autistic children. However, the new algorithms show great improvements in specificity (Gotham et al., 2007).

Despite its ability to distinguish between those with and without autism, the *ADOS* continues to have a number of limitations. The extensive administration and scoring techniques require intensive training and supervised practice (Lord et al., 1989). Unfortunately, while the *ADOS* is a tool to measure autistic symptomatology, the standardized activities do not assess specific motor behaviors, sensory abnormalities, or restricted and/or repetitive behaviors or interests (Lord et al., 1989; Matson & Minshawi, 2006). Rather, *ADOS* items relating to these areas are worded very broadly (e.g., “sensory interests,” “repetitive behaviors”).

Prelinguistic Autism Diagnostic Observation Schedule

As previously mentioned, the *ADOS* is only administered to children as young as 5 years of age. This is due to its focus on verbal abilities, lengthy administration time, and conversational administration style. Thus, the *Prelinguistic Autism Diagnostic Observation Schedule (PL-ADOS)*, designed in 1995, is a version of the *ADOS* designed for infants, toddlers, and nonverbal children (DiLavore, Lord, & Rutter, 1995). The *PL-ADOS* is comprised of 12 standardized examiner-child interactions based upon nonverbal symptoms of autism (e.g., eye contact, joint attention, imitation, pretend play).

To administer, the examiner assesses each task during natural play activities rather than structured tasks between the examiner and child at a table as in the standard form of the *ADOS*. This measure can be administered in about 30 minutes. The 12 tasks include: (1) free play, (2) imitation of child, (3) mechanical animal or car play, (4) play with bubble gun, (5) play with balloons, (6) social routines, (7) play with a toy drum, (8) having a birthday party, (9) snack time, (10) dropping papers, (11) simple actions with objects, and (12) adapting to a strange situation. Each task is then scored as either 0 (no abnormality), 1 (neither clearly typical nor clearly indicative of autism), or 2 (definite abnormality). As with the *ADOS*, the *PL-ADOS* also

has overall scores that the examiner applies to the task ratings. Cutoffs were determined by the score that correctly classified the most children. As a result, a score of 12+ on the social/communication domain plus a score of 2+ on the restricted/repetitive behaviors domain will place the child in the range for a diagnosis of autism (DiLavore et al., 1995).

DiLavore and colleagues (1995) found the *PL-ADOS* to have good interrater reliability with coefficients ranging from .63-.95 for the individual tasks, .60-.94 for the general ratings, and .86 for the total autism score. Nine of the individual task scores were able to reliably discriminate between young children with and without a diagnosis of autism. Recently, the *PL-ADOS* has been adapted for older individuals with severe to profound ID. When using a cutoff score of 15, Berument and colleagues (2005) found the *PL-ADOS* to have a sensitivity of .82 and a specificity of .85. Similar to the *ADOS*, the *PL-ADOS* requires extensive training and practice. The *PL-ADOS* is also unable to reliably differentiate between those with and without autism in children with verbal abilities.

Autism Diagnostic Observation Schedule – Generic

As a result of the problem of accommodating verbal ability on both the *ADOS* and the *PL-ADOS*, diagnostic accuracy was compromised. Children with lower language abilities assessed with the *ADOS* were being over-diagnosed, while children with higher linguistic abilities who were able to complete the tasks on the *PL-ADOS* yet still exhibited autistic symptoms were being under-diagnosed (Lord et al., 2000). Ultimately, this led to the creation of the *Autism Diagnostic Observation Schedule – Generic (ADOS-G)*. Like its two predecessors, the *ADOS-G* uses social “presses” to assess various activities and tasks of social interaction, communication, play, and imaginative use of objects. The *ADOS-G* is comprised of four 30-minute modules, each one appropriate for different age groups and developmental levels.

Module selection for each individual is based upon their expressive language abilities as opposed to their chronological or mental age (Lord, Rutter, DiLavore, & Risi, 2002).

Module 1 is designated for children with an expressive language level of less than 3 years of age. Module 2 is used with children who speak in short phrases but have an expressive language level of less than 4 years of age. Based on the *ADOS*, Module 3 is intended for verbally fluent children. Finally, Module 4 is intended for verbally fluent adults and for adolescents who are uninterested in toy play. Items are scored on a 3-point scale (0, 1, or 2) similar to the *ADOS* and *PL-ADOS* to form two domain scores (communication and social interaction) and one total score. Some items allow the examiner to rate the behavior as a 3 if it interferes with the observation.

Interrater reliability was established for Modules 1-4 with mean weighted kappas of .78, .70, .65, and .66, respectively (Lord et al., 2000). For the social domain, intraclass correlations ranged from .88 to .97. On the communication domain intraclass correlations ranged from .74 to .90, and for the total score intraclass correlations ranged from .84 to .98. Diagnostic classification rates between autism versus no autism were 100% for Modules 1 and 3, 91% for Module 2, and 90% for Module 4. These rates dropped when PDD-NOS participants were included in the classification process. Moderate to high internal consistency was also established for the Social domain (.86-.91), the communication domain (.74-.84), for stereotyped behaviors and restricted interests (.47-.65), and the total score (.91-.94).

Like the *ADOS* and *PL-ADOS*, the *ADOS-G* requires extensive training and practice prior to administration of this measure. Additionally, the *ADOS-G* does not have a domain for one of the core features of autism, restricted and repetitive behaviors and interests (Gotham et al.,

2007). Furthermore, the *ADOS-G* does not assess for age of onset. These limitations may lead to under-diagnosis of certain individuals (Lord et al., 2000).

Autism Diagnostic Observation Schedule, Second Edition

In an effort to condense, simplify, and improve upon the three former ASD assessments (i.e., *ADOS*, *PL-ADOS*, *ADOS-G*), the *Autism Diagnostic Observation Schedule, Second Edition* (*ADOS-2*) was developed (Lord, Luyster, Gotham, & Guthrie, 2012; Lord, Rutter, et al., 2012). In this edition, the diagnostic algorithms used in Modules 1-3 have been updated, and a new Toddler Module was added. The examiner determines which of the five modules to use based upon the individual's level of expressive language and/or their chronological age. Additional descriptions of the examiner prompts and behaviors to observe were also included. Finally, the previous three domains in the *ADOS* (i.e., socialization, communication, and RRBI) were condensed into two domains (i.e., social affect and restricted and repetitive behaviors) to better reflect the current *DSM-5* diagnostic criteria.

Only very slight modifications were made to the administration procedures for the *ADOS-2*. Administration time for the measure is still approximately 40 to 60 minutes, depending on which module is used. Scores are produced for the following areas: (a) language and communication, (b) reciprocal social interaction, (c) play and imagination, (d) stereotyped behaviors and restricted interests, and (e) other behaviors.

With regard to psychometric properties, internal consistency for Modules 1-3 ranged from .51 to .92. Internal consistency for Module 4 ranged from .47 to .85, and internal consistency for the Toddler Module ranged from .50 to .90. Overall, all modules demonstrated lower internal consistencies for the restricted/repetitive behavior domain than the social affect domain. Test-retest reliability for Modules 1 – 3 and the Toddler Module ranged from .64 to .92.

To date, no test-retest reliability has been conducted for Module 4. Interrater reliability for all five modules ranged from .79 to .98. The measure was also found to have sufficient predictive validity with sensitivities ranging from 60% to 95% and specificities ranging from 75% to 100% (Lord, Luyster, et al., 2012; Lord, Rutter, et al., 2012).

Screening Test for Autism in Two-year-olds

Unlike the previously reviewed assessments, the *Screening Test for Autism in Two-year-olds (STAT)* is intended to be used solely as a screener for autism. This brief, interactive assessment measures autistic symptomatology in children 2 to 3 years of age. The *STAT* can be administered in approximately 20 minutes. The examiner observes behaviors exhibited by the child along four domains: (1) play, (2) imitation, (3) requesting, and (4) attention directing skills. These behaviors are scored on 12 items as either pass or fail (W. Stone, Coonrod, & Ousley, 2000; W. Stone & Ousley, 1997).

Stone and colleagues (2004) evaluated 29 children with autism, PDD-NOS, and developmental delays. Interrater reliability for categorizing the participants into either a high risk or low risk group was 1.00, indicating perfect agreement. Test-retest reliability was also excellent (.90). Thirty nine children were then assessed with the *STAT* as either high risk or low risk and assessed with the *ADOS-G* as either autism or no autism. The *STAT* had a concurrent validity of .95 with the *ADOS-G* after the removal of the participants with PDD-NOS.

Unfortunately, the *STAT* is not without some limitations. Replication of the *STAT*'s psychometric studies is needed using larger, community-based samples (Stone et al., 2000; Stone et al., 2004). The *STAT* has also shown to under-diagnose milder forms of autism. For example, in a sample of 24 children with PDD-NOS diagnoses, the *STAT* classified 64% of the sample as low risk and the remaining 36% as high risk (Matson & Minshawi, 2006; Stone et al., 2004).

Autism Observation Scale for Infants

One of the most recently developed ASD observation scales is the *Autism Observation Scale for Infants (AOSI)*, a semi-structured observation for children between 6 to 18 months of age. Examiners engage in interactive play with the infant across a small table with the child in his/her parent's lap. The examiner carries out a set of seven standardized social "presses" including: (1) visual tracking, (2) disengagement of attention, (3) orientation to name, (4) reciprocal social smiling, (5) differential response to facial emotion, (6) social anticipation, and (7) imitation. Each press is administered a certain number of times, and activities can be repeated if the infant is distracted or inattentive. Behaviors exhibited in response to these activities are scored along 18 different items. Eye contact, atypical motor behavior, and atypical sensory behavior are rated as either a 0 (typical) or 2 (atypical). All other target behaviors are scored along a 4-point scale (0-3). The *AOSI* can be administered in about 20 minutes (Bryson, Zwaigenbaum, McDermott, Rombough, & Brian, 2008).

Bryson and colleagues examined the interrater reliability of the *AOSI* with children at 6, 12, and 18 months of age with correlation coefficients at .74, .93, and .94, respectively. Most individual items showed good to excellent reliability ($>.65$), while reliability ratings were lower in the 6 month age group. Across all ages, the reliability of the *AOSI* total score is .92. Test-retest reliability is fair to good for the total marker counts (.68) and the total scores (.61).

The psychometric studies for the *AOSI* have included small sample sizes and used multiple examiners/raters which may have influenced the reliability data. Similarly, no studies examining the validity of the *AOSI* have yet been conducted. Researchers have also yet to determine the specificity and sensitivity of the *AOSI* when differentiating between ASD and other DDs. Finally, this measure has yet proven to be effective for clinical purposes as the *AOSI*

was developed as a research tool (Bryson et al., 2008). See Table 1 for an outline of the general properties for all of the aforementioned ASD observation measures.

Table 1. Observation Measures of ASD

Measure	Age Range	Admin Time	Cost of Instrument	Cost of Training	Interrater Reliability	Test-retest Reli	Concurrent Validity	Sensitivity	Specificity
BRIAAC	All ages	2 hours	\$130 + \$80 for reproducible masters	Not Specified	0.85 to 0.93	n/a	0.69	n/a	n/a
BOS	45 - 56 months	30 min observations: One per day for 3 days	n/a	Not Specified	>.70 for 16/24 items	n/a	n/a	n/a	n/a
CARS	2+ years	30 min	n/a	Not Specified	0.84	0.88	0.80 - 0.84	0.88	0.86
CARS-2	2+ years	10 min (after observational data are collected)	\$170 + \$1.62 per protocol	Not Specified	CARS-2 High Functioning Form (0.95)	n/a	0.38 - 0.79	0.81	0.87
ADOS	Toddler to Adult	45 min - administration; 1 hour - scoring	\$1480 + \$4.50 each per protocol	\$360	0.58 - 0.87	0.58 - 0.92	0.77	0.85 - 1.00	0.45 - 0.60
ADOS-2	1+ years	40 - 60 min	\$1995 + \$5.10 each per protocol	\$475 – 2-day training course; \$999 - training DVDs	0.79 - 0.98	0.64 - 0.92	n/a	0.60 - 0.95	0.75 - 1.00

(Table 1 continued)

Measure	Age Range	Admin Time	Cost of Instrument	Cost of Training	Interrater Reliability	Test-retest Reli	Concurrent Validity	Sensitivity	Specificity
PL-ADOS	<5 years	30 min	n/a	Not Specified	0.86	n/a	n/a	0.82	0.85
ADOS-G	All ages	30 min	n/a	Not Specified	0.65 - 0.78	0.59 - 0.82	0.63	0.80 - 1.00	0.68 - 1.00
STAT	24 - 35 months	20 min	\$500 + \$1.00 each per protocol	\$200 online certification	1.0	0.90	0.95	0.92	0.85
AOSI	6 - 18 months	20 min	n/a	Not Specified	0.74 - 0.94	0.61 - 0.68	n/a	n/a	n/a
ASD-OC	1 - 16 years	Approx. 20 min	Not Specified	n/a	0.96	n/a	0.83	0.58 - 0.90	0.81 - 0.96

Note. Admin Time=Administration Time; Reli=Reliability; BRIAAC=Behavior Rating Instrument for Autistic and Atypical Children; BOS=Behavior Observation System for Autism; CARS=Childhood Autism Rating Scale; ADOS=Autism Diagnostic Observation Schedule; PL-ADOS=Prelinguistic Autism Diagnostic Observation Schedule; ADOS-G= Autism Diagnostic Observation Schedule-Generic; STAT=Screening Test for Autism in Two-year-olds; AOSI=Autism Observation Scale for Infants; ASD-OC=Autism Spectrum Disorder-Observation for Children

Purpose

In applied settings, clinicians often rely primarily on unstructured interviews and loose interpretations of diagnostic criteria when making ASD diagnoses (Matson & Minshawi, 2006). Many times, the use of psychometrically sound instruments during clinical diagnostic assessments is waived, often to avoid the expenditure of resources (e.g., time, money, etc.; Soares & Patel, 2012). This oversight, however, has improved over time with the development of various assessment tools to assist diagnosticians.

With the significant rise in autism diagnoses, clinicians are becoming more astute during the diagnostic process and in their understanding of ASDs. Researchers have found that early intervention leads to better outcomes, thereby making early diagnosis a priority among today's clinicians (Dawson, 2013; Matson & Konst, 2013; Matson, Wilkins, et al., 2008; Reichow et al., 2012; Volkmar & Pauls, 2003; Wiggins, Baio, & Rice, 2006). Most clinicians assert that the most comprehensive and reliable diagnoses are ones that are based upon assessments involving multiple informants (e.g., parent, teacher, clinician) and multiple methods of data collection (e.g., parent-report measures, diagnostic interviews, direct observation; Charman, 2008; Doss, 2005; Manning-Courtney et al., 2003; Matson, Mahan, Hess, Fodstad, & Neal, 2010; Matson, Nebel-Schwalm, & Matson, 2007; Zwaigenbaum et al., 2009a). Each of these various types of assessment tools has their own strengths and weaknesses, which is why using multiple types of data collection is important. Direct observation measures are particularly advantageous because they allow the examiner to directly see the interactions between an individual and their environment. Direct observation also circumvents any personal biases that may arise during parent interviews or on an informant-based measure. The *ASD-OC* is an example of such measure and holds promise in the field of ASD assessment.

The *ASD-OC* is a clinician-rated, direct observation measure assessing symptoms of ASD in children (Neal, Matson, & Hattier, 2012). Many of the already-existing ASD observation tools have several shortcomings, and the *ASD-OC* has the ability to satisfy a number of these weaknesses. Eight main limitations of these pre-existing observation tools, which the *ASD-OC* can address, have been identified as: (1) weak psychometric properties, (2) inability to differentiate between ASDs and atypical development, (3) difficult and lengthy administration, (4) complexity of scoring procedures, (5) extensive examiner training requirements, (6) lack of correlation with formal diagnostic criteria, (7) inability to individualize the assessment for each client, and (8) lack of corresponding parent-report rating scales.

First, many of the ASD observation measures are lacking in sound psychometric properties. This is problematic and may lead to either false positives or false negatives when making diagnostic decisions. For example, the *BRIAAC* has outdated norms since all of the studies examining its psychometric properties have only focused on the original version of the *BRIAAC* (Ruttenberg et al., 1977). Similarly, the test-retest reliability and internal consistency of the *BOS* have yet to be established, and much of the existing psychometric research on the *BOS* has not been updated since 1984 (Freeman et al., 1984; Freeman & Schroth, 1984). Studies examining the validity of the *AOSI* have yet to be conducted (Bryson et al., 2008). Findings from the psychometric studies of the *STAT* and *AOSI* should also be interpreted with caution, as these studies are based upon small sample sizes (Bryson et al., 2008; Stone et al., 2000). Compared to the aforementioned tools, excellent interrater reliability (.96), excellent internal consistency (.96), and strong convergent validity (.83) have already been established for the *ASD-OC* (Neal et al., 2013; Neal et al., 2014). Cicchetti and Sparrow (1981) have outlined qualitative ranges for adequate item coefficients to use when interpreting reliability findings.

Item coefficients below .40 are considered poor, .40-.59 are fair, .60-.74 are good, and .75-1.00 are considered excellent. With regard to validity findings, correlation coefficients should be interpreted as .1 to .29 as small, .3 to .49 as medium, and .5+ as large (Cohen, 1988). Based upon these standards, the *ASD-OC* is psychometrically superior to other observation tools measuring autistic symptomatology.

Second, the ability to differentiate between various forms of ASDs and other DDs or atypical development has always been an objective of clinicians and researchers; however, this task has proven to be difficult. While the *BRIAAC* and *STAT* can discriminate between those with and without autism, these measures are unable to differentiate between autism and other DDs or atypical development (Ruttenberg et al., 1977; Ruttenberg et al., 1991; Stone & Ousley, 1997). Although the *BOS* can differentiate between autism and ID, there are no classification cutoffs to distinguish autism from other ASDs or other DDs (Freeman et al., 1984). Similarly, the *CARS* and *ADOS-G* do not differentiate between autism and other ASDs or atypical development (Lord et al., 2000; Schopler et al., 1988). While the *ADOS-G* does provide cutoffs for autism and for ASD (referring to possible diagnoses of autism, PDD-NOS, and Asperger's disorder), this measure has only a 33% correct classification rate when distinguishing between children with autism and PDD-NOS (Lord et al., 2002). Additionally, the *AOSI* cannot differentiate between ASDs and atypical development and is only used for research and monitoring purposes (Bryson et al., 2008). The current study aims to provide such valuable cutoffs for the *ASD-OC*.

Third, with the ever-increasing rise in the detection of ASDs (Matson & Kozlowski, 2011), clinicians are strained to provide brief and efficient services without compromising the quality or integrity of the diagnostic assessments (Gallo, 2010). Many insurance companies

place considerable restrictions on the time clinicians can spend with clients providing assessments or treatment services (Gallo, 2010; Manning-Courtney et al., 2013), making assessment tools that are brief and easy to administer even more desirable. Unfortunately, some of the already established ASD observation measures have still yet to overcome this obstacle. For example, the *BRIAAC* takes at least 2 hours to administer (Ruttenberg et al., 1991), and the *ADOS* can last up to 90 minutes to administer and score (Molloy et al., 2011). Even the second edition of the *ADOS* requires extensive time (i.e., 40-60 minutes) to administer (Lord, Rutter, et al., 2012)

Fourth, clinicians also benefit from more simplistic scoring procedures for their assessment tools, thus saving time and cost. The *BOS* has a complex scoring procedure - the examiner must first videotape the child, then study the recording and code certain behaviors observed during the videotaped session, and then score these codes (Freeman et al., 1984). Again, the administration and scoring of the *ADOS* and *ADOS-2* can be quite lengthy (Lord, Rutter, et al., 2012; Molloy et al., 2011). Unlike these tools, the *ASD-OC* offers practitioners a simple and brief method of scoring.

Fifth, the amount of time that a clinician or examiner must spend on training in order to become proficient in administering and scoring these ASD observation tools is also pertinent. The *BRIAAC* requires 40 to 80 hours of training before the examiner can actually carry out the 2-hour long observation period necessary to administer the *BRIAAC* (Ruttenberg et al., 1991). Similarly, examiners must undergo up to 2 months of training on the *BOS* in order to reliably administer the measure, including memorization of the coding system (Freeman et al., 1984). Some have also claimed that the training for the administration of the *CARS* is extensive; however, the training level can vary depending on the school district (Wormald, 2011). The

ADOS also requires that the examiner have previous experience with children with ASDs in addition to substantial training and supervised practice with the measure (Lord et al., 1989).

Similar requirements are also expected for examiners of the *ADOS-2*.

Sixth, it is often helpful to clinicians when assessment tools are developed in parallel to the current formal diagnostic criteria for the disorder in question. This assists the clinician in making informed decisions when integrating information from various sources (e.g., observation measure, parent-report measure, medical history record review) into a comprehensive assessment to diagnose an individual. Unfortunately, some of the ASD observation tools fail to map onto the *DSM* diagnostic criteria for autism and neglect to directly assess some behaviors pertinent to a diagnosis of autism (i.e., repetitive and restricted behaviors and/or interests). Specifically, the *ADOS* includes items which assess the social and communicative behaviors of the child; however, stereotyped/restricted behaviors and nonspecific abnormal behaviors are only scored at the end of the assessment with one overall clinician-reported rating (Lord et al., 1989).

Restricted interests and repetitive behaviors are also not included in the algorithm for the *ADOS-G*, possibly causing children with PDD-NOS to not meet the cutoff score on this measure (Lord et al., 2000). Additionally, the development of the *CARS* predates the *DSM-IV*; therefore, the scale was developed based upon a number of various definitions for autism including ones from Kanner (1943), Creak (1961), Rutter (1978), and Ritvo (1978). These definitions were not consistent in noting repetitive and restricted behaviors and/or interests as a core feature of the disorder. Thus, items in reference to this feature of autism were not included in the *CARS* (Maygar & Pandolfi, 2007), nor does the *CARS-2* specifically probe for these behaviors (Schopler, Van Bourgondien, Wellman, & Love, 2010). More importantly, with the vast changes that have been made to ASD diagnostic criteria with recent the publication of the *DSM-*

5 (Frazier et al., 2011; Matson, Kozlowski, et al., 2012; McPartland et al., 2012), it will be beneficial to have a measure which can also correspond to this new set of criteria to aid in an easy transition from the fourth to fifth edition of the *DSM* for diagnosticians, and the *ASD-OC* fulfills this need.

Seventh, the ability of an assessment tool to be flexible in order to individualize the assessment for each client is important. Autistic symptoms can vary in behavioral presentation among different individuals (Charman, 2008; Kim & Lord, 2013; Landa & Garrett-Mayer, 2006; Piven, Harper, Palmer, & Arndt, 1996); therefore, it is beneficial to tailor an assessment to each child's abilities. Although this is not consistent across all ASD observation tools, some of the current observation measures are able to provide examiners with the freedom of individualizing the assessment. For example, the *BOS* allows clinicians to modify the toys/items used during the assessment based upon the child's developmental level. The *ASD-OC* is also among this group. Based upon the child's needs, the clinician can tailor the type of prompt used to assess each item, the stimuli utilized throughout the observation (i.e., toys), the length of the observation, and the setting in which the observation takes place.

Finally, the *ASD-OC* has been developed alongside corresponding parent-report measures (i.e., the *Autism Spectrum Disorder – Diagnostic for Children [ASD-DC]* and the *Baby and Infant Screen for Children with aUtism Traits [BISCUIT]*) to allow the clinician to conduct a comprehensive, multiple-informant-based assessment, which is considered to be a best practice by most clinicians and researchers (Charman, 2008; Manning-Courtney et al., 2003; Risi et al., 2006; Zwaigenbaum et al., 2009a). This is a feature of the *ASD-OC* that is lacking in many other ASD observation tools (i.e., *BRIAAC*, *BOS*, *AOSI*, *STAT*, *CARS*).

In sum, Neal and colleagues have already examined many psychometric properties and other features of the *ASD-OC*. First, Neal, Matson, and Hattier (2012) compared autistic severity in children diagnosed according to the *DSM-IV-TR* and children diagnosed according to the *DSM-5*. No significant differences were found between these two groups; however, both groups demonstrated significantly higher scores than the control group. Second, Neal, Matson, and Belva (2012) conducted a discriminant analysis on both the total *ASD-OC* score and the individual item scores. It was determined that the *ASD-OC* was able to significantly discriminate between those with ASD and those with atypical development. Third, Neal, Matson, and Belva (2013) found the *ASD-OC* to be a measure with robust reliability. Fourth, Neal, Matson, and Hattier (2014) established convergent and criterion-related validity for the measure. The current paper continues this line of research into the *ASD-OC*.

This paper was divided into three separate studies. The factor structure of the *ASD-OC* was assessed in Study 1. Clinical cutoff scores were developed for the *ASD-OC* according to the *DSM-IV-TR* diagnostic criteria in Study 2, and cutoff scores based upon the current *DSM-5* diagnostic criteria were developed in Study 3. This line of research advances the diagnostic tools available to clinicians, which may improve the likelihood that children will be diagnosed and receive appropriate services as early as possible. Considering the previously established reliability and validity of this measure, the *ASD-OC* appears to be a promising tool for assessing autistic symptoms in children.

Study 1

Method

Participants. A total of 179 children aged 1 to 16 years ($M = 6.0$; $SD = 3.83$) and their caregivers served as the participants for the current studies. Based on Donner's (1982) justification of retaining a participant if less than 10% of the items were missing and replacing these with the mean for that item, children who were missing no more than three *ASD-OC* item scores were included in the study and those missing items were replaced with the mean for that item. Participants missing greater than 10% of their data were removed from the sample (Bennett, 2001; Donner, 1982; Field, 2005). The initial sample included 867 children and their caregivers; however, 688 were removed due to insufficient data. Given that much of the sample was archival data from a large database which had been gathered over an extended period of time, many of these participants were excluded from this study simply because they had not been administered the *ASD-OC*. All participants were assessed either through a university outpatient clinic in Louisiana or a local school providing specialized services to children with ASD and other developmental disabilities. Primary referrals for the outpatient clinic included ASD, anxiety, learning disorders, behavior problems, and gifted and talented testing. A breakdown of demographic variables for the sample can be found in Table 2.

Table 2. Demographic Characteristics

	Total Sample (N = 179)
Age (years)	
Mean (SD)	6.0 (3.83)
Range	1-16
Gender	
Male	125 (69.8%)
Female	51 (28.5%)
Ethnicity	
Caucasian	124 (69.3%)
African American	21 (11.7%)

(Table 2 continued)

	Total Sample (N = 179)
Hispanic	5 (2.8%)
Other	18 (10.1%)
Not Reported	11 (6.1%)

Measures. The *ASD-OC* is a recently-developed observation measure for assessing autistic symptomatology. The *ASD-OC* initially consisted of 54 total items; however, items with poor interrater reliability (i.e., intraclass correlations less than .40) and little to no variance were removed in a previous analysis of the measure by Neal and colleagues (2013). Following the removal of these items, the *ASD-OC* currently consists of 35 items total and 10 supplementary items for verbal individuals only. Items were developed by a clinical psychologist with more than 30 years of experience with ASDs and other DDs through a review of ASD literature, current *DSM* and *ICD* diagnostic criteria, and other ASD assessment measures (e.g., *ASD-DC*, *ADOS*, *CARS*). A second clinical psychologist with extensive experience with this population reviewed the original item pool, suggested minor changes, and developed additional items. Following pilot administrations of the *ASD-OC* to several children referred for an ASD assessment at an outpatient clinic, minor revisions were made to the scale (i.e., deletion and clarification of items and prompts).

The *ASD-OC* is administered by the clinician conducting a brief observational play period with the child. While examiners are expected to be trained on the topic of ASD and on the administration of the *ASD-OC*, formalized training programs are not required for this measure unlike other ASD observation scales. These formal training requirements are often time-consuming (e.g., 40-80 hours for BRIAAC training; 2 months for BOS training) and costly (e.g., \$475 for 2-day training course for *ADOS-2*; \$999 for *ADOS-2* training DVDs).

The observation session should be individualized for each child based upon their age, developmental level, cooperation, and clinical judgment. Some sample items include: looks when name is called, uses gestures to communicate, can imitate simple movements, eye contact, and abnormal repetitive movements. Most items are supplemented with optional prompts that the examiner can utilize to assess that item. For example, for the item *initiates make-believe or pretend play*, the suggested prompt is “present plastic dishware and doll and see if child ‘makes food’ or feeds the doll; present a toy spacecraft to see if the child ‘flies’ it.” For the item *can imitate simple physical gestures or movements*, the suggested prompt is “waving; tapping on the table; making a toy jump.” Other item examples include: *looks when name is called*, *initiates joint attention*, *eye contact when communicating expressively*, *reaction when transitioning between activities*.

Following the observation, the clinician is instructed to compare the child to other same-age, typically developing peers. Items are then rated on a 3-point scale to the extent that the behavior was a problem during the observation: 0 (no impairment), 1 (mild impairment), or 2 (severe impairment). Neal, Matson, and Belva (2013) examined the reliability of the *ASD-OC* and observed excellent interrater reliability of .96 and internal consistency of .96. A significantly large correlation ($r = .83$) between the *ASD-OC* and *CARS* established its strong convergent validity (Neal et al., 2014). Criterion validity has also been established for the *ASD-OC* by comparing mean total scores of different diagnostic groups; there was a statistically significant difference ($p < .001$) between the groups. Specifically, children with an ASD scored significantly higher, indicating worse impairment, than both typically and atypically developing children (Neal et al., 2014).

Procedure. The *ASD-OC* was administered by doctoral level graduate students studying clinical psychology. All examiners received extensive training on the topic of ASD and on the administration of the *ASD-OC* to ensure standardized administrations. Examiners were also supervised by a senior student experienced in administering the *ASD-OC* to ensure reliable administrations. For participants recruited from the university outpatient clinic, the *ASD-OC* was administered as part of a full ASD assessment battery; however, the *ASD-OC* was not included in the diagnostic formulation of each participant.

The parents and/or legal guardians of the children participating in this study have provided informed consent for participation. Informed assent was also obtained from many children within the sample as a way for them to acknowledge that they understand what it means to participate and that their participation is voluntary. While informed assent is not a legally endorsed process (Lambert & Glacken, 2011), examiners, with the help of most parents, attempted to inform many of the children with at least minimally developed competencies about the study and invited them to participate. Although age is often used as an influential factor in determining a child's competence to provide assent, many researchers have found there to be no relationship between a child's age and competence for assent (Kumpunen, Shipway, Taylor, Aldiss, & Gibson, 2012; Miller, Drotar, & Kodish, 2004). Thus, the decision to obtain informed assent was based upon the discretion of each examiner. Additionally, this study has received prior approval from the Louisiana State University Institutional Review Board (See Appendix).

Research Design. Study 1 inspected the factor structure of the *ASD-OC* as a means to construct subscales. Factor analysis is a useful tool for summarizing information gathered from individual items by grouping the items or variables that show a significant relation to one another. Conducting a factor analysis can be informative, particularly for developing new

clinical measures. However, this analysis is not without certain issues that must first be addressed, including: sample size, the number of factors to retain, the extraction method to use, and the type of rotation to implement (Field, 2005).

When conducting a factor analysis, Fabrigar, Wegener, MacCallum, and Strahan (1999) advise that researchers avoid utilizing “samples whose selection is related to measured variables in the analysis” (i.e., autistic severity). Therefore, all participants with sufficient *ASD-OC* data regardless of diagnosis, or lack thereof, were included in the analysis. This prevented from having a more homogeneous sample than the larger population, which could lead to a restricted range in the measures, correlation among variables, and falsely low estimates of factor loadings (Fabrigar et al., 1999).

It is important to note that this factor analysis did not include the 10 supplementary items for children with verbal abilities due to limited data. Less than 100 participants had sufficient data for these 10 supplementary items, which would not have yielded an adequate participant to item ratio for these analyses. Rather, a total sample size of 179 participants was used for the present study, while only including the 35 primary items of the *ASD-OC*. This sample size yielded a participant to item ratio of at least 5:1. Many researchers recommend this ratio guideline of 5 participants to 1 item for exploratory factor analyses (Conway & Huffcutt, 2003; Costello & Osborne, 2005; Fabrigar et al., 1999; Field, 2005; Floyd & Widaman, 1995; Gorsuch, 1983). Streiner (1994) recommends that adequate results from a factor analysis will be obtained with 5 participants per variable as long as there are 100 participants in the sample. Floyd & Widaman (1995) even state, “until recently, a general rule of thumb regarding sample size for principal components and common factor analysis has been ‘the more participants, the better’” (p. 289). However, Costello and Osborne (2005) note that “strict rules regarding sample size for

exploratory factor analysis have mostly disappeared” (p. 4). Instead, adequate sample size should be somewhat determined by the stability of the data (Costello & Osborne, 2005). For example, Guadagnoli and Velicer (1988) took another approach to determining the appropriate sample size for factor analyses by using a Monte Carlo analysis. Based upon the results of this study, the authors determined that the stability of one’s findings and the necessary sample size to obtain those findings were more so related to the factor loadings of each variable, rather than adhering to an arbitrary participant to item ratio. For example, Guadagnoli and Velicer were able to conclude that with factor loadings of .80, solutions were determined to be highly stable regardless of the sample size, even with as few as 50 participants. Furthermore, when at least 10 variables had factor loadings of .40, sample sizes of 150 participants produced accurate solutions. With regard to the current study, it should be noted that all *ASD-OC* items that were retained had factor loadings above .40.

In addition to meeting these aforementioned sample size recommendations, the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was also computed to inspect the adequacy of the sample size for conducting this factor analysis. The KMO test calculates the ratio of the squared correlation between variables to the squared partial correlation between variables. The KMO statistic varies between 0 and 1, with larger values indicating that the patterns of correlations are relatively compact and so factor analysis should yield distinct and reliable factors (Kaiser, 1974). Hutcheson and Sofroniou (1999) qualitatively describe the KMO values as: 0.0 – 0.5 (unacceptable); 0.5 – 0.7 (mediocre); 0.7 – 0.8 (good); 0.8 – 0.9 (great); and 0.9+ (superb).

To determine the number of factors to retain during the exploratory factor analysis, eigenvalues were calculated for each component in the data. Eigenvalues measure the amount of

variance accounted for by that factor (Floyd & Widaman, 1995). A scree plot was then graphed using these eigenvalues (Field, 2005). Any factors to the left of the point of inflexion (i.e., factors with high eigenvalues) were retained (Cattell, 1966). Field (2005), along with other researchers (e.g., Costello & Osborne, 2005; Fabrigar et al., 1999), recommend that this method be supplemented with another technique (i.e., parallel analysis) to determine how many factors to retain.

Therefore, a parallel analysis was also conducted using SPSS syntax (O'Connor, 2000) available at <https://people.ok.ubc.ca/briocconn/nfactors/nfactors.html>, since there currently is no method for parallel analysis built into SPSS. A parallel analysis essentially extracts eigenvalues from random datasets that parallel the actual dataset with respect to the number of cases and variables. In other words, the parallel analysis constructs a second correlation matrix from normally distributed random numbers, using the same number of variables/items and the same number of participants that are in the researcher's study. A factor analysis is then conducted using the squared multiple correlations from the diagonal of this newly created "random" matrix. Eigenvalues from the parallel analysis and eigenvalues from the actual current study are then compared to one another. The eigenvalues from the actual current dataset which correspond to the 95th percentile of the distribution of random data eigenvalues were retained (O'Connor, 2000). Montanelli and Homphreys (1976) explain that "this method is based on the idea that a researcher would not be interested in a factor which does not account for more variance than the corresponding factor obtained from distributions of random numbers" (p. 341).

Next, the normality of the dataset was examined prior to choosing the type of exploratory factor analysis for the data. To determine if the dataset was normally distributed, a Kolmogorov-Smirnov test of normality was performed to test for significant differences between the

distribution of scores from this dataset and a normal distribution (Field, 2005). The *ASD-OC* scores, $D(179) = 0.19, p < .05$, were found to be significantly non-normal, which is a common problem in the case of social sciences (Costello & Osborne, 2005). Since the assumption of normality was violated, the principal axis factors (PAF) method was chosen as the factor extraction model because this method does not adhere to any distributional assumptions (Costello & Osborne, 2005; Fabrigar et al., 1999).

The exploratory factor analysis was then conducted using a promax rotation, which is a type of oblique rotation. An oblique rotation is appropriate for this dataset, considering that the psychological constructs which the *ASD-OC* assesses have previously been found to be highly correlated with one another (Austin, 2005; Costello & Osborne, 2005; Hurst, Mitchell, Kimbrel, Kwapil, & Nelson-Gray, 2007; Matson, Boisjoli, Hess, & Wilkins, 2011; Matson, Boisjoli, Rojahn, & Hess, 2009). The resulting factors represented subscales of the *ASD-OC* and were named accordingly. Items were applied to the factor with the greatest factor loading. Only items with a factor loading exceeding 0.4 were assigned to particular subscales (Costello & Osborne, 2005).

Results

A PAF analysis was conducted on the 35 items with oblique rotation (promax). Upon analysis, the KMO measure verified the sampling adequacy for the analysis, $KMO = .95$ ('superb' according to Field, 2009), and all KMO values for individual items were $> .78$, which is well above the acceptable limit of .5 (Field, 2009). Bartlett's test of sphericity, $\chi^2(595) = 5746.726, p < .001$, indicated that correlations between items were sufficiently large for PAF.

An initial analysis was conducted to obtain eigenvalues for each component in the data. The scree plot showed an inflexion that would justify retaining two factors (see Figure 1).

Likewise, the results of the parallel analysis indicated that only two factors were statistically significant at the .05 level. Therefore, a two-factor solution was retained in the final analysis, which in combination accounted for 59.46% of the variance. Upon inspection of the factor correlation matrix, the two factors were found to be highly correlated, $r = .745$. Therefore, the use of the oblique rotation (e.g., promax) was justified.

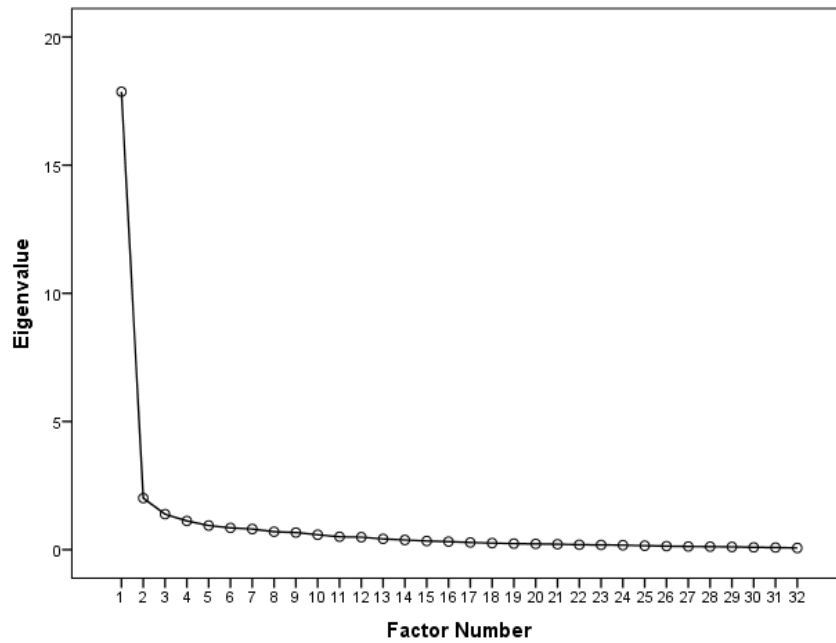


Figure 1. Scree plot of eigenvalues.

Three items failed to meet the .40 criteria for inclusion and were consequently removed. These three items included: (1) *respect for others' personal space*, (2) *repetitive sniffing, touching, feeling, licking, mouthing, tapping of objects or surfaces*, (3) *walks or runs on toes/balls of feet*. Table 3 shows the factor loadings for each item after rotation. The items that cluster on factor 1 primarily included items relating to socialization and communication (e.g., use of gestures to communicate, initiates joint attention, eye contact when communicating expressively). Thus, factor 1 was titled “Social/Communicative Behaviors.” The second factor reflects items relating to restricted and repetitive behaviors and interests (e.g., abnormal,

repetitive hand or arm movements; rituals, insistence on sameness, lining things up, arranging things), so factor 2 was titled “Restricted/Repetitive Behaviors.”

Table 3. Factor Loadings for ASD-OC

Item	Factor 1	Factor 2
9 Can imitate simple sounds, words, phrases	1.079	
10 Can imitate simple sentences	1.063	
15 Can imitate facial expressions of emotion by clinician upon request	1.023	
11 Can imitate simple physical gestures or movements	.932	
12 Can imitate complex physical gestures or movements	.928	
14 Can make facial expression of emotion when asked	.920	
7 Initiates joint attention	.885	
1 Looks when name called	.772	
21 Shares enjoyment, interests, or achievement with others	.748	
3 Use of gestures to communicate	.721	
19 Reaction to praise	.720	
17 Interest in another person’s side of the conversation	.686	
8 Follows along with joint attention	.665	
5 Initiates make-believe or pretend play	.655	
35 Shows empathy	.650	
6 Follows along/participates in make-believe or pretend play	.639	
16 Able to understand the subtle cues/gestures/body language of others	.630	
24 Eye contact when communicating expressively	.601	
22 Reaction to sounds	.547	

(Table 3 continued)

Item	Factor 1	Factor 2
20 Reaction to correction	.529	
4 Awareness of unwritten or unspoken rules of social play	.484	
25 Eye contact when being spoken to	.461	
13 Asks for help	.443	
18 Understanding of age appropriate jokes, figures of speech, or sayings	.422	
27 Excessive interest in inanimate objects		.849
31 Abnormal preoccupation with the parts of an object(s)		.706
32 Rituals, insistence on sameness, lining things up, arranging things		.647
2 Curiosity with surroundings		.568
28 Reaction when transitioning between activities		.558
23 Facial expression corresponds to environmental events		.551
30 Abnormal, repetitive motor movements involving the entire body		.470
29 Abnormal, repetitive hand or arm movements		.454

Discussion

An exploratory factor analysis of the *ASD-OC* using a promax rotation yielded a 2-factor solution. The first factor entitled, ‘Social/Communicative Behaviors,’ consisted of 24 items. The remaining eight items of the measure fell under the ‘Restricted/Repetitive Behaviors’ factor. These two factors easily map onto the new diagnostic criteria outlined in the *DSM-5*. This study adds to the psychometric literature supporting the adequacy and relevancy of the *ASD-OC*. Additionally, the other aforementioned practical qualities of the measure further support the *ASD-OC* to be considered a valuable diagnostic tool.

It is important, however, to consider the possible limitations of this current study while interpreting the results. For example, while many researchers consider a 5:1 participant to item ratio sufficient (Gorsuch, 1983), others advocate for much larger sample sizes when conducting exploratory factor analyses (Fabrigar et al., 1999; Nunnally, 1978). Therefore, future researchers are encouraged to replicate this study with a larger sample to substantiate these findings. Results of exploratory factor analyses which utilize larger samples are innately more generalizable and replicable (Costello & Osborne, 2005).

Additionally, it is important to consider that the participants of this study are likely qualified as a clinical sample, meaning that they were gathered from clinical settings only (i.e., outpatient psychological service clinic or school/treatment program specifically designed for children with developmental disabilities). As previously stated, Fabrigar et al. (1999) suggest using a more heterogeneous sample when conducting an exploratory factor analysis in order to prevent a restricted range in the measures, correlation among variables, and falsely low estimates of factor loadings. While a wide range of total *ASD-OC* scores were evident within the dataset, suggesting a less homogeneous sample, it would be informative to replicate the current study with additional participants recruited from non-clinical settings (e.g., mainstream schools, pediatricians' offices, local churches, other community centers). This would expand the generalizability of these findings to a larger population. Nevertheless, using a primarily clinical population is a common occurrence in the ASD literature. Many researchers examining the factor structure for a measure of autistic symptomatology have used similar populations from strictly clinical settings. For example, many have used participants gathered from the Autism Genetic Resource Exchange (AGRE), where the majority of children in the database are from families with at least two members with an ASD diagnosis (Frazier, Youngstrom, Kubu, Sinclair,

& Rezai, 2008; Norris, Lecavalier, & Edwards, 2012; Snow, Lecavalier, & Houts, 2009).

Several other researchers have only used an ASD population when conducting such analyses (Boomsma et al., 2008; Lecavalier et al., 2006; Lecavalier, Gadow, DeVincent, Houts, & Edwards, 2009; Robertson, Tanguay, L'Ecuyer, Sims, & Waltrip, 1999; Tadevosyan-Leyfer et al., 2003).

Finally, it is crucial to remember that the nature and design of exploratory factor analyses is just that – *exploratory*. It is designed to explore a dataset, rather than test theories (Costello & Osborne, 2005). Therefore, future researchers should conduct a confirmatory factor analysis in order to test theories via inferential techniques. A confirmatory factor analysis can be used to ensure that a 2-factor solution is the most appropriate in describing the structure of ASD.

Study 2

Method

Participants. The sample for Study 2 consisted of 114 children aged 1 to 16 years ($M = 6.06$; $SD = 3.70$) and their caregivers who served as their informants for the current study. The initial sample included 867 children; however, 753 were removed. This sample was collected from a large database of archival data; thus, many participants were excluded from the study simply because they had not been administered the *ASD-OC* and/or the *DSM-IV-TR/ICD-10 Symptom Checklist*. Others were excluded due to insufficient, missing, or inappropriately coded data. Additionally, children with 10% or more of their *ASD-OC* data missing were removed from the sample (Bennett, 2001; Donner, 1982; Field, 2005; Matson, Wilkins, et al., 2009). Just as in Study 1, participants were recruited from either a university outpatient clinic in Louisiana or a local school providing specialized services to children with ASD and other developmental disabilities. With regard to gender, 70.2% of the sample was male and 29.8% was female. Additionally, 66.7% of participants identified as Caucasian, 14.0% identified as African American, 3.5% identified as Hispanic, 10.5% identified as other, and 5.3% did not report their ethnicity. Each individual was included in only one of the four diagnostic groups; no participant was eligible for inclusion in multiple diagnostic groups. See Table 4 for a breakdown of demographic variables by diagnostic group.

Diagnostic group classification was based upon scores gathered from the *DSM-IV-TR/ICD-10 Symptom Checklist*. Given the criteria for categorization, which is outlined below in the Procedures section, children were assigned to the following conditions: Autistic Disorder (AD; $n = 43$), Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS; $n = 19$), Atypically Developing (AtypDev; $n = 34$), and Typically Developing (TypDev; $n = 18$). The

Atypically Developing group consisted of participants who failed to meet criteria for an ASD according to the *DSM-IV-TR/ICD-10 Symptom Checklist* but had another diagnosed/parent-reported Axis I diagnosis, developmental delay, or genetic condition, which are listed in Table 5.

Table 4. Demographic Characteristics by Diagnostic Group.

	Total Sample (N = 114)	ASD Group (n=62)		Non-ASD Group (n=52)	
		AD (n=43)	PDD-NOS (n=19)	AtypDev (n=34)	TypDev (n=18)
Age (years)					
Mean (SD)	6.06 (3.70)	3.98 (2.83)	3.95 (1.98)	8.91 (3.01)	7.89 (3.69)
Range	1-16	1-11	2-7	2-16	2-14
Gender					
Male	80 (70.2%)	33 (76.7%)	16 (84.2%)	18 (52.9%)	13 (72.2%)
Female	34 (29.8%)	10 (23.3%)	3 (15.8%)	16 (47.1%)	5 (27.8%)
Ethnicity					
Caucasian	76 (66.7%)	24 (55.8%)	12 (63.2%)	25 (73.5%)	15 (83.3%)
African American	16 (14.0%)	7 (16.3%)	1 (5.3%)	6 (17.6%)	2 (11.1%)
Hispanic	4 (3.5%)	1 (2.3%)	1 (5.3%)	2 (5.9%)	0
Other	12 (10.5%)	7 (16.3%)	3 (15.8%)	1 (2.9%)	1 (5.6%)
Not Reported	6 (5.3%)	4 (9.3%)	2 (10.5%)	0	0

Note. AD=Autistic Disorder; PDD-NOS=Pervasive Developmental Disorder-Not Otherwise Specified; AtypDev=Atypically Developing group; TypDev=Typically Developing Group

Table 5. Diagnoses of Atypically Developing Group.

Diagnosis	Atypically Developing group (n=34)
Anxiety Disorders	16
ADHD	14
ODD	4
Intellectual Disability	4
Enuresis	3
Other Developmental Delays	3
Learning Disorders	2
Impulse Control Disorders	1
Adjustment Disorder	1

Note. Some participants had more than one diagnosis. ADHD=Attention Deficit Hyperactivity Disorder; ODD=Oppositional Defiant Disorder

Measures.

ASD-OC. See Measures section in Study 1. However, it is important to note that the 32-item version of the *ASD-OC* was used for Study 2 following the removal of three items in Study 1 due to low factor loadings.

DSM-IV-TR/ICD-10 Symptom Checklist. The *DSM-IV-TR/ICD-10 Symptom Checklist* is a parent/caregiver report measure consisting of 19 items (Matson, Gonzalez, Wilkins, & Rivet, 2008). These items assess diagnostic criteria for ASD based on the *DSM-IV-TR* and the *ICD-10*. Respondents indicate “yes” or “no” if the specific item is applicable to their child or not. The checklist contains items pertaining to symptoms from the three core areas of ASD impairment: (1) socialization, (2) communication, and (3) restricted, repetitive, and stereotyped patterns of behavior. Most items on the checklist were supplemented with examples taken from the text of either the *DSM-IV-TR* or the *ICD-10*, as a means to assist the respondent’s understanding of the items. Caregivers were also asked to report if delays or impairments in at least one of these three areas were present prior to 3 years of age. Previous researchers have shown the *DSM-IV-TR/ICD-10 Symptom Checklist* to have excellent interrater reliability ($r = .90$), test-retest reliability ($r = .97$), and internal consistency ($\alpha = .95$) (Matson, Gonzalez, Wilkins, & Rivet, 2008). Face validity of the checklist is also considered to be excellent, considering the checklist is based on the diagnostic criteria from the two most widely used diagnostic manuals. For the purposes of the current study, this checklist was used to assign participants into various diagnostic groups, which is explained in detail in the Procedure section below.

Procedure. Since scales are typically validated based on the comparison to a widely recognized standard (Charman, 2005; de Bilt et al., 2003; Freeman & Schroth, 1984; Lord et al., 2000; Moore & Goodson, 2003), the *DSM-IV-TR/ICD-10 Symptom Checklist* was used as the

criterion from which ASD diagnoses were made (Matson, Boisjoli, Gonzalez, Smith, & Wilkins, 2007). Participants for Study 2 were collected from the same sample used in Study 1 and were assigned to one of the following diagnostic groups: Autistic Disorder, PDD-NOS, atypical development, or typical development. Group assignment for the Autistic Disorder group was based on whether participants meet criteria for this disorder according to the *DSM-IV-TR* algorithm (at least 2 socialization impairments; at least 1 communication impairment; at least 1 RRBI). Participants were assigned to the PDD-NOS group if they had more than three total endorsements on the *DSM-IV-TR/ICD-10 Symptom Checklist* but did not meet the full criteria for Autistic Disorder (Matson, Boisjoli, Gonzalez, Smith, & Wilkins, 2007; Walker et al., 2004). This method of diagnosis allows for continuity across participants for research purposes and has been utilized in previous ASD research (see Matson, Boisjoli, Gonzalez, Smith, & Wilkins, 2007).

The atypically developing group consisted of children who did not have a primary ASD diagnosis according to the *DSM-IV-TR/ICD-10 Symptom Checklist* but met criteria for one or more Axis I diagnoses (e.g., attention-deficit/hyperactivity disorder [ADHD], depression, generalized anxiety disorder, separation anxiety disorder, obsessive compulsive disorder [OCD], enuresis, selective mutism, learning disorder, social phobia, specific phobia) and/or had reported developmental delays or genetic conditions (e.g., Down's syndrome, epilepsy, cerebral palsy, mild ID). The typically developing group consisted of participants who did not meet criteria for an ASD, other Axis I diagnoses, genetic conditions, intellectual disabilities, or other developmental delays. See the Procedure section of Study 1 for further information regarding the administration of the *ASD-OC*, consent/assent, and Institutional Review Board approval.

Research Design. Receiver operating characteristics (ROC) analyses first appeared in the 1960s as a means to detect radar signals in the fields of engineering and psychophysics (Green & Swets, 1966). More recently, ROC analyses have begun to be used in a number of other areas, including medical imaging, weather forecasting, information retrieval, polygraph lie detection, aptitude testing, machine learning, data mining research, prediction of violence, and diagnostic testing (Compton, Fuchs, Fuchs, & Bryant, 2006; Fawcett, 2006; Green & Swets, 1966; Metz, 1978; Mossman, 1994; Rice & Harris, 1995; Swets, 1979, 1988; Swets, Dawes, & Monahan, 2000; Swets & Pickett, 1982; Swets, Tanner, & Birdsall, 1964; Tanner & Swets, 1954). For the purposes of this study, this analysis was used as a means to develop clinical cutoff scores for a measure of autistic symptomatology.

By conducting a series of ROC analyses, cutoff scores were developed for the *ASD-OC* in order to discriminate between: (a) ASD and no ASD, (b) autism and PDD-NOS (within the ASD group), and (c) atypical development and typical development (within the No ASD group). This type of analysis allows the researcher to evaluate the ability of a test to discriminate between various groups by plotting the sensitivity of each score against the specificity of each score (Fawcett, 2006; Fresco, Mennin, Heimberg, & Turk, 2003; Hanley & McNeil, 1982; Kumar & Indrayan, 2011). Sensitivity refers to the rate at which a measure correctly identifies individuals with the disorder as having the disorder, while specificity is the degree to which a test is able to correctly identify those without the disorder as not having the disorder (Compton et al., 2006). Thus, the y-axis is a plot of the true-positive rate for each value and the x-axis plots the false-positive rate for each possible value. If a test were to function at a rate of exactly chance performance then the ROC curve would appear as a diagonal line from the lower left-hand corner of the graph to the upper right-hand corner, as seen in Figure 2 (i.e., dotted line).

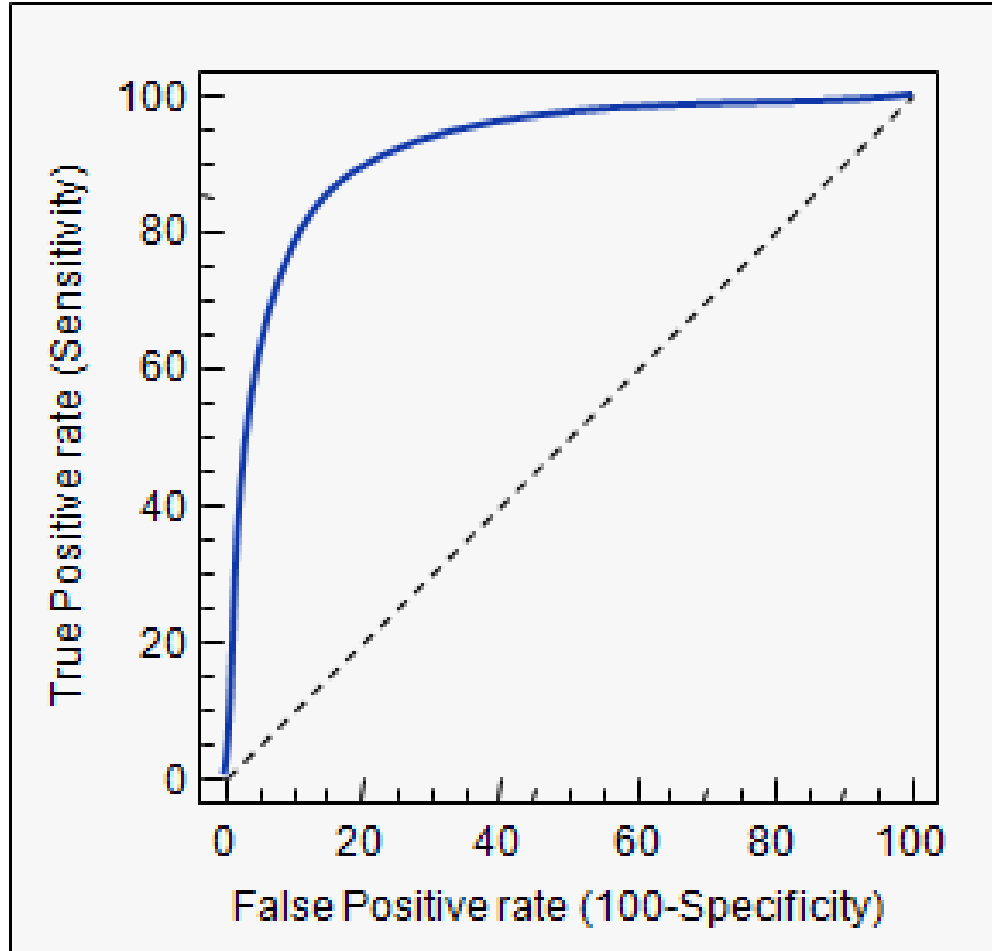


Figure 2. ROC curve example.

ROC curves are interpreted by inspecting the proportion of the total area of the graph that lies under the curve. The greater the area, the greater the difference at each point between the true-positive and false-positive rates, and the better the prediction of the instrument (Hanley & McNeil, 1982; M. E. Rice & Harris, 1995). The ability of the *ASD-OC* to reliably discriminate between groups was determined by inspecting the Area Under the Curve (AUC) statistic. The AUC statistic ranges from 0.5 (i.e., chance performance) to 1.0 (i.e., perfect performance; Compton, Fuchs, Fuchs, & Bryant, 2006; Fombonne, 1991; Swets, 1988) and has been said to be the most commonly used global index of diagnostic accuracy (Fluss, Faraggi, & Reiser, 2005;

Martinez-Cambolor, 2013). Fawcett (2006) describes the AUC as “the probability that the classifier will rank a randomly chosen positive instance higher than a randomly chosen negative instance” (p. 868). For example, an AUC of 0.90 would indicate that the test of interest had a 90% chance of making a correct group assignment. Qualitatively, the AUC statistic ranges are described as: .90+ (excellent); .80 to .90 (good); .70 to .80 (fair); and below .70 (poor) (Compton et al., 2006). A .05 alpha level was used when determining the significance of each AUC in order to identify if the *ASD-OC* performed significantly better than chance (i.e., $AUC = 0.5$) for the target population. For each comparison with a significant AUC, the cutoff score that maximized sensitivity and specificity was identified using the Youden Index.

The Youden Index is a commonly used criterion for choosing the optimal threshold value (Youden, 1950). This index is defined as $J = \max_c [Sensitivity(c) + Specificity(c) - 1]$ and ranges between 0 and 1. Fluss, Faraggi, and Reiser (2005) describe the Youden Index to be the most streamlined method to obtain an optimal threshold, as it does not require any extra information (e.g., prevalence rates, decision error costs). For this study, the cutoff point that maximized sensitivity and specificity was established using the Youden Index, representing the overall accuracy of the test (Krzanowski & Hand, 2009; Kumar & Indrayan, 2011; Perkins & Schisterman, 2005; Youden, 1950).

Finally, for the comparison using the full sample (i.e., ASD vs. non-ASD) the positive predictive value (PPV) and negative predictive value (NPV) of the *ASD-OC* were calculated according to the cutoff score determined by the Youden Index. PPV is the probability that the disorder (i.e., ASD) is present when the test is positive, whereas NPV is the probability that the disorder (i.e., ASD) is not present when the test is negative when using the specified cutoff score.

It is important to note that these procedures, using the ROC analysis, have frequently been used in previous research when assessing the accuracy of diagnostic tools within the area of autism and other childhood disorders. For example, ROC analyses were used when developing the scoring algorithm for the *ADOS-T* (Luyster et al., 2009). Cohen and colleagues (2010) also used ROC analyses to assess the ability of the *Pervasive Developmental Disorder Behavior Inventory (PDDBI)* to differentiate between the following comparisons: (1) Autism vs. no ASD, (2) PDD-NOS vs. no ASD, and (3) Autism + PDD-NOS vs. no ASD. The significance of the resulting AUC statistic was tested at the .05 alpha level. The Youden Index (*J*) was also calculated in order to identify the optimal cutoff score for the measure. Likewise, these same analyses were conducted to identify cutoff scores for the *BISCUIT* (Matson, Wilkins, Sharp, Knight, Sevin, Boisjoli, 2009), along with age-based scoring cutoffs for this measure as well (Horovitz & Matson, 2014). Chen, Faraone, Biederman, & Tsuang (1994) utilized ROC analyses to determine the diagnostic accuracy of the *Child Behavior Checklist (CBCL)* for identifying ADHD. These procedures have also been used as a means to identify the ability of a test to correctly differentiate between second graders with and without a reading disorder (Compton et al., 2006). Although not within a child population, Matson, Boisjoli, Gonzalez, Smith, and Wilkins (2007) also utilized ROC analyses to develop cutoff scores for the *Autism Spectrum Disorder-Diagnosis for Adults (ASD-DA)*. Additionally, Fresco, Mennin, Heimberg, & Turk (2003) used these analyses to identify adults with Generalized Anxiety Disorder when using the *Penn State Worry Questionnaire*. Given the amount of research supporting the use of ROC analyses not only within the field of autism, but within a variety of other areas as well, it is believed that these procedures will produce the most accurate and reliable outcomes for determining cutoff scores for the *ASD-OC*.

Results

Preliminary Analyses. To determine the sample size required to conduct a ROC analysis, an *a priori* power analysis was conducted. With an alpha of .05, power of .80, and an estimated AUC of .80 (i.e., good discriminative ability), the required sample size was identified as 13 per group (MedCalc, 2011). The groups for the current study exceeded these limits, as the smallest group (i.e., the typically developing group) had a total of 18 participants. Elsewhere, it has also been recommended that a total sample size of at least 100 be utilized for ROC analyses (Metz, 1978), which this dataset exceeded as well ($N = 114$). Additionally, considering that the groups in the current study were all of unequal sizes, it is important to note that one particular strength of a ROC analysis is that the test is robust even with unequal group sizes (Fresco, Mennin, Heimberg, & Turk, 2003; Rice & Harris, 1995).

Main Analyses.

ASD vs. No ASD. First, the ROC analysis was conducted for the entire sample to establish the optimal cutoff to discriminate between the ASD group ($n = 62$) and the non-ASD group ($n = 52$). The resulting curve had an $AUC = .981$, $p < .01$. An AUC above .9 represents excellent discriminating ability (Compton et al., 2006; Swets, 1988). Figure 3 shows the curve from this analysis. The Youden Index (J) was then calculated to determine the cutoff point that maximized sensitivity and specificity. J is defined as the maximum (sensitivity + specificity - 1) for all possible cutoff values (Youden, 1950). The diagonal line depicted in the ROC curve represents chance performance, and J is essentially the cutoff point that falls farthest from this line. The optimal cutoff for differentiating between ASD and no ASD for the full sample was found to be a score equal to or greater than 10, $J = .87$. Sensitivity and specificity for this cutoff point were .90 and .96, respectively.

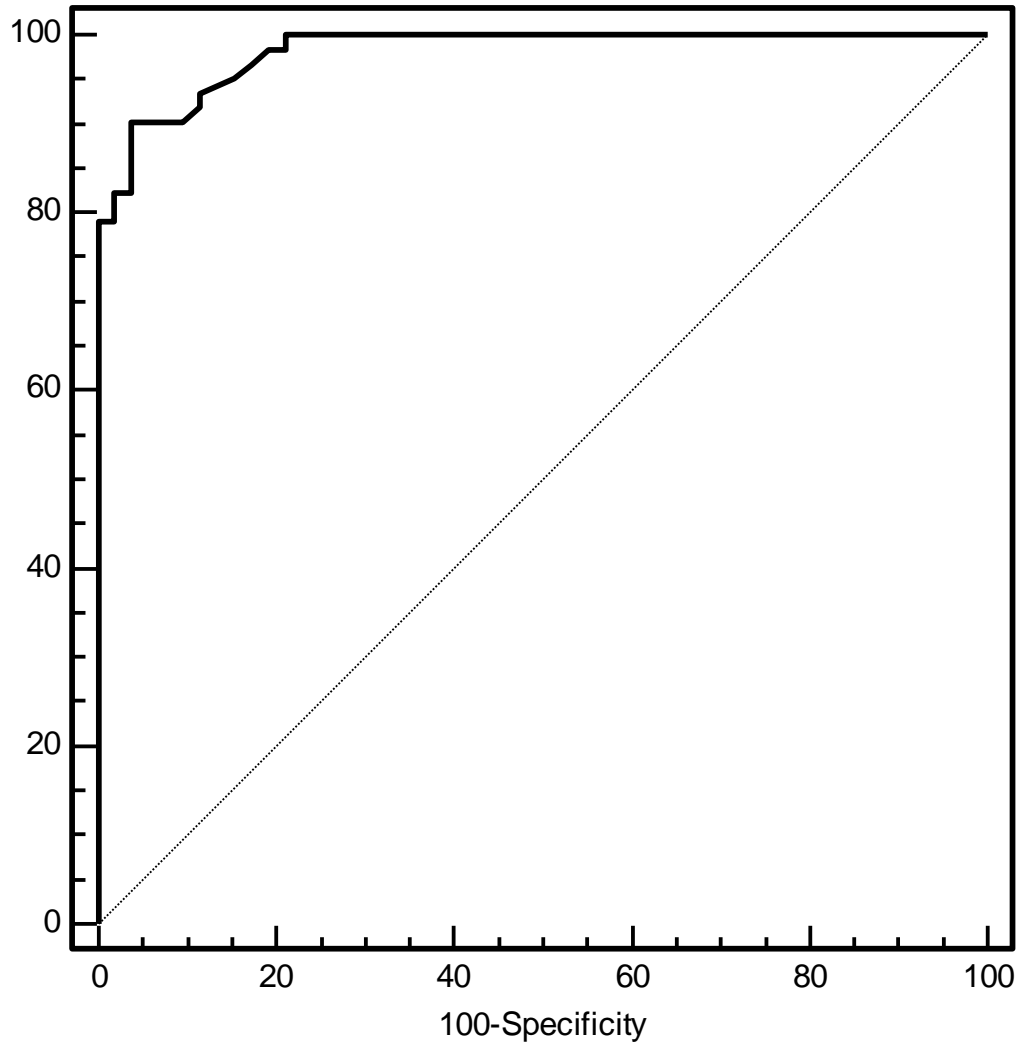


Figure 3. ROC curve plotting the sensitivity and 100-specificity of each possible cutoff point discriminating the ASD group vs. the No ASD group according to the *DSM-IV-TR*.

The sensitivity and specificity for each possible cutoff point on the ASD-OC were then plotted against one another. This plot can be seen in Figure 4. The point at which the two lines cross marks the *ASD-OC* total score which maximizes sensitivity and specificity.

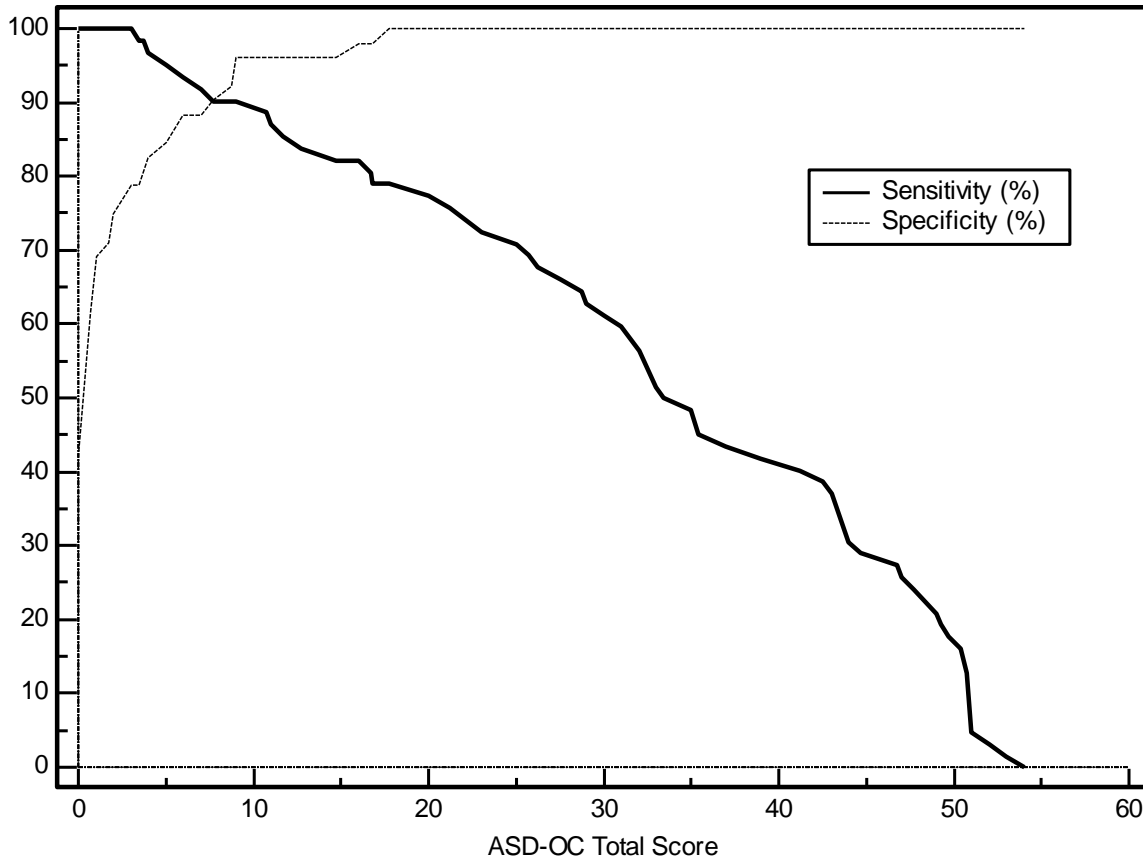


Figure 4. Plot of sensitivity and specificity for each possible cutoff point on the ASD-OC when discriminating between the ASD group vs. the No ASD group according to the *DSM-IV-TR*.

Finally, the positive predictive value (PPV) and negative predictive value (NPV) of the *ASD-OC* according to the cutoff score of 10 were calculated. PPV is the probability that the disorder is present when the test is positive, and NPV is the probability that the disorder is not present when the test is negative. With a sensitivity of 90%, a specificity of 96%, and a disease prevalence of 54.39%, results yielded a PPV of 96.55% and a NPV of 89.29% when using the cutoff score of ≥ 10 .

Autistic Disorder vs. PDD-NOS. These same ROC analyses were then repeated using only the ASD group ($n = 62$) in order to determine the optimal cutoff point for differentiating between Autistic Disorder ($n = 43$) and PDD-NOS ($n = 19$). The *ASD-OC* demonstrated fair

discriminating ability with an $AUC = .718$, $p < .01$. Figure 5 shows the curve from this analysis. The optimal cutoff for differentiating between Autistic Disorder and PDD-NOS was a score equal to or greater than 38, $J = .48$. Sensitivity and specificity for this cutoff point were .58 and .90, respectively.

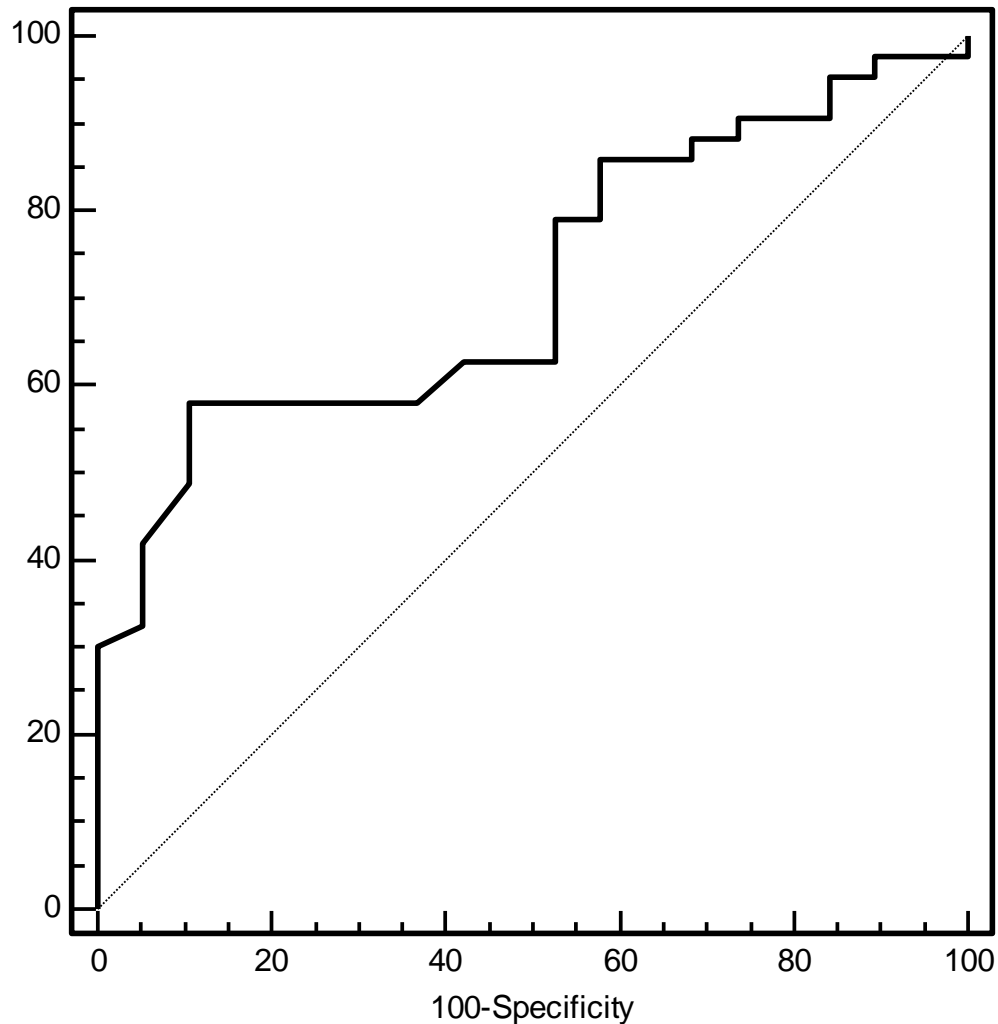


Figure 5. ROC curve plotting the sensitivity and 100-specificity of each possible cutoff point discriminating the AD group vs. the PDD-NOS group according to the *DSM-IV-TR*.

The sensitivity and specificity for each possible cutoff point on the ASD-OC were then plotted against one another. This plot can be seen in Figure 6. The point at which the two lines cross marks the ASD-OC score which maximizes sensitivity and specificity when discriminating between Autistic Disorder and PDD-NOS.

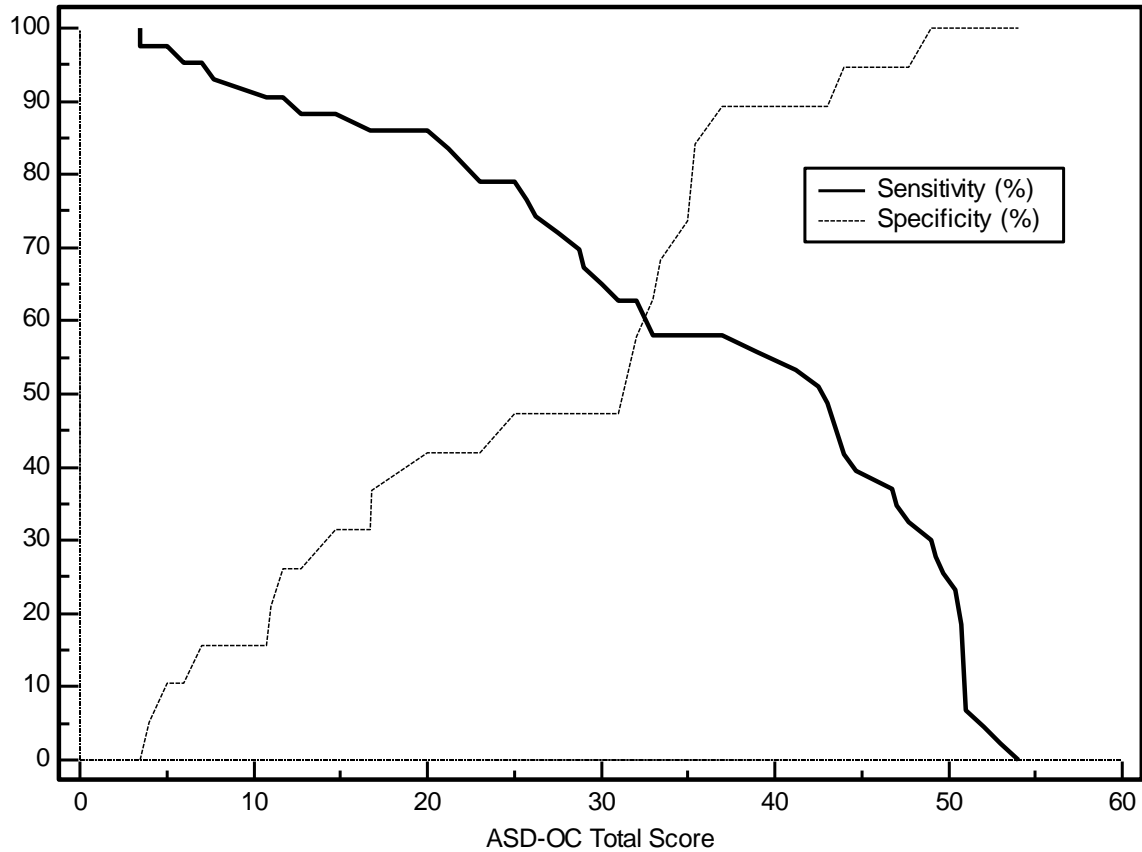


Figure 6. Plot of sensitivity and specificity for each possible cutoff point on the ASD-OC when discriminating between AD and PDD-NOS according to the *DSM-IV-TR*.

Atypically Developing vs. Typically Developing. These same ROC analyses were then repeated using only the non-ASD group ($n=52$) in order to determine the optimal cutoff point for differentiating between the Atypically Developing group ($n=34$) and the Typically Developing group ($n=18$). The $AUC=.508$ (i.e., poor discriminating ability), $p=.923$. Figure 7 shows the curve from this analysis. The Youden Index and corresponding optimal cutoff for differentiating between these two groups were not calculated due to the nonsignificant AUC.

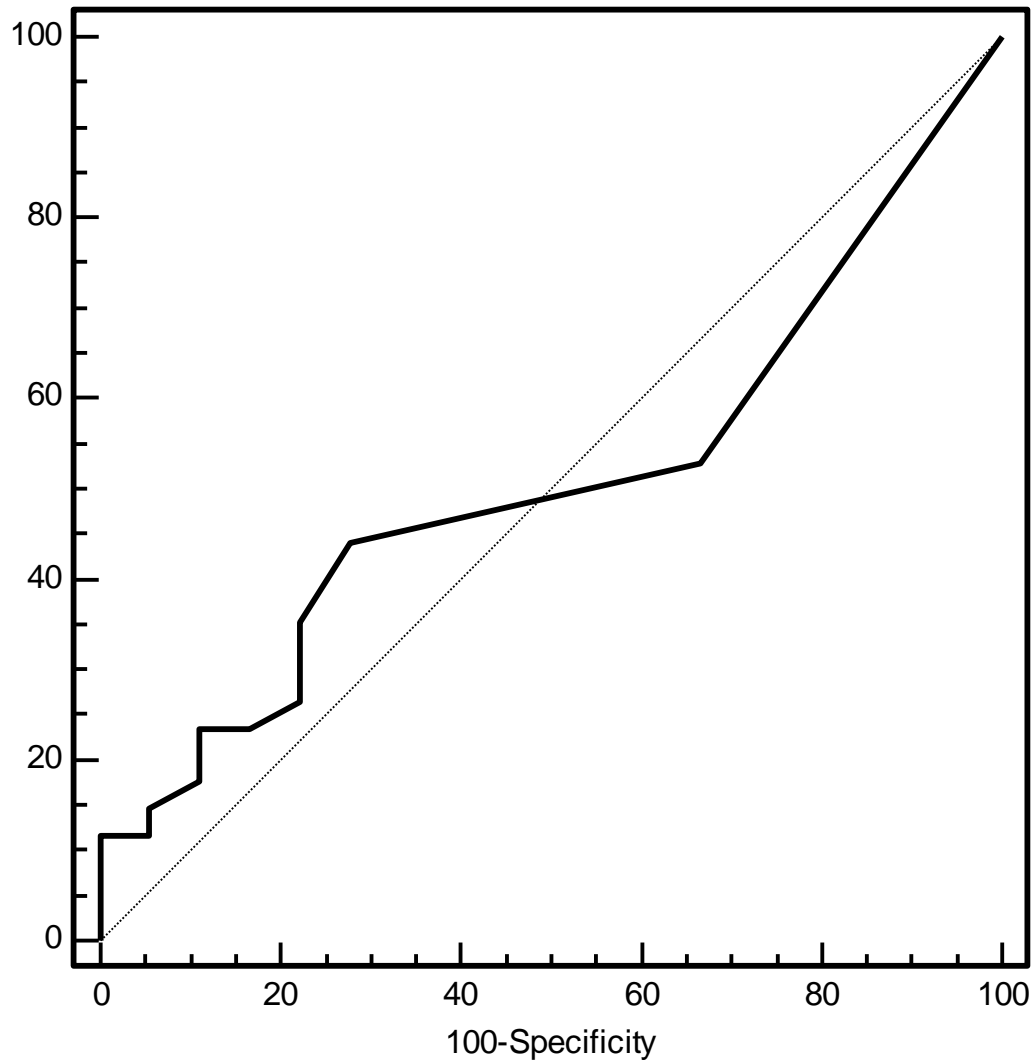


Figure 7. ROC curve plotting the sensitivity and 100-specificity of each possible cutoff point discriminating the Atypically Developing group vs. the Typically Developing group according to the *DSM-IV-TR*.

Discussion

The purpose of Study 2 was to establish cutoff scores based on ROC analyses for the total score of the *ASD-OC*. Given the increasing focus on early intervention and treatment of children with developmental disabilities, such research is essential. The *ASD-OC* was able to discriminate between those with and without ASD with excellent precision. Additionally, the *ASD-OC* demonstrated a fair ability to discriminate between PDD-NOS and autism. For both of

these comparisons, the AUC statistic was significant for each curve, implying that the *ASD-OC* performed significantly better than chance.

It was to be expected that the *ASD-OC* would be better at discriminating between ASD and non-ASD than it would be at discriminating between Autistic Disorder and PDD-NOS. According to the *DSM-IV-TR*, there are no explicit criteria that must be met in order to diagnose one with PDD-NOS. Examples that may justify this diagnosis include a later age of onset, a composition of symptoms that fails to reach the threshold level for autism, or an atypical set of symptoms failing to meet the criteria for another ASD (APA, 2000). This ambiguity leads to great variability in the symptom presentation across all children with PDD-NOS, thereby directly influencing clinician ratings on an observation measure, such as the *ASD-OC*. On the other hand, some researchers have found more stability in broader classifications of ASD or no ASD made in early childhood (Moore & Goodson, 2003), making a measure's ability to discriminate between these two groups less complicated.

Unfortunately, the *ASD-OC* was unable to reliably differentiate between the Atypically Developing group and the Typically Developing group. As previously mentioned, the Atypically Developing group was made up of participants who did not meet criteria for an ASD according to the *DSM-IV-TR/ICD-10 Symptom Checklist* but had another diagnosed/parent-reported Axis I diagnosis, developmental delay, or genetic condition. Upon further inspection of the dataset, the majority of the Atypically Developing group did not have a reported intellectual disability or other developmental concern. Rather, most participants of this group had one or more Axis I diagnoses that are unlikely to produce elevated scores on the *ASD-OC* in comparison to those with typical development, simply due to the validity of the measure. For example, anxiety disorders and ADHD were two of the most common disorders among the Atypically Developing

group, and it is unlikely that ASD-specific items (e.g., items assessing joint attention, communication problems, restricted interests, or hypersensitivities) on the *ASD-OC* would yield elevated scores in these children. Please refer to Table 5 for a list of the diagnoses of the Atypically Developing group.

Study 3

Method

Participants. This study is an extension of Study 2; therefore, the same initial sample pool was used ($N = 867$). Those with missing, insufficient, or improperly coded data were removed from the sample ($n = 758$). Most of these excluded participants were ones who simply had not been administered the ASD-OC and/or the *DSM-IV-TR/ICD-10 Symptom Checklist*, yet were still included in the original large database of archival data from which this sample was collected. Children with no more than 10% of their *ASD-OC* data missing were retained and the missing values were replaced with the mean for that item (Bennett, 2001; Donner, 1982; Field, 2005; Matson, Wilkins, et al., 2009). The final sample for this study included 109 caregivers and their children aged 1-16 years ($M = 6.21$, $SD = 3.71$). Method of recruitment for Study 3 was identical to Study 1 and 2 (i.e., a university outpatient clinic in Louisiana and a local school providing specialized services to children with ASD and other developmental disabilities). Overall, the sample was comprised of 71.6% males and 28.4% females. Ethnicity, as reported by caregivers, was 67.0% Caucasian, 12.8% African American, 3.7% Hispanic, 11.0% other, and 5.5% not reported. Demographic information for this sample according to diagnostic group is presented in Table 6. Participants were categorized into one of two diagnostic groups. Group classification was based upon scores gathered from the *DSM-IV-TR/ICD-10 Symptom Checklist*. Given the criteria for categorization, which is outlined below in the Procedures section, children were assigned to the following conditions: ASD ($n = 31$) or non-ASD ($n = 78$).

Table 6. Demographic Characteristics by Diagnostic Group

	Total Sample (N=109)	ASD (n=31)	Non-ASD (n=78)
Age (years)			
Mean (SD)	6.21 (3.71)	3.90 (2.67)	7.13 (3.67)
Range	1-16	1-11	2-16

(Table 6 continued)

	Total Sample (N=109)	ASD (n=31)	Non-ASD (n=78)
Gender			
Male	78 (71.6%)	27 (87.1%)	51 (65.4%)
Female	31 (28.4%)	4 (12.9%)	27 (34.6%)
Ethnicity			
Caucasian	73 (67.0%)	17 (54.8%)	56 (71.8%)
African American	14 (12.8%)	5 (16.1%)	9 (11.5%)
Hispanic	4 (3.7%)	0	4 (5.1%)
Other	12 (11.0%)	5 (16.1%)	7 (9.0%)
Not Reported	6 (5.5%)	4 (12.9%)	2 (2.6%)

Measures.

ASD-OC. See Measures section of Study 1 for a description of the *ASD-OC*. Again, it should be noted that the 32-item version of the *ASD-OC* was used for Study 3 following the removal of three items in Study 1 due to low factor loadings.

DSM-IV-TR/ICD-10 Symptom Checklist. See the Measures section of Study 2 for a description of the *DSM-IV-TR/ICD-10 Symptom Checklist*.

Procedure. As most scales are validated against a widely recognized standard, the *DSM-5* criteria for ASD were used as the basis from which ASD diagnoses were made for Study 3 (Matson et al., 2007). Participants for Study 3 were collected from the same sample as used in Study 1 and 2 and were assigned to one of the following diagnostic groups: ASD or non-ASD. Group assignment was based upon the *DSM-IV-TR/ICD-10 Symptom Checklist* scores. In previous research, this diagnostic checklist has been slightly modified in order to provide *DSM-5* ASD diagnoses for research purposes (Matson, Belva, et al., 2012; Neal, Matson, & Hattier, 2012). In order for a participant to meet criteria for ASD according to the *DSM-5*, a minimum of five total “yes” endorsements were required on the *DSM-IV-TR/ICD-10 Symptom Checklist*. Three of these five endorsements must have been from the social interaction portion of the

checklist, and at least two endorsements were required from the repetitive and restricted interests and behaviors section.

The *DSM-IV-TR/ICD-10 Symptom Checklist* contains items assessing all three of the social communication/interaction symptoms listed in the *DSM-5*. The checklist also contains items assessing three of the four symptoms within the repetitive and restricted interests and behaviors criterion of the *DSM-5*. The one symptom within this domain that is not included in the checklist is “hyper- or hypo-reactivity to sensory input or unusual interests in sensory aspects of the environment.” Therefore, it was possible that some participants did not meet the criteria for ASD according to the *DSM-5* as stated above (i.e., at least 3 social impairments and 2 restricted/repetitive behavior and interest impairments) but actually should have met the criteria because they had sensory abnormalities which were not assessed on the *DSM-IV-TR/ICD-10 Symptom Checklist*. To account for this disparity, all participants meeting the socialization criterion (i.e., at least three endorsements) but only had one endorsement on the restricted/repetitive behaviors and interests criterion were excluded from the study. This was done to eliminate the possibility of including participants who should have been given an ASD diagnosis from falling within the non-ASD group, given the possibility that they may have had hyper- or hypo-reactivity to sensory input. A total of five participants were removed from the sample to account for this potential problem. Please see the Procedure section of Study 1 for further information regarding the administration of the *ASD-OC*, informed consent and assent, and Institutional Review Board approval.

Research Design. The same analyses from Study 2 were replicated in Study 3; however, the cutoff scores were calculated to identify ASD according to the current *DSM-5* criteria (APA, 2013). Having cut-off scores which map onto both the current *DSM-IV-TR* diagnostic criteria

and the *DSM-5* diagnostic criteria will be useful for clinicians when attempting to easily transition from one edition of the classification system to another.

Additionally, the current *DSM-5* ASD diagnostic criteria are accompanied by severity ratings: Level 1 (requiring support); Level 2 (requiring substantial support); and Level 3 (requiring very substantial support). Thus, a complementary severity rating scale for the *ASD-OC* was developed. These varying levels of severity (i.e., high, moderate, low) can be used by clinicians as a guideline when determining the child's *DSM-5* ASD severity level (APA, 2013). ROC analyses were not able to be used as the means by which severity cutoff scores were developed, because differing levels of severity are not assessed by the *DSM-IV-TR Symptom Checklist*. Thus, these severity cutoff scores were established using the standard deviation method by calculating measures of central tendency. The mean was selected as the norm for the purposes of this study. One standard deviation above and one standard deviation below this score was used to determine clinical significance (Kendall & Grove, 1988).

Results

Preliminary Analyses. As in Study 2, an *a priori* analysis was conducted in order to establish the required sample size needed to conduct a ROC analysis. It was determined that at least 13 participants per group were necessary with the following set limitations, an alpha of .05, power of .80, and an estimated AUC of .80 (i.e., good discriminative ability). Others have also set general sample size guidelines when conducting ROC analyses. For example, Metz (1978) noted that a total sample size of at least 100 participants is sufficient. The sample for Study 3 exceeded these requirements ($N = 109$). Furthermore, it has also been suggested that one particular strength of a ROC analysis is that the test is robust even with unequal group sizes (Fresco, Mennin, Heimberg, & Turk, 2003).

Main Analyses. A ROC analysis was conducted to establish the optimal cutoff to discriminate between the ASD group and the non-ASD group according to the *DSM-5*. The resulting curve had an $AUC = .906$, $p < .0001$, indicating excellent discriminating ability (Compton et al., 2006). Figure 8 shows the curve from this analysis. The Youden Index (J) was then calculated to establish a cutoff point maximizing sensitivity and specificity. The optimal cutoff was found to be a score equal to or greater than 22, $J = .68$. Sensitivity and specificity for this cutoff point were .87 and .81, respectively. See Table 7 for an outline of the cutoff scores generated from both Study 2 and Study 3, along with each score's sensitivity, specificity, and Youden index.

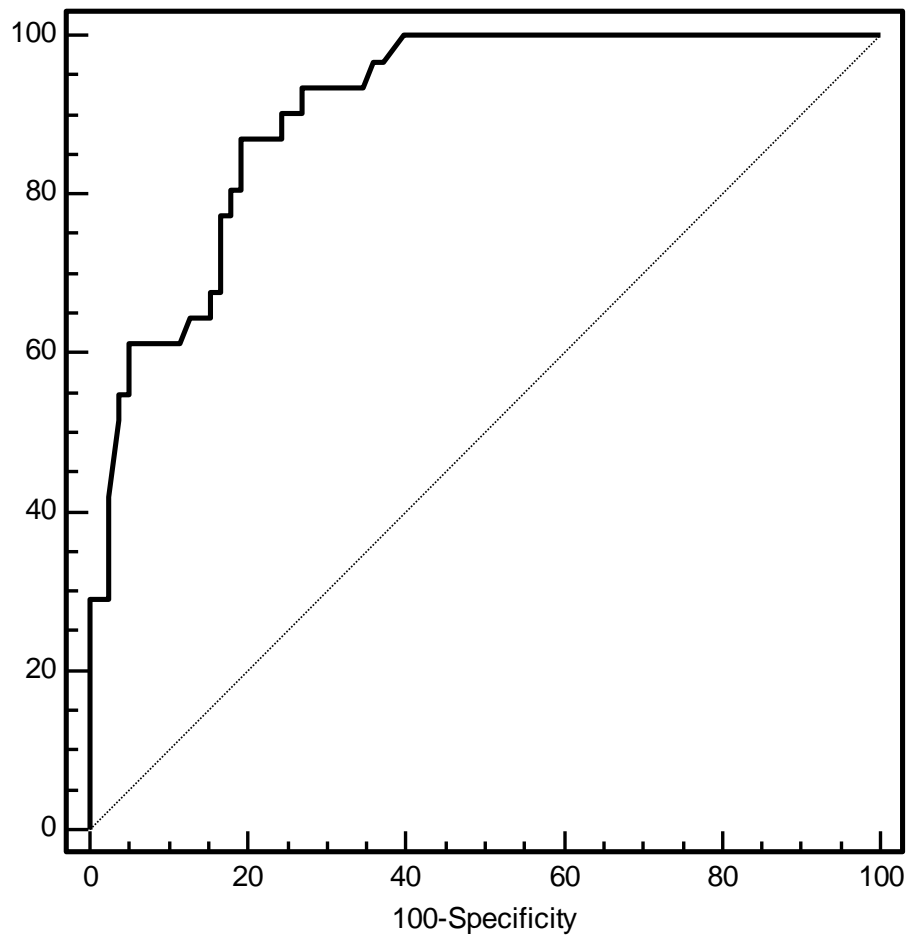


Figure 8. ROC curve plotting the sensitivity and 100-specificity of each possible cutoff point discriminating the ASD group vs. the No ASD group according to the *DSM-5*.

Table 7. Selected cutoffs and psychometric properties for each sample

	Cutoff	Sensitivity	Specificity	Youden Index (J)
DSM-IV-TR				
Autism vs. PDD-NOS	≥ 38	58%	90%	.48
ASD vs. No ASD	≥ 10	90%	96%	.87
DSM-5				
ASD vs. No ASD	≥ 22	87%	81%	.68

The sensitivity and specificity for each possible cutoff point on the *ASD-OC* were then plotted against one another. This plot can be seen in Figure 9. The point at which the two lines cross is essentially the score on the *ASD-OC* which maximizes sensitivity and specificity when discriminating between those with and without ASD according to the *DSM-5*.

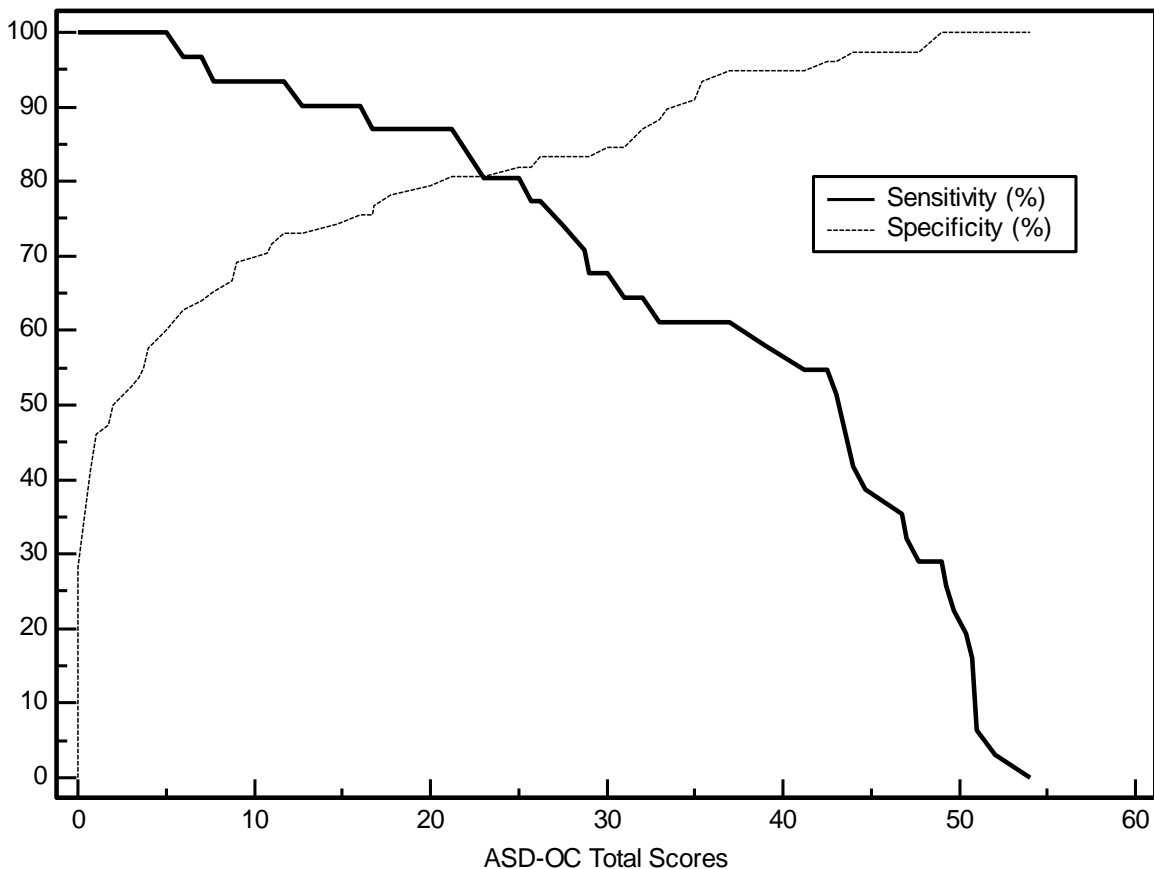


Figure 9. Plot of sensitivity and specificity for each possible cutoff point on the *ASD-OC* when discriminating between ASD and no ASD using the *DSM-5*.

Next, the PPV and NPV of the *ASD-OC* according to the cutoff score of 22 were calculated. As previously stated, PPV is the probability that the disorder is present when the test is positive, and NPV is the probability that the disorder is not present when the test is negative. With a sensitivity of 87%, a specificity of 81%, and a disease prevalence of 28.44%, results yielded a PPV of 64.29% and a NPV of 94.03% when using the cutoff score of ≥ 22 .

A final analysis was conducted to determine severity cutoff scores within the ASD range (i.e., *ASD-OC* total scores from 22 to 64). The standard deviation method was used to determine three levels of severity (i.e., high, moderate, low). The mean score of participants meeting the ASD cutoff (i.e., ≥ 22) was 39.53, with a standard deviation of 9.43. Thus, the high severity cutoff was any score equal to or greater than 49. The moderate severity cutoff was any score falling within the range of 31 to 48, and participants fell within the low severity range if they had an *ASD-OC* total score from 22 to 30.

Discussion

Based upon the results of Study 3, the *ASD-OC* demonstrated excellent discriminating ability when differentiating between the ASD group and the No ASD group when diagnoses were based upon the *DSM-5* criteria. Unsurprisingly, the cutoff score generated (i.e., ≥ 22) from Study 3 was greater than the cutoff score generated in Study 2 discriminating between ASD and no ASD (i.e., ≥ 10). Upon further inspection of the dataset, 23.8% ($n = 26$) of the *DSM-5* No ASD group met criteria for an ASD when diagnosed according to the *DSM-IV-TR*. This may explain why many of the participants in the *DSM-5* No ASD group had elevated *ASD-OC* total scores despite failing to meet criteria for ASD when using the *DSM-5* as the basis for classification. This corroborates much of the current literature. Based upon previous research, approximately 30 – 45% of individuals diagnosed with an ASD based upon the *DSM-IV-TR* no

longer meet criteria for ASD due to the increased diagnostic stringency of the *DSM-5* (Matson, Belva, et al., 2012; Matson, Kozlowski, et al., 2012; McPartland et al., 2012; Worley & Matson, 2012).

Conclusion

The study of ASD has received great attention over recent years among researchers in the scientific community. The disorder has been studied with respect to its etiology, prevalence, behavioral presentation, gender differences, comorbidities, early identification, and diagnosis, among many other areas. With the help of this extensive research, clinicians have become more accurate when identifying and diagnosing ASD. To increase diagnostic reliability, many practitioners turn to the use of well-studied and psychometrically sound assessment tools. Over time with the progression of ASD literature, these tools have evolved and new ones have been developed in order to remain relevant and informative. Given the fact that ASD diagnostic criteria has largely advanced with the release of the latest edition of the *DSM* in 2013, continued research in the field of assessment tool development for ASD is vital not only for clinicians, but for researchers, caregivers, and children with ASD, alike.

The *ASD-OC* is a clinician-rated observation measure intended for children suspected of having ASD. As previously discussed there are eight primary shortcomings of the pre-existing observation tools, which the *ASD-OC* has the ability to address. To review, these eight areas include: (1) weak psychometric properties, (2) inability to differentiate between ASDs and atypical development, (3) difficult and lengthy administration, (4) complexity of scoring procedures, (5) extensive examiner training requirements, (6) lack of correlation with formal diagnostic criteria, (7) inability to individualize the assessment for each client, and (8) lack of corresponding parent-report rating scales. The goals of the current studies were to examine the factor structure of the *ASD-OC* and develop two sets of clinical cutoff scores which map onto both the *DSM-IV-TR* and *DSM-5* diagnostic criteria. This research will provide further support

for the use of the *ASD-OC* as a reliable diagnostic tool consistent with the latest diagnostic criteria.

An EFA was conducted, and the factor structure resulted in two domains of ASD symptomatology. Over recent years, researchers have provided reliable findings which warrant the collapse of the communication and socialization domains into one feature of autism (Dube, MacDonald, Mansfield, Holcomb, & Ahern, 2004; Frazier et al., 2011; Guthrie, Swingford, Wetherby, & Lord, 2013; So Hyun Kim & Catherine Lord, 2013). Based upon the results of the EFA, the two factors of the *ASD-OC* correlate well with recent literature, which suggests that ASD consists of two basic domains: (a) social/communicative impairments and (b) repetitive/restricted behaviors and interests. The two-component factor structure of the *ASD-OC* explained 59.46% of the total variance of the items. The removal of three items was necessary due to their considerably low factor loadings (below .40). Inspecting the factor structure of the *ASD-OC* ensures that this measure remains relevant and consistent with the diagnostic criteria being used by clinicians today, thereby satisfying one of the eight previously mentioned limitations of other ASD observation scales.

Many clinicians and diagnosticians strongly encourage integrating the utilization of multiple methods and informants to achieve a comprehensive assessment and more accurate diagnoses. Nevertheless, it remains imperative that every assessment process is built on the foundation of psychometrically sound diagnostic instruments. This research enhances the psychometric properties of the *ASD-OC* by providing clinicians with diagnostic guidelines when inspecting a child's *ASD-OC* total score. Based upon the results of ROC analyses, clinical cutoff scores were developed for the *ASD-OC*. As hypothesized, the *ASD-OC* performed significantly better than chance, as evidenced by AUC statistics significantly different from 0.5. Cutoff scores

that were chosen were ones that exhibited the optimal trade-off between sensitivity and specificity when differentiating between the specified groups for this sample. Cutoff scores were divided into ones specific to the *DSM-IV-TR* and ones specific to the *DSM-5* diagnostic criteria. Please refer to Table 7 for a comparison of these cutoff scores, along with each score's sensitivity, specificity, and Youden index. These scoring procedures were designed to be simple and efficient for clinicians, thus satisfying another one of the eight aforementioned limitations of other ASD observation measures.

Finally, severity guidelines according to the *DSM-5* criteria were designed. One of the many changes that were made to the diagnosis of ASD in the *DSM-5* was the inclusion of a complementary severity score that clinicians can provide alongside a child's ASD diagnosis. These severity scores can be used for future clinicians or service providers to quickly determine a child's level of functioning for assessment or treatment purposes. With regard to the *ASD-OC*, these severity cutoff markers will assist clinicians when determining a child's *DSM-5* severity score.

As previously mentioned, these studies are not without limitations. One major limitation of these studies is that participants were classified into diagnostic groups based upon informant ratings of the *DSM-IV-TR/ICD-10 Symptom Checklist*. While this measure has previously shown to have sound psychometric properties, there will always be the possibility of inaccurate ratings. For example, informants may have misinterpreted certain items of the measure or may have been biased when completing the checklist. Future researchers are encouraged to utilize clinical diagnoses provided by licensed psychologists as the basis for group classification. Additionally, the predictive validities reported in Study 2 and Study 3 should be interpreted with some caution. PPV and NPV scores will vary across different research laboratories or different samples, since

these values are dependent on the base rate of ASD for each sample. Therefore, false positive rates will increase as the base rate of ASD decreases.

There are several avenues for future research following these studies. The current study demonstrated the utility of *DSM*-based scoring procedures for the *ASD-OC*. Future researchers, however, are encouraged to investigate the development of *ASD-OC* cutoff scores based upon the child's age. Age-based scoring has previously been found to increase the accuracy of certain assessment instruments (Achenbach & Rescorla, 2000; Horovitz & Matson, 2014; Kim & Lord, 2012). Second, it would be sensible for future researchers to replicate the severity cutoff scores, as done in Study 3, by using the ROC analysis method rather than the standard deviation method. To do this, the administration of an independent measure of ASD severity would be required for the study. Future researchers should consider replicating these studies with the inclusion of the ten supplementary items for verbal children only. The current studies were unable to include these items into the analyses due to limited data. Finally, an examination of the incremental validity of adding the *ASD-OC* to its corresponding parent-report measures (i.e., *ASD-DC*, *BISCUIT*) would also provide further evidence supporting the use of this assessment battery when conducting comprehensive diagnostic assessments. In sum, the *ASD-OC* offers a number of valuable qualities for clinicians. This is a tool that proves to be a meaningful and psychometrically sound observation instrument of autistic symptomatology which warrants further use and investigation by researchers today.

References

- Achenbach, T., & Rescorla, L. (2000). *Child behavior checklist*. Burlington: ASEBA.
- ADDMN. (2012). Prevalence of autism spectrum disorders. *MMWR Surveillance Summaries, 14 sites. United States, 61*, 1-19.
- American Psychiatric Association. (1980). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed.)*. Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed. revised)*. Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders 4th ed. text revision (4th ed. text revision ed.)*. Washington, DC: Author.
- American Psychiatric Association. (2011). Autism Spectrum Disorder. DSM-V development. Retrieved February 16, 2012, from <http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=94>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Washington, DC: American Psychiatric Association.
- Arndt, T. L., Stodgell, C. J., & Rodier, P. M. (2005). The teratology of autism. *Int J Dev Neurosci, 23*(2-3), 189-199. doi: 10.1016/j.ijdevneu.2004.11.001
- Asperger, H. (1991). Autistic psychopathy in childhood (U. Frith, Trans.). In U. Frith (Ed.), *Autism and Asperger syndrome*. New York: Cambridge University Press.
- Austin, E. J. (2005). Personality correlates of the broader autism phenotype as assessed by the Autism Spectrum Quotient (AQ). *Personality and Individual Differences, 38*, 451-460.
- Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9-12 months of age. *Journal of Autism and Developmental Disorders, 29*, 213-224.
- Barbaro, J., & Dissanayake, C. (2009). Autism spectrum disorders in infancy and toddlerhood: A review of the evidence on early signs, early identification tools, and early diagnosis. *Journal of Developmental and Behavioral Pediatrics, 30*, 447-459.
- Baron-Cohen, S., Lombardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B., & Knickmeyer, R. (2011). Why are autism spectrum conditions more prevalent in males? *PLoS Biology, 9*, 1-10.
- Barton, M. L., Orinstein, A., Troyb, E., & Fein, D. A. (2013). Early Manifestations of Autism Spectrum Disorders. In J. D. Buxbaum & P. R. Hof (Eds.), *The Neuroscience of Autism Spectrum Disorders*. (pp. 39-53). Oxford, UK: Academic Press.

- Bedell, J. R., & Lennox, S. S. (1997). *Handbook for communication and problem-solving skills training: A cognitive-behavioral approach*. New York, NY: Wiley.
- Beitchman, J. (2006). Language development and its impact on children's psychosocial and emotional development *Encyclopedia of Language and Literacy Development* (pp. 1-7). London, ON: Canadian Language and Literacy Research Network.
- Ben-Itzhak, E., & Zachor, D. A. (2007). The effects of intellectual functioning and autism severity on outcome of early behavioral intervention for children with autism. *Research in Developmental Disabilities, 28*, 287-303.
- Bennett, D. A. (2001). How can I deal with missing data in my study? *Australian and New Zealand Journal of Public Health, 25*(5), 464-469.
- Bertoglio, K., & Hendren, R. L. (2009). New developments in Autism. *Psychiatric Clinics of North America, 32*, 1-14.
- Berument, S. K., Starr, E., Pickles, A., Tomlins, M., Papanikolaou, K., Lord, C., & Rutter, M. (2005). Pre-linguistic Autism Diagnostic Observation Schedule adapted for older individual with severe to profound mental retardation: A pilot study. *Journal of Autism and Developmental Disorders, 35*, 821-829.
- Bettelheim, B. (1967). *The Empty Fortress*. New York, NY: The Free Press.
- Bishop, S. L., Richler, J., & Lord, C. (2006). Association between restricted and repetitive behaviors and nonverbal IQ in children with autism spectrum disorders. *Child Neuropsychology, 12*, 247-267.
- Bleuler, E. (1913). Autistic thinking. *The American Journal of Insanity, 69*, 873-886.
- Bleuler, E. (1919). *Das autistisch-undisziplinierte Denken in der Medizin und seine Ueberwindung*. Berlin: Springer.
- Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders, 30*, 237-243.
- Boomsma, A., van Lang, N. D. J., de Jonge, M. V., de Bildt, A. A., van Engeland, H., & Minderaa, R. B. (2008). A new symptom model for autism cross validated in an independent sample. *Journal of Child Psychology and Psychiatry, 49*, 809-816.
- Boyd, B. A., Odom, S. L., Humphreys, B. P., & Sam, A. M. (2010). Infants and toddlers with Autism Spectrum Disorders: Early identification and early intervention. *Journal of Early Intervention, 32*, 75-98.
- Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders, 37*, 12-24.

- Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2008). The Autism Observation Scale for Infants: Scale development and reliability data. *Journal of Autism and Developmental Disabilities*, 38, 731-738.
- Buitelaar, J. K., & Van der Gaag, R. J. (1998). Diagnostic Rules for Children with PDD-NOS and Multiple Complex Developmental Disorder. *Journal of Child Psychology and Psychiatry*, 39, 911-918.
- Campbell, C. A., Davarya, S., Elsabbagh, M., Madden, L., & Fombonne, E. (2011). Prevalence and the controversy. In J. L. Matson & P. Sturmey (Eds.), *International Handbook of Autism and Pervasive Developmental Disorders* (pp. 25-35). New York: Springer.
- Campbell, W. (2011). Autism Spectrum Disorders. *Berman's Pediatric Decision Making*, 514-517.
- Centers for Disease Control and Prevention. (2009a). Prevalence of Autism Spectrum Disorders - Autism and developmental disabilities monitoring network, United States, 2006. *Surveillance Summaries*, 58(1-24).
- Centers for Disease Control and Prevention. (2009b). Prevalence of autism spectrum disorders – Autism and developmental disabilities monitoring network, United States, 2006. *Surveillance summaries. Morbidity and mortality weekly report*, 58, SS-10.
- Centers for Disease Control and Prevention. (2011). Autism Spectrum Disorders. from <http://www.cdc.gov/ncbddd/autism/index.html>
- Centers for Disease Control and Prevention. (2012). Prevalence of Autism Spectrum Disorders - Autism and Developmental Disabilities Monitoring Network, 14 sites, United States, 2008. *Morbidity and Mortality Weekly Report*. Retrieved March 23, 2014, from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6103a1.htm?s_cid=ss6103a1_w
- Cederlund, M., Hagberg, B., & Gillberg, C. (2010). Asperger syndrome in adolescent and young adult males. Interview, self- and parent assessment of social, emotional, and cognitive problems. *Research in Developmental Disabilities*, 31(2), 287-298. doi: 10.1016/j.ridd.2009.09.006
- Chakrabarti, S. (2001). Pervasive Developmental Disorders in Preschool Children. *JAMA: The Journal of the American Medical Association*, 285(24), 3093-3099. doi: 10.1001/jama.285.24.3093
- Chakrabarti, S., & Fombonne, E. (2005). Pervasive developmental disorders in preschool children: Confirmation of high prevalence. *American Journal of Psychiatry*, 162(1133-1141).
- Chamak, B. (2010). Autism: Overestimation of the genetic origins. *Medecine Sciences*, 26, 659-662.
- Charman, T. (2008). Autism Spectrum Disorders. *Psychiatry*, 7, 331-334.

- Charman, T., Taylor, E., & Drew, A. (2005). Outcome at 7 years of children diagnosed with autism aged 2: Predictive validity of assessments conducted at 2 and 3 years of age and symptom pattern over time. *Journal of Child Psychology and Psychiatry*, 46, 500-513.
- Charman, T. (2005). Autism spectrum disorders. *Psychiatry*, 4(8), 81-84. doi: 10.1383/psyt.2005.4.8.81
- Chen, W. J., Faraone, S. J., Biederman, J., & Tsuang, M. T. (1994). Diagnostic accuracy of the Child Behavior Checklist Scales for Attention-Deficit Hyperactivity Disorder: A receiver-operating characteristics analysis. *Journal of Counseling and Clinical Psychology*, 62, 1017-1025.
- Chowdhury, M., Benson, B. A., & Hillier, A. (2010). Changes in Restricted Repetitive Behaviors with age: A study of high-functioning adults with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders*, 4(2), 210-216. doi: 10.1016/j.rasd.2009.09.006
- Cicchetti, D. V., & Sparrow, S. S. (1981). Developing criteria for establishing interrater reliability of specific items: Applications to assessment of adaptive behavior. *American Journal of Mental Deficiency*, 86, 127-137.
- Cohen, D. J., Caparulo, B. K., Gold, J. R., Waldo, M. C., Shaywitz, B. A., Rutterberg, B. A., & Rimland, B. (1978). Agreement in diagnosis: Clinical assessment and behavior rating scales for pervasively disturbed children. *Journal of the American Academy of Child Psychiatry*, 17, 589-603.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.
- Compton, D. L., Fuchs, D., Fuchs, L., & Bryant, J. D. (2006). Selecting at-risk readers in first grade for early intervention: A two-year longitudinal study of decision rules and procedures. *Journal of Educational Psychology*, 98, 394-409.
- Conway, J. M., & Huffcutt, A. I. (2003). A Review and Evaluation of Exploratory Factor Analysis Practices in Organizational Research. *Organizational Research Methods*, 6, 147-168.
- Costello, A. B., & Osborne, J. W. (2005). Best practices in exploratory factor analysis: Four recommendations for getting the most from your analysis. *Practical Assessment, Research & Evaluation*, 10, 1-9.
- Creak, M. (1961). Schizophrenic syndrome in children: Progress report of a working party. *Cerebral Palsy Bulletin*, 3, 501-504.
- Croen, L. A., Grether, J. K., Hoogstrate, J., & Selvin, S. (2002). The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders*, 32, 207-215.
- Cunningham, A. B., & Schreibman, L. (2008). Stereotypy in autism: The importance of function. *Research in Autism Spectrum Disorders*, 2, 469-479.

- Dawson, G. (2013). Early intensive behavioral intervention appears beneficial for young children with autism spectrum disorders. *The Journal of Pediatrics*, *162*, 1080-1081.
- Dawson, G., Mottron, L., & Gernsbacher, M. A. (2008). Learning in Autism. In J. H. Byrne & H. Roediger (Eds.), *Learning and Memory: A Comprehensive Reference*. New York: Elsevier.
- Dawson, G., & Murias, M. (2009). Autism. *Encyclopedia of Neuroscience*, 779-784.
- de Bilt, A., Sytema, S., Ketelaars, C., Kraijer, D., Volkmar, F. R., & Minderaa, R. (2003). Measuring pervasive developmental disorders in children and adolescents with mental retardation: A comparison of two screening instruments used in a study of the totally mentally retarded population from a designated area. *Journal of Autism and Developmental Disabilities*, *33*, 595-605.
- DiLavore, P. C., Lord, C., & Rutter, M. (1995). The Pre-Linguistic Autism Diagnostic Observation Schedule. *Journal of Autism and Developmental Disorders*, *25*, 355-379.
- Donner, A. (1982). The relative effectiveness of procedures commonly used in multiple regression analysis for dealing with missing values. *The American Statistician*, *36*, 378-381.
- Doss, A. J. (2005). Evidence-based diagnosis: Incorporating diagnostic instruments into clinical practice. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*, 947-952.
- Dube, W. V., MacDonald, R. P. F., Mansfield, R. C., Holcomb, W. L., & Ahern, W. H. (2004). Toward a behavioral analysis of joint attention. *The Behavior Analyst*, *27*, 197-207.
- Duffy, C., & Healy, O. (2011). Spontaneous communication in autism spectrum disorder: a review of topographies and interventions. *Research in Autism Spectrum Disorders*, *5*, 977-983.
- Eisenberg, L., & Kanner, L. (1956). Childhood schizophrenia; symposium, 1955. VI. Early infantile autism, 1943-55. *American Journal of Orthopsychiatry*, *26*, 556-566.
- Elsabbagh, M., & Johnson, M. H. (2007). Infancy and Autism: Progress, prospects, and challenges. *Progress in Brain Research*, *164*, 355-383.
- Evans, M., Stoddart, H., Condon, L., Freeman, E., Grizzell, M., & Mullen, R. (2001). Parent's perspectives on the MMR immunisation: A focus group study. *British Journal of General Practice*, *51*, 904-910.
- Eveloff, H. H. (1960). The autistic child. *Archives of General Psychiatry*, *3*, 66-81.
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, *4*, 272-299.

- Farrington, C. P., Miller, E., & Taylor, B. (2001). MMR and autism: Further evidence against a causal association. *Vaccine, 19*, 3632-3635.
- Fawcett, T. (2006). An introduction to ROC analysis. *Pattern Recognition Letters, 27*, 861-874.
- Ferster, C. B. (1961). Positive reinforcement and behavioral deficits of autistic children. *Child Development, 32*, 437-456.
- Field, A. (2005). *Discovering statistics using SPSS*. London: Sage Publications.
- Floyd, F. J., & Widaman, K. F. (1995). Factor analysis in the development and refinement of clinical assessment instruments. *Psychological Assessment, 7*, 286-299.
- Fluss, R., Faraggi, D., & Reiser, B. (2005). Estimation of the Youden Index and its associated cutoff point. *Biometrical Journal, 47*(4), 458-472.
- Fodstad, J. C., Matson, J. L., Hess, J., & Neal, D. (2009). Social and communication behaviours in infants and toddlers with autism and pervasive developmental disorder-not otherwise specified. *Developmental Neurorehabilitation, 12*, 152-157.
- Folstein, S. E., & Rosen-Sheidley, B. (2001). Genetics of autism: Complex aetiology for a heterogeneous disorder. *Nature Reviews Genetics, 2*, 943-955.
- Folstein, S., & Rutter, M. (1977). Infantile autism: A genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry, 18*, 297-321.
- Fombonne, E. (1991). The use of questionnaires in child psychiatry research: Measuring their performance and choosing an optimal cut-off. *Journal of Child Psychology and Psychiatry, 32*, 677-693.
- Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities, 18*, 281-294.
- Frances, A. (2010). Opening pandora's box: The 19 worst suggestions for DSM5. *Psychiatric Times*.
- Frazier, T. W., Youngstrom, E. A., Kubu, C. S., Sinclair, L., & Rezai, A. (2008). Exploratory and confirmatory factor analysis of the autism diagnostic interview-revised. *Journal of Autism and Developmental Disorders, 38*, 474-480.
- Frazier, T. W., Youngstrom, E. A., Speer, L., Embacher, R., Law, P., Constantino, J., . . . Eng, C. (2011). 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., & Schroth, R. (1984). Behavior assessment of the syndrome of autism: Behavior observation system. *Journal of the American Academy of Child Psychiatry, 23*, 588-594.

- Freeman, B. J., & Schroth, P. C. (1984). The development of the Behavior Observation System. *Behavioral Assessment, 6*, 177-187.
- Fresco, D. M., Mennin, D. S., Heimberg, R. G., & Turk, C. L. (2003). Using the Penn State Worry Questionnaire to identify individuals with generalized anxiety disorder: A receiver operating characteristic analysis. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 283-291.
- Frith, U., & Happe, F. (2005). Autism Spectrum Disorder. *Current Biology, 15*, R786-R790.
- Gallo, D. P. (2010). *Diagnosing Autism Spectrum Disorders: A Lifespan Perspective*. UK: Wiley-Blackwell.
- Gardner, F. (2000). Methodological issues in the direct observation of parent-child interaction: Do observational findings reflect the natural behavior of participants? *Clinical Child and Family Psychology, 3*, 185-198.
- Geschwind, D. H., & Konopka, G. (2009). Neuroscience in the era of functional genomics and systems biology. *Nature, 461*, 908-915.
- Ghaziuddin, M. (2010). Should the DSM-5 drop Asperger syndrome? *Journal of Autism and Developmental Disorders, 40*, 1146-1148.
- Gillberg, C., Ehlers, S., Schaumann, H., Jakobsson, G., Dahlgren, S. O., Lindblom, R., . . . Blinder, E. (1990). Autism under age 3 years: A clinical study of 28 cases referred for autistic symptoms in infancy. *Journal of Child Psychology and Psychiatry, 31*, 921-934.
- Gorsuch, R. L. (1983). *Factor Analysis (2nd ed)*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The Autism Diagnostic Observation Schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders, 37*, 613-627.
- Gray, K. M., & Tonge, B. J. (2001). Are there early identifying features of autism in infants and preschool children? *Journal of Paediatrics and Child Health, 37*, 221-226.
- Green, D. M., & Swets, J. A. (1966). *Signal detection theory and psychophysics*. New York: Wiley.
- Grossman, T., & Johnson, M. H. (2007). The development of the social brain in human infancy. *European Journal of Neuroscience, 25*, 909-919.
- Guadagnoli, E., & Velicer, W. F. (1988). Relation of sample size to the stability of component patterns. *Psychological Bulletin, 103*, 265-275.
- Guinchat, V., Chamak, B., Bonniau, B., Bodeau, N., Perisse, D., Cohen, D., & Danion, A. (2012). Very early signs of autism reported by parents include many concerns not specific to autism criteria. *Research in Autism Spectrum Disorders, 6*, 589-601.

- Guthrie, W., Swingford, L. B., Wetherby, A. M., & Lord, C. (2013). Comparison of DSM-IV and DSM-5 factor structure models for toddlers with autism spectrum disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52, 797-805.
- Hanley, J. A., & McNeil, B. J. (1982). The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology*, 143, 29-36.
- Hansen, R., Ozonoff, S., Krakowiak, P., Angkustsiri, K., Jones, C., Deprey, L. J., . . . Hertz-Picciotto, I. (2008). Regression in Autism: Prevalence and associated factors in the CHARGE study. *Ambulatory Pediatrics*, 8, 25-31.
- Happe, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, 9, 1218-1220.
- Harrington, J. W., Patrick, P. A., Edwards, K. S., & Brand, D. A. (2006). Parental beliefs about autism: Implications for the treating physician. *Autism*, 10, 452-462.
- Hartmann, D. P., Barrios, B. A., & Wood, D. D. (2004). Principles of Behavioral Observation. In S. N. Haynes & E. M. Heiby (Eds.), *Comprehensive Handbook of Psychological Assessment: Behavioral assessment* (Vol. 3, pp. 108-127). Hoboken, NJ: John Wiley & Sons, Inc.
- Haynes, S. N., & O'Brien, W. H. (2000). *Principles and practice of behavioral assessment*. New York: Plenum Publishing Corporation.
- Hebert, J. D., Sharp, I. R., & Gaudiano, B. A. (2002). Separating fact from fiction in the etiology and treatment of autism: A scientific review of the evidence. *The Scientific Review of Mental Health Practice*, 1, 23-43.
- Hingtgen, J. H., & Bryson, C. Q. (1972). Recent developments in the study of early childhood psychoses: Infantile autism, childhood schizophrenia, and related disorders. *Schizophrenia Bulletin*, 1, 8-54.
- Hornig, M., Briese, T., Buie, T., Bauman, M. L., Lauwers, G., Siemetzki, U., . . . Lipkin, W. I. (2008). Lack of association between measles virus vaccine and autism with enteropathy: a case-control study. *PLoS One*, 3(9), e3140. doi: 10.1371/journal.pone.0003140
- Horovitz, M., & Matson, J. L. (2014). The Baby and Infant Screen for Children with aUtism Traits-Part 1: Age-based scoring procedures. *Journal of Developmental and Physical Disabilities*, 26, 1-22.
- Hoshino, Y., Kaneko, M., Yashima, Y., Kumashiro, H., Volkmar, F. R., & Cohen, D. J. (1987). Clinical features of autistic children with setback course in their infancy. *Japanese Journal of Psychiatry and Neurology*, 41, 237-246.
- Howlin, P. (2006). Autism spectrum disorders. *Psychiatry*, 5, 320-324.

- Hurst, R. M., Mitchell, J. T., Kimbrel, N. A., Kwapil, T. K., & Nelson-Gray, R. O. (2007). Examination of the reliability and factor structure of the Autism Spectrum Quotient (AQ) in a non-clinical sample. *Personality and Individual Differences, 43*, 1938-1949.
- Hutcheson, G., & Sofroniou, N. (1999). *The multivariate social scientist*. London: Sage.
- Inglese, M. D., & Elder, J. H. (2009a). Caring for children with Autism Spectrum Disorder, part I: Prevalence, etiology, and core features. *Journal of Pediatric Nursing, 24*(41-48).
- Inglese, M. D., & Elder, J. H. (2009b). Caring for children with Autism Spectrum Disorder, part II: Screening, diagnosis, and management. *Journal of Pediatric Nursing, 24*(49-59).
- Johnson, C. P., & Myers, S. M. (2008). Autism spectrum disorders. In M. L. Wolraich, D. D. Drotar, P. H. Dworkin & E. C. Perrin (Eds.), *Developmental-Behavioral Pediatrics: Evidence and Practice* (pp. 519-577). Philadelphia, PA: Mosby, Inc.
- Johnson, M. H., Grossman, T., & Farroni, T. (2010). The social cognitive neuroscience of infancy: Illuminating the early development of social brain functions. In R. Kail (Ed.), *Advances in Child Development and Behavior* (Vol. 36).
- Kaiser, H. F. (1974). An index of factorial simplicity. *Psychometrika, 39*, 31-36.
- Kaland, N., Mortensen, E. L., & Smith, L. (2011). Social communication impairments in children and adolescents with Asperger syndrome: Slow response time and the impact of prompting. *Research in Autism Spectrum Disorders, 5*, 1129-1137.
- 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.
- Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioral Science, 4*, 412-420.
- Kanner, L., & Eisenberg, L. (1956). Early infantile autism, 1943-1955. *American Journal of Orthopsychiatry, 26*, 556-566.
- Kanner, L., & Lesser, L. I. (1958). Early infantile autism. *Pediatric Clinics of North America, 5*, 711-730.
- Kaye, J. A., del Melero-Montes, M., & Jick, H. (2001). Mumps, measles, and rubella vaccine and the incidence of autism recorded by general practitioners: A time trend analysis. *British Medical Journal, 322*, 460-463.
- Kazdin, A. (1982). Observer effects: Reactivity of direct observation. *New direction for methodology of social and behavioral science, 14*, 5-19.
- Kendall, P. C., & Grove, W. M. (1988). Normative comparisons in therapy outcome. *Behavioral Assessment, 10*, 147-158.

- Kim, S. H., & Lord, C. (2013). The behavioral manifestations of Autism Spectrum Disorders. *The Neuroscience of Autism Spectrum Disorders*.
- Kim, S., & Lord, C. (2012). New Autism Diagnostic Interview-Revised algorithms for toddlers and young preschoolers from 12 to 47 months of age. *Journal of Autism and Developmental Disabilities, 42*, 82-93.
- Kim, S. H., & Lord, C. (2013). The Behavioral Manifestations of Autism Spectrum Disorders. 25-37. doi: 10.1016/b978-0-12-391924-3.00002-8
- Kogan, M. D., Blumberg, S. J., Schieve, L. A., Boyle, C. A., Perrin, J. M., Ghandour, R. M., ... & van Dyck, P. C. (2009). Prevalence of parent-reported diagnosis of autism spectrum disorder among children in the US, 2007. *Pediatrics, 124*, 1395-1403.
- Kozlowski, A. M., Matson, J. L., Horovitz, M., Worley, J. A., & Neal, D. (2011). Parents' first concerns of their child's development in toddlers with autism spectrum disorders. *Developmental Neurorehabilitation, 14*, 72-78.
- Krzanowski, W. J., & Hand, D. J. (2009). *ROC Curves for Continuous Data*. Boca Raton, FL: CRC Press.
- Kumar, R., & Indrayan, A. (2011). Receiver Operating Characteristic (ROC) curve for medical researchers. *Indian Pediatrics, 48*, 277-287.
- Kumpunen, S., Shipway, L., Taylor, R. M., Aldiss, S., & Gibson, F. (2012). Practical approaches to seeking assent from children. *Nurse Researcher, 19*, 23-27.
- L., Matson J., & Boisjoli, J. A. (2007). Differential diagnosis of PDDNOS in children. *Research in Autism Spectrum Disorders, 1*, 75-84.
- Lambert, V., & Glacken, M. (2011). Engaging with children in research: Theoretical and practical implications of negotiating informed consent/assent. *Nursing Ethics, 18*, 781-801.
- Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: a prospective study. *J Child Psychol Psychiatry, 47*(6), 629-638. doi: 10.1111/j.1469-7610.2006.01531.x
- Lecavalier, L., Aman, M., Scahill, L., McDougle, C. J., McCracken, J. T., Vitiello, B., . . . Kau, A. S. M. (2006). Validity of the autism diagnostic interview-revised. *American Journal on Mental Retardation, 111*, 199-215.
- Lecavalier, L., Gadaw, K. D., DeVincent, C. J., Houts, C., & Edwards, M. C. (2009). Deconstructing the PDD clinical phenotype: Internal validity of the DSM-IV. *The Journal of Child Psychology and Psychiatry, 50*, 1246-1254.

- Leonard, H., Dixon, G., Whitehouse, A. J. O., Bourke, J., Aiberti, K., Nassar, N., . . . Glasson, E. J. (2010). Unpacking the complex nature of the autism epidemic. *Research in Autism Spectrum Disorders, 4*, 548-554.
- Lewis, F. M., Murdoch, B. E., & Woodyatt, G. C. (2007). Linguistic abilities in children with autism spectrum disorder. *Research in Autism Spectrum Disorders, 1*, 85-100.
- Lewis, M. H., & Baumeister, A. A. (1982). Stereotyped mannerisms in mentally retarded persons: Animal models and theoretical analyses. In N. R. Ellis (Ed.), *International review of research in mental retardation* (pp. 123-161). New York: Academic Press.
- Lipinski, D. P., & Nelson, R. O. (1974). Problems in the use of naturalistic observation as a means of behavioral assessment. *Behavior therapy, 5*, 341-351.
- Llaneza, D. C., DeLuke, S. V., Batista, M., Crawley, J. N., Christodulu, K. V., & Frye, C. A. (2010). Communication, interventions, and scientific advances in autism: a commentary. *Physiol Behav, 100*(3), 268-276. doi: 10.1016/j.physbeh.2010.01.003
- Lockyer, L., & Rutter, M. (1968). A five to fifteen-year follow-up study of infantile psychosis. *The British Journal of Psychiatry, 115*, 865-882.
- Lord, C. (1995). Follow-up of two-year olds referred for possible autism. *Journal of Child Psychology and Psychiatry, 36*, 1365-1685.
- Lord, C., & Corsello, C. (2005). Diagnostic Instruments in Autistic Spectrum Disorders. In F. R. Volkmar, R. Paul, A. Klin & D. Cohen (Eds.), *Handbook of Autism and Pervasive Developmental Disorders* (Vol. 2).
- Lord, C., & Luyster, R. (2006). Early diagnosis of children with Autism Spectrum Disorders. *Clinical Neuroscience Research, 6*, 189-194.
- Lord, C., Luyster, R., Gotham, K., & Guthrie, D. (2012). *Autism diagnostic observation schedule, second edition (ADOS-2) manual (Part II): Toddler module*. Torrance, CA: Western Psychological Services.
- Lord, C., & Risi, S. (1998). Frameworks and methods in diagnosing autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 90-96.
- Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., . . . Rutter, M. (2000). The Autism Diagnostic Observation Schedule-Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30*, 205-223.
- Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism Diagnostic Observation Schedule-Generic*. Los Angeles: Western Psychological Services.

- Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. (2012). *Autism diagnostic observation schedule, second edition*. Torrance, CA: Western Psychological Services.
- Lord, C., Rutter, M., Goode, S., Heemsbergen, S., Jordan, J., Mawhood, L., & Schopler, E. (1989). Autism diagnostic observation schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders*, *19*, 185-212.
- Lovaas, O. I., & Smith, T. (1989). A comprehensive behavioral theory of autistic children: Paradigm for research and treatment. *Journal of Behavior Therapy and Experimental Psychiatry*, *29*, 17-29.
- Luyster, R., Gotham, K., Guthrie, D., Coffing, M., Petrak, R., Pierce, K., . . . Lord, C. (2009). The Autism Diagnostic Observation Schedule – Toddler Module: A new module of a standardized diagnostic measure for autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *39*, 1305-1320.
- MacDonald, R., Green, G., Mansfield, R., Geckeler, A., Gardenier, N., Anderson, J., . . . Sanchez, J. (2007). Stereotypy in young children with autism and typically developing children. *Res Dev Disabil*, *28*(3), 266-277. doi: 10.1016/j.ridd.2006.01.004
- Madsen, K. M., Lauritsen, M. B., Pedersen, C. B., Thorsen, P., Plesner, A. M., Anderson, P. H., & Mortensen, P. B. (2003). Thimerosal and the occurrence of autism: Negative ecological evidence from Danish population-based data. *Pediatrics*, *112*, 604-606.
- Maestro, S., Muratori, F., Cavallaro, M. C., Pei, F., Stern, D., Golse, B., & Palacio-Espasa, F. (2002). Attentional skills during the first 6 months of age in autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 1239-1245.
- Manning-Courtney, P., Brown, J., Molloy, C. A., Reinhold, J., Murray, D., Sorensen-Burnworth, R., . . . Kent, B. (2003). Diagnosis and treatment of Autism Spectrum Disorders. *Current Problems in Pediatric and Adolescent Health Care*, *23*, 283-304.
- Manning-Courtney, P., Murray, D. S., Currans, K., Johnson, H., Bing, N., Kroeger-Geoppinger, K., . . . Messerschmidt, T. (2013). Autism Spectrum Disorders. *Current Problems in Pediatric and Adolescent Health Care*, *43*, 2-11.
- Martinez-Cambor, P. (2013). Area under the ROC curve comparison in the presence of missing data. *Journal of the Korean Statistical Society*, *42*(4), 431-442.
- Martinez-Pedraza, F. L., & Carter, A. S. (2009). Autism spectrum disorders in young children. *Child and Adolescent Psychiatric Clinics in North America*, *18*, 645-663.
- Matson, J. L. (2007a). Current status differential diagnosis for children with autism spectrum disorders. *Research in Developmental Disabilities*, *28*, 109-118.

- Matson, J. L. (2007b). Determining treatment outcome in early intervention programs for autism spectrum disorders: A critical analysis of measurement issues in learning based interventions. *Research in Developmental Disabilities, 28*, 207-218.
- Matson, J. L., Belva, B. C., Horovitz, M., Kozlowski, A. M., & Bamburg, J. W. (2012). Comparing symptoms of autism spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR diagnostic criteria and the proposed DSM-5 diagnostic criteria. *Journal of Developmental and Physical Disabilities, 24*, 403-414.
- Matson, J. L., Boisjoli, J. A., Gonzalez, M. L., Smith, K. R., & Wilkins, J. (2007). Norms and cut offs 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., Hess, J., & Wilkins, J. (2011). Comorbid psychopathology factor structure on the Baby and Infant Screen for Children with aUtism Traits-Part 2 (BISCUIT-Part 2). *Research in Autism Spectrum Disorders, 5*, 426-432.
- Matson, J. L., Boisjoli, J. A., Rojahn, J., & Hess, J. (2009). A factor analysis of challenging behaviors assessed with the Baby and Infant Screen for Children with aUtism Traits (BISCUIT-Part 3). *Research in Autism Spectrum Disorders, 3*, 714-722.
- Matson, J. L., Dempsey, T., & Fodstad, J. C. (2009). Stereotypies and repetitive/restrictive behaviours in infants with autism and pervasive developmental disorder. *Developmental Neurorehabilitation, 12*, 122-127.
- Matson, J. L., Dempsey, T., & LoVullo, S. V. (2009). Characteristics of social skills for adults with intellectual disability, autism, and PDD-NOS. *Research in Autism Spectrum Disorders, 3*, 207-213.
- Matson, J. L., Dempsey, T., Lovullo, S. V., Fodstad, J. C., Knight, C., Sevin, J. A., & Sharp, B. (2012). The moderating effects of intellectual development on core symptoms of autism and PDD-NOS in toddlers and infants. *Res Dev Disabil, 34*(1), 573-578. doi: 10.1016/j.ridd.2012.03.031
- Matson, J. L., Gonzalez, M., Wilkins, J., & Rivet, T. T. (2008). Reliability of the Autism Spectrum Disorder-Diagnostic for Children (ASD-DC). *Research in Autism Spectrum Disorders, 2*, 533-545.
- Matson, J. L., Hattier, M., & Williams, L. (2012). How does relaxing the algorithm for autism affect DSM-V prevalence rates? *Journal of Autism and Developmental Disorders, 42*, 1549-1556.
- Matson, J. L., & Konst, M. J. (2013). What is the evidence for long term effects of early autism interventions? *Research in Autism Spectrum Disorders, 7*, 475-479.
- Matson, J. L., & Kozlowski, A. M. (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., & Neal, D. (2010). Differentiating communication disorders and autism in children. *Research in Autism Spectrum Disorders, 4*, 626-632.
- Matson, J. L., & Nebel-Schwalm, M. (2007). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities, 28*, 567-579.
- 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. (2007). A critical review of assessment targets and methods for social skills excesses and deficits for children with autism spectrum disorders. *Research in Autism and Developmental Disability, 23*, 137-145.
- Matson, J. L., Wilkins, J., & Gonzalez, M. (2008). Early identification and diagnosis in Autism Spectrum Disorders in young children and infants: How early is too early? *Research in Autism Spectrum Disorders, 2*, 75-84.
- Matson, J.L. (2008). *Autism spectrum disorders: Evidence based assessment and intervention across the lifespan*. London: Academic Press.
- Matson, J.L. (2009). *Practitioner's guide to applied behavior analysis for children with autism spectrum disorders*. New York: Springer.
- Matson, J.L., & Minshawi, N.F. (2006). *Early intervention for autism spectrum disorders: A critical analysis*. Oxford, England: Elsevier Science, Inc.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of American Academy of Child & Adolescent Psychiatry, 51*, 368-383.
- MedCalc. (2011). MedCalc Software. Mariakerke, Belgium.
- Memari, A. H., Ziaee, V., Beygi, S., Moshayedi, P., & Mirfazeli, F. S. (2012). Overuse of psychotropic medications among children and adolescents with autism spectrum disorders: perspective from a developing country. *Res Dev Disabil, 33*(2), 563-569. doi: 10.1016/j.ridd.2011.10.001
- Metz, C. E. (1978). Basic principles of ROC analysis. *Seminars in Nuclear Medicine, 8*, 283-298.
- Miller, V. A., Drotar, D., & Kodish, E. (2004). Children's competence for assent and consent: A review of empirical findings. *Ethics & Behavior, 14*, 255-295.

- Molloy, C. A., Murray, D. S., Akers, R., Mitchell, T., & Manning-Courtney, P. (2011). Use of the Autism Diagnostic Observation Schedule (ADOS) in a clinical setting. *Autism, 15*, 143-162.
- Montanelli, R. G., & Humphreys, L. G. (1976). Latent roots of random data correlation matrices with squared multiple correlations on the diagonal: A Monte Carlo study. *Psychometrika, 41*, 341-347.
- Moore, V., & Goodson, S. (2003). How well does early diagnosis of autism stand the test of time? Follow-up study of children assessed for autism at age 2 and development of early diagnostic service. *Autism, 7*, 47-63.
- Mossman, D. (1994). Assessing predictions of violence: Being accurate about accuracy. *Journal of Consulting and Clinical Psychology, 62*, 783-792.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Neal, D., Matson, J. L., & Belva, B. C. (2012). Discriminant analysis of the autism spectrum disorder observation for children. *Developmental Neurorehabilitation, 15*, 267-273.
- Neal, D., Matson, J. L., & Belva, B. C. (2013). An examination of the reliability of a new observation measure for autism spectrum disorders: The Autism Spectrum Disorder Observation for Children. *Research in Autism Spectrum Disorders, 7*, 29-34.
- Neal, D., Matson, J. L., & Hattier, M. (2012). A comparison of diagnostic criteria on the Autism Spectrum Disorder Observation for Children (ASD-OC). *Developmental Neurorehabilitation, 15*, 329-335.
- Neal, D., Matson, J. L., & Hattier, M. A. (2014). Validity of the Autism Spectrum Disorder Observation for Children (ASD-OC). *Journal of Mental Health Research in Intellectual Disabilities, 7*, 14-33.
- 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.
- Nunnally, J. C. (1978). *Psychometric Theory* (2nd ed.). New York: McGraw-Hill.
- O'Connor, B. P. (2000). SPSS and SAS programs for determining the number of components using parallel analysis and Velicer's MAP test. *Behavior Research Methods, Instrumentation, and Computers, 32*, 396-402.
- Offit, Paul A., & Coffin, Susan E. (2003). Communicating science to the public: MMR vaccine and autism. *Vaccine, 22*(1), 1-6. doi: 10.1016/s0264-410x(03)00532-2

- Osterling, J. A., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home video tapes. *Journal of Autism and Developmental Disorders*, 24, 247-257.
- Ozonoff, S., Ana-Maria, I., Bagulo, F., Cook, I. C., Hill, M. M., Hutman, T., . . . Young, G. S. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, 49, 256-266.
- Ozonoff, S., Iosif, A., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., . . . Young, G. S. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49, 256-266.
- Pennington, B. F. (2009). *Diagnosing learning disorders: A neuropsychological framework*. New York: Guilford Press.
- Perkins, N. J., & Schisterman, E. F. (2005). The inconsistency of 'optimal' cut points obtained using two criteria based on the receiver operating characteristic curve. *American Journal of Epidemiology*, 163, 670-675.
- Peters-Scheffer, N., Didden, R., Korzilius, H., & Sturmey, P. (2011). A meta-analytic study on the effectiveness of comprehensive ABA-based early intervention programs for children with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders*, 5, 60-69.
- Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: A retrospective study of high-IQ adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 523-529.
- Pry, R., Peterson, A. F., & Baghdadli, A. (2009). Developmental changes of expressive language and interactive competencies in children with autism. *Research in Autism Spectrum Disorders*, 3, 98-112.
- Rapp, J. T., & Vollmer, T. R. (2005). Stereotypy I: a review of behavioral assessment and treatment. *Res Dev Disabil*, 26(6), 527-547. doi: 10.1016/j.ridd.2004.11.005
- Reichow, B., Barton, E. E., Boyd, B. A., & Hume, K. (2012). Early intensive behavioral intervention (EIBI) for young children with autism spectrum disorders (ASD). *Cochrane Database Systematic Review*, 10.
- Rice, C. (2007). Prevalence of autism spectrum disorders-autism and developmental disabilities monitoring network, 14 sites, United States, 2002. *Morbidity and Mortality Weekly Report*, 56, 12-28.
- Rice, C., Nicholas, J., Baio, J., Pettygrove, S., Lee, L., Van Naarden Braun, K., . . . Yeargin-Allsopp, M. (2010). Changes in autism spectrum disorder prevalence in 4 areas of the United States. *Disability and Health Journal*, 3, 186-201.
- Rice, M. E., & Harris, G. T. (1995). Violent Recidivism: Assessing Predictive Validity. *Journal of Consulting and Clinical Psychology*, 63(5), 737-748.

- Rimland, B. (1964). *Infantile autism: the syndrome and its implications for a neural theory of behavior*: Prentice-Hall.
- Risi, S., Lord, C., Gotham, K., Corsello, C., Chrysler, C., Szatmari, P., . . . Pickles, A. (2006). Combining information from multiple sources in the diagnosis of autism spectrum disorders. *J Am Acad Child Adolesc Psychiatry*, 45(9), 1094-1103. doi: 10.1097/01.chi.0000227880.42780.0e
- Ritvo, E. R. (1978). National society for autistic children definition of the syndrome autism. *Journal of Autism and Developmental Disorders*, 8, 162-167.
- Robertson, J. M., Tanguay, P. E., L'Ecuyer, S., Sims, A., & Waltrip, C. (1999). Domains on social communication handicap in autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38, 738-745.
- Rogers, S. (2000). Diagnosis of autism before the age of 3. *International Review of Research in Mental Retardation*, 23, 1-31.
- Romanczyk, R. G., Kent, R. N., Diament, C., & O'Leary, K. D. (1973). Measuring the reliability of observational data: A reactive process. *Journal of Applied Behavior Analysis*, 6, 175-184.
- Rosenberg, R. E., Law, J., Yenokyan, G., McGready, J., Kaufmann, W. E., & Law, P. A. (2009). Characteristics and concordance of autism spectrum disorders among 277 twin pairs. *Archives of Pediatrics and Adolescent Medicine*, 163, 907-914.
- Ruttenberg, B. A., Dratman, M. L., Franknoi, J., & Wenar, C. (1966). An instrument for evaluating autistic children. *Journal of the American Academy of Child Psychiatry*, 5, 453-578.
- Ruttenberg, B. A., Kalish, B. I., Wenar, C., & Wolf, E. G. (1977). *The behavior rating instrument for autistic and other atypical children*. Philadelphia: Developmental Center for Autistic Children.
- Ruttenberg, B. A., Wolf-Schein, E. G., & Wenar, C. (1991). *The behavior rating instrument for autistic and other atypical children: Instruction manual*. Wood Dale, IL: Stoelting.
- Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry*, 9, 1-25.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2, 315-337.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Developmental Disorders*, 8, 139-161.
- Rutter, M. (2000). Genetic studies of autism: From the 1970s into the millennium. *Journal of Abnormal Child Psychiatry*, 28, 3-14.

- Rutter, M. (2002). [Address to the Second Annual International Meeting for Autism Research].
- Rutter, M. (2005). Incidence of autism spectrum disorders: Changes over time and their meaning. *Acata Pediatrica*, *94*, 2-15.
- Rutter, M., & Bartak, L. (1971). Causes of infantile autism: Some considerations from recent research. *Journal of Autism and Childhood Schizophrenia*, *1*, 20-32.
- Rutter, M., & Lockyer, L. (1967). A five to fifteen-year follow-up study of infantile psychosis I - Description of sample. *The British Journal of Psychiatry*, *113*, 1169-1182.
- Schopler, E., Reichler, R. J., DeVellis, R., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood Autism Rating Scale (CARS). *Journal of Autism and Developmental Disorders*, *10*, 91-103.
- Schopler, E., Reichler, R. J., & Renner, B. R. (1988). *The Childhood Autism Rating Scale (CARS), Revised*. Los Angeles, CA: Western Psychological Services.
- Schreibman, L. (2005). *The Science and Fiction of Autism*. Cambridge, MA: Harvard University Press.
- Sevin, B. M., Knight, C. L., & Braud, S. A. (2007). Autism and Pervasive Developmental Disorders. *34*, 163-196. doi: 10.1016/s0074-7750(07)34005-6
- Shorter, E. (1997). *A History of Psychiatry: From the Era of the Asylum to the Age of Prozac*. New York: John Wiley & Sons, Inc.
- Sigafoos, J. (2000). Communication development and aberrant behavior in children with developmental disabilities. *Educational and Training in Mental Retardation and Developmental Disabilities*, *35*, 168-176.
- Smith, K. R., & Matson, J. L. (2010). Social skills: differences among adults with intellectual disabilities, co-morbid autism spectrum disorders and epilepsy. *Res Dev Disabil*, *31*(6), 1366-1372. doi: 10.1016/j.ridd.2010.07.002
- Snow, A., Lecavalier, L., & Houts, C. (2009). Structure of the autism diagnostic interview-revised: Diagnostic and phenotypic implications. *Journal of Child Psychology and Psychiatry*, *50*, 734-742.
- Soares, N. S., & Patel, D. R. (2012). Office screening and early identification of children with autism. *Pediatric Clinics of North America*, *59*, 89-102.
- Stone, W., Coonrod, E. E., & Ousley, O. Y. (2000). Brief report: Screening tool for autism in 2-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders*, *30*, 607-612.

- Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders*, 34, 691-701.
- Stone, W., & Ousley, O. Y. (1997). *STAT Manual: Screening Tool for Autism in Two-Year-Olds*: Unpublished manuscript, Vanderbilt University.
- Streiner, D. L. (1994). Figuring out factors: The use and misuse of factor analysis. *Canadian Journal of Psychiatry*, 39, 135-140.
- Swets, J. A. (1979). ROC analysis appliet to the evaluation of medical imaging techniques. *Invest Radiology*, 14, 109-121.
- Swets, J. A. (1988). Measuring the accuracy of diagnostic systems. *Science*, 240, 1285-1293.
- Swets, J. A., Dawes, R. M., & Monahan, J. (2000). Better decisions through science. *Scientific American*, 283, 82-87.
- Swets, J. A., & Pickett, R. M. (1982). *Evaluation of Diagnostic Systems: Methods from Signal Detection Theory*. New York: Academic Press.
- Swets, J. A., Tanner, W. P., & Birdsall, T. G. (1964). Decision process in perception. *Psychological Review*, 68, 301-340.
- Szatmari, P. (2000). The classification of autism, Asperger's syndrome, and pervasive developmental disorder. *Canadian Journal of Psychiatry*, 45, 731-738.
- Tadevosyan-Leyfer, O., Dowd, M., Mankoski, R., Winklosky, M. A., Putnam, S., McGrath, L., . . . Folstein, S. (2003). A principal components analysis of the autism diagnostic interview-revised. *Journal of the American Academy of Child & Adolescent Psychiatry*, 42, 864-872.
- Tager-Flusberg, H. (2001). Understanding the language and communicative impairments in autism. *International Review of Research in Mental Retardation*, 23, 185-205.
- Tager-Flusberg, H. (2002). Language acquisition. *Encyclopedia of the Human Brain*, 2, 617-629.
- Tager-Flusberg, H. (2010). The origins of social impairments in autism spectrum disorders: Studies of infants at risk. *Neural Networks*, 23, 1072-1076.
- Tager-Flusberg, H., & Caronna, E. (2007). Language Disorders: Autism and other Pervasive Developmental Disorders. *Pediatric Clinics of North America*, 54, 469-481.
- Tanner, W. P., & Swets, J. A. (1954). A decision-making theory of visual detection. *Psychological Review*, 61, 401-409.

- Taylor, B., Miller, E., Farrington, C. P., Petropoulos, M., Favot-Mayaud, I., Li, J., & Waight, P. (1999). Autism and measles, mumps, and rubella vaccine: No epidemiological evidence for a causal association. *The Lancet*, *353*, 2026-2029.
- Thelen, E. (1979). Rhythmical stereotypies in normal human infants. *Animal Behavior*, *27*, 699-715.
- Thomas, J. (2010). Paranoia strikes deep: MMR vaccine and autism. *Psychiatric Times*, *27*, 1-6.
- Thurm, A., Bishop, S., & Shumway, S. (2011). Developmental issues and milestones. In J. L. Matson & P. Sturmey (Eds.), *International Handbook of Autism and Pervasive Developmental Disorders*. New York: Springer.
- Tidmarsh, L., & Volkmar, F. R. (2003). Diagnosis and epidemiology of autism spectrum disorders. *The Canadian Journal of Psychiatry*, *48*, 517-525.
- Turner, M. (1999). Repetitive behaviour in autism: A review of psychological research. *Journal of Child Psychology and Psychiatry*, *40*, 839-849.
- Vaughan, C. A. (2011). Test Review: Childhood Autism Rating Scale (2nd ed.). *Journal of Psychoeducational Assessment*, *25*, 489-493.
- Volkmar, F. R. (1996). Childhood and adolescent psychosis: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry*, *35*, 843-851.
- Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin & D. Cohen (Eds.), *Handbook of Autism and Pervasive Developmental Disorders 3rd Edition* (pp. 5-41). Hoboken, NJ: John Wiley & Sons, Inc.
- Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., . . . Kline, W. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry*, *151*, 1361-1367.
- Volkmar, F. R., & Pauls, D. (2003). Autism. *The Lancet*, *362*, 1133 – 1142.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., . . . Walker-Smith, J. A. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, *351*, 637-641.
- Walker, D. R., Thompson, A., Zwaigenbaum, L., Goldberg, J., Bryson, S. E., Mahoney, W. J., . . . Szatmari, P. (2004). Specifying PDD-NOS: A comparison of PDD-NOS, Asperger syndrome, and autism. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*, 172-180.
- Waterhouse, L., Wing, L., Spitzer, R., & Siegel, B. (1989). Pervasive developmental disorders: From DSM-III to DSM-III-R. *Journal of Autism and Developmental Disorders*, *22*, 525-549.

- Waterhouse, Lynn. (2013). Increasing Prevalence and the Problem of Diagnosis. 345-398. doi: 10.1016/b978-0-12-415961-7.00007-1
- Wenar, C., & Ruttenberg, B. A. (1976). The use of BRIAAC for evaluating therapeutic effectiveness. *Journal of Autism and Childhood Schizophrenia*, 6, 175-191.
- Werner, E., Dawson, G., Osterling, J. A., & Dinno, N. (2000). Brief report: Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. *Journal of Autism and Developmental Disorders*, 30, 157-162.
- Wiggins, L. D., Baio, J., & Rice, C. (2006). Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. *Developmental and Behavioral Pediatrics*, 27, 79-87.
- 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.
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews*, 8, 151-161.
- Worley, J. A., & Matson, J. L. (2012). Comparing symptoms of autism spectrum disorders using the current DSM-IV-TR diagnostic criteria and the proposed DSM-V diagnostic criteria. *Research in Autism Spectrum Disorders*, 6, 965-970.
- World Health Organization. (1992). *International classification of diseases* (10th ed.). Geneva, Switzerland: Author.
- World Health Organization. (2001). Recent concerns regarding MMR vaccine. <http://www.who.int/inf-pr-2001/en/state2001-02.html>
- Wormald, A. M. (2011). *Autism Spectrum Disorder: Examining current diagnosis strategies and assessment tools*. (Master of Arts in Education), Biola University.
- Youden, W. J. (1950). An index for rating diagnostic test. *Cancer*, 3, 32-35.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., . . . Yirmiya, N. (2009a). Clinical assessment and management of toddlers with suspected autism spectrum disorder: Infants from studies of high-risk infants. *Pediatrics*, 123, 1383-1391.
- Zwaigenbaum, L., Bryson, S., Lord, C., Rogers, S., Carter, A., Carver, L., . . . Yirmiya, N. (2009b). Clinical assessment and management of toddlers with suspected autism spectrum disorder: insights from studies of high-risk infants. *Pediatrics*, 123(5), 1383-1391. doi: 10.1542/peds.2008-1606
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, 23, 143-152.

Zwaigenbaum, L., Thurm, A., Stone, W., Baranek, G., Bryson, S., Iverson, J., . . . 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

IRB Approval

Project Report and Continuation Application

(Complete and return to IRB, 131 David Boyd Hall, Direct questions go to IRB Chairman Robert Mathews 578-8692.)



Institutional Review Board
Dr. Robert Mathews, Chair
131 David Boyd Hall
Baton Rouge, LA 70803
P: 225.578.8692
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IRB#: 2609 Current Approval Expires On: 09/14/2012
Review Type: Expedited Risk Factor: small
PI: Johnny Matson Dept: Psychology Phone: 225-578-8745
Student/Co-Investigator:
Project Title: Developing the Autism Spectrum Disorder
Number of Subjects Authorized: 2000

Please read the entire application. Missing information will delay approval
IRB Security of Data Agreement: <http://research.lsu.edu/files/item26774.pdf>

I. PROJECT FUNDED BY: N/A LSU Proposal #:

II. PROJECT STATUS: Check the appropriate blank(s) and complete the following:

- 1. Active, subject enrollment continuing; # subjects enrolled: 676
- 2. Active, subject enrollment complete; # subjects enrolled: _____
- 3. Active, subject enrollment complete; work with subjects continues.
- 4. Active, work with subjects complete; data analysis in progress.
- 5. Project start postponed; date: _____
- 7. Project cancelled: no human subjects used.
- 6. Project complete; end date: _____

III. PROTOCOL: (Check one).

- Protocol continues as previously approved
- Changes are requested*
--List (on separate sheet) any changes to approved protocol.

IV. UNEXPECTED PROBLEMS: (did anything occur that increased risks to participants):

- State number of events since study inception: 0 since last report: 0
- If such events occurred, describe them and how they affect risks in your study, in an attached report
- Have there been any previously unreported events? Yes/No: N

V. CONSENT FORM AND RISK/BENEFIT RATIO:

- Do new knowledge or adverse events change the risk/benefit ratio? Yes/No: N
- Is a corresponding change in the consent form needed? Yes/No: N

VI. ATTACH A BRIEF, FACTUAL SUMMARY of project progress/results to show continued participation of subjects is justified; or to provide a final report on project findings.

VII. ATTACH CURRENT CONSENT FORM (only if subject enrollment is continuing); and check the appropriate blank;

- 1. Form is unchanged since last approved
- 2. Approval of revision requested herewith: (Identify changes)

Signature of Principle Investigator: *Johnny P. Matson*

Date: *Sept. 4, 2012*

IRB Action: Continuation approved; Approval Expires: *9, 9, 13*
 Disapproved
 File Closed
Signed: *Robert Mathews* Date: *9/10/12*

Print Form

Vita

Megan A. Hattier was born in New Orleans, Louisiana, in 1987. Following the completion of her Bachelor of Science degree in psychology from Louisiana State University in 2009, she enrolled in Louisiana State University's Clinical Psychology Doctoral Program under the supervision of Dr. Johnny L. Matson. Her clinical and research interests include the assessment and treatment of autism spectrum disorders and other developmental disabilities among children and adults. She completed her master's thesis entitled *An Examination of the Relationship between Communication and Socialization Deficits in Infants and Toddlers with Autism and Pervasive Developmental Disorder-Not Otherwise Specified* and received her Master of Arts degree in 2011. She has completed her pre-doctoral internship at Kennedy Krieger Institute, Johns Hopkins School of Medicine. While training at Kennedy Krieger Institute, she further developed her clinical skills and knowledge of applied behavior analysis while working at the Neurobehavioral Outpatient Program and the Pediatric Feeding Disorders Program.