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Construct Validity of the Autism Spectrum Disorders-Child Version (ASD-C) and the Child Behavior Checklist (CBCL)

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CONSTRUCT VALIDITY OF THE AUTISM SPECTRUM DISORDERS-CHILD VERSION
(ASD-C) AND THE CHILD BEHAVIOR CHECKLIST (CBCL)

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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TABLE OF CONTENTS

LIST OF TABLES	iv
LIST OF FIGURES	v
ABSTRACT.....	vi
CHAPTER 1. INTRODUCTION	1
1.1 Early Definitions of Autism.....	1
1.2 Evolution of the Current Definition of ASD.....	7
1.2.1 Asperger’s Disorder	11
1.2.2 Childhood Disintegrative Disorder (CDD).....	15
1.2.3 Rett’s Syndrome.....	16
1.2.4 Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS).....	18
1.3 Current Definitions of ASD	20
1.4 Prevalence of ASD.....	24
1.5 Current Definitions of ASD	27
1.5.1 Learning Theories	28
1.5.2 Genetics.....	29
1.5.3 Neurobiology	31
1.5.4 MMR Vaccine.....	34
1.6 Comorbid Disorders.....	36
1.6.1 Attention-Deficit/Hyperactivity Disorder (ADHD).....	36
1.6.2 Anxiety Disorders	38
1.6.3 Mood Disorders	39
1.6.4 Motor Disorders and Tourette Syndrome	41
1.6.5 Disruptive Behavior Disorders	42
1.7 Co-occurring Challenging Behavior	43
1.8 Assessing Comorbidity	45
CHAPTER 2. PURPOSE	50
CHAPTER 3. METHOD	52
3.1 Participants.....	52
3.2 Measures	53
3.2.1 Child Behavior Checklist.....	53
3.2.2 Autism Spectrum Disorders-Child Version.....	55
3.3 Procedure	58
3.4 Statistical Analyses	58
3.5 Hypotheses.....	59
CHAPTER 4. Results	61
CHAPTER 5. DISCUSSION	74

REFERENCES	83
APPENDIX	
A Internal Review Board Approval	110
B ASD Study Consent Form.....	111
VITA.....	113

LIST OF TABLES

1. Demographic Characteristics by Diagnostic Group.....	53
2. Diagnoses for Children in the Psychopathology Group.....	53
3. Correlations between Individuals Items on the ASD-C and CBCL.....	61
4. Multitrait Multimethod Matrix for Convergent Validity of ASD-C and CBCL.....	65

LIST OF FIGURES

1. ROC Curve for ASD-C Diagnostic Total.....	66
2. ROC Curve for Withdrawn/Depressed Subscale from CBCL.....	67
3. ROC Curve for Social Problems Subscale from CBCL.....	67
4. ROC Curve for Thought Problems Subscale from CBCL.....	68
5. ROC Curve for ASD Scale from CBCL.....	68
6. ROC Curve for Anxious/Depressed Subscale from CBCL.....	69
7. ROC Curve for Internalizing Composite from CBCL.....	70
8. ROC Curve for Worry/Depressed Subscale from ASD-C.....	70
9. ROC Curve for Attention Problems Subscale from CBCL.....	71
10. ROC Curve for Externalizing Problems Composite from CBCL.....	71
11. ROC Curve for Rule-Breaking subscale from CBCL.....	72
12. ROC Curve for Aggressive Behavior Subscale from CBCL.....	72
13. ROC Curve for Conduct Subscale from CBCL.....	73
14. ROC Curve for Tantrum Behavior Subscale from CBCL.....	73

ABSTRACT

Previous researchers have demonstrated that Autism Spectrum Disorder (ASD) is related to increased prevalence of other psychological disorders. Comorbid disorders increase the difficulties in socialization and communication already experienced by children with ASD. These symptoms often require additional intervention techniques and should be identified as early as possible to beget the best prognosis. The *Autism Spectrum Disorders – Child Version (ASD-C)* is an informant based assessment battery specifically designed to measure symptoms of ASD, comorbid disorders, and challenging behavior in children with ASD. The reliability and validity of this assessment has been established in previous studies, but there is no research to date that compares the measure to the *Child Behavior Checklist (CBCL)* to establish construct validity of both measures. Participants in the current study included 114 children classified into three categories: ASD (n=14), psychopathology (n=71), and controls (n=29). Parents completed the *ASD-C* and *CBCL* as part of comprehensive psychological evaluations. Following a content analysis assessing correlations between items on the two measures, a Multitrait-Multimethod Matrix was constructed to assess convergent and divergent validity. All subscales and composites from both measures demonstrated convergence. Additionally, receiving operator characteristic (ROC) curves were utilized to examine the diagnostic validity of the two measures for ASD, Anxiety Disorder, Attention-Deficit/Hyperactivity Disorder (ADHD), and Oppositional Defiant Disorder (ODD). Both measures demonstrated diagnostic utility for clinicians to screen for psychopathology in children with and without ASD. Time and cost effective parent report measures would fill a gap in the provision of mental health services to low SES children and their families.

CHAPTER 1. INTRODUCTION

1.1 Early Definitions of Autism

The disorder that we conceptualize as Autism Spectrum Disorder (ASD) today has undergone numerous modifications from its original form. Leo Kanner (1943) is credited with being the first psychiatrist to provide a detailed description of the childhood disorder that he labeled *autistic disturbances of affective contact* or *autism*. In his seminal paper, Kanner (1943) provided a detailed case study of 11 children who demonstrated disturbances in social interaction and communication (e.g., muteness, echolalia, or nonfunctional speech). Additionally, these children exhibited repetitive or ritualistic patterns of behavior, restricted interests in specific objects or parts of objects, and insistence on sameness in their environment. Kanner's use of the term autism was meant to reflect the self-centered, isolating quality of these children (Volkmar & Klin, 2005). He described these children as demonstrating evidence of these impairments since birth, and thus, the disorder was likely biological in origin. Kanner revised his initial observations to highlight the importance of self-isolation and insistence on sameness when distinguishing autism from other childhood disorders (Eisenberg & Kanner, 1956). Moreover, Kanner proposed that these autistic symptoms must be evident prior to 2 years of age.

Much of Kanner's (1943) initial conceptualization of autism is still considered to be central to the disorder as we know it today. For example, impairments in socialization, communication, and restricted, repetitive behaviors or interests (RRBIs) are still the core symptoms. However, Kanner's paper proposed that individuals with autism were not intellectually disabled, but rather, these children tended to demonstrate poor performance on intelligence tests, especially verbal tasks, and in everyday activities due to a lack of motivation. In subsequent decades, researchers had provided evidence that as many as 75% of children with

autistic disorder (i.e., those with the most severe form of ASD) do actually have an intellectual disability (ID; i.e., IQ below 70; Lockyer & Rutter, 1970; Matson & Nebel-Schwalm, 2007a; Rutter, 1968).

Other early misconceptions about autism have also been dispelled by researchers, although they can make it difficult to accurately track research findings in the literature. In particular, early researchers often referred to the disorder as childhood schizophrenia, a term that was utilized until the 1970s (Rutter, 1978). This confusion originated from Kanner's (1943) use of the term autism, which had previously been coined by Bleuler, a Swiss psychiatrist, to describe the disconnection from external reality and social isolation displayed by people with schizophrenia (Bleuler, 1913). The seeming overlaps between the two disorders and the poor prognoses for those with autism and those with schizophrenia led many in the field to conceptualize autism as an early form of schizophrenia (Bender, 1946). In 1961, Creak provided a diagnostic outline of a disorder he referred to as *early childhood psychosis* that overlapped with the diagnostic criteria for autism. Early childhood psychosis was characterized by nine main symptoms: 1) impairments in interpersonal relationships (e.g., social isolation); 2) regression in speech, complete failure to acquire language, or abnormal speech (e.g., echolalia, pronoun reversal, speaking in monotone); 3) unawareness of personal identity (e.g., self-injurious behavior, failure to use personal pronouns, and abnormal body postures); 4) preoccupation with parts of objects as opposed to the intended function of the object; 5) abnormal perceptual experiences and responses to environmental stimuli (e.g., hyper- or hypo-sensitivity to pain or noises); 6) insistence on sameness; 7) intellectual disability, (although this was not true of all children with the disorder); 8) excessive anxiety and resistance to environmental change; and 9) abnormal motor movements (e.g., abnormal gait, poor coordination, repetitive movements).

Unfortunately, Creak did not specify how these behavior patterns were unique and separate from autism, and thus, some of these symptoms (i.e., abnormal sensory experiences) have been incorporated into assessment measures for autism over the years despite lack of support from scientific research (Matson & Minshawi, 2006).

As late as 1977, the *International Classification of Diseases, Ninth Edition (ICD-9;* WHO, 1977) still grouped autism with the childhood psychotic disorders, despite scientific research that clearly delineated the differences between autism and schizophrenia. Of note, the two disorders are inconsistent in terms of language and cognitive development, which are normal in those with schizophrenia and atypical in those with autism (Kolvin, 1971; Rutter & Bartak, 1971). Additionally, the experience of hallucinations or delusions is only present in those with schizophrenia and not a characteristic of autism (Kolvin, 1971). The two disorders differ in terms of distribution between the two genders, with autism more common in males and no gender difference observed in the rate of schizophrenia (Rutter & Bartak, 1971). Of most importance in terms of diagnosis is the differing ages of onset, with ASD apparent much earlier than schizophrenia. To highlight this difference, Rutter and Bartak (1971) added onset of symptoms prior to 30 months of age to Kanner's (1943) original three criteria for autism.

More than 20 years after Kanner's seminal paper, the research of Michael Rutter and Edward Ritvo, chairman of The National Society for Autistic Children (NSAC), served to clarify the core symptoms of autism and allow for more widespread diagnosis by physicians and psychologists (Schopler, 1978). Despite this push for clarity, the two men proposed separate and not completely compatible definitions. Rutter largely advocated a return to Kanner's initial definition of autism with the caveat that the scientific community should empirically test these hypotheses (Rutter, 1978). His conceptualization grouped autistic symptoms into three broad

categories: 1) impairment in socialization leading to a failure to develop relationships that are developmentally appropriate; 2) impairment in communication (delayed or impaired language development, idiosyncratic speech, impaired nonverbal communication); and 3) RRBIs.

Conversely, Ritvo (1978) and the NSAC consensus provided the following core features of autism: 1) disturbances in the ability to relate to people, objects, and environmental stimuli; 2) disturbances in language and communication abilities; 3) abnormal development (i.e., motor, adaptive, cognitive); and 4) disturbances in responses to sensory experiences. Both Rutter (1978) and Ritvo (1978) proposed that these features must be present prior to 30 months of age. They noted that intellectual impairment, epilepsy, and self-injurious behaviors were commonly co-occurring conditions. The NSAC description also included mood lability (e.g., unexpected crying or laughing) and inappropriate fears. While, Rutter (1978) specified that children with autism often exhibit a shortened attention span and delayed bowel control, although these are not characteristic of all children with autism. He made the suggestion that clinicians take into account mental age (i.e., intellectual level) and presence of neurological disorders when assessing behaviors, because this may impact judgments of developmental appropriateness.

The definitions put forth by Rutter (1978) and Ritvo (1978) are largely consistent in their descriptions of the three core features of autism. However, the process behind their conceptualization and even their motivating factors are divergent. Rutter's definition developed out of the historical perspective dating back to Kanner (1943) and took into account recent scientific findings. Whereas Ritvo's definition was motivated by a desire to increase public awareness and incite political action to fund treatments and research for autism (Matson & Minshawi, 2006). Despite their differences, these two definitions have contributed greatly to advances in research and the development of empirically supported treatments for autism.

Critical to past and current definitions of autism is the criterion for impairment in social interaction, which is evident from infancy. Children with autism frequently lack affection, cuddling, or emotional attachment to their caregivers. They are often described by their caregivers as self-isolating and will not seek out their parent to be comforted when they are in pain or upset. These children regularly fail to develop social-communicative behaviors (e.g., holding arms up when they want to be held, eye-to-eye gaze). Moreover, children with autism may experience stranger anxiety, fail to make eye contact, and seem uninterested or unwilling to participate in simple interaction games (e.g., peek-a-boo). As the children grow older, other social impairments become evident in terms of failure to make friends, lack of empathy, and inability to engage in cooperative play. These deficits in socialization and communication can lead to the child engaging in socially inappropriate speech or actions towards others (Rutter, 1978). Children with autism often appear immature and have difficulty understanding social cues (i.e., turn taking, signs of disinterest).

The second key component of autism is impairment in communication. Children with autism typically have delayed language acquisition or fail to develop any functional language. Paralleling the socialization deficits, this abnormality is present from infancy, as children with autism often fail to develop pre-language skills (e.g., social imitation, reciprocal smiling, and joint attention). Additionally, children with autism are frequently impaired in their use of gestures to communicate their wants and needs. Even when language does begin to develop, it may be echolalia, include pronoun reversals, involve abnormal inflection, or a singsong rhythm. These children may have a limited capacity to understand abstract terms, concepts, or reasoning. Likewise, a number of children with the most severe form of autism never develop functional speech (Rutter, 1978).

RRBIs comprise the third component of autism. Children may exhibit rigid or ritualistic play patterns that lack imagination and flexibility. Examples of such behavior include lining up toys, collecting objects, compulsive touching, or playing with only parts of a toy (Rutter, 1978). Often, these children will engage in tantrums or other challenging behavior if the toy or object is removed. Another common RRBIs is insistence on sameness, to the extent that the child will exhibit extreme distress if routines or stimuli in the environment are altered. Children with autism also exhibit stereotyped repetitive behaviors (e.g., spinning, hand-flapping, body rocking, touching) at higher rates than individuals with ID, although the behavior is common to children with autism and those with ID (Bodfish, Symons, Parker, & Lewis, 2000).

Aside from these three primary categories of disturbances in children with autism, there are other abnormalities in development and perceptual experiences that commonly occur in children with autism. These disturbances often involve delayed or impaired development of adaptive skills. One common area of associated adaptive impairment involves atypical motor movements (e.g., motor apraxia, gross motor delay, toe-walking) that impact functioning (Ming, Brimacombe, & Wagner, 2007). For example, a child with autism may exhibit typical gross motor development, but experience difficulties with tasks requiring fine motor skills. On the other hand, a child with autism may exhibit normal development until a certain age and then experience cessation or regression of skills. For example, a child may begin talking within normal limits, but then experience a regression in language skills or even a complete loss of functional speech. Conversely, a child may be delayed in the development of language and then rapidly acquire a large number of communication skills (Ritvo, 1978).

Another associated symptom of autism is abnormal responsiveness to sensory stimuli (Klintwall et al., 2011). This can involve abnormal auditory responses (i.e., hyper- or hypo-

sensitivity to sounds or non-responsiveness to certain sounds); tactile sensations (i.e., hypo- or hyper-sensitivity to pain, temperature, and certain textures of clothing); vestibular experiences (i.e., self-spinning without dizziness or preoccupation with spinning objects); olfactory and gustatory sensations (i.e., sniffing and/or licking people or objects and food preferences involving specific textures); proprioceptive senses (i.e., body postures, hand flapping, grimacing); and visual disturbances (i.e., poor eye-contact, staring at objects, sensitivity to light, or paying attention to parts of objects instead of the whole). Additionally, children with autism may exhibit abnormal activity levels (i.e., hyperactivity or psychomotor retardation), which can fluctuate over the lifespan (Zwaigenbaum, Bryson, Rogers, Roberts, Brian, & Szatmari, 2005).

1.2 Evolution of the Current Definition of ASD

The earliest prevalence estimates were established by Wing and Gould (1979), who conducted an epidemiological study of autism based on Kanner's (1943) early definition and more recent revisions. They surveyed the caregivers of one hundred thirty-two children who exhibited one or more of the three core symptoms (i.e., impaired socialization, abnormal language development and/or communication deficits, and RRBI) and/or met criteria for an ID (as measured by a standardized intelligence test). They analyzed general rates of impairment, how these symptoms were related to presence or absence of ID, and how the symptoms could be categorized. Caregivers completed structured interviews about the children's symptoms, while the children's behaviors were rated by trained observers. Wing and Gould (1979) found that the nature of the children's ability to interact (i.e., sociable, aloof, passive but odd) could reliably discriminate between a "socially impaired" group and a "sociable severely mentally retarded" group. Additionally, all the children in the "socially impaired" group exhibited repetitive behaviors and most lacked functional language.

There was a significant positive correlation between severity of ID and being classified as “socially impaired.” However, idiosyncratic use of language, elaborate routines, and pronoun reversals reliably distinguished children with autism from children with ID (Wing and Gould, 1979). Notably, only 17 of the 74 children in the “socially impaired” group met criteria for autism based on Kanner’s definition. Thus, Wing and Gould (1979) suggested that the definition of autism be broadened to encompass a larger number of impairments in social interaction. They proposed a triad of deficits that became a hallmark of research about autism. These deficits included: impairments in social interaction; impairments or complete absence of language and communication; and impairment in flexibility and imagination (i.e., insistence on sameness and presence of repetitive and stereotyped behaviors and interests).

These landmark research findings led to the creation of a new category of disorders that included autism and several other childhood disorders in the publication of the *Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition (DSM-III)*; American Psychiatric Association [APA], 1980). The title Pervasive Developmental Disorders (PDDs) was given to the chapter that encompassed infantile autism (i.e., Rutter’s 1978 definition), residual infantile autism, childhood onset pervasive developmental disorder (COPDD), and atypical pervasive developmental disorder. Residual infantile autism was a diagnosis meant to encapsulate children who no longer met criteria for autism, but still required services. COPDD was the diagnosis that accounted for children who developed autism symptoms after 30 months of age, but was considered to be extremely rare (Burd, Fisher, & Kerbeshian, 1988). Atypical pervasive developmental disorder was the original version of pervasive developmental disorder not otherwise specified, and was a diagnosis to account for individuals who met some of the criteria for autism, but did not cross the diagnostic threshold for a specific PDD. Overall, the category of

PDDs was utilized to diagnose children and adults who met some or all of the diagnostic criteria outlined by Rutter (1978) and explicitly excluded those with hallucinations and delusions solidifying the split between autism and childhood schizophrenia.

As with all diagnostic manuals, the DSM is constantly under scrutiny in response to new research findings and new assessment tools. Thus, revisions to the *DSM-III* began not long after its publication (Volkmar & Klin, 2005). Revisions to the definition of autism and the broader PDD category were largely influenced by Wing and Gould's (1979) triad of impairment. The most prominent and enduring change in the *DSM-III-R* (APA, 1987) was to use the term Autistic Disorder instead of infantile autism. This revision was made to highlight that autism is not merely a disorder of childhood and results in lifelong impairments (Volkmar, Cohen, & Paul, 1986). The revision also removed the age of onset, effectively eliminating the COPDD diagnosis. Unfortunately, this also led to over diagnosis of autism in many cases, and the false-positive rate was increased by approximately 40% (Spitzer & Siegel, 1990). The decision to remove age of onset was inconsistent with empirical evidence and made it more difficult to conduct longitudinal research. Additionally, the *DSM-III-R* definition of autism was inconsistent with the definition in the ICD, which was more stringent (Volkmar & Klin, 2005).

In response to this inconsistency, the development of the *DSM-IV* (APA, 1994) focused on more closely matching diagnostic criteria with the soon to be published *ICD-10* (WHO, 1992). The revisions to the PDD diagnoses in the *DSM-IV* focused on increasing reliability and validity, as well as making the criteria more easily understood by clinicians (Volkmar et al., 1994). A comprehensive field trial was conducted to evaluate the new diagnostic criteria and reduce the number of false-positives. Twenty-one sites and 125 raters across multiple countries participated in the study. In total, 977 individuals were categorized according to the proposed

criteria for Autistic Disorder, Asperger's Disorder, Childhood Disintegrative Disorder (CDD), and Rett's Disorder. The authors concluded that the new diagnostic criteria was reliable and demonstrated sufficient convergent validity with the *ICD-10* criteria (Volkmar & Klin, 2005).

The field trial also revealed that age of onset of symptoms was positively correlated with IQ. This is in accordance with findings that individuals with Asperger's Disorder, who tended to have higher IQs than those with Autistic Disorder, were diagnosed at the average age of 7.2 years (Mandell, Novak, & Zubritsky, 2005). The decision to include broader categories (e.g., Asperger's Disorder, Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), CDD, and Rett's Disorder) helped account for the presence of autism symptoms across the life span. Additionally, by increasing the age of onset criteria to 36 months of age for Autistic Disorder, the sensitivity of the diagnosis was increased. The reliability of the criteria was also increased when more experienced raters made the diagnoses (Klin, Lang, Cicchetti, & Volkmar, 2000). Despite the increased reliability and stability of diagnoses in the short-term (i.e., under a year), the criteria were still unstable among toddlers with lower levels of intellectual and adaptive functioning (Volkmar & Klin, 2005). Volkmar and colleagues (1994) found that placing more emphasis on socialization deficits increased sensitivity. This shift in diagnostic emphasis was also consistent with Kanner's definition of autism, which categorized social impairments as the primary deficit (Volkmar & Klin, 2005).

The *DSM-IV* introduced broader categories that conceptualized autism symptoms as occurring across a spectrum of disorders. Wing (1988) proposed that autistic symptoms and the associated disorders may be better understood as a continuum, or spectrum, of disorders. Conceptualizing autism as a spectrum of disorders was supported by developmental research demonstrating that there are not clearly defined boundaries between classical autism and the

related disorders (Dahl, Cohen, & Provence, 1986; Waterhouse et al., 1996; Waterhouse, Wing, & Fein, 1989; Wing, 1988). Although the different diagnostic categories are now subsumed under the heading Autism Spectrum Disorder, the history of Asperger's Disorder, PDD-NOS, CDD, and Rett's Disorder will now be reviewed in subsequent sections to highlight the heterogeneity of individuals with ASD.

1.2.1 Asperger's Disorder

Unbeknownst to Kanner, Hans Asperger, an Austrian physician, published a case study of 4 children with similar impairments in socialization and communication. Asperger (1944) termed the disorder autistic psychopathology and identified six main symptoms: 1) impairments in verbal communication, 2) impairments in nonverbal communication, 3) difficulties with socialization and specific interests that were abnormal in intensity, 4) motor clumsiness and odd body postures, 5) conduct problems, and 6) intellectualization (i.e., suppression of affective response).

These children differed in important ways from the children described by Kanner (1943). For example, Asperger (1944) described children who had developed functional language, but whom often spoke in an odd, incoherent, or tangential manner. Other common characteristics were the tendency not to modulate volume when speaking and/or singsong speech. Additionally, the children appeared unaware of social cues and tended to have one-sided conversations focused on a topic of interest to them. When speaking about these topics, the children would often sound pedantic leading to difficulties making friends. Their singular focus would consume large amounts of time and lead to the neglect of self-care, further hindering social interaction. Another interesting characteristic of the children Asperger identified was their superior rote memory of factual knowledge (e.g., bus schedules, historical dates, mathematical equations). These children

were able to recite facts that were beyond their educational level, although they often could not utilize these facts in a meaningful way.

The socialization deficits that were central to Kanner's (1943) definition of autism were also present in the children identified by Asperger, although the specific deficits differed. Asperger described children who could communicate at a basic level, but struggled to understand other's emotions or idiosyncratic use of language. These children would intellectualize their affect, meaning they would turn to facts and logic when confronted with uncomfortable feelings (Volkmar, 2011). In social situations, where the children were unable to isolate themselves from these emotions, these children had a tendency to evince challenging behaviors including physical aggression (Bjorkly, 2009; Ghaziuddin, Tsai, Ghaziuddin, 1991; Katz & Zemishlany, 2006; Simpson & Myles, 1998). These children also seemed to have the associated characteristics of clumsiness, odd posture, abnormal gait, and poor handwriting (Green, Baird, Barnett, Henderson, Huber, & Henderson, 2002). These abnormalities in motor skills combined with their circumscribed interests resulted in these children being bullied by their peers at higher rates than typical children (Sofronoff, Dark, & Stone, 2011).

Asperger published his original findings in German, and it was not until 1991 that an English translation of his work was available to a wide-spread audience (Frith, 1991). In the meantime, Van Krevelen (1971) endeavored to delineate the differences between the disorder described by Kanner (1943) and the one described by Asperger (1944). He proposed that the two disorders, while similar, were distinct. Early infantile autism, as described by Kanner, was characterized by the following: evident from infancy, involves delayed language development and language is often nonfunctional when it does develop, lack of eye contact, and social withdrawal. Whereas autistic psychopathology, as described by Asperger, was characterized by

the following: manifests after age 3 years, involves delays in motor functioning, normal language development, and the individual's desire to interact with others. Kreveken referred to autism as "a psychotic process" and Asperger's Disorder as "a personality trait." Overall, children who were best characterized by Asperger's description had a better prognosis than the children who fit Kanner's definition.

The debate about whether to classify Asperger's Disorder as a form of autism or a distinct disorder continues until the present day. In 1981, Wing clarified the core features of Asperger's Disorder and described the syndrome as falling into a larger group of disorders that was characterized by impairments in social interaction, communication, and imagination. She suggested some modifications and additions to the current diagnostic criteria for Asperger's Disorder. For example, Wing (1981) noted that as many as half of children with Asperger's Disorder had some delays in speech acquisition. Although they eventually obtained functional language skills, these children still struggled with pragmatics and relating to same-aged peers. Additionally, Wing (1981) pointed out that children with Asperger's Disorder often lacked pretend play as young children, and as adults they were best described as pedantic, literal, and logical instead of creative.

Wing's (1981) account of Asperger's Disorder also highlighted some of the main differences between Asperger's Disorder and autism. The take home message was that autism involved more severe impairments in socialization and communication than Asperger's Disorder. While children with autism were "aloof and indifferent," children with Asperger's Disorder were "passive or inappropriate" towards others. Children with autism appeared uninterested in social interaction, whereas children with Asperger's Disorder tried to socialize, but often experienced rejection or bullying. As for communication deficits, children with autism often failed to

develop functional language, whereas children with Asperger's Disorder tended to develop appropriate grammar and vocabulary, but struggled with comprehension and enriched content. Additionally, while both groups of children were described as having restricted interests, children with autism tended to exhibit repetitive behaviors, insistence on sameness, preoccupation with parts of objects, and rituals. On the other hand, children with Asperger's Disorder tended to have circumscribed interests in specific topics or activities.

The *DSM-IV-TR* and *ICD-10* were criticized for not accurately distinguishing between children with high-functioning autism (HFA) and children with Asperger's Disorder (Freeman, Cronin, & Candela, 2002; Mayes, Calhoun, & Crites, 2001). Empirical research failed to support the diagnostic validity of the *DSM-IV-TR* criteria for Asperger's Disorder (Mayes & Calhoun, 2004; Tryon, Mayes, Rhodes, & Waldo, 2006). More specifically, researchers demonstrated that delay in speech development is not a reliable predictor of symptom severity and adaptive functioning in children with HFA or Asperger's Disorder (Eisenmajer et al., 1998; Mayes & Calhoun, 2001). This has led some to conclude that there was not a meaningful diagnostic distinction between HFA and Asperger's Disorder (Howlin, 2003; Miller & Ozonoff, 2000; Ozonoff, South, & Miller, 2000).

After decades of debate, the distinction between autism and Asperger's Disorder became a moot point with the publication of the *DSM-5* (APA, 2013). The new diagnostic manual groups Autistic Disorder, Asperger Disorder, CDD, and PDD-NOS into one diagnosis called Autism Spectrum Disorder (APA, 2013; Ozonoff, 2012). When researchers have applied the *DSM-5* criteria to children currently diagnosed with Asperger's Disorder according to *DSM-IV-TR* they found that only 25.0% still met criteria for ASD (McPartland, Reichow, & Volkmar, 2012). To account for this discrepancy, children and adults previously diagnosed with

Asperger's Disorder will be assigned the diagnosis of Autism Spectrum Disorder according to the new criteria.

1.2.2 Childhood Disintegrative Disorder (CDD)

Theodore Heller, an Austrian educator, was the first to describe children with the regression in skills that are characteristic of CDD (Heller, 1908). Heller described six children who were typically developing until around age 3 or 4 when they exhibited a drastic regression in language, social, and motor skills. He referred to the disorder as 'dementia infantilis' and his case study predated Kanner's (1943) description of infantile autism by 35 years. CDD was the most recent terminology for this long recognized disorder, as it was referred to by other names prior to the publication of the *DSM-IV* (APA, 1994). The symptoms described by Heller were initially classified as 'disintegrative psychosis of childhood' in the *ICD-9* (WHO, 1977) and a subtype of dementia in the *DSM-III* (APA, 1980).

Heller (1930) outlined the following criteria for CDD: 1) onset between ages 3 and 4; 2) progressive deterioration of intellect and behavior with loss or marked impairment of language abilities apparent from onset; 3) associated symptoms (e.g., fear, overactivity) and possible hallucinations; and 4) absence of clear organic cause or neurological dysfunction. Children with CDD appeared similar to children with autism in many ways, such as impairments in socialization, communication deficits, and presence of RRBI. However, they were distinct in their late age of onset following a period of typical development. These children experienced loss of language and deterioration in adaptive skills. Following the regression, children with CDD were described as having profound mental retardation and making no further developmental gains (Burd et al., 1988). These children also regularly developed odd behaviors including stereotypies and insistence on sameness (Malhotra & Singh, 1993).

CDD was an extremely rare disorder, with prevalence estimated to be 1.7 per 100,000 (Fombonne, 2002). Further, the disorder occurred in only one out of 175 children diagnosed with a PDD. Children with CDD tended to have lower adaptive functioning than matched cases of autism (Mouridsen, Rich, & Isager, 1998). This is partially attributed to the high co-occurrence of epilepsy in children previously diagnosed with CDD (Mouridsen, 2003; Tuchman & Rapin, 1997). The disorder was removed from the *DSM-5* and individuals with the diagnosis will be subsumed into the category of Autism Spectrum Disorder according to the new criteria.

1.2.3 Rett's Syndrome

Rett's syndrome is the only former ASD that occurred almost exclusively in females (Comings, 1986). The X-linked genetic disorder was first identified by Andreas Rett (1966), an Austrian pediatric neurologist, in a case study of 22 girls with similar characteristics. Rett described these girls as engaging in repetitive hand movements (i.e., wringing and/or repeatedly putting hands into the mouth), dementia, communication deficits, ataxia, and hyperammonemia (i.e., metabolic disturbance causing excess ammonia in the blood). The disorder was initially termed *cerebroatrophic hyperammonemia*, but this metabolic condition was not found to be consistently characteristic of the disorder and rather an associated feature in some individuals (Burd, Kemp, Knull, & Loveless, 1990). Rett's research received limited consideration until Hagberg, a Swedish pediatrician whom was unfamiliar with Rett's findings, described similar symptoms among a group of 16 girls in a paper he presented to the European Federation of Child Neurology Societies (1980, June). His findings led to increased research and interest in this disorder, which became commonly referred to as Rett's syndrome (Van Acker, Loncola, & Van Acker, 2005).

Rett's syndrome is not typically identified at birth, despite recent research that has identified a genetic etiology. Retrospective interviews with parents revealed some early warning signs in infancy including: involuntary finger or hand movements and autistic-like behavior (Trevvarthen & Daniel, 2005). However, in most instances, the symptoms of Rett syndrome do not become apparent until between 6 and 18 months. These females appeared to be developing normally until this age, and then experienced a regression. This period of regression is characterized by deceleration in the rate of head growth (sometimes resulting in microcephaly), loss of purposeful use of hands, failure to meet developmental milestones (e.g., speech, motor, social), and jerky motor movements (Hagberg, Hanefeld, Percy, & Skjeldal, 2002). Due to these physical and behavioral changes, individuals with Rett syndrome typically meet criteria for severe to profound intellectual disability. Additionally, the rate of comorbid epilepsy in this population is estimated to be as high as 90% (Huppke, Kohler, Brockmann, Stettner, & Gartner, 2007; Steffenburg, Hagberg, & Hagberg, 2001). Recent estimates of prevalence indicate that the disorder is extremely rare, occurring at a rate of 0.65 for every 10,000 births (Hagberg, 2008). Of note, Hagberg (2008) reported that Rett syndrome accounts for between one-fourth and one-third of cases of severe intellectual disability in females.

Rett syndrome is an X-linked dominant disorder, which is typically fatal in males. The disorder is due to a methyl CpG binding protein 2 (MECP2) mutation on the X chromosome. Females are able to survive, because they have two X chromosomes and the non-mutant chromosome mediates transcriptional repression during the development of the central nervous system (Goffin et al., 2011). Thus, the non-mutant chromosome is expressed in some cells, while the mutant chromosome is expressed in other cells leading to neurodevelopmental abnormalities (Amir et al., 1999). Schwartzman, Bernardino, Nishimura, Gomes, and Zatz

(2001) reported a case of Rett syndrome in a male with a XXY karyotype. This chromosomal constitution is referred to as Klinefelter's syndrome, and can result in hypogonadism and reduced fertility. Only a few studies have been published on Rett syndrome in males so the prevalence is unknown, but thought to be extremely rare (Zeev et al., 2002). Rett syndrome was removed from the *DSM-5*, because a specific etiology was identified in 1999 and thus, the disorder no longer needs to be defined by behavioral symptoms (Ozonoff, 2012). This change has been controversial and anxiety provoking for the families of children diagnosed with Rett syndrome as not all individuals presenting with these symptoms have the MECP2 mutation (Mao & Yen, 2010).

1.2.4 Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS)

The term PDD-NOS was first introduced in the *DSM-III-R* as a sub-threshold category for children who failed to meet the full criteria for a developmental disorder (Towbin, 2005). Prior to this revision, this diagnosis was referred to as atypical autism and was considered to be a less severe form of autism (Buitelaar, Van der Gaag, Klin, & Volkmar, 1999). PDD-NOS served as a catch-all for individuals who exhibited some deficits in socialization or communication, but were not as severely impaired as individuals with autism. More specifically, the *DSM-IV* (APA, 1994) defined PDD-NOS as a severe impairment in socialization, communication, or presence of RRBI, that does not meet criteria for a specific PDD. One example was late age of onset, because age of onset had to be prior to 36 months of age to meet criteria for autism. The criteria in the *DSM-IV-TR* (APA, 2000) was more precise and required a clear impairment in socialization as evidenced by impairments in verbal communication, nonverbal communication, or RRBI.

The subthreshold category of atypical autism arose following clinical accounts of individuals who were similar to those with autism, but did not exactly match Kanner's (1943) definition of autism. More specifically, these children had less severe language impairments and less impairing RRBI (Despert & Sherwin, 1958). Despite these diagnostic differences between children with PDD-NOS and autism, the inclusion of a "not otherwise specified" category received many criticisms. Clinicians have argued that the diagnosis allowed for too much heterogeneity of symptom type and severity, and is unnecessary if autism is conceptualized as a spectrum of disorders. The *DSM-IV-TR* was criticized for failing to clearly delineate the level of impairment that is needed for a child to meet criteria for ASD. Towbin (2005) argued that the diagnostic criteria required clinicians to use categorical symptom descriptions to diagnose a dimensional variable. In the end, clinicians were often left to use their own discretion to make final judgments. Mahoney and colleagues (1998) found that clinicians could reliably distinguish between ASDs and non ASDs ($\kappa = .67$, 91% agreement); however, they were much less reliable in distinguishing between different categories of ASD ($\kappa = .51$, 73% agreement). In particular, they found that clinicians were most likely to disagree about diagnoses involving PDD-NOS.

Mandy, Charman, Gilmour, and Skuse (2011) studied 66 individuals with PDD-NOS and found that, contrary to the popular belief that this group was completely heterogeneous, 97% of those with PDD-NOS fell within a single, distinct symptom presentation. Those individuals all had impairments in social reciprocity and communication, without the presence of significant RRBI. Mandy and colleagues (2011) described the PDD-NOS group as a less severe presentation of autistic psychopathology. They surmised that the *DSM-5* criteria for Autism Spectrum Disorder would effectively exclude many of the individuals with PDD-NOS, because they would fail to meet the criteria for RRBI.

In the past couple of years, a number of different research groups have specifically investigated changes to rates of ASD diagnosis by applying *DSM-5* criteria in both retrospective and prospective manners. McPartland and colleagues (2012) used a retrospective technique and found that only 28.3% of individuals diagnosed with PDD-NOS using *DSM-IV-TR* criteria would still cross the diagnostic threshold for Autism Spectrum Disorder using the *DSM-5* criteria. Conversely, Huerta, Bishop, Duncan, Hus, and Lord (2012) argued that the *DSM-5* criteria have improved specificity compared to the criteria for PDD-NOS in the *DSM-IV-TR*.

1.3 Current Definitions of ASD

As of 2013, the two main diagnostic standards are the *DSM-5* (APA, 2013) and the *ICD-10* (WHO, 1992). The *ICD-10* category for pervasive developmental disorders includes: Childhood Autism, Rett's Syndrome, CDD, Asperger's Syndrome, Atypical Autism, Other pervasive developmental disorder, Pervasive developmental disorder unspecified, and Overactive disorder associated with retardation and stereotyped movements. Atypical autism in the *ICD-10* refers to individuals with late age of onset or subthreshold symptomatology. PDD unspecified in the *ICD-10* is used when an individual fits the general description for PDD, but the clinician either does not have the necessary information to diagnose a specific PDD or is presented with contradictory information.

The fifth edition of the DSM was published in May 2013 and includes numerous changes to the diagnostic criteria for ASD (Mandy, Charman, & Skuse, 2012). The first major change involves re-conceptualizing ASD as a dyad of impairments as opposed to the well-established triad that was developed by Wing and Gould (1979). The two previously separate categories of socialization and communication were combined to form the new category 'social communication.' The second component of the dyad is RRBI's. Another major change to the

diagnostic criteria involves eliminating the categories of ASD (e.g., Asperger's Disorder and PDD-NOS) and subsuming all individuals under the heading 'Autism Spectrum Disorder' (Kaland, 2011). This change is highly controversial, especially among individuals with Asperger's Disorder who object to being group with individuals who have different symptom presentations (i.e., Autistic Disorder) and require different treatment modalities (Wing, Gould, & Gillberg, 2011). The *DSM-5* also introduced a new category called Social (Pragmatic) Communication Disorder, which consists of all the social and communication deficits seen in ASD without the presence of RRBI (APA, 2013). Researchers have hypothesized that some of the children who no longer meet criteria for ASD with the *DSM-5* will meet criteria for this new diagnosis, which is grouped with the other communication disorders (Gibbs, Aldridge, Chandler, Witzlsperger, & Smith, 2012).

The exact criteria for Autism Spectrum Disorder in *DSM-5* requires that the individual present with persistent deficits in both social interaction and social communication in multiple contexts (APA, 2013). Examples of symptoms include: deficits in social-emotional reciprocity (e.g., inability to maintain a conversation, lack of interest in initiating social contact); deficits in nonverbal communication that impairs social interaction (e.g., poor eye contact, lack of facial expressions, abnormal use of gestures); and deficits in developing age appropriate relationships (e.g., lack of pretend play, not interested in peers, difficulties making friends). Secondly, the individual must exhibit two or more RRBI out of four: 1) stereotypies (e.g., repetitive motor movements, abnormal use of objects, echolalia); 2) insistence on sameness (e.g., adherence to routines or rituals, difficulties with transitions, rigid thinking patterns); 3) restricted interests that are abnormal in focus or intensity; or 4) hyper- or hyposensitivity to visual, auditory, olfactory,

or tactile stimuli. Additionally, the individual's symptoms must be evident during the developmental period, effectively eliminating a rigid age-of-onset criterion.

Finally, the diagnostic criteria require the clinician to specify the current severity level of the individual's symptoms. Level 1 would be ascribed to an individual "requiring support," but whom is able to communicate to some degree and whose attempts to make friends are usually unsuccessful due to odd interaction style. Level 2 would be ascribed to an individual "requiring substantial support" due to lack of social interaction and limited verbal and nonverbal communication skills. Level 3 is the most severe and would be ascribed to an individual "requiring very substantial support" due to minimal or lack of speech, failure to initiate social interaction, and marked impairment from RRBI's.

Numerous studies have been published evaluating the validity of the *DSM-5* criteria for ASD. There are studies that support both sides of the argument. For example, Mandy and colleagues (2012) evaluated 708 children with ASD and conducted a confirmatory factor analysis to determine if the *DSM-5* model was superior to the triad approach of *DSM-IV-TR*. They reported that among individuals with autism who are higher functioning, ASD fits into a dyad model better than the previous triad of impairments. Frazier and colleagues (2012), likewise, analyzed data from 14,744 siblings with and without ASD. They applied the proposed *DSM-5* algorithm and found that the *DSM-5* had superior specificity (0.97 compared to 0.86); however, it had lower sensitivity (0.81 compared to 0.95). The researchers then relaxed the *DSM-5* criteria by requiring one less symptom criterion and this increased the sensitivity to 0.93, although the specificity was reduced to 0.95. They concluded that the *DSM-5* criteria had increased specificity and with slightly more relaxed criteria could allow for more accurate diagnoses.

On the other side were the studies that raised concern about the *DSM-5* criteria excluding a large portion of individuals who were previously diagnosed with ASD. Mattila and colleagues (2011) applied the proposed criteria to a group of eight-year-old Finnish children and found that the *DSM-5* was less sensitive, especially in regards to individuals previously diagnosed with Asperger's Disorder and high-functioning individuals. As stated previously in the section on Asperger's Disorder, McPartland and colleagues (2012), likewise, found that when the *DSM-5* criteria was applied to individuals from the *DSM-IV* field trial, only 60.6% of the individuals who were previously diagnosed with ASD still received the diagnosis. Worley and Matson (2012) assessed 281 children utilizing both the *DSM-IV-TR* and *DSM-5* criteria for purposes of comparison. They found that the children who no longer met criteria for ASD using the *DSM-5* criteria had similar symptom severity to those children who still met criteria. Further, the children no longer meeting criteria differed significantly from typically developing children. Thus, the new diagnostic criteria appears to have decreased sensitivity and the broader classification used by the *DSM-IV-TR* appears to be superior for identifying all children with significant impairments in these core areas.

In another study, researchers evaluated the rates of diagnosis using the new and old criteria and they found that 36.53% of adults who met criteria for ASD according to the *DSM-IV-TR* no longer met criteria according to the *DSM-5* (Matson, Belva, Horovitz, Kozlowski, & Bamburg, 2012). Likewise, Taheri and Perry (2012), found that 37% of previously diagnosed individuals no longer met criteria using the *DSM-5*. More specifically, only 17% of individuals previously diagnosed with PDD-NOS met criteria for ASD. To account for these discrepancies, the *DSM-5* includes the caveat that individuals who were previously diagnosed with ASD with

the *DSM-IV-TR* should be given a *DSM-5* diagnosis of ASD regardless of whether they meet the new criteria.

1.4 Prevalence of ASD

ASD was once considered to be relatively rare, but is now described by some as epidemic in the United States. In the last decade alone, there has been a 78% increase in the prevalence rate (Baio, 2012). There are numerous hypotheses for the increased rate of diagnosis, with many researchers arguing that the numbers do not denote an actual increase in incidence. Wing and Potter (2002) proposed some specific explanations for the changing rates including: 1) frequently updated diagnostic criteria; 2) differences in methods used for diagnosis between prevalence studies; 3) research to support that ASD can co-occur with intellectual disability, physical disability, or psychological disorder; 4) increased availability of public and private services for individuals with ASD; and 5) increased awareness about the disorder among physicians and caregivers. They also provided the caveat that the increasing numbers could represent a true increase in the prevalence of the disorder.

As per the first two proposed explanations, there have been numerous revisions to the DSM and ICD criteria for ASD since the disorder was initially introduced by Kanner in 1943. For instance, the disorder was not officially recognized in the DSM until the publication of the *DSM-III* in the 1980s. Additionally, early definitions of ASD did not allow for a diagnosis if the individual had subthreshold symptomatology in any of the three core deficits. To account for heterogeneity and the conceptualization of autism as a “spectrum disorder,” each revision to the DSM has included different subgroups under the PDD heading. This constant shifting in diagnostic categories led to variability in which diagnostic criteria were used for any given prevalence study. Additionally, different researchers utilized different participants (i.e., age,

gender, socioeconomic status [SES]). Fombonne (2003) indicated that studies conducted with a smaller number of participants resulted in higher rates of ASD prevalence. Higher rates were also found in studies with a higher percentage of immigrants (Wing & Potter, 2002)

As per the third point presented by Wing and Potter (2002), ASD was initially described by Kanner (1943) as occurring solely in children of average intelligence. Researchers now consider ASD to occur at fairly high rates in individuals with ID. Baird and colleagues (2006) found that 50% to 70% of individuals with ASD also had ID. Further, diagnostic criteria now allows for a diagnosis of ASD in children with above average intelligence. These children are most often diagnosed with Asperger's Disorder, which was not introduced to the DSM until 1994. Moreover, researchers have only recently begun to focus on comorbidity with other psychiatric disorders (Clark, Feehan, Tinline, Vostanis, 1999; De Bruin, Ferdinand, Meester, de Nijs, & Verheij, 2007; Ghaziuddin, Ghaziuddin, & Greden, 2002; Ghaziuddin, Tsai, & Ghaziuddin, 1992; Ghaziuddin, Weidmer-Mikhail, & Ghaziuddin, 1998; Leyfer et al., 2006; Matson & Nebel-Schwalm, 2007b; Smith & Matson, 2010). Specific studies of comorbidity will be discussed in more detail in subsequent sections.

With increases in prevalence have come increased demand for specialized services in the schools and larger community that address the needs of children with ASD. Unfortunately, some children are more likely than others to gain access to such services, and this may partly explain discrepancies between demographic groups in rates of diagnosis. Thomas, Ellis, McLaurin, Daniels, and Morrissey (2007) found that access to care was more limited for families from racial and ethnic minority groups. Likewise, Liptak and colleagues (2008) found that ASD was likely being underdiagnosed in children from racial minorities, especially those from families of low SES who are traditionally underserved in the health care system. Despite these disparities,

there has been a marked improvement in the availability of services since the Individuals with Disabilities Education Act (IDEA) officially recognized ASD in 1991. In the 10 years following the introduction of this legislation, the rates of ASD went from 3 per 10,000 to 52 per 10,000 (Gurney, Fritz, Ness, Sievers, Newschaffer, & Shapiro, 2003). In order to best serve the child, both parents and professionals are more likely to consider a diagnosis of ASD when there are services available to improve that child's prognostic outcome (Wing & Potter, 2002).

Moreover, increased public awareness of ASD has led to the formation of parent support groups and advocacy groups that aid parents in identifying qualified professionals to evaluate and treat their children. These groups have pushed for research on the etiology and treatment of these disorders leading to increased public and private funding for empirical studies (Singh, Illes, Lazerroni, & Hallmayer, 2009). As of yet, there is no research that draws any causal relationships between these factors and the increased prevalence of ASD. Wing and Potter (2002) suggest that future research is needed to more fully address these proposed explanations.

The most recent estimates from the Center for Disease Control place the rate of ASD as 1 in 88 children (Baio, 2012). The Autism and Developmental Disabilities Monitoring Network collected data on children aged 8 years from 14 different sites across the United States. In total, 337,093 children were reviewed and 3,820 met criteria for an ASD. The rates ranged between sites from 4.8 per 1,000 in Alabama to 21.2 in 1,000 in Utah. Prevalence rates significantly differed according to gender, with ASD occurring in one in 54 males as opposed to one in 252 females. Additionally, the sites identified significantly more non-Hispanic white children with ASD than non-Hispanic black children and Hispanic children. In comparison to the 2006 surveillance year, ASD prevalence increased 23%.

Kim and colleagues (2011) evaluated the prevalence of ASD in South Korea among 7 to 12 year-old children. The researchers screened 36,886 children attending elementary schools in the Islan district of Goyang City, South Korea. The study consisted of a 2 step diagnostic process. All children were screened for possible ASD, and then those who screened-positive (1,214 children) received further evaluation. The crude prevalence of all ASDs was found to be 0.36%, however, following statistical adjustment for nonparticipants (37%) the overall ASD prevalence was estimated to be 2.64% with 1.89% in the general sample and 0.75% in the high-probability group. The researchers acknowledge that this may be a slight overestimate considering previous estimates ranged from 0.6% to 1.8% prevalence (Fombonne, 2009). However, an estimated two-thirds of the children meeting criteria for ASD were previously undiagnosed and untreated, bringing into question the comprehensiveness of the established detection methods.

1.5 Etiology of ASD

Increased public interest in the disorder has led to increased empirical research on the etiology of ASD. Prior to the last few decades, explanations of etiology were largely theoretical as opposed to evidence-based. Kanner (1943) initially theorized that autism had a biological origin, but could not identify a clear precipitant. He included a more thorough description of some of the interpersonal difficulties he observed between the children with autism and their parents. These interpersonal factors mapped easily into the psychodynamic theories of the era and became the main focus of early descriptions of etiology. Eveloff (1960), for example, crafted his case studies with a direct focus on parent-child relational problems. He described the mothers as cold, impersonal, and rigid, while the fathers were perfectionistic and detached from their children. These parental characteristics were thought to both contribute to and maintain the

child's symptoms. This theory was expanded upon by Bettelheim (1967), who coined the term "refrigerator mothers" to describe this pattern of maternal relational characteristics and rearing style that supposedly led to autistic behavior in children. Empirical studies proved time and again that these theories were unfounded, and that there was no link between child-rearing practices and the development of autism (Pitfield & Oppenheim, 1964). Despite these findings, parents and especially mothers of children with autism still often blame themselves for their child's difficulties (Matson & Minshawi, 2006).

1.5.1 Learning Theories

Behavioral theories gained popularity in the first part of the 20th century, but were not immediately applied to research on ASD. Philips (1957) summarized the literature to date linking ASD with learning theory formulations and found there to be a dearth of research in the area. Ferster (1961), who worked with Skinner on the operant conditioning model of learning (Ferster & Skinner, 1957), acknowledged this insufficiency and made the first true attempts to explain the etiology of ASD using learning principles. He proposed that ASD symptomology resulted from the persistent failure to provide social praise or parental attention in response to desired behaviors. Therefore, the child never learned to associate these social responses with positive reinforcement. However, Ferster noted that children with ASD could learn new behaviors, as evidenced by the effectiveness of primary reinforcers (i.e., food) to foster learning in an operant conditioning procedure (Ferster & DeMeyer, 1961). Again, the idea that parental reactions to behavior caused ASD has been refuted by empirical research, but these behavioral principles became key components of treatments for children with ASD.

This early work was the basis for Lovaas and Smith's (1989) behavioral theory of ASD, which is broken down into four basic principles. First, they offered that learning theory accounts

for the patterns of behaviors characteristic of children with ASD and the basic laws of learning can be adapted into interventions tailored to these behaviors. Second, the researchers acknowledged that the behaviors are not all learned and that some are due to developmental delays, which might never be remediated even with intensive treatment. Third, they proposed that children with ASD can learn skills when they are taught in a controlled environment adhering to specific reinforcement schedules. Finally, the researchers stated that a child with ASD is capable of mastering new learning when environmental variables are highly controlled and tailored to the individual child. Using these tenets for guidance, the researchers developed a behavior therapy that focuses on specific behaviors and how they can be changed in the present as opposed to where they originated from in the past. Thus, behavioral views of ASD are fairly unique among other etiological theories in that they are present focused.

1.5.2 Genetics

The new information garnered from behavioral theories led to a shift in the other types of research being conducted on ASD. In the first part of the 20th century, little attention was paid to possible genetic explanations for ASD, because qualitative research indicated that children with ASD were not typically born from parents with ASD and there was not a clear chromosomal anomaly that could account for the disorder (Rutter, 1968). Further, early studies did not find high rates of concordance among siblings. However, more recent studies have focused on twins and found relatively high rates of concordance indicative of genetic factors being involved in the etiology of the disorder. For example, Folstein and Rutter (1977) examined 11 pairs of monozygotic and 10 pairs of dizygotic twins. They found a 36% pair-wise concordance rate for ASD in monozygotic twins as compared to no concordance in dizygotic twins. This finding is thought to be a low estimate based on the stricter diagnostic criteria of the time period (Folstein

& Rutter, 1988). A later study using a larger sample by Ritvo, Freeman, Mason-Brothers, Mo, and Ritvo (1985), reported a much higher concordance rate of 95.7% among monozygotic twins and 23.5% concordance among dizygotic twins. There were admittedly some methodological concerns with these early studies (e.g., no random sampling and use of parental report to determine zygosity), but the findings provided early evidence that ASD occurred in both types of twins at a higher rate than would predicted by chance. A more recent study found similarly high rates of genetic heritability for autistic traits. Ronald and colleagues (2006) screened 3,419 eight-year old twin pairs in the United Kingdom. In 75% of the cases, zygosity was determined using DNA samples. They found overall concordance rates of 80.0% for monozygotic twins and 21.5% for dizygotic twins. Further, genetic modeling allowed them to investigate correlations between the three main components of ASD. The researchers found that distinct genetic influences were responsible for each of the components lending support to the theory that the genetic factors involved in ASD are highly heterogeneous.

An alternative method for studying genetic factors is to study the non-twin siblings of individuals with ASD. Early studies reported rates that ranged between 3% and 10% for recurrence in the sibling of a child with ASD (August, Stewart, & Tsai, 1981; Bolton et al., 1994; Chakrabarti & Fombonne, 2001; Lauritsen, Pedersen, & Mortensen, 2005). More recently, Constantino, Zhang, Frazier, Abbacchi, and Law (2010) found a recurrence rate of 10.9%. They also reported that an additional 20% of the non-ASD siblings had a history of speech delay. Ozonoff and colleagues (2011) conducted a prospective longitudinal study of 664 infants at risk for ASD whom had an older sibling with ASD. They found that 18.7% of these at risk infants developed ASD by 36 months of age. Some possible explanations for these discrepancies in recurrence rates are that early studies were limited by small sample sizes and biases in reporting.

The association between ASD and specific genetic disorders is a third piece of evidence in support of a genetic etiology of ASD (Klauck, 2006). The relationship between Fragile X Syndrome, an X-linked recessive disorder, and ASD is well established. Fragile X has been identified as the second most common known genetic cause of intellectual disability after Down syndrome (Brown et al., 1982). Brown and colleagues (1986) compiled the results from 12 studies, including their own, where a total of 614 males with ASD were screened for Fragile X. They reported that 47 of the males were positive for the syndrome, which equates to a 7.7% frequency of Fragile X among males with ASD. Further, an estimated 12.3% of males with Fragile X met criteria for ASD. The researchers suggest genetic testing for all individuals with ASD with specific emphasis on screening for the Fragile X marker.

A recent literature review identified 44 genetic disorders and 103 disease genes that are found in individuals with ASD or associated with autistic symptoms (Betancur, 2011). Each of these account for only a small number of ASD cases and suggest a highly heterogeneous genetic etiology. Some of the disorders identified as being highly comorbid with ASD include 22q13 deletion syndrome, tuberous sclerosis, and adenylosuccinate lyase deficiency. Despite increased research into genetic factors, many causes of ASD have yet to be identified and researchers caution against placing too much emphasis on genetic testing (Herman, Henninger, Ratliff-Schaub, Pastore, Fitzgerald, & McBride, 2007).

1.5.3 Neurobiology

With increased knowledge about the structure and function of areas in the brain, researchers have suggested that neurobiological factors play an important role in explaining the etiology of ASD. The high rate of comorbid ID in individuals with ASD lends further support to the notion that abnormalities in neurobiology are probable causal mechanisms in ASD (Matson

& Minshawi, 2006). Specific neurobiological deficits in ASD include gait disturbances, clumsiness, sensitivity to sensory stimuli, and epilepsy (Ben-Sasson, Hen, Fluss, Cermak, Engel-Yeger, & Gal, 2009; Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; Levisohn, 2007; Vilensky, Damasio, & Maurer, 1981). The connection between epilepsy and ASD has been a focus of neurological research in recent years. The disorder is estimated to affect 5% to 38% of individuals with ASD (Danielsson, Gillberg, Billstedt, Gillberg, & Olsson, 2005; Tuchman & Rapin, 2002). Moreover, the risk of epilepsy is higher in those with comorbid ID or cerebral palsy (Tuchman & Rapin, 2002).

At present, multiple areas of the brain have been suggested as the center of dysfunction in ASD including the amygdala, basal ganglia, cerebellum, and thalamus (Vargas, Nascimbene, Krishnan, Zimmerman, & Pardo, 2005). Schumann, Barnes, Lord, and Courchesne (2009) investigated the developmental trajectory of the amygdala in children with ASD and found that the amygdala was enlarged in comparison to typically developing toddlers. Further, they found that in male toddlers who developed ASD, the degree of enlargement was correlated with the severity of their socialization and communication impairments. The amygdala is involved in the production and recognition of emotions as well as modulating social behavior (Adolphs, 2008). Thus, the pattern of impairments in ASD may be attributable to amygdala dysfunction. As for the basal ganglia, researchers have found that abnormalities in the structure are associated with motor, communication, and social deficits in male children with ASD (Qui, Adler, Crocetti, Miller, & Mostofsky, 2010). The basal ganglia has also been implicated in postural abnormalities of the trunk and head that are common in individuals with ASD (Rinehart et al., 2006). The cerebellum has also been linked to some of the characteristic motor impairments in children with ASD, especially abnormalities in gait function (Rinehart et al., 2006). Cerebellar

dysfunction in children with ASD is thought to be due to decreased vermis volume; however, the data is inclusive at present (Scott, Schumann, Goodlin-Jones, & Amaral, 2009). Finally, the thalamus has been identified as playing a possible role in the pathophysiology of ASD, and more specifically the development of abnormal sensory responses (Harden et al., 2008).

Aside from structural abnormalities, researchers have also investigated the role of neurotransmitters in ASD. The most commonly researched neurochemical in individuals with ASD is serotonin (Matson & Minshawi, 2006), which is likely due to its role in psychotropic medications (i.e., selective serotonin reuptake inhibitors [SSRIs]) that are commonly prescribed to individuals with ASD. Abnormalities in serotonin levels in the bloodstream were first linked to ASD by Schain and Freedman (1961), who reported elevated levels of 5-hydroxyindole in children with ASD. This finding is critical, because serotonin plays a major role in cortical development and specifically areas of the brain that are impaired in children with ASD (Chugani, 2004). The abnormal serotonin levels in individuals with ASD have been linked to a variety of behavioral symptoms. Kolevzon and colleagues (2010), for example, reported that serotonin level was inversely related to presence of self-injurious behavior (SIB) in individuals with ASD. However, the studies on serotonin and behavioral symptoms of ASD have been inconsistent overall and controversy remains over the use of SSRIs with this population (Hranilovic, Bujas-Petkovic, Vragovic, Vuk, Hock, & Jernej, 2007).

Another neurotransmitter that has been linked to ASD is gamma-aminobutyric acid (GABA), which is the main inhibitory chemical in the brain (Collins et al., 2006). The high rates of epilepsy in individuals with ASD may be related to a reduced number of GABA receptors (Fatemi, Folsom, Reutiman, & Thuras, 2009). Researchers have proposed that the brain of

individuals with ASD is overstimulated and unable to filter out unnecessary external stimuli due to a lack of GABA (Hussman, 2001).

A third group of neurotransmitter that has been linked to ASD are endogenous opioids, which have been specifically associated with SIB in individuals with ASD (Gillberg, 1995). One theory for this link is related to the finding that high levels of endorphins in children with ASD is associated with reduced pain reactivity (Tordjman et al., 2009). Additionally, Tordjman and colleagues (2009) found that the level of endorphins in blood plasma was positively correlated with severity of ASD symptoms. Endogenous opioids have also been linked to prosocial behaviors including social bonding, with these social interactions resulting in an endorphin release (ElChaar, Maisch, Augusto, & Wehring, 2006; Wink, Plawecki, Erikson, Stigler, & McDougle, 2010). Thus, the socialization impairments that are characteristic of children with ASD may be due to high levels of endogenous opioids that negate the opioid reward that comes from social interaction (Machin & Dunbar, 2011). Further research is needed to elucidate the exact roles of neurotransmitters in the etiology of ASD.

1.5.4 MMR Vaccine

In 1998, Wakefield and colleagues published their theory that autism was caused by the measles, mumps, and rubella (MMR) immunizations. The initial purpose of their study was to investigate the relationship between gastrointestinal conditions (i.e., chronic enterocolitis) and regressive ASD in 12 children aged 3 to 10 years. Wakefield and colleagues (1998) reported that in 8 of the 12 children, administration of the MMR vaccine was followed by loss of acquired skills (i.e., language abilities) and development of gastrointestinal symptoms (i.e., diarrhea, food intolerance, and abdominal pain). Parents reported that the first behavioral symptoms occurred an average of 6.3 days after the immunization. The researchers hypothesized that some

component of the vaccine caused increased permeability of the intestines leading to excessive absorption of gut-derived peptides. They proclaimed this phenomenon to be the basis of a new syndrome, which they termed ‘autistic enterocolitis,’ because the absorption of peptides led to the development of autistic behaviors in these children. Wakefield and colleagues (1998) were careful not to suggest a causal link between the MMR vaccine and autism; however, they advocated that parents stop allowing their children to receive the vaccination. The publication caused immediate controversy and led to an increase in the number of parents refusing the vaccine despite subsequent evidence against Wakefield’s findings (Brown et al., 2012).

In the years following the article’s publication, numerous studies were published that found no evidence to support a link between ASD and the MMR vaccine (Honda, Shimizu, & Rutter, 2005; Madsen et al., 2002; Taylor, Miller, Lingam, Andrews, Simmons, & Stowe, 2002). Furthermore, numerous concerns were brought against Wakefield regarding the methodology of the study. First, no control group was utilized in the study, so all the findings could be merely coincidental. Second, only one child included in the study was later found to meet the criteria for ‘regressive autism,’ and three of the children did not even have ASD (Deer, 2011). Further, more evidence surfaced that some of the children had gastrointestinal problems prior to the MMR vaccine. Finally, the only children included in the study were those who had developmental delays and gastrointestinal concerns, as Wakefield did not use random sampling to recruit participants. Following a thorough investigation by Deer (2011), Wakefield and colleagues’ (1998) article was retracted from the *Lancet* in 2010. Additionally, Wakefield was found guilty of subjecting the children in his study to intrusive procedures that disregarded ethical standards and stripped of his medical license by the United Kingdom General Medical Council (Dyer, 2010).

The past two decades have resulted in a great influx of research on the etiology of ASD. There is evidence to support multiple causalities of this highly heterogeneous disorder. Thus, it appears to be most likely the case that ASD is the result of multiple etiological factors and these may differ between individuals.

1.6 Comorbid Disorders

Comorbidity is the term used when an individual presents with two or more disorders. In the context of the current research, comorbidity refers to an individual who is diagnosed with ASD and another disorder. In these instances, ASD is typically considered the primary disorder and the comorbid disorders are considered secondary (Matson & Nebel-Schwalm, 2007b). ASD is primary because it tends to be the most interfering on functional domains. Simonoff, Pickles, Charman, Chandler, Loucas, and Baird (2008) assessed 112 children with ASD and reported that 70% had at least one comorbid disorder and 41% met criteria for two or more disorders. In some instances, researchers and clinicians have debated whether these disorders should even be diagnosed in individuals with ASD or instead considered associated symptoms.

1.6.1 Attention-Deficit/Hyperactivity Disorder (ADHD)

The most commonly studied comorbid disorder in individuals with ASD is attention-deficit/hyperactivity disorder (ADHD) as the behavioral symptoms of the two disorders often overlap leading to difficulties with differential diagnosis (Lovell, Moss, & Wetherell, 2012; Matson & Nebel-Schwalm, 2007b). ADHD is a neurobehavioral disorder that is estimated to occur in 2% to 17% of the total population (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Froehlich, Lanphear, Epstein, Barbaresi, Katusic, & Kahn, 2007). The disorder is characterized by hyperactivity, inattention, and impulsivity (Barkley, 1981; Barkley, 1997; Burns, Boe, Walsh, Sommers-Flannagan, & Teegarden, 2001). These symptoms are only considered clinically

significant when they occur at a frequency or intensity that is greater than would be expected for an individual at that developmental level (Luk, 1985). Children with ADHD are at increased risk for functional impairments including poor coordination (Fliers et al., 2008; Kadesjo & Gillberg, 1998; Wilson, 2005), social skill deficits (Booster, DuPaul, Eiraldi, & Power, 2012; de Boo & Prins, 2007; DuPaul, McGoey, Eckert, & VanBrakle, 2001), and learning difficulties (Hinshaw, 1992; Rapport, Scanlan, & Denney, 1999). These difficulties begin in early childhood and can persist into adulthood (Gadow, Sprafkin, & Nolan, 2001; Hechtman, 2000; Wilens, Biederman, & Spencer, 2002).

Prevalence estimates have found ADHD to be one of the most commonly comorbid disorders in individuals with ASD. Simonoff and colleagues (2008) reported a rate of 28.2% of children with ASD who had comorbid ADHD. Likewise, Leyfer and colleagues (2006) reported a rate of 30.6% among children with ASD. Other researchers report even higher rates of comorbid ADHD ranging from 59% to 78% (Goldstein & Schwebach, 2004; Holtmann, Bolte, & Poustka, 2007; Lee & Ousley, 2006). The two disorders have some shared symptoms, but they also have numerous disorder-specific symptoms, supporting the argument that they are distinct and separable diagnoses (Chnstakou et al., 2013; Di Martino et al., 2013; van der Meer et al., 2012). Identifying comorbid ADHD symptoms in children with ASD is critical, because they compound the impairments leading to poorer prognosis (Frazier et al., 2001). Sinzig, Walter, and Doepfner (2009) investigated ADHD symptoms in 83 children with ASD. They found that symptoms of hyperactivity were significantly correlated with severity of communication deficits and symptoms of inattention were significantly correlated with stereotyped behaviors.

Additionally, children with ASD and ADHD exhibit significantly more externalizing symptoms

(Tureck, Matson, May, & Turygin, 2013) and functional impairments (Yerys, Wallace, Sokoloff, Shook, James, & Kenworthy, 2009) than children with only one of the disorders.

1.6.2 Anxiety Disorders

Anxiety and phobic disorders are the most commonly comorbid disorders in individuals with ASD (Leyfer et al., 2006; Simonoff et al., 2008). However, some researchers have argued that anxiety is not always a true comorbid disorder, but rather a manifestation of ASD symptomatology (Kerns & Kendall, 2013). In particular, social avoidance, as well as ritualistic and compulsive behavior are central to ASD and some anxiety disorders (Wood & Gadow, 2010). Leyfer and colleagues (2006) proposed that social avoidance in ASD and social anxiety are separable, because children with ASD avoid social situations due to non-social factors (e.g., loud noises) as opposed to intentionally avoiding social interactions. Further, generalized anxiety disorder (GAD) was found to be different from the anxiety inherent to ASD symptomatology, because GAD involves anxiety about multiple things as opposed to anxiety about transitions or insistence on sameness. Diagnosing anxiety disorders in children with ASD is further confounded by communication deficits, which prohibit many children with ASD from expressing internalizing symptoms (Davis et al., 2011).

The prevalence of anxiety disorders in children with ASD is estimated to be between 11% and 84% (White, Oswald, Ollendick, & Scahill, 2009). The rates are highly variable due to the unfortunate tendency for clinicians not to diagnose anxiety disorders (especially social phobia and obsessive compulsive disorder) in children with ASD (White et al., 2009). However, they are still consistently higher than in typically developing children, where the prevalence is estimated to be between 2.2% and 27% (Costello, Egger, & Angold, 2005). Leyfer and colleagues (2006) reported that specific phobias were the most common comorbid disorder with

ASD occurring at a rate of 44.3%. Obsessive compulsive disorder (OCD) was the second most common, occurring at a rate of 37.2%. Simonoff and colleagues (2008) conversely found that social phobia was the most commonly comorbid disorder, occurring in 29.2% of children with ASD. Despite these diagnostic discrepancies, researchers are in agreement that anxiety disorders occur at a high rate in children with ASD and cause additional behavioral difficulties (Kim, Szatmari, Bryson, Steiner, & Wilson, 2000).

1.6.3 Mood Disorders

In an early study on comorbidity in individuals with Asperger's Disorder, Wing (1981) reported that depression was the most common comorbid psychiatric condition. Depression is more common in adolescents and adults with ASD than children (Ghaziuddin et al., 1998). One explanation for this discrepancy might be related to how symptoms present at different ages. For example, Leyfer and colleagues (2006) reported that only 10% of children with ASD met criteria for Major Depressive Disorder (MDD); however, this rate increased to 24% for 'subsyndromal' (i.e., falls just short of the diagnostic criteria, but is significantly impairing) major depression. These subsyndromal cases are important to consider, because some diagnostic criteria may not be applicable to children with ASD due to cognitive, developmental, or language limitations (Kendler & Gardner, 1998). This discrepancy was confirmed by Simonoff and colleagues (2008), who reported that only around 1% of children with ASD met criteria for MDD; however, an additional 10.9% had experienced a significant period of subsyndromal depression or irritability.

Diagnosing depression in individuals with ASD can pose a challenge to clinicians as the child will rarely report depressed mood, so the clinicians must rely on parent report of increased irritability, loss of interest, and other features of the disorder (Stewart, Barnard, Pearson, Hasan,

& O'Brien, 2006). Another concern is that symptoms of ASD may mask some of the symptoms of depression (Magnuson & Constantino, 2011). For example, children with ASD who develop depression exhibit increases in ritualistic behavior, obsessions, and hyperactivity (Ghaziuddin et al., 2002). Thus, caregivers must be observant of increasing ASD symptom severity, because few children with ASD are able to effectively communicate feelings of depressed mood or loss of interest. Researchers have also noted that depression in children with ASD often leads to an increase or onset of challenging behavior (e.g., SIB, aggression) highlighting the need for effective diagnostic measures tailored to this population (Clarke, Baxter, Perry, & Prasher, 1999; Long, Wood, & Holmes, 2000). One theory is that the increase in SIB is related to the child's increasingly negative self-view (Skinner, Ng, McDonald, & Walters, 2005; Stewart et al., 2006).

Bipolar disorder in children with ASD has just recently begun to receive attention from researchers. Frazier, Doyle, Chiu, and Coyle (2005) presented the case of a 13-year-old boy with Asperger's Disorder who was exhibiting extreme violence and aggression. Upon evaluation, the clinicians reported sleep disturbances, irritability, obsessions, pressured speech, and racing thoughts. Most concerning, he had recently presented with symptoms of depression and expressed suicidal ideation. The patient was diagnosed with Bipolar Disorder, Mixed, with Psychotic Features. Upon the introduction of Lithium treatment, the patient began exhibiting more regulated mood symptoms and fewer aggressive behaviors. Frazier and colleagues provided this case presentation to encourage researchers and clinicians to consider diagnosing comorbid bipolar disorder in adolescents with ASD as opposed to attributing the symptoms to the ASD diagnosis.

Overall rates of hypomanic/manic disorders (i.e., bipolar disorder, hypomanic episodes, manic episodes, and mixed episodes) are relatively low in children with ASD with less than 1%

meeting diagnostic criteria (Leyfer et al., 2006). The prevalence is considerably higher in adolescents with high-functioning ASD. Munesue, Ono, Mutoh, Shimoda, Nakatani, and Kikuchi (2008) assessed 44 adolescents with ASD and an IQ greater than 70 and reported that 27% met criteria for bipolar disorder. This is in line with other researchers who reported that rates of mood disorders significantly increased in adolescence as compared to early childhood (Ming, Briacombe, Chaaban, Zimmerman-Bier, & Wagner, 2008).

1.6.4 Motor Disorders and Tourette Syndrome

The rates of motor disorders are higher in children with ASD than among typically developing children. At present, these associated features are not diagnostic, but can cause impairments in activities of daily living. Researchers have long reported that children with ASD experience clumsiness, postural abnormalities, and fine or gross motor deficits at higher rates than typically developing children (Bauman, 1992; Fournier et al., 2010; Rapin, 1997). These deficits are noticeable prior to 2 years of age and often persist across the lifespan (Teitelbaum, Benton, Shah, Prince, Kelly, & Teitelbaum, 2004). Green and colleagues (2009) evaluated movement skills in 101 children with ASD and found that 79% exhibited significant impairments and an additional 10% exhibited borderline impairments.

Tourette syndrome (TS) is a specific type of motor disorder that is characterized by chronic motor and vocal tics that occur at random times. Researchers estimate that the current rate of TS is between 0.3 and 1% of the population (Robertson, Eapen, & Cavanna, 2009; Scharf, Miller, Matthews, & Ben-Shlomo, 2012). In the past few decades, researchers have focused on the relationship between TS and ASD working under the assumption that the two disorders likely have some common etiological factors (Canitano & Vivanti, 2007). Some of the behavioral symptoms of the two disorders directly overlap, including echolalia, palilalia (i.e., involuntary

repetition of syllables, words, or phrases using contextually correct speech), and obsessions/compulsions (Stern & Robertson, 1997). Moreover, individuals with both disorders exhibit repetitive motor movements, although these are typically spontaneous in those with TS and more stereotyped in those with ASD (Rapin, 2001). Canitano and Vivanti (2007) evaluated the prevalence of tic disorders and TS in 105 children with ASD. Overall, 22% of the children presented with tic disorders, which was broken down to 11% with TS and 11% with chronic motor tics. Simonoff and colleagues (2008) reported a much lower rate of 4.8% of children with ASD meeting criteria for TS. Again, an additional 9% of children with ASD exhibited chronic tic disorder. Considering this high degree of comorbidity, more research is needed to delineate the association between these two disorders.

1.6.5 Disruptive Behavior Disorders

Oppositional defiant disorder (ODD) is a disruptive behavior disorder characterized by a pattern of disobedient, hostile, and purposefully defiant behavior toward adults (APA, 2013). Children with ODD often exhibit temper tantrums, deliberately annoy others, blame others for own mistakes, and are irritable (Hamilton & Armando, 2008). Leyfer and colleagues (2006) reported that ODD has a prevalence of 7% in children with ASD, which is similar to the prevalence in the overall population of 8.5% (Kessler, Beglund, Demler, Jin, Merikangas, & Walters, 2005). However, other researchers have estimated the prevalence of ODD in ASD to be much higher at around 28% (Gadow, DeVincent, Pomeroy, & Azizian, 2004; Simonoff et al., 2008). Gadow, DeVincent, and Drabick (2008) investigated whether ODD was separable from other symptoms of ASD and found that ODD is a distinguishable behavioral syndrome resulting in more severe challenges than in children with ASD who do not have ODD. Conduct disorder is a more severe form of disruptive behavior where the child engages in a pattern of behavior that

violates the basic rights of others and other societal norms. Simonoff and colleagues (2008) estimated the rate of conduct disorder to be 3.2% of children with ASD.

1.7 Co-Occurring Challenging Behavior

Children with ASD often exhibit one or more challenging behaviors requiring intervention (Matson & Minshawi, 2007). Challenging behavior is defined as “culturally abnormal behavior(s) of such intensity, frequency or duration that the physical safety of the person or others is likely to be placed in serious jeopardy, or behavior which is likely to seriously limit use of, or result in the person being denied access to, ordinary community facilities” (Emerson, 2001, p. 3). Specific examples of challenging behavior are physical aggression, pica (i.e., eating inedible objects), SIB, tantrum behavior, and stereotypies. These behaviors are considered to co-occur with ASD, because they are central to conceptualizations of the disorder but are not core diagnostic features (Matson & Nebel-Schwalm, 2007a). An estimated 82% of children with ASD display challenging behavior and 96% of these exhibit more than one form (Murphy, Healy, & Leader, 2009). Furthermore, severity of ASD symptoms is significantly and positively correlated with the number and intensity of challenging behavior, such that those with more severe ASD exhibit more challenging behavior (Matson, Wilkins, & Macken, 2008).

Researchers have investigated specific risk factors for the development of challenging behavior in individuals with ASD. One factor appears to be the age of the individual, with challenging behavior reaching a peak in adolescence and early adulthood (Holden & Gitlesen, 2006). However, this finding does not appear to be universally true, as SIB actually tends to decrease with age (Baghdadli, Pascal, Grisi, & Aussilloux, 2003). One theory for this decrease is that children with ASD develop better adaptive skills that result in a decrease in SIB. Hartley,

Sikora, and McCoy (2008) found that non-Caucasian children exhibited more challenging behaviors than Caucasian children; however they did not control for SES.

SIB has been a main focus of research on individuals with ASD and ID since the 1960s due to the life-threatening nature of some of the behaviors (Carr, 1977). The term is used to describe any behavior that produces physical damage to the individual's own body (Tate & Baroff, 1966). Some examples include eye-gouging, head banging, self-scratching, hand biting, and skin picking. Pica is typically considered to be a form of SIB, as it can cause significant harm to the individual (Falcomata, Roane, & Pabico, 2007; Roane, Kelly, & Fisher, 2003). SIB often occurs in a repetitive manner leading to debilitation, disfigurement, or in extreme cases—death (Rojahn, Schroeder, & Hoch, 2008). Researchers have hypothesized that there is a biological etiology of SIB, but there is no definitive explanation at present (Matson & LoVullo, 2008). SIBs are currently conceptualized in terms of a functional assessment and then treated with behavioral techniques.

Aggression is the most common challenging behavior among children with ASD, occurring at a rate of between 15% and 68% (Kanne & Mazurek, 2011; Lecavalier, 2006; Mazurek, Kanne, & Wodka, 2013). This potentially dangerous behavior is also the most common reason that caregivers seek behavioral or psychotropic treatment for individuals with ASD (Parikh, Kolevzon, & Hollander, 2008; Tsakanikos, Costello, Sturmey, & Bouras, 2007). Numerous explanations have been offered to account for the high rate of aggression among these individuals, including sleep problems (Chervin, Dillon, Archbold, & Ruzicka, 2003) and sensory impairments (Shochat, Tzischinsky, & Engel-Yeger, 2009). In children with ASD, aggression serves as a functional behavior that manipulates their environment and is often inadvertently reinforced by their caregivers (Foxy & Meindl, 2007). For example, when a child acts

aggressively towards a peer at school, the child gets attention from the teacher, principal, and his parents. Tantrum behavior, which can include kicking, screaming, and throwing objects, is often grouped under the broader category of aggression (Matson, 2009). Both tantrum behavior and other forms of aggression appear to be primarily maintained by attention (Dawson, Matson, & Cherry, 1998).

Stereotyped behaviors are considered to be the least problematic of the challenging behaviors as they rarely result in injury or damage (Matson, Benavidez, Compton, Paclawski, & Baglio, 1996). However, it is important to note that stereotypic behavior can interfere with skill acquisition and has been hypothesized to be a pre-cursor to SIB (Morrison & Rozales-Ruiz, 1997; Oliver, Murphy, & Corbett, 1987). Examples of stereotypies are repetitive vocalizations, rocking body, heel and toe walking, rubbing body parts, spinning body, hand flapping, and hand or finger posturing. Overall, stereotypic behaviors are highly heterogeneous and occur in individuals with ASD of all ages (Cunningham & Schreibman, 2008; Smith & Van Houten, 1996). At present, they are the only challenging behavior that is included in the diagnostic criteria for ASD (APA, 2013) and stereotyped behavior is more common in lower functioning individuals (Matson, Kiely, & Bamburg, 1997; Rojahn, Matlock, & Tasse, 2000). The most commonly mentioned maintaining variable for stereotypy is self-stimulation (i.e., automatic reinforcement); however, it can occur for other reasons (Rapp & Vollmer, 2005; Rogers & Oznoff, 2005).

1.8 Assessing Comorbidity

As previously stated, when individuals are diagnosed with ASD this is typically the primary disorder and all other conditions would be considered secondary. However, regardless of their secondary status, comorbid disorders often require tailored treatments above and beyond

the standard behavioral treatments for the core symptoms of ASD (Matson & Nebel-Schwalm, 2007). Assessment of comorbidity in this population has been problematic for clinicians as the majority of measures were developed for use with typically developing children (Leyfer et al., 2006). In many instances, these measures are insufficient to truly determine if the child's symptoms are due to a comorbid disorder or better accounted for by symptoms of ASD. Thus, researchers have worked to develop measures that are tailored to the ASD population, but still screen for a variety of emotional and behavioral concerns. Examples of specific assessment scales are provided in more detail in this section.

The *Child Behavior Checklist (CBCL)* was originally developed in the 1970s by Thomas Achenbach as a broadband screener of psychopathology in school-aged children. He developed this scale as a standardized method for parents to quickly report on observable behaviors that may be problematic (Achenbach, 1978). It served as an expansion upon the *Behavior Problem Checklist* (Achenbach, 1966; Quay & Peterson, 1967), which was designed for research purposes. The *CBCL* reflected diagnostic changes that allowed for the diagnosis of a larger number of psychological disorders in children, as prior to the publication of the *DSM-II* (APA, 1968) the only childhood disorders were adjustment reaction of childhood and childhood schizophrenia (Achenbach, 1978).

The most recent version of the *CBCL* (Achenbach & Rescorla, 2001) has been evaluated in multiple child populations for diagnostic validity. One of these populations was children with ASD, especially those who are higher functioning (i.e., $IQ > 70$). Biederman and colleagues (2010) investigated the effectiveness of the *CBCL* clinical scales in discriminating between children with and without ASD. They found that children with ASD consistently exhibited elevations on the Withdrawn/Depressed, Social Problems, and Thought Problems scales. Ooi,

Rescorla, Ang, Woo, and Fung (2011) reported similar findings from their study that compared children with ADHD, children with ASD, typically developing children, and children who were referred but did not receive a diagnosis. They constructed an ASD scale that consisted of nine items from those three subscales of the *CBCL*. Further evidence is needed to evaluate the psychometrics of a possible ASD clinical scale; however, elevations on those three subscales may indicate to clinicians that the child needs further assessment to rule out a possible ASD.

In practice, the *CBCL* is often used to assess for possible comorbid disorders in children already diagnosed with ASD. Pandolfi, Magyar, and Dill (2012) evaluated the psychometric properties of the measure in 122 children with ASD. They found that the *CBCL* had good sensitivity, but lacked specificity in detecting comorbid psychopathology in this population. Thus, the results of the *CBCL* should be used in conjunction with data from clinical interview and further assessment when diagnosing comorbid disorders in children with ASD. The psychometric properties of the *CBCL* are discussed in more detail in the description of the current study.

The *Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004)* was also developed to assess for problem behaviors and adaptive skills in children. The researchers included children and adolescents diagnosed with ASD in their initial sample, and the scale has since been commonly used with children with ASD. The measure provides nine clinical scales (i.e., aggression, anxiety, attention problems, atypicality, conduct problems, depression, hyperactivity, somatization, and withdrawal) and five adaptive scales (i.e., functional communication, activities of daily living, adaptability, leadership, and social skills). The scales load onto four composites: Behavioral Symptoms Index, Adaptive Skills, Externalizing Problems, and Internalizing Problems. The parent report form of the *BASC-2* had

internal consistency scores ranging from .90 to .95 (Reynolds & Kamphaus, 2004). Furthermore, alpha coefficients for the different scales had a mean of .84. The *BASC-2* demonstrated convergent validity with the *CBCL*.

Volker and colleagues (2010) utilized the *BASC-2* with a population of children with high-functioning ASD. These children were significantly elevated on the Atypicality, Withdrawal, and Behavioral Symptoms Index in comparison to typically developing children. When evaluating a broader ASD group with the *BASC-2*, Mahan and Matson (2011a) found that children with ASD scored significantly higher on all clinical subscales and composites than typically developing children. Likewise, the children with ASD scored significantly lower on the adaptive subscales and composite.

Contrary to the *BASC-2* and *CBCL*, the *Autism Comorbidity Interview-Present and Lifetime Perspective (ACI-PL)*; Leyfer et al., 2006) was developed to specifically assess for psychopathology in children with ASD. The authors of the measure modified the *Kiddie Schedule for Affective Disorders and Schizophrenia* (Puig-Antich & Chambers, 1978) to reflect the discrepancies in the presentation of comorbid symptoms in this population. The authors emphasized the importance of establishing a baseline for each child and then asking the parents about changes from baseline that might indicate the presence of a comorbid disorder (Leyfer et al., 2006). The interview also allows for flexibility in the questioning to make the measure more applicable to the cognitive capacities of the child (i.e., only probing about guilt in higher functioning individuals). The psychometric properties of this measure are still unknown at this time.

The *Developmental Behavior Checklist (DBC)*; Einfeld & Tonge, 1992) was developed to assess behavioral and emotional disturbances in children with ID and developmental disabilities.

The parent report measure produces a Total Behavior Problem Score and subscale scores (disruptive, antisocial, self-absorbed, communication disturbance, anxiety, and social relating). The measure has been shown to have high inter-rater reliability, test-retest reliability, and internal consistency for identifying comorbid disorders in children with ID (Einfeld & Tonge, 1995). The *DBC* also has an Autism Screening Algorithm consisting of 26 items that can be utilized for diagnostic purposes. Bereton, Tonge, Mackinnon, and Einfeld (2002) evaluated 180 children with ASD and 180 matched controls using the algorithm and found that a cutoff score of 17 could reliably discriminate between children with ASD and controls. However, Witwer and Lecavalier (2007) found that the Autism Screening Algorithm had poor specificity, especially in instances where the child exhibited a high number of challenging behaviors.

The *Autism Spectrum Disorders-Child Version (ASD-C)*; Matson & Gonzalez, 2007a,b,c) was developed to be a cost-efficient screen for atypically developing children with possible ASD. It is the first measure to be specifically designed to assess comorbid emotional and behavioral disorders in children with ASD. The assessment battery serves multiple purposes: 1) assesses symptoms of ASD; 2) assesses for possible comorbid disorders; 3) identifies challenging behavior; and 4) monitors changes in presentation over time. Specific items for the measure were generated from a comprehensive literature review and incorporation of diagnostic standards. The items were modified after initial psychometric evaluation to increase the reliability and validity of the measure. Both typically developing children and children with ASD were included in the standardization sample to allow for a broader application. The psychometric properties of this assessment battery are discussed in more detail in the description of the current study.

CHAPTER 2. PURPOSE

In many settings, especially outpatient clinics and the school system, comprehensive clinical evaluations are not always feasible. Thus, parent rating scales are the most commonly used measures of child psychopathology in current practice (Hunsley & Mash, 2008). This represents a shift from the extensive test batteries that were previously employed by psychologists working with children, which consisted of intelligence testing, achievement testing, projective personality tests, and specific measures of psychopathology (Mash & Hunsley, 2004). These lengthy evaluations were time and cost intensive, which made such testing unattainable for many families with children at risk for developmental disabilities, behavioral, and affective disorders (Percevic, Lambert, & Kordy, 2004). Data from the National Survey of American Families revealed that only 21% of children in need of services were actually being seen by mental health professionals (Kataoka, Zhang, & Wells, 2002). Newer, broadband measures have been developed to allow clinicians to effectively screen for a variety of psychological disorders as the first step in a comprehensive assessment (Mash & Hunsley, 2005). These measures increase the likelihood that children, especially from underserved areas, will receive necessary mental health services.

The first psychometrically sound broadband measure of psychopathology in children was the *CBLC* (Achenbach, 1978). The revised versions of this measure are still frequently administered to parents as a brief screen for a range of psychological and behavioral problems. In cases where the child is suspected to have a developmental disability, the child should also be administered measures that are tailored to the ASD population. Thus, the *ASD-C* (Matson & Gonzalez, 2007a,b,c) was developed to address the need for a screening measure for symptoms of ASD, comorbid disorders, and challenging behavior in children with possible ASD. To date,

numerous publications have examined the reliability and validity of both of these measures, but none have compared the two measures.

The goal of the study was to evaluate the construct validity of both measures. Both the broadband (e.g., Externalizing and Internalizing) and narrowband (e.g., *ASD-C* Worry/Depressed and *CBCL* Anxious/Depressed) scales of the *ASD-C* and *CBCL* were examined to evaluate similarities and differences in how these measures assess behaviors. Additionally, the diagnostic accuracy of various subscales and composites from the *ASD-C* and *CBCL* was evaluated. As broad screeners for psychopathology, the ability of these measures to correctly identify disorders is critical. Early identification precipitates the best long-term outcomes for children with developmental disabilities or psychological disorders. Numerous studies have documented that low SES is a risk factor for psychological disorders in children (Bringewatt & Gershoff, 2010; Knitzer, 1996; McLearn, Knitzer, & Carter, 2007). Thus, establishing the validity of these inexpensive screening measures helps to provide services to children and their families whom face numerous barriers to care. These measures are especially useful for clinicians who are not specifically trained in identifying developmental disabilities and comorbid disorders. They allow a pediatrician or teacher to identify that a child is at risk based on his elevation on a particular subscale. This child can then be referred to a psychologist or psychiatrist for further evaluation. Evaluating the diagnostic validity of these measures ensures their utility as low-cost broadband screeners of psychopathology in children.

CHAPTER 3. METHOD

3.1 Participants

Participation in this study was based on participation in a larger research study, so the data had been collected over a period of multiple years. Participants for this study were 114 children who received a developmental disability, gifted, or psychoeducational assessment at a university clinic in the southeastern United States between the ages of 6 and 16. A power analysis program, G*Power (Erdfelder, Faul & Buchner, 1996), was utilized to determine the number of participants needed to have enough power to run this study. When looking at a two-tailed correlation utilizing Spearman's Rho with a medium effect size of .30, a total sample size of 84 was needed. The standard in the behavioral sciences is to set the significance level at $\alpha = .05$, which sets the power at .80 (Hinkle, Wiersma, & Jurs, 2003). A two-tailed test was utilized, because both positive and negative correlations could have been significant. In order to be included in the current study, parents/guardians of participants must have completed the *CBCL* and *ASD-C*. Participants were excluded if more than 3 items were missing on either of the measures. Additionally, participants were excluded if there was demographic information missing (i.e., age, gender, race).

The sample consisted of 74 males (64.9%) and 40 females (35.1%). Participants ranged in age from 6-16 years old ($M = 9.72$, $SD = 2.76$). The majority of participants self-identified as White, non Hispanic (81.6%). Table 1 presents the demographic characteristics of the participants. Overall, 14 children were diagnosed with ASD, 29 children had no diagnosis, and 71 children were diagnosed with some form of psychopathology yet still typically developing. Table 2 presents a breakdown of the children with psychopathology without ASD. Diagnoses of psychopathology were made using a psychoeducational battery that included the *Anxiety*

Disorders Interview Schedule for DSM IV – Child and Parent version (Silverman & Albano, 1996), normative child, parent, and teacher rating scales (e.g., Achenbach scales; Achenbach & Rescorla, 2001; Conners’ scales; Conners, 1997), and standardized intellectual and achievement tests.

Table 1
Demographic Characteristics by Diagnostic Group (N=114)

Demographic Characteristics	ASD (n=14)	Non-ASD (n=29)	Psychopathology (n=71)
Age (in years)			
Mean (SD)	9.93 (3.22)	9.66 (3.00)	9.70 (2.60)
Range	6-16	6-16	6-15
Gender, %			
Male	92.9%	69.0%	57.7%
Female	7.1%	31.0%	42.3%
Race/Ethnicity, %			
Caucasian	85.7%	82.8%	80.3%
African-American	7.1%	13.8%	14.1%
Hispanic	7.1%	0%	1.4%
‘Other’	0%	3.4%	4.2%

Table 2
Diagnoses for Children in the Psychopathology Group (N=71)

Psychological Diagnoses	
Anxiety Disorder	33.8%
Mood Disorder	2.8%
Attention-Deficit/Hyperactivity Disorder	33.8%
Oppositional Defiant Disorder	14.1%
Other	15.5%

3.2 Measures

3.2.1 Child Behavior Checklist

The *Child Behavior Checklist (CBCL;* Achenbach & Rescorla, 2001) is a parent rating scale that assesses for a broad range of possible psychological disorders in children and

adolescents. The measure is available in two forms for different age groups. Only the School Age form for children 6-18 years old was utilized in the current research. The scale consists of 112 items that were rated by the parents and/or guardians on how true the statement is about their children: 0 = “Not true,” 1 = “Somewhat or Sometimes True,” and 2 = “Very True or Often True.” The informants were prompted to assess their children’s behavior over the past 6 months. The scale requires between 10 and 20 minutes to complete, with the discrepancy reflecting how quickly the informant can read each item. In this study, the measure was computer scored by doctoral level graduate students working under the supervision of a licensed clinical psychologist. The computer scoring program produces norm-referenced T-scores for symptom scales, DSM-oriented syndrome scales, and behavior problem scales. However, raw scores were utilized for the purposes of this study to allow for comparison with the *ASD-C*.

A confirmatory factor analysis produced an eight-factor structure (i.e., Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, and Aggressive Behavior) for the symptom scales (Achenbach & Rescorla, 2001). All eight subscales were considered for the current research. The behavior problem scales consist of Internalizing Problems, Externalizing Problems, and Total Problems, of which the Internalizing and Externalizing composites were also utilized in the current study. The six DSM-oriented syndrome scales and the Competence scales were not considered in the current analyses.

The *CBCL* has been shown to have excellent psychometric properties. The inter-rater reliability was .96, test-retest reliability was .95, and internal consistency ranged from .78 to .97 (Achenbach & Rescorla, 2001). The test creators used structural equation modeling to assess the effect of demographic variables on syndrome ratings. They found a significant effect of SES,

such that children of lower SES had higher problem scores. To assess the construct validity of the measure, researchers compared *DSM-IV* diagnoses and significant syndrome scale elevations. Kappa coefficients ranged from .27 for the Anxious/Depressed scale and diagnoses of Anxiety disorder to .80 for the Attention Problems scale and diagnoses of ADHD (Achenbach & Rescorla, 2001). To establish convergent and discriminant validity, the *CBCL* was compared to the Conners scales (Conners, 1997) and the *BASC* (Reynolds & Kamphaus, 1992) with results indicating that the measures assessed similar underlying constructs.

The ASD scale is an unofficial subscale of the *CBCL* comprised of 9 items: 1. Acts too young for his/her age; 25. Doesn't get along with other kids; 29. Fears certain animals, situations; 42. Would rather be alone than with others; 46. Nervous movements or twitching; 66. Repeats certain acts over and over; 79. Speech problems; 84. Strange behavior; 111. Withdrawn, doesn't get involved with others (Ooi et al., 2011). This was included in this study for the purposes of assessing the diagnostic accuracy of the *CBCL* in identifying ASD.

3.2.2 Autism Spectrum Disorders – Child Version

The *Autism Spectrum Disorders – Child Version (ASD-C)* consists of three separate scales that can be administered in combination to provide a comprehensive assessment of core symptoms, comorbid disorders, and behavior problems in children with ASD. The *Autism Spectrum Disorder – Diagnostic Child Version (ASD-DC; Matson & Gonzalez, 2007c)* consists of 40 items that assess symptoms of ASD in children from ages 2-16 years. Informants, who were the parent and/or guardian of the child, rated the items on a 3-point scale where 0 = “not different; no impairment,” 1 = “somewhat different; mild impairment,” and 2 = “very different; severe impairment.” The measure has been shown to have good psychometrics with test-retest and inter-rater reliability at .77 and .67 respectively (Matson, Gonzalez, Wilkins, & Rivet, 2007).

Additionally, the *ASD-DC* was able to distinguish between children with atypical development and no diagnosis at a rate of 84.3% and between atypical development and ASD at a rate of 87.8% (Matson, Gonzalez, & Wilkins, 2009). Matson, Boisjoli, and Dempsey (2009) established a four factor structure for the items: social relationships, nonverbal communication/socialization, verbal communication, and restricted interests/insistence on sameness.

The convergent validity of the *ASD-DC* was examined utilizing the *Childhood Autism Rating Scales (CARS)* (Schopler, Reichler, & Rochen-Renner, 1988) and the *Autism Diagnostic Interview Revised (ADI-R)* (Lord, Rutter, & LeCouteur, 1994). The *CARS* and *ASD-DC* had all significant correlations for related factors ranging from .37 to .68 and overall convergent validity was moderately high, $r = .54$ (Matson, Mahan, Hess, Fodstad, & Neal, 2010). Furthermore, the *ASD-DC* was found to have higher sensitivity and specificity, 76.5% and 95.0% respectively, than the *CARS* (58.8% and 85.0% respectively). Likewise, all correlations between the *ASD-DC* and *ADI-R* were statistically significant ranging from .48 to .61 and confirming convergent validity (Matson, Hess, Mahan, & Fodstad, 2010). In addition, the *ASD-DC* had a sensitivity of 73% and specificity of 67%, while the *ADI-R* had a sensitivity of 46%, but a specificity of 100%.

The *Autism Spectrum Disorders-Comorbidity Child (ASD-CC)* (Matson & Gonzalez, 2007b) was developed to measure commonly comorbid symptoms in children with ASD. The measure consists of 39 items that are rated by the caregiver as 0 = “not a problem or impairment; not at all”; 1 = “mild problem or impairment”; 2 = “severe problem or impairment”; or X = “does not apply or don’t know.” Specific items screen for conditions including ADHD, depression, eating disorders/difficulties, OCD, specific phobias, tic disorders, and conduct disorder. The results of an exploratory factor analysis revealed a seven-factor structure: Tantrum Behavior, Repetitive Behavior, Worry/Depressed, Avoidant Behavior, Under-Eating, Conduct, and Over-

Eating (Matson, LoVullo, Rivet, & Boisjoli, 2009). The internal consistencies of the factors ranged from .70 to .86 (Matson et al., 2009). The measure also demonstrated convergent and discriminant validity with the *BASC-2* (Matson et al., 2009). There are established cut-off scores for the *ASD-CC* that categorize a score as either “no/minimal impairment,” “moderate impairment,” or “severe impairment” (Thorson & Matson, 2012). These cut-offs allow the measure to be used more readily as an initial screening instrument for psychopathology in children with ASD.

The *Autism Spectrum Disorders-Behavior Problems Child (ASD-BPC)*; Matson & Gonzalez, 2007a) was designed to identify common problem behaviors in children with ASD. Specific items target SIB, aggression, pica, stereotypies, and property destruction. Caregivers rate each behavior as 0 = “not different; no impairment,” 1 = “somewhat different; mild impairment,” or 2 = “very different; severe impairment.” An exploratory factor analysis revealed a two-factor structure with Externalizing Behavior accounting for 36.72% of variance and Internalizing Behavior accounting for 9.94% of variance (Matson, Gonzalez, & Rivet, 2008). Mean inter-rater reliability was .49 with 92% agreement, while mean test-retest reliability was .64 with 92% agreement. Intercorrelations between items ranged from .04 to .63 with a mean of .32. Moreover, the *ASD-BPC* had an internal consistency of $\alpha = .90$. The *ASD-BPC* has been demonstrated to have convergent and discriminant validity with the *BASC-2* (Mahan & Matson, 2011b). The aggression, hyperactivity, and atypicality subscales of the *BASC-2* were all significantly correlated with the *ASD-BPC* externalizing scale, $r = .61$, $r = .52$, and $r = .48$, respectively. The atypicality subscale was also significantly correlated with the *ASD-BPC* internalizing scale, $r = .51$.

3.3 Procedure

Informed consent from the parents and/or guardians and assent from the participants (when developmentally appropriate) were obtained prior to enrollment in the study. The *CBCL* and *ASD-C* were completed by the parents and/or guardians of the participants. Directions were printed clearly on all measures and the informants were encouraged to discuss any questions or issues with their clinicians when they arose. Graduate clinicians trained in the administration and scoring procedures for all measures made follow-up calls when necessary to fill in missing items. The measures were scored by the clinicians and entered into a larger research database. Supervision was provided throughout the data collection process by a licensed clinical psychologist. The data used in this study was collected from 2008 to the present. The study was approved by the Louisiana State University Institutional Review Board.

3.4 Statistical Analyses

Prior to calculating statistical findings, item content was compared between the *CBCL* and *ASD-C* composites and subscales to demonstrate convergent validity. Items were considered a match if they had the same operational definition or if the item represented a closely related behavior to an item from the other measure when there was not an exact match.

To assess the convergent and divergent validity of the *CBCL* and *ASD-C* a correlational matrix similar to the Multitrait-Multimethod Matrix developed by Campbell and Fiske (1959) was employed. The content analysis helped guide the selection of scales and subscales to compare for assessing convergent validity. Spearman's Rho was utilized instead of Pearson's R, because the sample had an abnormal distribution. The distribution was specifically analyzed using the Kolmogorov-Smirnov (KS) tests of normality for all subscales and scales utilized in

this study and found to violate assumptions of normality. Bivariate correlations were calculated using $p < .05$ to determine significance.

To further assess the construct validity of both measures, receiver operating characteristic (ROC) curves were generated by plotting the relationship of the true positivity (sensitivity) and the false positivity (1 - specificity) of the subscales. ROC analyses result in an area under the curve (AUC), which indicates the degree to which the subscale predicted a binary classification (e.g., with or without disorder) (Hanley & McNeil, 1982). For the current study, AUC values were interpreted according to the following designations: .50-.70 = poor, .70-.80 = fair, .80-.90 = good, and .90-1.00 = excellent. Subscales and composites from the *CBCL* and *ASD-C* were analyzed using ASD, Anxiety Disorder, ADHD, and ODD as the positive actual states.

3.5 Hypotheses

Based on the existing literature on the validity of the both the *CBCL* and *ASD-C* separately, several predictions were made in regards to the outcomes of this study. First, convergent validity would be confirmed by a high level of correlation between similar subscales. The Withdrawn/Depressed, Social Problems, and Thought Problems scales from the *CBCL* were hypothesized to correlate highly with overall ASD severity as measured by the *ASD-DC*. Children with high scores on the *ASD-DC* were predicted to have higher scores on the three subscales than children with none or only a small number of ASD symptoms. It was further expected, that the Worry/Depressed subscale of the *ASD-CC* would correlate positively with the Anxious/Depressed and Internalizing Problems subscales of the *CBCL*. The Avoidant subscale of the *ASD-CC* would correlate positively with the Withdrawn/Depressed and Internalizing Problems subscales of the *CBCL*. The Tantrum Behavior subscale of the *ASD-CC* was expected to correlate positively with the Aggressive Behavior and Externalizing Problems subscales of the

CBCL. The Conduct subscale of the *ASD-CC* was likewise expected to correlate positively with the Rule-Breaking Behavior and Externalizing Problems subscales of the *CBCL*. Further, the Externalizing subscale of the *ASD-BPC* was expected to correlate positively with the Externalizing Problems subscale of the *CBCL*. Second, discriminant validity would be confirmed by negative or low correlations between dissimilar subscales. The Internalizing Problems subscale of the *CBCL* was hypothesized to demonstrate discriminant validity with the Internalizing and Externalizing subscales of the *ASD-BPC*. Additionally, the Repetitive Behaviors subscale of the *ASD-CC* would demonstrate divergent validity with the Internalizing Problems subscale of the *CBCL*.

As for the diagnostic accuracy of the measures, the Diagnostic Total (i.e., sum of items from *ASD-DC*) of the *ASD-C* was hypothesized to accurately predict which participants were diagnosed with ASD by the licensed psychologist. Second, it was predicted that the Withdrawn/Depressed, Social Problems, and Thought Problems subscales from the *CBCL* would accurately identify children diagnosed with ASD. The ASD scale from the *CBCL* was also predicted to accurately identify children with ASD.

Furthermore, the Anxious/Depressed subscale and Internalizing composite of the *CBCL* were predicted to accurately identify children diagnosed with an Anxiety Disorder. Likewise the Worry/Depressed subscale of the *ASD-C* was predicted to accurately identify children diagnosed with an Anxiety Disorder. As for ADHD, the Attention Problems subscale and Externalizing composite of the *CBCL* were predicted to correctly identify children with the disorder. Further, the Rule-Breaking Behavior subscale of the *CBCL* and the Conduct and Tantrum Behavior subscales of the *ASD-C* were predicted to correctly identify children with ODD.

CHAPTER 4. RESULTS

Content analysis revealed that operationally similar items from the *ASD-C* and *CBCL* were significantly correlated. Refer to Table 3 for a breakdown of the correlations between items. Convergence was demonstrated for all the predicted pairings of subscales from the two measures. There was a significant positive correlation between the Diagnostic Total of the *ASD-DC* and the Withdrawn/Depressed subscale of the *CBCL*, $\rho = .685, p < .01$. The Diagnostic Total of the *ASD-DC* was also positively and significantly correlated with the Thought Problems subscale of the *CBCL*, $\rho = .534, p < .01$, and the Social Problems subscale of the *CBCL*, $\rho = .697, p < .01$. There was a significant positive correlation between the Worry/Depressed subscale of the *ASD-CC* and the Anxious/Depressed subscale of the *CBCL*, $\rho = .562, p < .01$. Additionally, the Worry/Depressed subscale of the *ASD-CC* was significantly correlated with the Withdrawn/Depressed subscale of the *CBCL*, $\rho = .615, p < .01$. The Conduct scale of the *ASD-CC* was significantly correlated with the Rule-Breaking Behavior subscale of the *CBCL*, $\rho = .569, p < .01$. Likewise, the Tantrum subscale of the *ASD-CC* was significantly correlated with the Aggressive Behavior subscale of the *CBCL*, $\rho = .764, p < .01$.

Table 3
Correlations between Individual Items on the *ASD-C* and *CBCL*

<i>CBCL</i> item	<i>ASD-C</i> item	Spearman's rho
#65. Refuses to talk	<i>ASD-DC</i> # 1. Communication skills	.433*
#79. Speech problems	<i>ASD-DC</i> # 1. Communication skills	.439*
#50. Too fearful or anxious	<i>ASD-CC</i> #36. Experiences excessive worry or concern	.500*
#65. Refuses to talk	<i>ASD-DC</i> #4. Verbal communication	.369*
#79. Speech problems	<i>ASD-DC</i> #4. Verbal communication	.477*
#24. Doesn't eat well	<i>ASD-DC</i> #5. Prefers food of a certain texture or smell	.230**
#25. Doesn't get along well with other kids	<i>ASD-DC</i> # 8. Social interaction with others his/her age	.315*

(Table 3 continued)

CBCL item	ASD-C item	Spearman's rho
#48. Not liked by other kids	ASD-DC # Social interaction with 8 others his/her age	.310*
#65. Refuses to talk	ASD-DC #10. Use of language in conversations with others	.343*
#79. Speech problems	ASD-DC #10. Use of language in conversations with others	.523*
#69. Secretive, keeps things to self	ASD-DC #11. Shares enjoyment, interests, or achievements with others	.057
#25. Doesn't get along well with other kids	ASD-DC #12. Ability to make and keep friends	.476*
#48. Not liked by other kids	ASD-DC #12. Ability to make and keep friends	.391*
#42. Would rather be alone than with others	ASD-DC # 13. Interest in participating in social games, sports, and activities	.618*
#111. Withdrawn, doesn't get involved with others	ASD-DC # 13. Interest in participating in social games, sports, and activities	.706*
#65. Refuses to talk	ASD-DC #18. Communicates effectively	.461*
#79. Speech problems	ASD-DC #18. Communicates effectively	.434*
#25. Doesn't get along well with other kids	ASD-DC #35. Socializes with other children	.436*
#42. Would rather be alone than with others	ASD-DC #35. Socializes with other children	.605*
#48. Not liked by other kids	ASD-DC #35. Socializes with other children	.317*
#14. Cries a lot	ASD-CC #1. Easily becomes upset	.320*
#53. Overeating	ASD-CC #2. Eats too much	.574*
#14. Cries a lot	ASD-CC #5. Crying	.322*
#24. Doesn't eat well	ASD-CC #6. Will eat only certain foods	.350*
#21. Destroys things belonging to his/her family or others	ASD-CC #7. Destroys others' property	.635*
#106. Vandalism	ASD-CC #7. Destroys others' property	.428*
#43. Lying or cheating	ASD-CC #9. Lies to obtain goods or favors	.423*
#66. Repeats certain acts over and over; compulsions	ASD-CC #11. Engages in repetitive behaviors for no apparent reason or to reduce stress	.512*
#22. Disobedience at home	ASD-CC #12. Compliance with demands	.503*
#23. Disobedience at school	ASD-CC #12. Compliance with demands	.407*
#28. Breaks rules at home, school, or elsewhere	ASD-CC #12. Compliance with demands	.464*
#24. Doesn't eat well	ASD-CC #13. Has a poor appetite	.547*

(Table 3 continued)

CBCL item	ASD-C item	Spearman's rho
#9. Can't get his/her mind off certain thoughts; obsessions	ASD-CC #14. Has persistent or recurring thoughts that cause distress	.310*
#24. Doesn't eat well	ASD-CC #16. Eats too little	.579*
#111. Withdrawn, doesn't get involved with others	ASD-CC # 17. Withdraws or removes him/her self from social situations	.767*
#76. Sleeps less than most kids	ASD-CC #18. Has trouble sleeping	.512*
#77. Sleeps more than most kids during day and/or night	ASD-CC #18. Has trouble sleeping	.140
#100. Trouble sleeping	ASD-CC #18. Has trouble sleeping	.696*
#21. Destroys things belonging to his/her family or others	ASD-CC #19. Damages property	.661*
#106. Vandalism	ASD-CC #19. Damages property	.414*
#14. Cries a lot	ASD-CC #22. Tearful or weepy	.384*
#52. Feels too guilty	ASD-CC #23. Feelings of worthlessness or excessive guilt	.266*
#35. Feels worthless or inferior	ASD-CC #23. Feelings of worthlessness or excessive guilt	.484*
#4. Fails to finish things he/she starts	ASD-CC #26. Finishes assigned tasks	.428*
#95. Temper tantrums or hot temper	ASD-CC #28. Easily becomes angry	.632*
#95. Temper tantrums or hot temper	ASD-CC #30. Tantrums	.588*
#10. Can't sit still, restless, or hyperactive	ASD-CC #31. Fidgets or squirms	.611*
#54. Overtired without good reason	ASD-CC #32. Low energy or fatigue	.766*
#86. Stubborn, sullen, or irritable	ASD-CC #35. Irritable mood	.474*
#112. Worries	ASD-CC #36. Experiences excessive worry or concern	.513*
#50. Too fearful or anxious	ASD-CC #36. Experiences excessive worry or concern	.500*

*Correlation is significant at $p < .01$ (2 tailed)

**Correlation is significant at $p < .05$ (2 tailed)

Contrary to the hypothesized outcomes, subscales that were not predicted to correlate (e.g., *ASD-C* Worry/Depressed and *CBCL* Rule-Breaking Behavior) were positively and

significantly correlated. Thus, there was no evidence of divergence between the two measures.

Please refer to Table 4 for the complete Multitrait-Multimethod Matrix.

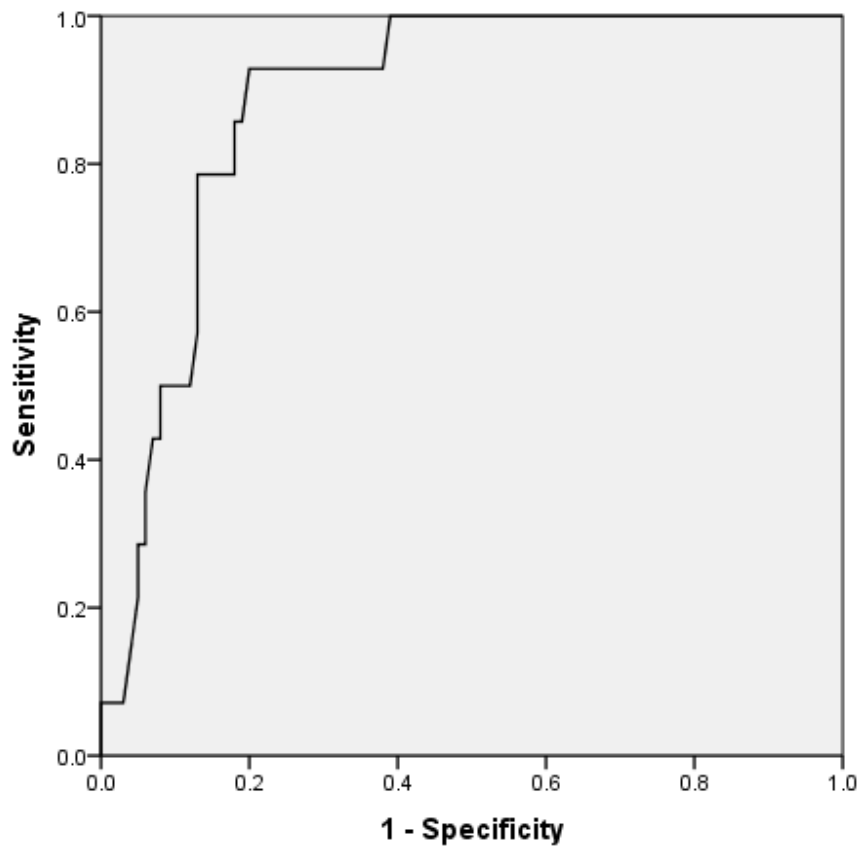
Table 4
Multitrait Multimethod Matrix for Convergent Validity of ASD-C and CBCL

Child Behavior Checklist											Autism Spectrum Disorder-Child Version							
	A/D	W/D	SC	SP	TP	AP	R-BB	AB	IP	EP	ASD-DC	TB	RB	WD	AB	CB	EB	IB
A/D	1																	
W/D	.558	1																
SC	.597	.431	1															
SP	.542	.607	.590	1														
TP	.490	.519	.606	.685	1													
AP	.293	.420	.489	.608	.620	1												
R-BB	.341	.399	.365	.562	.437	.478	1											
AB	.295	.421	.422	.654	.525	.609	.735	1										
IP	.893	.776	.788	.692	.651	.458	.433	.434	1									
EP	.325	.449	.428	.670	.541	.618	.852	.978	.462	1								
ASD-DC	.385	.685	.262	.697	.534	.473	.357	.496	.532	.487	1							
TB	.346	.488	.368	.662	.472	.529	.551	.764	.472	.741	.690	1						
RB	.312	.441	.405	.629	.564	.612	.456	.616	.456	.600	.613	.646	1					
WD	.562	.615	.553	.670	.597	.554	.430	.518	.680	.525	.611	.696	.599	1				
AB	.583	.732	.480	.697	.647	.493	.475	.527	.728	.548	.747	.543	.589	.617	1			
CB	.191	.362	.237	.550	.404	.489	.569	.709	.318	.704	.605	.756	.624	.546	.502	1		
EB	.327	.401	.265	.528	.473	.467	.621	.715	.460	.461	.619	.675	.567	.480	.555	.675	1	
IB	.352	.577	.240	.552	.548	.475	.342	.459	.396	.723	.744	.550	.660	.457	.608	.484	.593	1

Note: A/D = CBCL Anxious/Depressed Symptom Scale, W/D = CBCL Withdrawn/Depressed Symptom Scale, SC = CBCL Somatic Complaints Symptom Scale, SP = CBCL Social Problems Symptom Scale, TP = CBCL Thought Problems Symptom Scale, AP = CBCL Attention Problems Symptom Scale, R-BB = CBCL Rule-Breaking Behavior Symptom Scale, AB = CBCL Aggressive Behavior Symptom Scale, IP = CBCL Internalizing Problems Subscale, EP = CBCL Externalizing Problems Subscale, ASD-DC = ASD-C Autism Severity Total Score, TB = ASD-C Tantrum Behavior Subscale, RB = ASD-C Repetitive Behavior Subscale, WD = ASD-C Worry/Depressed Subscale, A = ASD-C Avoidant Behavior Subscale, CB = ASD-C Conduct Behavior Subscale, EB = ASD-C Externalizing Behavior Subscale, IB = ASD-C Internalizing Behavior Subscale

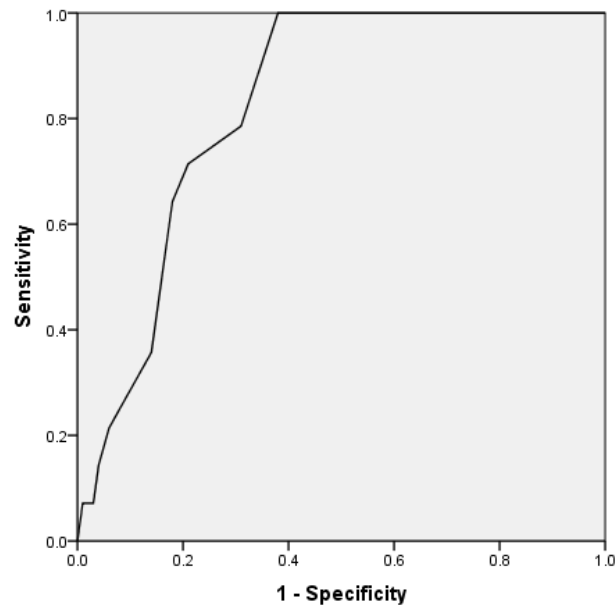
All correlations were significant at $p < .05$

ROC curve analyses were conducted with ASD as the positive actual state. The ROC curve analysis for the Diagnostic Total of the *ASD-DC* resulted in an AUC of .885, which is classified as good. Please refer to Figure 1 for the ROC curve. As for the *CBCL* subscales, the AUC for the Withdrawn/Depressed subscale was .827, which is classified as good. Please refer to Figure 2 for the ROC curve. However, the Social Problems (AUC = .790) and Thought Problems (AUC = .786) were in the fair range. Please refer to Figures 3 and 4 for the ROC curves. As for the ASD scale from the *CBCL*, the AUC was .890, which is classified as good. Please refer to Figure 5 for the ROC curve.



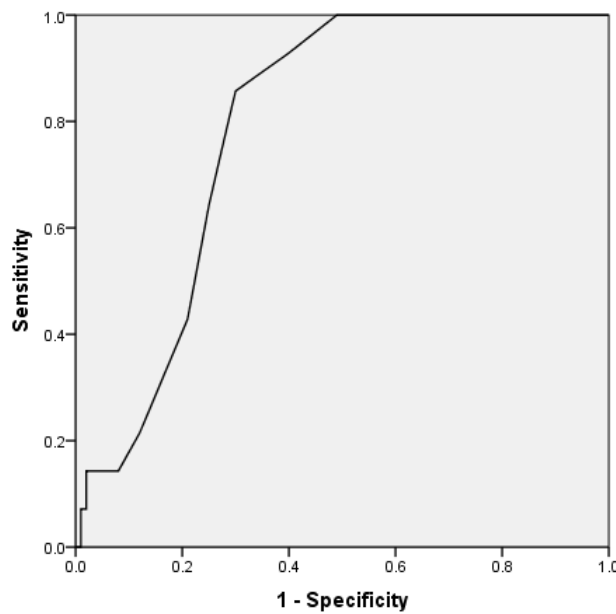
Diagonal segments are produced by ties.

Figure 1. ROC Curve for ASD-C Diagnostic Total



Diagonal segments are produced by ties.

Figure 2. ROC Curve for Withdrawn/Depressed Subscale from CBCL



Diagonal segments are produced by ties.

Figure 3. ROC Curve for Social Problems Subscale from CBCL

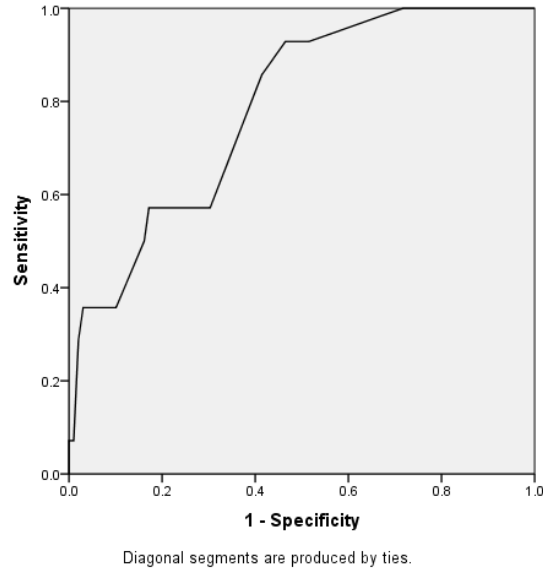


Figure 4. ROC Curve for Thought Problems Subscale from CBCL

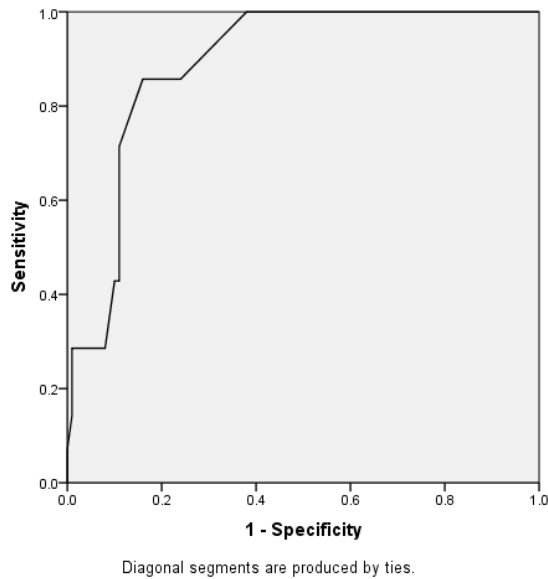
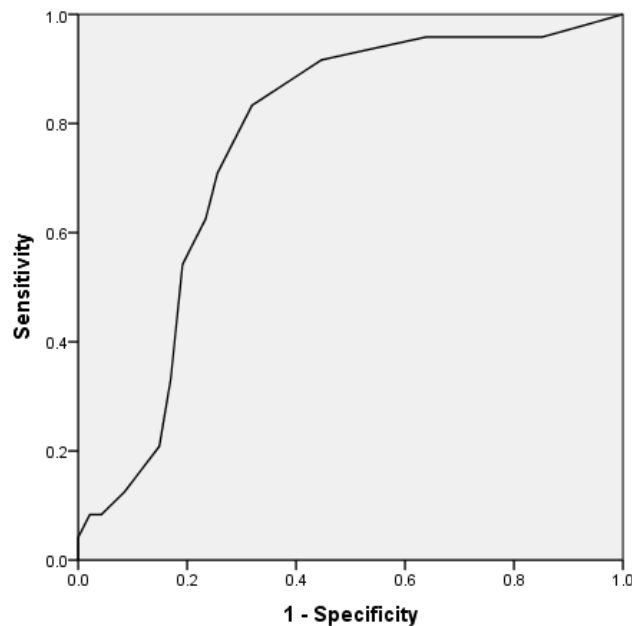


Figure 5. ROC Curve for ASD Scale from CBCL

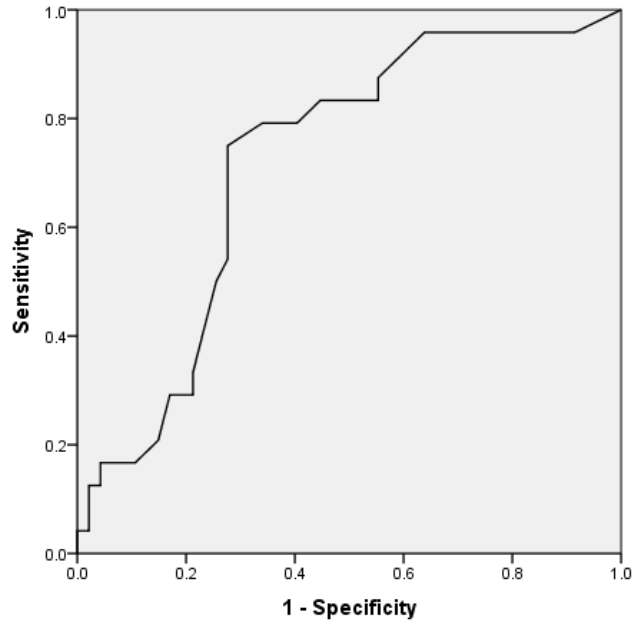
ROC curve analyses were also conducted for other diagnostic categories (e.g., Anxiety Disorder, ADHD, and ODD). Using Anxiety Disorder as the positive actual state, the *CBCL* Anxious/Depressed subscale (AUC = .762) and *CBCL* Internalizing composite (AUC = .718) were both determined to be fair at correctly identifying individuals diagnosed with an Anxiety

Disorder. Please refer to Figures 6 and 7 for the ROC curves. Whereas, the Worry/Depressed subscale of the *ASD-C* had an AUC of .545, which is classified as poor. Please refer to Figure 8 for the ROC curve. Using ADHD as the positive actual state, the *CBCL* Attention Problems subscale (AUC = .637) was found to be poor at identifying children diagnosed with ADHD, while the *CBCL* externalizing composite (AUC = .471) failed to identify children with ADHD beyond what would be predicted by chance. Please refer to Figures 9 and 10 for the ROC curves. Using ODD as the positive actual state, the *CBCL* Rule-Breaking Behavior (AUC = .854) and Aggressive Behavior (AUC = .814) subscales were classified as good. Please refer to Figures 11 and 12 for the ROC curves. Whereas, the *ASD-C* Conduct subscale (AUC = .707) was classified as fair and the *ASD-C* Tantrum Behavior subscale (AUC = .652) was classified as poor. Please refer to Figures 13 and 14 for the ROC curves.



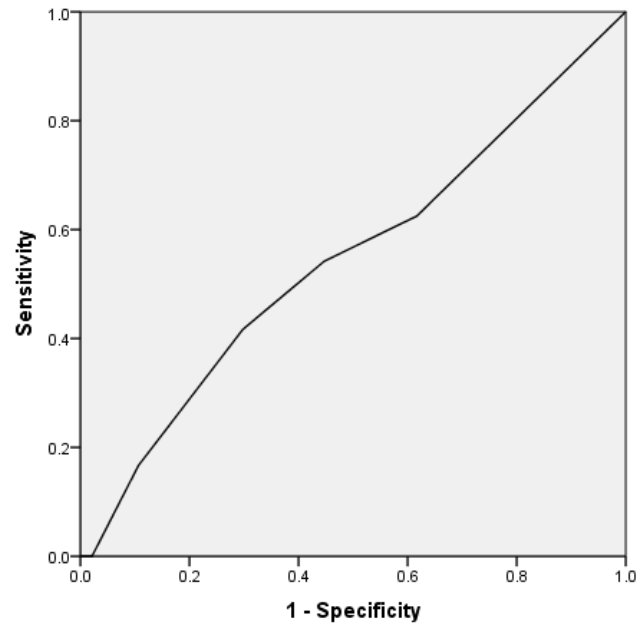
Diagonal segments are produced by ties.

Figure 6. ROC Curve for Anxious/Depressed Subscale from CBCL



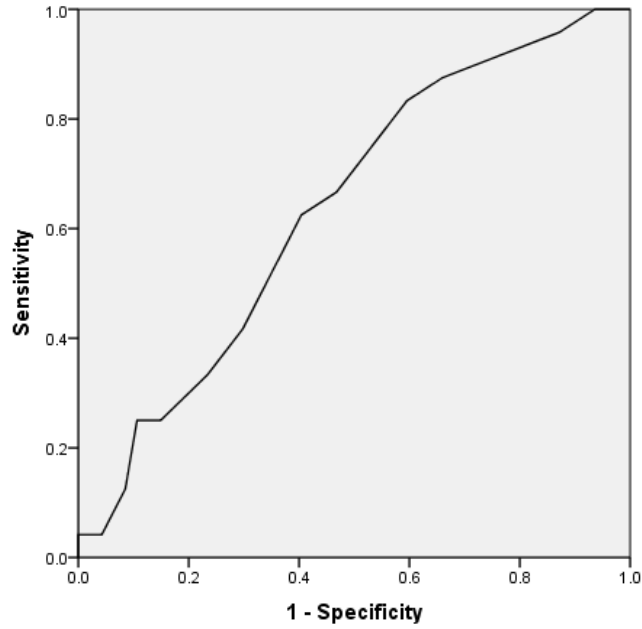
Diagonal segments are produced by ties.

Figure 7. ROC Curve for Internalizing Composite from CBCL



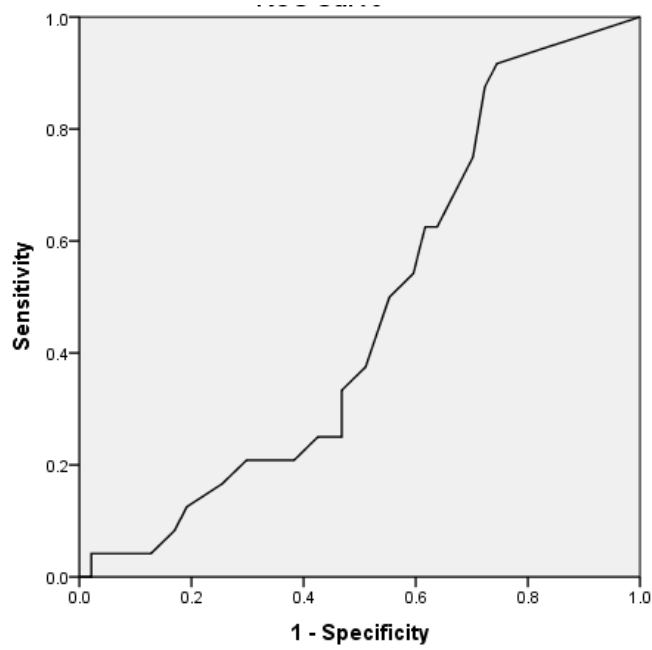
Diagonal segments are produced by ties.

Figure 8. ROC Curve for Worry/Depressed Subscale from ASD-C



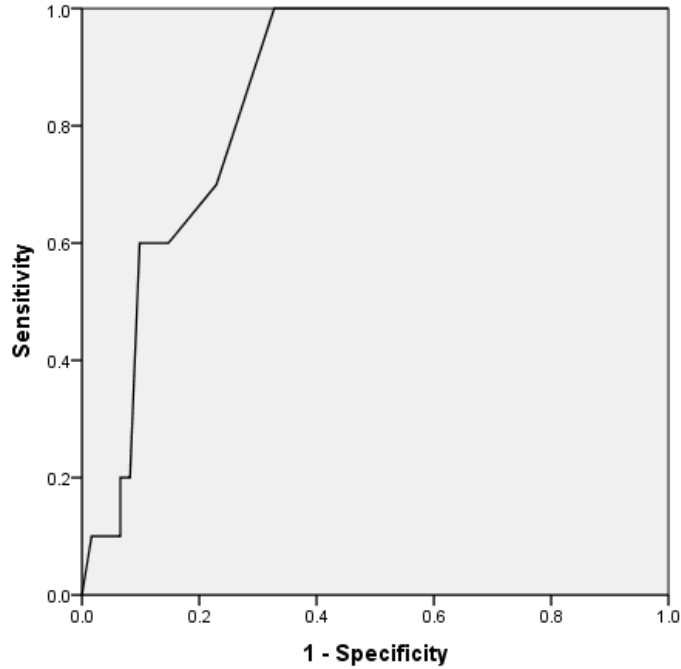
Diagonal segments are produced by ties.

Figure 9. ROC Curve for Attention Problems Subscale from CBCL



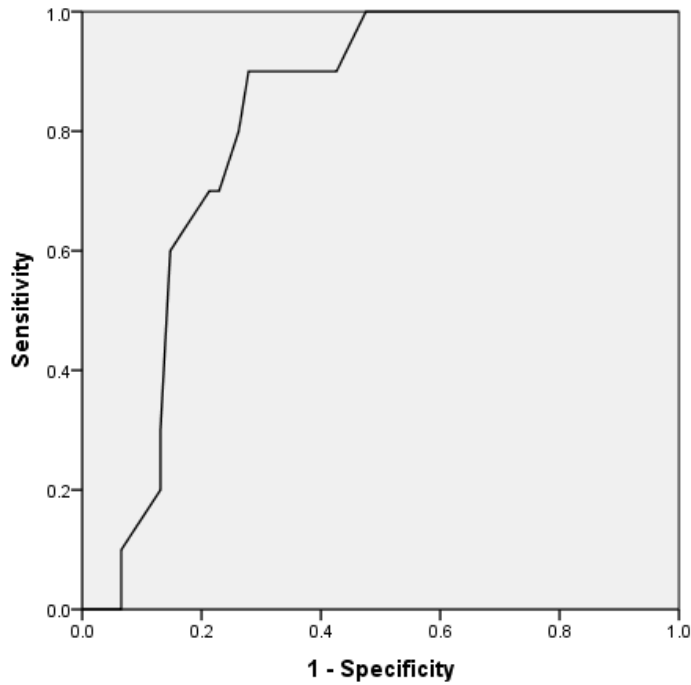
Diagonal segments are produced by ties.

Figure 10. ROC Curve for Externalizing Problems Composite from CBCL



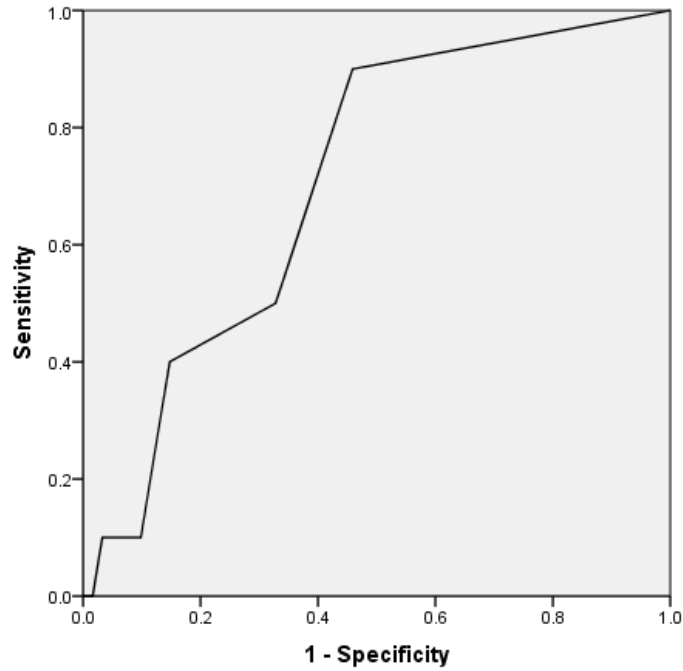
Diagonal segments are produced by ties.

Figure 11. ROC Curve for Rule-Breaking subscale from CBCL



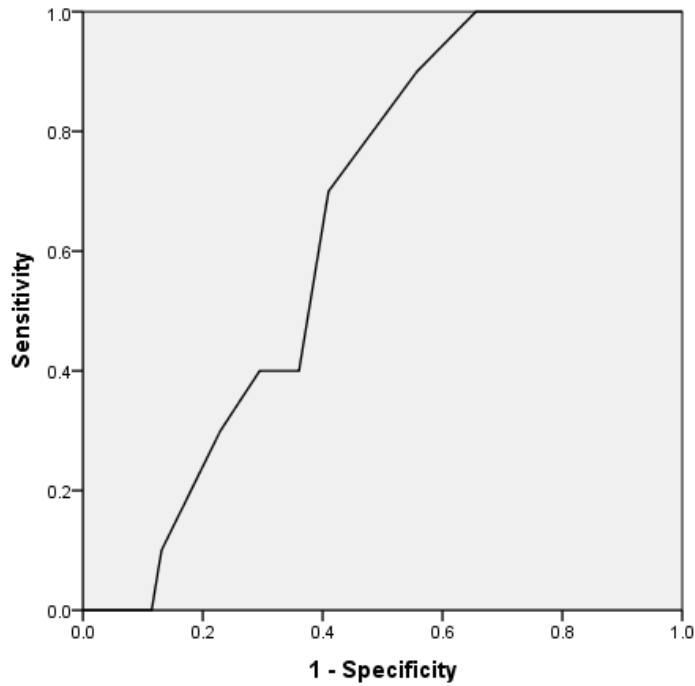
Diagonal segments are produced by ties.

Figure 12. ROC Curve for Aggressive Behavior Subscale from CBCL



Diagonal segments are produced by ties.

Figure 13. ROC Curve for Conduct Subscale from CBCL



Diagonal segments are produced by ties.

Figure 14. ROC Curve for Tantrum Behavior Subscale from CBCL

CHAPTER 5. DISCUSSION

The rationale behind the present study was to examine the construct validity of the *Autism Spectrum Disorder – Child Version* and the *Child Behavior Checklist* in children with and without ASD. Parent rating scales are time and cost effective measures that are commonly used to screen for psychological disorders (Hunsley & Mash, 2008). They allow clinicians to provide affordable services to children who may otherwise be unable to access mental health care due to financial constraints (Kataoka, Zhang, & Wells, 2002). The *CBCL* is one of the most widely used broadband screeners of psychopathology in clinical practice. However, the measure was developed for typically developing children. In cases where the child is suspected to have a developmental disability, the child should also be administered measures that are tailored to the ASD population. Thus, the *ASD-C* was specifically developed to address the need for a screening measure for core symptoms of ASD, related disorders, and challenging behaviors in children with possible developmental disabilities. As broadband screeners for psychopathology, the ability of these measures to correctly identify disorders is critical. Thus, establishing the validity of these inexpensive screening measures helps to provide mental health services to a broader range of children and their families.

Based on the results of the Multitrait-Multimethod Matrix, the two measures were shown to demonstrate convergent validity. There were significant positive correlations both between individual items with similar operational definitions and subscales assessing similar constructs. Contrary to hypotheses, operationally dissimilar subscales did not demonstrate divergent validity. This could potentially be attributed to the high rates of concurrent comorbidity among the participants. Previous researchers found that between 20% to 25.5% of children with a psychological disorder also met criteria for one or more concurrent diagnoses (Costello et al.,

2003; Merikangas et al., 2010). Further, children with ASD have even higher rates of comorbidity with estimates as high as 70% (Simonoff et al., 2008). With these high rates of psychiatric symptoms, the children included in this study likely elevated on subscales measuring dissimilar disorders (e.g., exhibited symptoms of anxiety and oppositional behavior). Conversely, the parents of children without any psychological disorders (i.e., controls) likely had no or few endorsements on items.

The measures were also evaluated for diagnostic validity for ASD, ADHD, Anxiety Disorder, and ODD. The Diagnostic Total of the *ASD-C*, Withdrawn/Depressed subscale of the *CBCL*, and ASD scale for the *CBCL* were determined to be good at identifying children with ASD. However, the Social Problems and Thought Problems subscales of the *CBCL* were only evaluated as fair at identifying children with ASD. Previous researchers had demonstrated that the *ASD-C* had an overall correct classification rate of 91.3% (Matson, Gonzalez et al., 2009). However, the current study only included 14 children diagnosed with ASD. Consequently, a larger sample would likely have resulted in improved diagnostic accuracy for the measure. The ASD scale from the *CBCL* had previously been demonstrated to have sensitivities ranging from 68% to 78% and specificities ranging from 73% to 92% (Ooi et al., 2011). Thus, the current study replicates the validity of the ASD scale. The Thought Problems subscale of the *CBCL* had been demonstrated to have sensitivities ranging from 82.9% to 94.3% and specificities ranging from 71% to 100% (Duarte, Bordin, de Oliveira, & Bird, 2003). Mazefsky, Anderson, Conner, and Minshew (2011) likewise found that the Social Problems subscale of the *CBCL* significantly predicted ASD diagnosis. Again, sample size likely contributed to the lesser findings in the current study.

Overall, the ASD scale of the *CBCL* and the *ASD-C* Diagnostic total were superior to the other subscales at identifying children diagnosed with ASD. Both of these scales were determined to be good screeners for ASD in a sample consisting of typically developing children with psychological disorders, children with ASD, and controls. An important implication of this study is that these measures accurately identified children with ASD while being economical, taking minimal administration time, and being quickly scored and interpreted by trained professionals with a master's level degree in an associated field. Poverty is a known risk factor for psychological disorders in children (Bringewatt & Gershoff, 2010; Knitzer, 1996; et al., 2007). Thus, parent-report measures that can be easily administered to low SES families will hopefully lead to increased access to services.

The current study also examined the diagnostic validity of the two measures for Anxiety Disorder. The *CBCL* Anxious/Depressed subscale and Internalizing composite were both determined to be fair at correctly identifying individuals diagnosed with an Anxiety Disorder. Whereas, the Worry/Depressed subscale of the *ASD-C* failed to correctly classify children with Anxiety Disorder beyond chance. For the purposes of this study and to account for the small sample size, anxiety disorders were lumped together into one overarching category. Thus, children grouped in this category had diagnoses ranging from Specific Phobia to Panic Disorder. Especially in the case of specific phobia where the anxiety symptoms are highly particular, their symptom presentation might not map onto a scale that measures more general worry and anxiety. Future researchers with access to a larger sample should consider separating the anxiety disorders into different diagnostic categories to address the heterogeneity of the disorders.

Diagnostic accuracy for externalizing disorders was also considered in the current research. The *CBCL* Attention Problems subscale was found to be poor at identifying children

diagnosed with ADHD, while the *CBCL* Externalizing composite failed to identify children with ADHD. This is contrary to the findings of previous researchers, who found that the Attention Problems subscale significantly predicted ADHD (Hudziak, Copeland, Stanger, & Wadsworth, 2004). The failure of these subscales to accurately identify clinical inattention may be attributable to the heterogeneous nature of ADHD. The clinical presentation of the disorder often shifts over an individual's lifespan from overt hyperactivity in early childhood to persistent inattention in adolescence and adulthood (Biederman et al., 1996). Additionally, the high rates of comorbidity between ASD and ADHD may have impacted the ability of the scale to differentiate between children with those disorders (Matson & Nebel-Schwalm, 2007b; Simonoff et al., 2008). This was further complicated by the *DSM-IV*'s prohibition of diagnosing children with ADHD if they met criteria for ASD (Gargaro, Rinehart, Bradshaw, Tonge, & Sheppard, 2011). As there is neither an attention or hyperactivity subscale on the *ASD-C*, the measure was not evaluated for diagnostic accuracy for ADHD.

As for identifying children diagnosed with ODD, the *CBCL* Rule-Breaking Behavior and Aggressive Behavior subscales were classified as good. Hudziak and colleagues (2004) also found the Aggressive Behavior subscale to be accurate at identifying children with ODD. However, previous researchers had demonstrated that the Rule-Breaking Behavior subscale was only fair at distinguishing children with ODD from CD, but good at identifying children with CD in the general population (Ebesutani, Bernstein, Nakamura, Chorpita, Higa-McMillan, & Weisz, 2010). Surprisingly, the *ASD-C* Conduct subscale was only classified as fair and the *ASD-C* Tantrum Behavior subscale was classified as poor. However, the Conduct subscale consists of only 4 items, and thus, may not have been able to accurately screen for all symptoms of ODD. Additionally, the Tantrum Behavior subscale included items that were likely endorsed by parents

of children with ASD as well as those with ODD. ASD has been demonstrated to exacerbate externalizing symptoms and children with ASD exhibit high rates of tantrum behavior (Goldin, Matson, Tureck, Cervantes, & Jang, 2013; Tureck et al., 2013). Future researchers should parse out which symptoms of the Tantrum Behavior subscale are more applicable to ODD and which are more generalized. Additionally, the small number of children with each of the psychological disorders may have affected the findings. In total, 24 children were diagnosed with an Anxiety Disorder, 24 children were diagnosed with ADHD, and 10 children were diagnosed with ODD. Future researchers should re-evaluate the diagnostic accuracy of these subscales with a larger sample.

It is important to note that other factors may have influenced symptom ratings leading to potentially biased reporting. Researchers have demonstrated that parent and child reports are typically discrepant with only low to moderate levels of agreement (De Los Reyes & Kazdin, 2005; Grills & Ollendick, 2002). This has been hypothesized to be due to an attribution bias, such that parents tend to report their children's behavior differently if they feel that the behavior warrants treatment (De Los Reyes & Kazdin, 2005). This highlights the impact of parent characteristics, including parental stress and psychopathology, on ratings (Smith, 2007). Stokes, Pogge, Wecksell, and Zaccario (2011) found that parenting stress is the factor most consistently associated with overreporting. This is especially important to consider when administering parent reports to children at risk for ASD, as Hayes and Watson (2013) found that parents of children with ASD experience significantly more stress than both parents of typically developing children and parents of children with other developmental disabilities (e.g., cerebral palsy and intellectual disability). In instances where parents are suspected to be overreporting their children's symptoms, the responsibility falls on the clinician to use direct observations to make

informed judgments about clinical diagnoses. This is especially important when using the *ASD-C* or *CBCL* as screening instruments, because neither of these two measures include a “lie scale.”

Another possible limitation of the current study is that the *CBCL* and *ASD-C* have different labels for the response ratings. The *CBCL* has parents rate each item as “not true,” “somewhat or sometimes true,” or “very true or often true.” Whereas the *ASD-C* has parents rate each item as “not a problem or impairment,” “mild problem or impairment,” or “severe problem or impairment.” How the ratings were worded could have impacted how parents interpreted the questions when responding. Previous researchers have demonstrated that when the wording of the response is more familiar to the responder, he/she is more likely to endorse that item. For example, Wiejters, Geuens, and Baumgartner (2013) found people were more likely to endorse “completely agree” than “strongly agree,” even though both ratings were expressing the same intensity of agreement.

Another limitation related to the differential designs of the two measures is that the *ASD-C* asks the extent an item was “ever” a problem on the diagnostic section and whether it was a “recent” problem on the comorbidity and problem behavior sections. Conversely, the *CBCL* asks how the item describes your child “now or within the past 6 months.” This difference in the timing of ratings is especially noteworthy, because many of the children undergoing evaluation were also receiving behavioral or pharmacological interventions that may have impacted the recent presentation of symptoms that were previously problematic. The actual impact of wording was not examined in the current research due to the ongoing nature of data collection. Future researchers should administer each measure with both sets of responses in order to compare response styles.

An additional implication of the current study is the importance of using valid measures of psychopathology when diagnosing children with developmental, behavioral, or affective disorders. This is increasingly important as doctors frequently prescribe numerous psychotropic medications to children following minimal assessment of symptoms. Olfson, Marcus, Weissman, and Jensen (2002) examined the difference in rates of psychotropic medication prescriptions among children and adolescents in the US in 1987 and 1996. They found that medication use increased from 1.4 per 100 children in 1987 to 3.9 per 100 children in 1996. This increase was largely attributed to more frequent prescription of stimulant medication to children between ages 6 and 14 years old. These rates have continued to increase over the past two decades (Matson & Hess, 2011). Negative side effects related to stimulants include insomnia, decreased appetite, headaches, and increased anxiety (Garcia, Logan, & Gonzalez-Heydrich, 2012). Thus, stimulant medications, such as methylphenidate, should be prescribed with caution and used only in instances when an ADHD diagnosis is confirmed by clinical testing and evaluation.

Furthermore, physicians frequently prescribe psychotropic medications including neuroleptics, antidepressants, antipsychotics, and stimulants to children with ASD (Mandell, Morales, Marcus, Stahmer, Doshi, & Polsky, 2008). These medications are being prescribed despite a lack of evidence that they address any of the core symptoms of ASD (Matson & Hess, 2011). Rather, psychotropics are used to manage symptoms related to ASD including aggression, self-injurious behaviors, and repetitive behaviors (Hollander, Phillips, & Yeh, 2003). Mandell and colleagues (2008) found that 56% of children with ASD receiving benefits through Medicaid were prescribed at least one psychotropic medication and 20% were prescribed three or more medications concurrently. This number may be a slightly high estimate for the general

population, as children who were uninsured or privately insured were found to be less likely to be prescribed three or more medications than those receiving Medicaid (Rosenberg, Mandell, Farmer, Law, Marvin, & Law, 2010). Unfortunately, psychotropic medications are often used as the first line of treatment for children whose families are unable to afford early intensive behavioral interventions (EIBI), which are both time and cost prohibitive in many cases (Matson & Hess, 2011). This leaves these individuals vulnerable to side effects ranging from irritability to weight gain to tardive dyskinesia (Campbell, Schopler, Cueva, & Hallin, 1996). Future research should address the need for cost effective behavioral interventions for children with ASD. This is especially relevant considering the increased availability and accuracy of low-cost diagnostic screens for children with ASD who are in need of treatment.

This study made strides toward identifying stand-alone screening instruments for identifying ASD and psychological disorders in children with and without a possible developmental disability. As previously discussed, there is significant controversy regarding the lack of available mental health services for children from low SES families (Bringewatt & Gershoff, 2010; Knitzer, 1996; McLearn et al., 2007). Many of these children are being prescribed psychotropic medications for behavioral symptoms without receiving comprehensive psychological evaluations. These medications are often unnecessary and result in adverse side effects (Matson & Hess, 2011). The current study demonstrated that the *ASD-C* and the *CBCL* contain disorder-specific scales that accurately identified children with developmental, behavioral, and affective disorders. The findings on the validity of the *ASD-C* are particularly important as the measure was developed specifically for an ASD population. The three-part measure is easy to administer, score, and interpret by a trained clinician. It could readily be utilized by pediatricians and other physicians who lack specific training in diagnosing ASD and

comorbid disorders (Meadows, Valleley, Haack, Thorson, & Evans, 2011). Once a child was identified as being at risk for a psychological disorder, the physician would then refer the child to a psychologist or psychiatrist for additional assessment before any diagnostic determinations were made. By screening more children in less time while maintaining good psychometric properties, the *ASD-C* and *CBCL* can identify children who would greatly benefit from behavioral treatments. Early diagnosis and intervention predicts the best possible outcomes for these children, while allowing for the provision of necessary supports to their families (Barbaro & Dissanayake, 2009; Matson, Wilkins, & Gonzalez, 2008; Volkmar, Chawarska, & Klin, 2005).

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APPENDIX A

Internal Review Board Approval

ACTION ON PROTOCOL CONTINUATION REQUEST



Institutional Review Board
Dr. Dennis Landin, Chair
130 David Boyd Hall
Baton Rouge, LA 70803
P: 225.578.8882
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TO: Johnny Matson
Psychology

FROM: Dennis Landin
Chair, Institutional Review Board

DATE: April 14, 2015

RE: IRB# 2609

TITLE: Developing the Autism Spectrum Disorder (ASD)

New Protocol/Modification/Continuation: Continuation

Review type: Full Expedited Review date: 4/14/2015

Risk Factor: Minimal Uncertain Greater Than Minimal

Approved Disapproved

Approval Date: 4/14/2015 Approval Expiration Date: 4/13/2016

Re-review frequency: (annual unless otherwise stated)

Number of subjects approved: 2,000

LSU Proposal Number (if applicable):

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

By: Dennis Landin, Chairman 

PRINCIPAL INVESTIGATOR: PLEASE READ THE FOLLOWING –
Continuing approval is **CONDITIONAL** on:

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

8. SPECIAL NOTE:

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

APPENDIX B

ASD Study Consent Form

1. Study Title: Developing the Autism Spectrum Disorder (ASD)

2. Performance Sites: Louisiana State University Psychological Services Center, preschools, grade schools, churches, hospitals or outpatient clinics, organizations, and internet websites.

3. Contact: Johnny L. Matson, Ph.D. (225) 578-8745

4. Purpose of Study: Several diagnostic instruments exist that are designed to determine the presence of emotional difficulties and behavior problems in children and adults. Currently there are no screening instruments that incorporate differential diagnosis of the developmental disorders. The purpose of this study is to develop assessment instruments designed to examine the social skills, challenging behaviors, and symptoms of emotional difficulties in children, as well as autistic traits in adults.

5. Participant Inclusion Criteria: Parents of children who are ≤ 18 years old receiving services at the Psychological Services Center; children who are receiving inpatient or outpatient medical/behavioral services, or currently attending preschools, grade schools, or church groups; children recruited via websites or organizations such as those for children with ASD or disabilities; and adults residing in the community. **Exclusion Criteria:** Parents, legal guardians, or informants unable or unwilling to provide informed consent or parental consent. **Maximum number of subjects:** 2000

6. Study Procedures: Assessment instruments designed to examine the social skills, challenging behaviors, and symptoms of emotional difficulties in individuals will be administered to the sample of 2000 adult participants (i.e., parents of child participants). Participants will receive information about the study and given an opportunity to volunteer through informational mail-outs at their child's school, church, clinic, etc. or information given to them when calling about services at the Psychological Services Center. Once consent is granted, participants will be given assessment packets regarding the following either in person at the outpatient clinic, mail, or internet link. Participants will provide information regarding the individual's: 1) demographics (e.g., age, gender, ethnicity, parents' names, number of siblings, etc.); 2) current psychotropic drug use and diagnoses; 3) developmental milestones; 4) social skills (e.g., turns head towards caregiver, initiates verbal communication, complains often, prefers to be alone, disturbs others, interacts positively with others, etc.); 5) challenging behavior (e.g., circumstances in which the target behavior occurs); and 6) symptoms of other difficulties (e.g., tantrums, excessive worry or concern, initiates fights, fidgets or squirms excessively, stereotypies, intellectual disability, impaired social interactions, language delays, etc.). Participants who receive the packet via mail will receive a follow-up phone call to ensure that they have received the packet and have the opportunity to ask questions. This study will take approximately 1 hour to 1.5 hours for each participant. Additionally, children (recruited from the outpatient clinic) of a subset of the sampled adult participants (i.e., parents of child participants) will be administered an abbreviated assessment of intellectual functioning.

7. Benefits: Participants under the age of 18 years may benefit from this study by taking advantage of reduced price assessment services at the Psychological Services Center in Baton Rouge, Louisiana. If parents decide to take advantage of this offered benefit, participants will be required to come into the clinic to complete a parent interview and child observation session. All

treatment services will be full price. Further, participants may benefit from professionals developing more reliable and valid assessment measures, suggesting improved diagnostic capabilities and more effective treatment interventions.

8. Risks/Discomforts: There is a small possibility of disclosure of personal information associated with this study. There are no other known risks resulting from participating in this study. Risks experienced should be those limited to those commonly experienced when receiving services from a public mental health clinic.

9. Measures taken to reduce risk: All participants will be given participant numbers. All data collected will be stored in reference to this number only. There will be one (1) master list which will list patient number by participant number to provide a means by which participants can choose to remove their data from the data set after participation. This list will be the only means by which data collected can be linked to personal information such as name or patient number. This list will be stored in a locked file cabinet, separately from the data collected.

10. Right to refuse: Participation is voluntary. Participants may change their mind and withdraw from the study at any time before the conclusion of the study without penalty or loss of any benefit to which they may otherwise be entitled.

11. Privacy: This study is confidential. Data will be kept confidential unless release is legally compelled.

12. Financial information: There is no cost to the participant and no payment will be provided for participation.

13. Withdrawal: There are no consequences for terminating participation in this study, which will last approximately 1 hour and 30 minutes in duration for each participant. To withdraw from the study, participants must inform the principle investigator of their desire to do so before the end of the study.

14. Removal: A participant's data may be removed from the study if it is discovered that there were errors in administration of any measure for that particular participant.

VITA

Kimberly Sheffield received her bachelor's degree in Neuroscience at Union College in 2010 and her master's degree from Louisiana State University in 2012. She completed an American Psychological Association accredited internship at Denver Health Medical Center in August 2015. She plans to begin a postdoctoral fellowship at Children's Hospital Colorado in September 2015.