2014

The Contribution of Comorbid Symptomology to the Social Skill Deficits in Infants and Toddlers with Autism Spectrum Disorder

Paige Elizabeth Cervantes
Louisiana State University and Agricultural and Mechanical College, pcerva2@tigers.lsu.edu

THE CONTRIBUTION OF COMORBID SYMPTOMOLOGY TO THE SOCIAL SKILL DEFICITS IN INFANTS AND TODDLERS WITH AUTISM SPECTRUM DISORDER

A Thesis

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
Master of Arts

in

The Department of Psychology

by
Paige Cervantes
B.A., Binghamton University, 2012
May 2015

TABLE OF CONTENTS

ABSTRACT

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication and the presence of restricted and repetitive patterns of behavior or interests. The onset of ASD symptomology occurs prior to 30 months of age; however, typical diagnosis is made at 3 to 4 years old. Early identification of ASD is imperative for more effective treatment and a bettered prognosis. The evaluation and treatment of ASD is complicated by high rates of comorbid psychopathology. When an individual presents with ASD and a co-occurring disorder, symptoms may manifest differently. Utilizing the *Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT)* and the *Battelle Developmental Inventory, Second Edition (BDI-2),* the current study explored the relationship of comorbid symptoms to socialization impairment in infants and toddlers with ASD. Specifically, this study examined the ability for comorbid symptoms to predict social skill impairment beyond the deficits accounted for by ASD symptom severity. Results indicated that although ASD severity significantly predicted social skill impairment, comorbid symptomology did not contribute to the social skill deficits seen in infants and toddlers with ASD. Implications are discussed.

INTRODUCTION

The accuracy of diagnosing psychopathologies in children has been argued for years, particularly in the realm of dual diagnosis. First came the debate over whether or not children can experience such disorders as depression; next, many researchers and clinicians refuted the claim that individuals with an intellectual disability could have a co-occurring mental health disorder (Matson & Nebel-Schwalm, 2007). Most recently, there has been a divide in the field of autism spectrum disorder (ASD). ASD is a heterogeneous, neurodevelopmental disorder characterized by deficits in social communication and the presence of restricted and repetitive patterns of behavior (American Psychiatric Association [APA], 2013). Some professionals believe dual diagnosis is not appropriate, arguing that symptoms such as anxiety and inattention in individuals with ASD are simply due to a variation in the manifestation of autism. However, there is a recent and extensive research base supporting the validity of the presence of comorbid psychopathologies within the ASD population (Davis III et al., 2010; de Bruin, Ferdinand, Meester, de Nijs, & Verheij, 2007; Dover & Le Couteur, 2007; Matson & Nebel-Schwalm, 2007; Matson & Cervantes, 2014; Mattila et al., 2010; Morgan, Roy, & Chance, 2003; Simonoff, Pickles, Charman, Chandler, & Baird, 2008).

In fact, Simonoff and colleagues (2008) have estimated that 70% of children with ASD had at least one comorbid disorder and 41% had two or more. Conditions found to most commonly co-occur with ASD include attention-deficit/hyperactivity disorder (ADHD), a variety of anxiety disorders, and oppositional defiant disorder (ODD; de Bruin et al., 2007; Leyfer et al., 2006; Simonoff et al., 2008).

Researchers suggest psychopathologies may present differently when co-occurring with ASD compared to when they occur alone; for instance, there is very little correspondence in the

types of specific phobias in children with ASD and the types of phobias in children of typical development (Davis III et al., 2010; Leyfer et al., 2006; Matson & Nebel-Schwalm, 2007). Additionally, the presentation of autism symptomology in individuals with ASD and a comorbid condition may be markedly different from individuals with ASD alone. For example, children with ASD and ADHD have been found to engage in significantly more stereotyped behavior than children with ASD only (Yerys et al., 2009); similarly, evidence suggests children with ASD and anxiety experience greater deficits related to the core symptoms of autism (Wood & Gadow, 2010).

The present study aimed to explore the interaction of ASD and comorbid psychopathologies, and examine the relationship of comorbid symptoms to social skill impairments in infants and toddlers with ASD. More specifically, when accounting for ASD severity, do symptoms of comorbid psychopathologies significantly contribute to socialization deficiencies? First, ASD will be discussed, followed by an overview of socialization deficits in children with ASD, and then finally a discussion of comorbid psychopathology in the ASD population.

AUTISM SPECTRUM DISORDER

History of ASD

Leo Kanner is accredited for laying the foundation for the literature on the condition we now know as autism in 1943. With the publication of his paper entitled "Autistic Disturbances of Affective Contact," Kanner provided detailed clinical accounts of 11 children presenting with a similar set of symptoms that could not be categorized into an existing disorder (Kanner, 1943). The eight males and three females were described as heterogeneous in several factors such as their degree of disturbance, manifestation of symptoms, and family and developmental history. However, all 11 children appeared to experience the same core deficits. Kanner identified the core symptoms of communication impairments, a desire for the maintenance of sameness, and severe socialization deficits. Among these symptoms, Kanner believed an "extreme autistic aloneness," or an inherent inability to relate to others, was the most fundamental. Socially, Kanner (1943) described the children as showing "no apparent affection;" they held minimal eye contact, preferred objects over people, and did not run to their parents for comfort. In his 1944 follow-up study, Kanner termed this set of symptoms, "infantile autism."

In regards to communication, the language abilities of Kanner's 1943 sample varied greatly. Of the 11 children, three never developed speech. The language of the eight verbal children, however, was described as nonfunctional and distorted. Language was rarely used to convey meaning. Echolalia, or repeating what was previously heard, and pronominal reversal were commonly observed in these children. Although echolalia does occur in typically developing toddlers and is seen as a normal stage of language development, it often persists in individuals with autism well beyond normal limits, into adolescence and adulthood (Van Houten, 1990). Researchers believe echolalic speech may be a mechanism to respond to verbal stimuli to

which individuals with autism have not learned an appropriate response (Van Houten, 1990). Pronominal reversal describes a confusion of first and second person speech; a child who demonstrates pronominal reversal will often refer to his or herself in the second person (i.e. you, he, she). Even in a child who could produce grammatically correct sentences, Kanner observed a tendency to respond only when the topic was a part of a restricted interest and the child's responses were more often peculiar in nature (Kanner, 1943).

In addition, all children presented with an "anxiously obsessive desire for the maintenance of sameness" (Kanner, 1943, p. 245). The children were repetitious and held very strict and ritualized daily routines. When a routine was broken or a change occurred, the children often displayed challenging behaviors and were said to become highly disturbed; the children in Kanner's sample would demonstrate severe tantrum behaviors when consistency was violated. Some of the associated symptoms Kanner (1943) identified in the 11 children with infantile autism included stereotyped behaviors, aggression, anxiety, and deficits in adaptive functioning.

Even after Kanner's publications describing this new syndrome, great confusion over whether autism had a place within the spectrum of schizophrenia or stands alone remained for years (Kanner, 1965). Prior to its identification, the symptoms of autism would most likely be categorized under the diagnosis of childhood schizophrenia. However, Kanner (1943; 1944) emphasized that infantile autism is different from schizophrenia. Much of the confusion over the new disorder is attributed to the name, "infantile autism." Eugen Bleuler, a Swiss psychiatrist, first used the term "autism." Bleuler (1913) defined autistic thinking as the withdrawal from the external world shown by individuals with schizophrenia, while Kanner (1943) used the word "autism" to signify a failure to form relationships with the external world.

Kanner (1943) argued the deficit in social functioning characteristic of infantile autism is not due to the same withdrawal from previous social contact seen in schizophrenics, but rather that it exists from the start as an innate inability to relate. Kanner (1943) reported that children with infantile autism display social deficits as early as infanthood when they fail to assume an anticipatory posture before being picked up, whereas schizophrenia typically has an age of onset closer to adulthood. Rutter (1968) provided a further distinction between the two disorders. Rutter (1968) reported that autism occurs at a higher rate in males compared to females, whereas schizophrenia occurs equally across genders. The parents of children with autism were found to be of a higher intelligence and at a greater socioeconomic status than the parents of children with schizophrenia. Also, schizophrenia was rare in the family history of children with autism. Further, autism and intellectual disabilities, or "mental subnormalities," commonly co-occurred whereas the same trend was not seen in schizophrenia. Children with autism have a similar pattern of intelligent quotient (IQ) scores that is not seen in children with schizophrenia. Children with autism do not experience delusions and hallucinations, and autism is steady across the lifespan while relapse and remissions are characteristic of schizophrenia (Rutter, 1968).

In his 1943 paper, Kanner explained that children with autism were likely to be misdiagnosed not only as having schizophrenia, but also as having intellectual disability. Kanner (1943) reported a similarity in repetitive behaviors in children with autism and intellectual disabilities; however, repetitive behavior with social "aloneness" helps to differentiate autism. The repetitive actions of individuals with autism have been reported more intricate and solitary than the repetitive actions of individuals with intellectual disability (Eveloff, 1960). Additionally, autism cannot be considered an intellectual disability because some children with autism have an average IQ. Those with autism with an average IQ display the same behavioral

tendencies as those with a low IQ, and children with autism have a characteristic IQ pattern not typical of individuals with intellectual disabilities (Rutter, 1968). The IQ pattern to which Rutter (1968) referred includes low scores on verbal subtests and comparatively high scores on tasks requiring visuospatial skills. This IQ pattern suggests that rather than a global intellectual deficit, the poor intellectual functioning of individuals with autism may be due more to impairment in language (Lockyer & Rutter, 1970; Rutter & Bartak, 1971). Rutter and Schopler (1988) reported that autism differs from intellectual disability in age of onset of comorbid seizures, associations with genetic conditions, and gender distribution. In individuals with an intellectual disability, if present, seizures will begin at an early age; while, individuals with autism who experience seizures will typically have an onset of seizures closer to adolescence. Down syndrome was the most common cause of intellectual disability; however, Down syndrome, at the time, was not correlated with autism. Lastly, intellectual disability has only a slightly higher prevalence rate in males while autism occurs four times more often in males than females (Rutter & Schopler, 1988).

There was also confusion over the cause of autism at this time. There seemed to be a rush to blame the parents of children with autism, particularly mothers. There was an emphasis on the effect of emotional coldness and obsessive qualities in parents, as well as a lack of affection from the parents to the affected child (Rutter, 1968). Rutter (1968) refuted this theory; if it were the characteristics of parents that caused autism, a higher rate of autism would be seen in the siblings of children with autism than observed. Others believed autism to be a defense mechanism in children with other pre-existing psychopathologies, like schizophrenia, where they withdraw to protect themselves from the conflict of reality versus their pathology; this theory was soon shown flawed as autism emerged early and persisted through the lifespan (Bender,

1959). Other theories involve autism as the result of faulty learning, of a malfunctioning reticular system or consistent overarousal, of a deprivation of social stimulation and contact, and finally, as the result of an inability to comprehend sound (Rutter, 1968). With all of these theories proving unlikely, both Kanner (1965) and Rutter (1968; 1978) recognized the etiology of autism must involve an interaction between innate and environmental factors.

Until recently however, the role of genetic factors in the etiology of autism was debated; this debate was due to the low rate of autism in parents and in siblings of children with autism, and the lack of identified chromosomal differences related to autism (Rutter, 2000). We now know the incidence of autism is significantly higher in siblings with autism compared to the general population (Rutter, 2000). The heightened comorbidity rates between autism and seizure disorder, intellectual disability, and several genetic conditions like Down syndrome also point to a genetic association. Researchers now recognize the significant role genetics may play in the etiology of ASD; however, more research addressing this role is necessary (Rutter, 2000).

Diagnostic Criteria

Despite the early work of Leo Kanner and Michael Rutter, it was not until 1980 that autism was recognized in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (*DSM-III*; APA, 1980; Rutter & Schopler, 1988). The *DSM-III* title for this set of symptoms was pervasive developmental disorders (PDDs). The creators of the *DSM-III* were careful in their word choice. Due to previous confusion, the creators wanted to clearly differentiate this condition from mental illness. Therefore, they chose to emphasize the developmental aspect of the disorder. The word pervasive was chosen to represent the extensive range of impairment across the domains of communication, socialization, and behavior (Rutter & Schopler, 1988). Under the category of PDDs, the *DSM-III* included diagnoses of Infantile

Autism representing the prototypical case described by Kanner, Atypical Autism for children who exhibit most symptoms in line with an autism diagnosis, and Childhood Onset PDD used to label children with a range of disturbances related to one cause rather than several comorbidities (APA, 1980; Cohen, Paul, & Volkmar, 1986). Seven years later in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (*DSM-III-R*; APA, 1987), the label of Infantile Autism was changed to Autistic Disorder and Atypical Autism to Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS; Spitzer & Siegel, 1990).

The classification of autism further evolved with the publication of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*; APA, 1994). The creators of the *DSM-IV* aimed to mirror the empirically-based conceptualization of autism in the *International Classification of Diseases, Tenth Revision* (*ICD-10*) published by the World Health Organization (WHO) in 1992 (Klin, Lang, Cicchetti, & Volkmar, 2000; WHO, 1992). The *DSM-IV* consisted of 5 separate subcategories including Autistic Disorder, Asperger's Disorder, PDD-NOS, Rett's Disorder, and Childhood Disintegrative Disorder. The *Diagnostic and Statistical Manual of Mental Disorders*, *Fourth Edition, Text Revised* (*DSM-IV-TR*; APA, 2000) published in 2000 preserved these five subcategories.

To have received a diagnosis of Autistic Disorder with the *DSM-IV-TR*, an individual must present with six or more of the diagnostic items. Endorsed items were required to include at least two items from the social domain, at least one from the communication domain, and at least one from the restricted, repetitive, and stereotyped patterns of behavior domain. The domain of social impairment included (a) impairment in nonverbal behaviors like eye contact, facial expression, gestures, etc., (b) lack of ability to develop developmentally appropriate relationships with peers, (c) failure to seek shared enjoyment, and (d) impairment in social or

emotional reciprocity (APA, 2000). The communication domain consisted of (a) delay in, or failure to develop, verbal language, (b) impairment in initiating and maintaining conversation, (c) use of stereotyped or idiosyncratic language, and (d) lack of developmentally appropriate pretend play skills (APA, 2000). Included in the restricted, repetitive, and stereotyped patterns of behavior domain was (a) abnormal preoccupation with one or more stereotyped or restricted interests, (b) strict insistence on specific, nonfunctional routines, (c) stereotyped and repetitive motor movements, and (d) unusual fixation on parts of objects (APA, 2000). For an Autistic Disorder diagnosis according to the *DSM-IV-TR*, individuals must have exhibited symptoms prior to 3 years of age and the symptoms could not be better explained with a diagnosis of Rett's Disorder or Child Disintegrative Disorder (APA, 2000).

Although Asperger's Disorder was not recognized as an autism subcategory until the *DSM-IV*, it was originally described by Hans Asperger in 1944 (Asperger, 1991). Asperger (1991) described several case studies of highly intelligent children exhibiting severe social disturbances and peculiar behaviors. Unfortunately, Asperger's findings were not widely recognized until Uta Frith translated the accounts into the English language in 1991. According to the *DSM-IV-TR*, a diagnosis of Asperger's Disorder was given if an individual exhibited no impairment in cognitive development, adaptive functioning, or language but demonstrated symptoms consistent with an ASD in the socialization and repetitive, restricted, or stereotyped behavior domains described above (APA, 2000).

Previously referred to as Atypical Autism, PDD-NOS held no explicit criteria in the *DSM-IV-TR*. To have received a diagnosis of PDD-NOS, an individual must have exhibited deficits in socialization as well as demonstrated impairments in communication or presented with restricted, repetitive, and stereotyped patterns of behavior consistent of an Autistic Disorder

diagnosis. The symptoms demonstrated by an individual with PDD-NOS would be similar to an individual with Autistic Disorder; however, criteria would not be met due to late age of onset or an unusual or subthreshold presentation of symptoms. The symptomology must have not been better accounted for by a specific PDD diagnosis, Schizophrenia, Schizotypal Personality Disorder, or Avoidant Personality Disorder (APA, 2000).

Current Diagnostic Criteria

In May of 2013, the APA released the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*) following some concern with the criteria presented in the *DSM-IV-TR* (APA, 2013; Matson, Belva, Horovitz, Kozlowski, & Bamburg, 2012; Worley & Matson, 2012). The authors of the *DSM-5* were concerned with possible ambiguity of the boundaries between autism subcategories as well as the vagueness of the Asperger's Disorder and PDD-NOS diagnostic criteria (Matson, Belva, et al., 2012; Worley & Matson, 2012). To account for these weaknesses, the APA established various changes to the criteria. Consequently, these changes instigated great controversy in the autism field for both researchers and clinicians (Frazier et al., 2012; Gibbs, Aldridge, Chandler, Witzlsperger, & Smith, 2012; Matson, Belva, et al., 2012; Matson, Hattier, & Williams, 2012; McPartland, Reichow, & Volkmar, 2012; Wing, Gould, & Gillberg, 2011).

In the *DSM-5*, the Autistic Disorder, Asperger's Disorder, and PDD-NOS subcategories were collapsed into a single Autism Spectrum Disorder category varying on level of severity. The diagnoses of Rett's Disorder and Childhood Disintegrative Disorder were removed from the ASD category. The social and communication domains included in the *DSM-IV-TR* were merged into a single diagnostic category labeled Communication and Social Interaction. Within the Communication and Social Interaction domain, individuals must endorse all three items to be

eligible for diagnosis. These items include (1) impairment in social-emotional reciprocity, (2) deficits in nonverbal communication, and (3) impairment in developing and maintaining relationships. The domain of restricted, repetitive patterns of behavior, interests, or activities was preserved in the *DSM-5*. An individual must evince at least two of the four items in this category including (1) stereotyped, repetitive speech, motor movements, or object manipulations, (2) strict adherence to routines, ritualized patterns of verbal or nonverbal behavior, or extreme resistance to change, (3) highly restricted, fixated interests that are unusual in intensity or focus, and (4) over or under reactivity to sensory input or abnormal interest in sensory aspects of the environment. The *DSM-IV-TR* required symptoms to be present before age 3 for diagnosis (APA, 2000); however, the *DSM-5* is less clear, stating symptoms must be exhibited in early childhood but "may not become fully manifest until social demands exceed limited capacities" (APA, 2013).

Prevalence of ASD

Once considered an infrequent, rare condition, original prevalence rates of autism and the disorders that make up the spectrum were estimated around four to five individuals per 10,000 in the general population (Dover & Le Couteur, 2007; Howlin, 2006; Koenig, Rubin, Klin, & Volkmar, 2000; Lotter, 1966; Matson & Kozlowski, 2011; Rutter, 1968). Epidemiology studies in the early 1990s, however, published prevalence estimates for the PDDs between 30 to 60 individuals per 10,000 with ten to 20 individuals per 10,000 meeting criteria for Autistic Disorder; this substantial climb from original calculations was a source of much concern and speculation of a possible "epidemic" (Inglese & Elder, 2009).

In response, the Autism and Developmental Disorders Monitoring Network (ADDM) of the Center for Disease Control and Prevention (CDC) began monitoring the prevalence of autism

and related disorders across the country and the world. In 2002, the CDC began a study examining ASD prevalence in 8-year-old children residing in 14 locations across the United States. The prevalence estimates published in 2007 were congruent with the findings in that time; 6.4 individuals per 1,000, or one in 150 individuals, were afflicted with an ASD (CDC, 2007; Inglese & Elder, 2009). Consistent with original gender differences reported by Kanner and Eisenberg (1957), the male to female ratio ranged from 3.4:1 to 6.5:1 (CDC, 2007). Significantly higher rates of ASDs were found in non-Hispanic white children (CDC, 2007).

The ADDM ASD prevalence estimate published in 2012 was 11.3 individuals per 1,000 or one in 88 (CDC, 2012). The study was similar to the 2002 procedure; data was collected in 2008 on 8-year-old children across 14 locations. In addition to an estimated 78% increase in overall prevalence from the 2007 study, the rate of ASD increased in all races. A 70% increase was calculated among non-Hispanic white children, a 91% increase among non-Hispanic black children, and a 110% increase among Hispanic children (CDC, 2012) compared to the 2007 study. These increases indicate a thinning of the ethnicity gap historically seen within the prevalence of ASD. The male to female ratio, though, remained stable at 4.65:1 (CDC, 2012).

The most recent ADDM study was published in 2014; data on 8-year-old children was collected in 2010 from 11 ADDM sites (CDC, 2014a). Overall prevalence of ASD was estimated at 14.7 per 1,000 children, or one in 68. There was a 29% increase in ASD prevalence compared to the 2008 study. Consistent with previous research, the male to female ratio was calculated at approximately 4.5:1. Notably, the estimated proportion of individuals with ASD and average or above average cognitive functioning (IQ > 85) was significantly greater than previous estimates. Compared to 32% in the 2002 study, 46% of 8-year-old children identified with ASD were found to have average or above average cognitive ability in 2010 (CDC, 2014a).

Examining the data collectively, it is obvious that autism rates have increased dramatically since its origination; however, it is difficult to pinpoint definitive reasons for this increase (Matson & Kozlowski, 2011). It is most likely that there are a variety of causes for the rise in ASD. First, the ASD diagnostic criteria are continually changing and the definition of autism has broadened significantly over the years (Fombonne, 2009; Koenig et al., 2000; Leonard et al., 2010; Matson & Kozlowski, 2011). When it was first described by Kanner in 1943, autism included a much narrower subset of individuals than those who would be placed on the spectrum today (Matson & Kozlowski, 2011). Another reason for the increase may be an increased awareness and acceptance of autism both in the lay and professional public (Fombonne, 2009; Inglese & Elder, 2009; Leonard et al., 2010; Matson & Kozlowski, 2011). An increased awareness of the symptoms of ASDs and of service availability may lead to better and earlier detection (Fombonne, 2009; Koenig et al., 2000; Leonard et al., 2010; Matson & Kozlowski, 2011).

While increased emphasis on autism in the general population can lead to better detection, it can also lead to increased misdiagnoses. Like childhood schizophrenia in the 1950s and 1960s (Mosse, 1958), it is possible autism diagnoses are being inappropriately and overly used (Leonard et al., 2010; Matson & Kozlowski, 2011). Correctly identifying and diagnosing autism can be very complicated due to heterogeneity and overlapping symptoms with other conditions, such as communication disorders (Matson & Kozlowski, 2011). It is not unlikely that inexperienced clinicians sometimes misdiagnosis autism. Diagnostic substitution may also contribute to the increasing ASD prevalence rates (Leonard et al., 2010). Diagnostic substitution involves replacing an original diagnosis with a more accurate diagnosis (Leonard et al., 2010). For example, historically, it has been customary to give an individual with an intellectual

disability and symptoms consistent with autism a primary diagnosis of intellectual disability. Today, it is more accurate to give a primary diagnosis of ASD and a secondary diagnosis of intellectual disability (Leonard et al., 2010). Other factors that may influence the ASD prevalence rates include the research methodology used to calculate estimates (Fombonne, 2009; Koenig et al., 2000; Leonard et al., 2010; Matson & Kozlowski, 2011) and cultural factors. For example, ASD is a relatively new concept in the Eastern world and particularly in Asian countries (Leonard et al., 2010; Matson & Kozlowski, 2011).

Lastly, changing environmental factors are thought to contribute to the increasing prevalence rates (Matson & Kozlowski, 2011). Although less is known regarding environmental causes of autism, there have been several speculations. Autism has been suggested to be associated with the use of medications, exposure to environmental contaminants, and high metal toxicity levels; however, research is limited regarding these factors (Inglese & Elder, 2009). One environmental factor that could be contributing is the improvement of prenatal and neonatal care (Matson & Kozlowski, 2011). With improved medical care, the survival rate for prematurity is heightened; premature infants have been found to have higher prevalence rates of ASD compared to the general population (Matson & Kozlowski, 2011). Further research regarding the involvement of environmental factors in autism is necessary.

With the recent introduction of the *DSM-5*, new prevalence rates are currently unspecified. However, the rate of ASDs will likely decrease with the tightening of criteria. With the diagnostic changes, researchers have found up to 40% of individuals with a *DSM-IV-TR* ASD diagnosis will no longer meet criteria (Frazier et al., 2012; Matson, Belva, et al., 2012; Matson, Kozlowski, et al., 2012; Mattila et al., 2011; McPartland et al., 2012; Worley & Matson, 2012). Individuals with ASD and no intellectual disability, individuals with PDD-NOS, and individuals

with Asperger's Disorder were particularly vulnerable for not meeting diagnostic criteria with the *DSM-5* (Mattila et al., 2011; Mayes, Black, & Tierney, 2013; Worley & Matson, 2012). The presentations of symptoms are also suspected to change with the *DSM-5*. Compared to a group meeting only *DSM-IV-TR* criteria for an ASD, those individuals with *DSM-5* diagnosable ASD have been found to have greater developmental delays, higher rates of challenging behavior, and evince greater levels of ASD core symptoms (Beighley et al., 2013; Matson, Belva, et al., 2012; Turygin et al., 2012). However, these individuals with less severe symptoms who do not meet *DSM-5* criteria still evince more ASD symptoms, more comorbid psychopathologies, and more challenging behaviors compared to an atypically developing population (Beighley et al., in press; Matson, Belva, et al., 2012; Matson, Hattier, et al., 2012; Rieske et al., in press; Turygin et al., 2012; Worley & Matson, 2012). While the *DSM-5* indicates that individuals diagnosed with an ASD using the *DSM-IV-TR* may keep the diagnosis, it will be interesting to see what will be done with individuals who do not yet have a diagnosis but present with an identical or more severe symptom set that meets *DSM-IV-TR* but not *DSM-5* criteria.

Assessment of ASD

With the rise in awareness and knowledge regarding the autism spectrum came an increase in instruments developed and utilized to identify ASD. Described below are a few of the most widely used instruments to help diagnose ASD in children and adults at this time.

Child Autism Rating Scale, Second Edition (CARS-2; Schopler, Van Bourgondien, Wellman, & Love, 2010). The *Child Autism Rating Scale, Second Edition (CARS-2)* is an observation scale designed to assess for ASD symptomology. The *CARS-2* consists of three different versions including the Standard Version (*CARS2-ST*), the High-Functioning Version (*CARS2-HF*), and the Questionnaire for Parents and Caregivers (*CARS2-QPC*). The *CARS2-ST*

can assess individuals aged 2 to 36 years old and consists of 15 categories. The categories are: Relating to People; Imitation; Emotional Response; Body Use; Object Use; Adaptation to Change; Visual Response; Listening Response; Taste, Smell, and Touch Response and Use; Fear or Nervousness; Verbal Communication; Nonverbal Communication; Activity Level; Level and Consistency of Intellectual Response; and General Impressions. Each category is scored based on direct observation; then, the category scores are summed for a total score. The individual being assessed can score in the range of 15 to 60. A score of 15 to 29.5 falls in the Minimal-to-No Symptoms of ASD cut-off range; a score of 30 to 36.5 indicates Mild-to-Moderate Symptoms of ASD range; and a score of 37 or above falls in the Severe Symptoms of ASD range. The *CARS2-HF* and the *CARS2-QPC* are new to the *CARS*. The *CARS2-HF* is designed for assessment of individuals with an IQ above 80, over 6 years old, and with fluent communication. The *CARS2-QPC* is based on parent report and is used in addition to observational scales (Schopler et al., 2010).

The *CARS2-ST* has been found reliable with an internal consistency coefficient of .92. Evidence suggests the *CARS2-ST* and *CARS2-HF* are valid measures; correlations with the *Autism Diagnostic Observation Schedule (ADOS*) were found at .79 for the *CARS2-ST* and .77 for the *CARS2-HF* (Vaughan, 2011). The *CARS-2* has sensitivity and specificity estimates above .85 (Vaughan, 2011).

Autism Diagnostic Observation Scale, Second Edition (ADOS-2; Gotham, Risi, Pickles, & Lord, 2007). The *Autism Diagnostic Observation Scale, Second Edition (ADOS-2*) includes a set of scenarios used to assess the communication and social interaction of an individual suspected of ASD. These scenarios are standardized and designed to help flag ASD symptomology. An individual being assessed using the *ADOS-2* is administered one of

five modules chosen based on age and language skills; one module takes about an hour to administer (Gotham et al., 2007).

The five modules included in the *ADOS-2* consist of: Module 1, intended for children 31 months and older who do not consistently use phrase speech; Module 2, intended for children of any age who do use phrase speech but are not verbally fluent; Module 3, intended for verbally fluent children and young adolescents; Module 4, intended for verbally fluent older adolescents and adults; and the Toddler Module, intended for use in children ages 12 to 30 months who do not consistently use phrase speech.

The assessor rates the individual's performance on each item in each scenario on a 3-point Likert scale throughout the administered module. A score of 0 indicates the individual is "within normal limits;" a score of 1 indicates an "infrequent or possible abnormality;" and, a score of 2 indicates a "definite abnormality." The total scores are put into a diagnostic algorithm according to the *DSM-5* criteria for ASD (Gotham et al., 2007).

Studies examining the psychometric properties of the *ADOS* indicate strong reliability estimates (Lord et al., 1989). Sensitivity estimates are found to be above .80 and specificity estimates about .68 (Kamp-Becker et al., 2013). The *ADOS*, however, is an expensive instrument, can be time-consuming, and the clinician administering the measure must be trained.

Autism Spectrum Disorder Battery – Child Version (ASD-C; Matson & Gonzalez, 2007). The *Autism Spectrum Disorder Battery – Child Version (ASD-C)* is designed to assess for core and associated symptoms of ASD in individuals aged 3 to 16 years. The *ASD-C* is an informant based assessment made up of three parts: the *Autism Spectrum Disorders – Diagnostic – Child Version* (*ASD-DC*), the *Autism Spectrum Disorders – Comorbid – Child Version* (*ASD- CC*), and *Autism Spectrum Disorders– Problem Behavior – Child Version (ASD-*

PBC). The *ASD-DC* is the diagnostic scale and assesses for core ASD symptomology. Informants are instructed to rate the child being assessed compared to children of his or her age to the extent an item is or was ever a problem. A rating of 0 suggests "not different; no impairment;" a rating of 1 suggests "somewhat different; mild impairment;" and a rating of 2 suggests "very different; severe impairment."

The *ASD-DC* demonstrates strong psychometric properties. Reliability estimates for the *ASD-DC* are adequate with an internal consistency coefficient of .99, an inter-rater reliability of .67, and a test-retest reliability of .77 (Matson, Gonzalez, Wilkins, & Rivet, 2008). Evidence of convergent validity with both the *ADI-R* and the *CARS* has been also found for the *ASD-DC* (Matson, Hess, Mahan, & Fodstad, 2010).

SOCIALIZATION IN INDIVIDUALS WITH ASD

Since Kanner's (1943) first accounts of autism, socialization impairment has been at the core of the disorder (Matson & Wilkins, 2007). Although it is recognized that the onset of ASD occurs by 30 months of age, a majority of children with ASD demonstrate social abnormalities from their first year of life (Rutter, 1978; Volkmar, Chawarska, & Klin, 2005). Symptoms are seen as early as 2 months old when children with ASD fail to assume a typical anticipatory posture in preparing to be held (Grossman, Carter, & Volkmar, 1997; Kanner, 1943; Rutter, 1978). While typically developing infants hold preferential attendance to faces, infants with ASD show very little interest in faces or people (Grossman et al., 1997; Rutter & Bartak, 1971). This lack of preference to social stimuli may develop further into a failure to comprehend and integrate interpersonal features that provide the basis for human interaction (Grossman et al., 1997).

Within the ASD population, deficits in eye contact are shown in early childhood by a lack of eye-to-eye gaze as well as abnormalities in the intensity of eye contact (Rutter & Bartak, 1971; Volkmar et al., 2005). The lack of appropriate eye contact also contributes to the substantial joint attention impairments experienced by individuals with ASD (Grossman et al., 1997; Rutter & Bartak, 1971). Grossman and colleagues (1997) defined joint attention as a "class of preverbal social communicative skills relating to sharing with another person the experience of a third object or event" (p. 442). Joint attention typically develops by 8 to 12 months of age and involves a series of behaviors such as pointing and gesturing, following a point, eye contact, and reading and expressing facial emotions (Grossman et al., 1997). The deficits in joint attention evinced by individuals with ASD are distinct and fairly consistent over every developmental level (Grossman et al., 1997).

Within joint attention, there are two behaviors of note: protoimperative gesturing and protodeclarative gesturing. Protoimperative gesturing involves the use of eye gaze or gesture to gain someone's help in obtaining a desired object or outcome. For example, if a child wanted a toy but the toy was very high on a shelf, the child might use protoimperative gesturing to point to the toy and ask for help. Protodeclarative gesturing is using eye gaze or gestures to gain someone's attention to an object or experience without the purpose of tangible gain. For example, if a child was interested in a show on television, they may make eye contact with their parents and point to the television to convey their enjoyment (Grossman et al., 1997). Young children with ASD may demonstrate protoimperative gestures; however, there is most typically a lack of protodeclarative gesturing within the ASD population (Grossman et al., 1997). A lack of joint attention skills places children with ASD at risk for debilitated interactions with others and formations of social relationships (Grossman et al., 1997).

In typically developing children, attachment behaviors develop by 12 months of age; these behaviors can be characterized by a child's desire for maintaining closeness to his/her caregiver and distress when the caregiver is absent (Grossman et al., 1997). Children with ASD do not form typical attachment patterns with their caregivers (Grossman et al., 1997). While attachment behaviors never emerge in some children with ASD (Rutter, 1978; Rutter & Bartak, 1971), other children develop abnormal, idiosyncratic attachment patterns (Grossman et al., 1997). Young children with ASD may not seek comfort when hurt or upset, may not show distress following a caregiver's departure or joy following a caregiver's return, may not show variation in facial expression, or may not respond to the speech of their parents (Grossman et al., 1997; Rutter, 1978; Rutter & Bartak, 1971).

Young children with ASD have been found to produce less spontaneous and elicited motor or vocal imitation (Grossman et al., 1997; Volkmar et al., 2005). Infants with ASD smile less frequently, vocalize less, and demonstrate less object exploration (Volkmar et al., 2005). Children with ASD are less likely to respond to their name and less likely to respond and orient themselves in the direction of speech (Volkmar et al., 2005). Young children with ASD typically do not demonstrate a range of facial expressions and lack nonverbal communication skills like nodding and gesturing (Volkmar et al., 2005).

Play skills normally emerge by 24 months of age in a typically developing child; however, age appropriate play skills may never develop in individuals with ASD (Grossman et al., 1997). Impairments in reciprocal social play are usually recognized by parents of children with ASD prior to age 2. Infants with ASD will often fail to imitate in games like peek-a-boo or show a lack of social interest and engagement (Grossman et al., 1997). Children with ASD rarely use toys in an appropriate manner; instead, they may engage in repetitive and stereotyped object manipulation (e.g., repetitively spinning wheels on a truck; Grossman et al., 1997). Additionally, individuals with ASD demonstrate a lack of spontaneous pretend play (e.g., pretending a banana is a phone; Grossman et al., 1997).

Children with ASD also display deficits in perspective taking; it is difficult for an individual with ASD to recognize the mental processes of other people (Grossman et al., 1997). To demonstrate this impairment, Baron-Cohen, Leslie, and Frith (1985) employed what became known as the Sally-Anne task. In the Sally-Anne task, a participant watches a video where Sally puts a marble in a certain place; she then leaves and Anne puts the marble in a different location. After viewing the scenario, participants are asked where Sally will look for her marble. To answer the study question correctly, the participants must recognize that because Sally did not

see Anne move the marble, she will still look in its original location (Baron-Cohen et al., 1985; Grossman et al., 1997). Results of the Sally-Anne task indicated that 80% of verbal children with ASD with mental ages over 4 years old failed the task compared to 14% of children with Down syndrome (Baron-Cohen et al., 1985). Therefore, these perspective taking deficits appear autism specific (Grossman et al., 1997).

As children with ASD get older and social demands increase, their deficits become more pronounced (White, Keonig, & Scahill, 2006). In conversation, children with ASD often have difficulty with turn taking and perspective taking, voice pitch and inflection that are necessary for conveying meaning, comprehending and expressing emotion, and understanding nonliteral language (White et al., 2006). Additionally, individuals with ASD tend to perseverate on certain topics of restricted interest in interactions (White et al., 2006). Children with ASD are less likely to approach peers to play or respond to peer initiations (Grossman et al., 1997). After age 5, a failure to make and maintain friendships, a lack of group involvement, and an increase in socially inappropriate behaviors are typical of the ASD population (Rutter, 1978; Rutter & Bartak, 1971).

Researchers have found children with ASD may process social situations differently (Klin Jones, Schultz, Volkmar, & Cohen, 2002). By tracking eye gaze while watching videos of social scenarios, Klin and colleagues (2002) identified various discrepancies between children with ASD and children with typical development. First, while typically developing participants had a tendency to fixate on the eyes of a conversationalist, the majority of children with ASD focused on the speaker's mouth (Klin et al., 2002). It has been theorized that because individuals with ASD have little ability to intuitively understand the social information revealed through the eyes, they rely on the verbal aspect of the mouth to comprehend social situations (Grossman,

Klin, & Volkmar, 2000). Despite the potential compensatory consequences of a verbal bias, eye contact is necessary to be considered competent in social interaction (Grossman et al., 2000). Next, Klin and colleagues (2002) found children with ASD did not search for social information in other people's reactions to the speaker. Recognizing the reaction of others is necessary for comprehending irony or embarrassment and for identifying context (Klin et al., 2002). Children with ASD also tended to ignore important social cues and attend more to physical cues; for example, children with ASD did not follow a character's point but instead moved their gaze between objects (Klin et al., 2002).

Social impairment and anxiety related to those impairments may increase when individuals with ASD approach adolescence; at this time, age appropriate social situations become much more complex and the individual may become aware of their social deficiencies (White et al., 2006). Individuals with ASD may report wanting more peer social interaction but may also report loneliness, peer rejection and social isolation (White et al., 2006). Socialization impairment plays a role in academic and occupational dysfunction and may also predict mood and anxiety problems later in life (White et al., 2006).

COMORBID PSYCHOPATHOLOGY

Although the possibility of comorbid psychopathology in individuals with ASD was previously disregarded, it is now known that co-occurring neurodevelopmental and mental health conditions are common in the autism population (de Bruin et al., 2007; Dover & Le Couteur, 2007; Matson & Nebel-Schwalm, 2007; Mattila et al., 2010; Morgan et al., 2003; Simonoff et al., 2008). Simonoff and colleagues (2008) conducted a large-scale study utilizing an epidemiological, population-derived sample of individuals and standardized assessments examining the rate of co-occurring conditions among the ASD population. The researchers estimated 70% of children with ASD met criteria for at least one comorbid condition and 41% of children with ASD had two or more co-occurring disorders (Simonoff et al., 2008). Included in these common comorbid disorders were a variety of anxiety disorders, ADHD, ODD and conduct disorder (CD), and mood and affective disorders (Simonoff et al., 2008).

While there is a developing and substantial research pool involving co-occurring disorders in autism, the concept of comorbidity remains debated and unclear (Wood & Gadow, 2010). Researchers and clinicians struggle with identifying a symptom set as a condition distinguishable from ASD that is similar to the condition in an individual without ASD, a condition distinguishable from ASD that may manifest differently due to ASD symptomology, or as symptoms inseparable from ASD representing the heterogeneity in the presentation of autism (Wood & Gadow, 2010). At this time, the majority of research indicates conditions co-occur with ASD and are not simply variants in the presentation of ASD (Wood & Gadow, 2010). Matson and Nebel-Schwalm (2007) emphasize that the symptomology of ASD remains stable across time, in contrast to the variation in intensity and presenting symptoms of other childhood psychopathologies seen even in individuals with ASD.

Recognizing and diagnosing comorbidities in individuals with ASD can be difficult. Symptoms of co-occurring psychopathologies are often overshadowed by the pervasiveness of ASD; these symptoms are often attributed to the ASD diagnosis (Simonoff et al., 2008). Additionally, communication impairments and intellectual deficits make clinical interview challenging (Leyfer et al., 2006; Simonoff et al., 2008; Volkmar & Cohen, 1991); therefore, psychopathology assessment modifications should be made to account for IQ and language skill deficiencies (Witwer & Lacavalier, 2010). Within individuals with ASD who have functional language, the ability to describe mental states and experiences is still impaired through deficits in perspective taking, information processing, and executive functioning (Leyfer et al., 2006).

Attention-Deficit/Hyperactivity Disorder

ADHD is characterized by an enduring pattern of inattention and/or hyperactivity and impulsivity (APA, 2013). The *DSM-5* criteria requires six or more symptoms of inattention and/or six or more symptoms of hyperactivity/impulsivity (five or more for individuals 17 years or older) present for at least 6 months that are inappropriate for the individual's developmental level. The inattention symptoms consist of: (1) often fails to pay attention to details, makes careless mistakes in work or activities, (2) difficulty maintaining attention on tasks or play, (3) does not seem to listen when spoken to directly, (4) difficulty following through on instructions, fails to finish schoolwork, chores, etc., (5) had trouble organizing, (6) avoids, dislikes, or is reluctant to do tasks that require maintained mental effort and attention, (7) often loses things, (8) easily distracted, and (9) is often forgetful (APA, 2013).

The hyperactivity and impulsivity symptoms include: (1) fidgets or squirms in seat, (2) displays inappropriate out of seat behavior, (3) runs around in situations where it is inappropriate, (4) unable to play quietly, (5) is often "on the go," (6) talks excessively, (7) often

yells out an answer before a question is completed, (8) has trouble waiting his/her turn, and (9) often interrupts or intrudes on others (APA, 2013). Additionally, symptoms must be present before 12 years of age, symptoms must be present in two or more settings, symptoms must interfere with daily functioning, and symptoms may not be better accounted for by another diagnosis (APA, 2013).

There are three different presentations of ADHD that can occur. Individuals can have the Combined Presentation (meeting the inattention and the hyperactivity/impulsivity criteria), the Predominately Inattentive Presentation (meeting the criteria for inattention but not hyperactivity/impulsivity), or the Predominantly Hyperactive-Impulsive Presentation (meeting the criteria for hyperactivity/impulsivity but not inattention). The *DSM-5* also emphasizes the presentation of ADHD may change over time (APA, 2013).

Individuals with ADHD commonly exhibit significant socialization impairment (Cervantes et al., 2013; Hoza et al., 2005; Miller, Nielson, & Shoen, 2012). These socialization deficits in ADHD often present as interrupting others, imposing on other's conversations, having difficulty navigating social conflict, and talking excessively (Miller, Nielson, & Shoen, 2012). Researchers have found that children with ADHD are more likely to be rejected by their peers (Hoza et al., 2005; Nijmeijer et al., 2008). Deficits in social skills associated with ADHD begin in childhood and continue through the lifespan; some researchers have found that socialization impairment may actually worsen over time in children with ADHD (Cervantes et al., 2013).

A high rate of comorbid ADHD has been identified in individuals with ASD (de Bruin et al., 2007; Leyfer et al., 2006; Mattila et al., 2010; Russell, Rodgers, Ukoumunne, & Ford, 2014; Simonoff et al., 2008.). Leyfer and colleagues (2006) found that 31% of children with ASD met diagnostic criteria for ADHD; further, 55% of children with ASD presented with elevated levels

of inattentive and hyperactive/impulsive symptoms. The common co-occurrence of ASD and ADHD symptoms has also been shown in infants and toddlers (Matson, Mahan, Sipes, & Kozlowski, 2010). A majority of individuals with ASD and ADHD have the Predominately Inattentive presentation (de Bruin et al., 2007; Leyfer et al., 2006). Researchers have also found that individuals with ASD who have intellectual disabilities are more likely to exhibit ADHD symptoms (Witwer & Lacavalier, 2010). Though ADHD is substantially more common in males than females in the general population, the gender differences are not seen within the ASD population (Simonoff et al., 2008).

When ASD and ADHD co-occur, the manifestation of symptoms can differ from the presentation of symptoms when ASD occurs alone or ADHD occurs alone (Holtmann et al., 2007; Matson, Mahan, et al., 2010; Matson, Hess, Neal, et al., 2010; Tureck, Matson, May, & Turygin, 2013; Yerys et al., 2009). Individuals with co-occurring ASD and ADHD experience exacerbated autism traits and may fall at a higher ASD severity rating (Yerys et al., 2009). Researchers have found children with comorbid ASD and ADHD are more impaired in executive functioning, daily living skills, and socialization when compared to children with ASD alone (Holtmann et al., 2007; Yerys et al., 2009). Co-occurring ASD and ADHD has also been correlated with greater rates of both internalizing and externalizing behavior, aggression, destruction, stereotypies, and tantrum behavior (Holtmann et al., 2007; Matson, Mahan, et al., 2010; Tureck et al., 2013; Yerys et al., 2009).

Anxiety Disorders and Obsessive Compulsive Disorder

The *DSM-5* category of Anxiety Disorders consists of various disorders characterized by excessive fear and anxiety as well as associated behavior disturbance; these disorders include separation anxiety disorder, selective mutism, specific phobia, social anxiety disorder,

agoraphobia, generalized anxiety disorder, and panic disorder (APA, 2013). Although OCD is categorized under the Obsessive-Compulsive and Related Disorders label (APA, 2013), many researchers lump OCD in with anxiety disorders; because of this, OCD will be addressed in this section. The three most prominent anxiety related disorders found in the ASD population are specific phobia, social anxiety disorder, and OCD (de Bruin et al., 2007; Leyfer et al., 2006; Simonoff et al., 2008; White, Oswald, Ollendick, & Scahill, 2009).

The *DSM-5* criteria for specific phobia includes (1) marked fear or anxiety about a specific object or situation, (2) the object or situation almost always instigates immediate fear or anxiety, (3) the object or situation is avoided or is endured with intense fear or anxiety, (4) the fear or anxiety is out of proportion to the actual danger posed and to the sociocultural context, (5) the fear, anxiety, or avoidance lasts 6 or more months, (6) the fear, anxiety, or avoidance causes clinically significant distress or impairment in functioning, and (7) the symptoms are not better explained by the symptoms of another mental disorder (APA, 2013).

Social anxiety disorder is defined within the *DSM-5* as (1) marked fear or anxiety about 1 or more social situations where an individual may experience scrutiny by others (social interactions, being observed, performing in front of others, etc.), (2) the individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (cause humiliation or embarrassment; lead to rejection; offend others), (3) the social situations almost always instigate fear or anxiety, (4) the social situations are avoided or endured with intense fear or anxiety, (5) the fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context, (6) the fear, anxiety, or avoidance lasts 6 or more months, (7) the fear, anxiety, or avoidance causes clinically significant distress and functioning impairment, (8) the fear, anxiety, or avoidance is not attributable to the effects of a substance or

medical condition, (9) the fear, anxiety, or avoidance is not better explained by another mental disorder, (10) the fear, anxiety, or avoidance is clearly unrelated or in excess if another medical condition is present (APA, 2013).

Lastly, OCD is characterized by (1) the presence of obsessions, compulsions, or both (APA, 2013). The *DSM-5* defines obsessions as (a) recurrent and persistent thoughts, urges, or images that are experienced at some time during the disturbance as intrusive and unwanted and that cause marked anxiety or distress, and (b) the individual attempts to ignore or suppress these intrusive thoughts, urges, or images, or to neutralize them by carrying out a compulsion (APA, 2013). Compulsions are defined by (a) repetitive behaviors that the individual is motivated to perform in response to an obsession or according to rigid rules and (b) the behaviors are aimed at preventing or diminishing anxiety or distress, or preventing some dreaded event; however, the behaviors are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive (APA, 2013). Additionally, diagnostic criteria states (2) the obsessions or compulsions must be time-consuming or cause clinically significant distress or impaired functioning, (3) the symptoms must not be attributable to the effects of a substance or medical condition, and (4) the symptoms are not better accounted for by another disorder (APA, 2013).

Children with anxiety symptoms have been found to demonstrate lower rates of appropriate social behavior and a lower overall social competence (Strauss, Lease, Kazdin, Dulcan, & Last, 1989). Anxious children are more likely to experience loneliness, be described by teachers and caregivers as socially maladjusted, and display shy and withdrawn social behavior (Strauss et al., 1989). Concerning peer relationships, children with anxiety are more

likely to be rejected, and are rated as less liked by classmates (Strauss, Lahey, Frick, Frame, & Hynd, 1988).

Social anxiety disorder is notable when discussing the relationship of anxiety disorders to socialization deficits. Social anxiety disorder, by definition, causes social impairment (i.e., avoidance or intense distress in social situations; APA, 2013). Children with social anxiety disorder have been found to have very few friends, experience extreme loneliness, and avoid extracurricular activities (Beidel, Turner, & Morris, 1999). Individuals with social anxiety disorder not only engage in social behaviors less often but have poorer social skills compared to individuals without social anxiety disorder; this skill deficit is evident through longer speech latencies which are characteristic of behavioral inhibition (Beidel et al., 1999).

It has been well established that anxiety occurs at substantially high rates in individuals with ASD; in fact, Kanner (1943) noted that significant problems with anxiety were very common among the cases he followed. Prevalence rates of comorbid anxiety disorders have been estimated between 11 and 84% (de Bruin et al., 2007; Leyfer et al., 2006; Simonoff et al., 2008; White et al., 2009). Among the most common co-occurring anxiety disorders in ASD are specific phobia, social anxiety disorder, and OCD (de Bruin et al., 2007; Leyfer et al., 2006; Simonoff et al., 2008; White et al., 2009). Several factors influence the manifestation of anxiety in individuals with ASD including severity of autism symptoms, level of cognitive functioning, and degree of social impairment (Davis III et al., 2012; White et al., 2009; Witwer & Lacavalier, 2010). Researchers have found individuals with ASD who have an IQ score above 70 may experience more anxiety symptoms (Witwer & Lacavalier, 2010). Additionally, communication deficits have been found to be correlated with higher rates of anxious behaviors in infants and toddlers with ASD (Davis III et al., 2012).

Leyfer and colleagues (2006) found the most frequently co-occurring anxiety disorder within the ASD population was specific phobia (44%). The most common phobias included needles and shots, crowds, and loud noises; while, the most frequent phobias in typically developing children (i.e., flying, store, standing in lines, bridges/tunnels) occurred at very low rates in children with ASD (Leyfer et al., 2006). Matson and Nebel-Schwalm (2007) found a similar pattern where fears in children with ASD had minimal correspondence with fears in age-matched, typically developing peers. An ASD diagnosis may be indicative of an increased risk for certain, specific fears and phobias, which may aid in appropriate clinical assessment and treatment of these phobias (Matson & Nebel-Schwalm, 2007).

While some researchers deny the comorbidity and contribute symptoms of social worry to the defining socialization deficits of ASD, others have found social anxiety disorder is a separate and distinguishable condition within ASD and co-occurs at significantly higher rates than in typically developing children (Leyfer et al., 2006; Simonoff et al., 2008; White et al., 2009; Wood & Gadow, 2010). Social fears and avoidance behaviors related to social situations may be associated but not indistinct from deficient social skills (Leyfer et al., 2006). Social anxiety occurs more often in higher functioning individuals with ASD (White et al., 2009); researchers believe individuals with high functioning ASD are more aware of their social impairments, and thus are more anxious about social interaction. This could cause a reciprocal relationship where an individual avoids engaging in social behavior due to social fear, thus further debilitating their social development by restricting opportunities to learn (White et al., 2009).

Prevalence estimates of OCD within the ASD population have been inconsistent (Leyfer et al., 2006; Wood & Gadow, 2010). Leyfer and colleagues (2006) reported 37% of children

with ASD met criteria for OCD. The most common compulsions of their participants with ASD involved the actions of other people; for example, requiring parents to ask the same question repetitively (Leyfer et al., 2006). Because individuals with ASD have difficulty communicating their thoughts and feelings, diagnoses of OCD are most often made solely on observable behaviors indicative of compulsions, rather than obsessions which are generally not able to be observed (Leyfer et al., 2006). Wood and Gadow (2010) warn clinicians of the difficulty in differential or dual diagnosis of OCD and ASD. The repetitive behaviors characteristic of ASD may be misrepresented as compulsions. Additionally, perseverations and preoccupations are common in children with ASD. However, the two disorders could be discriminated by the child's reactions to thoughts. While repetitive thoughts and behaviors associated with ASD are often enjoyable for an individual, the consuming obsessions of OCD are unpleasant and usually related to perceived danger (Wood & Gadow, 2010).

Higher rates of anxiety in individuals with ASD are associated with increased deficits in daily functioning and greater clinical impairment related to core ASD symptoms (Wood & Gadow, 2010). Children with ASD with elevated ratings of anxiety also report experiencing more social loneliness (White & Robserson-Nay, 2009). Although anxiety is common in individuals with ASD, anxiety is not a defining feature of the disorder and is not experienced by everyone with ASD. Therefore, anxiety should be considered a comorbid psychopathology and not another aspect within the autism spectrum (Leyfer et al., 2006).

Oppositional or Conduct Disorders

The ODD criteria in the *DSM-5* involve 3 domains of symptoms: Angry/Irritable Mood, Argumentative/Defiant Behavior, and Vindictiveness (APA, 2013). To meet for the disorder, an individual must present with at least four symptoms from any of the three domains; and, these

symptoms must have lasted at least 4 months (APA, 2013). The Angry/Irritable Mood domain symptoms include: (1) often loses temper, (2) touchy or easily annoyed, and (3) angry or resentful. The symptoms in the Argumentative/Defiant Behavior domain consist of: (1) argues with authority figures or adults, (2) actively noncompliant with requests from authority figures and adults or with rules, (3) deliberately annoys others, and (4) blames others for mistakes and misbehavior. Within the Vindictiveness domain is: (1) demonstrated spiteful or vindictive behavior at least twice in past 6 months (APA, 2013).

CD has been found to be less likely to co-occur with ASD (de Bruin et al., 2007); therefore, less emphasis will be placed on CD in this section. Of note, CD is defined as "a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated," (APA, 2013, p. 469). The four criteria domains of CD include: Aggression to People and Animals, Destruction of Property, Deceitfulness or Theft, and Serious Violations of Rules (APA, 2013).

The presence of ODD has been found to be a significant predictor of social problems at school, deficits in peer relations, and difficulties in parent-child relations (Green et al., 2002). Children with ODD experience high levels of interpersonal conflict and may perceive social exchanges as more hostile than children without ODD (Coy, Speltz, DeKlyen, & Jones, 2001). Researchers have found individuals with ODD are particularly impaired in social problem solving and are more likely to act aggressively in response to conflict (Coy et al., 2001). Because of these behavioral tendencies, children with ODD are more likely to be socially rejected by peers (Coy et al., 2001). Additionally, the high comorbidity of ODD with ADHD may add to a child's socialization deficits (Coy et al., 2001; Greene et al., 2002).

Children with ODD have been found to be rated higher on ASD symptoms compared to children without ODD (Gadow & Drabick, 2012). Researchers suggest the difficulty with emotion control and interpersonal relationships seen in individuals with ODD may coincide with the social skill deficits typical of individuals with ASD. Both individuals with ODD and individuals with ASD find socialization difficult; therefore, socializing in both populations can cause a range of challenging behaviors and emotional reactions (Gadow & Drabick, 2012).

Comorbidity rates of ASD and ODD have been estimated as high as 37.2% and rates of ASD and CD as high as 9.6% (de Bruin et al., 2007; Simonoff et al., 2008). Even in those children without a diagnosable ODD or CD, a large portion of the ASD population still exhibit symptoms consistent with ODD characterized by a pattern of hostile and defiant behavior directed towards adults and authority figures (Gadow, DeVincent, & Drabick, 2008). The symptoms of anger and irritability, and the subsequent emotional outbursts commonly seen in children with ASD are associated with increased use of pharmacotherapy, peer isolation, and family stress (Gadow & Drabick, 2012).

However, estimates of comorbidity rates have been inconsistent; Leyfer and colleagues (2006) found only 7% of children with ASD met criteria for ODD. The researchers reasoned that due to deficits in perspective taking and executive functioning, individuals with ASD may not fully comprehend the concepts of spitefulness, vindictiveness, and intentionality that make up the symptoms of the ODD diagnostic criteria (Leyfer et al., 2006). The trend of symptoms of ODD has been found to differ in the ASD population compared to the general population. While symptoms of ODD tend to either decrease with age or progress in severity to meet CD criteria, the behavioral manifestation of ODD appears relatively permanent throughout childhood in individuals with ASD (Mattila et al., 2010). Despite this, ODD in a child with ASD is very

similar in many other respects to ODD in a child without ASD. Therefore, ODD is still seen as a separate and distinguishable disorder in children with ASD (Gadow et al., 2008).

Assessment of Comorbid Psychopathology

As previously stated, the assessment of comorbid psychopathology in individuals with ASD can be very difficult. Described below are several of the instruments used to identify comorbid conditions in ASD including the *Child Behavior Checklist (CBCL;* Achenbach & Rescorla, 2000), the *Behavioral Assessment System for Children, Second Edition* (*BASC-2*; Reynolds & Kamphaus, 2004), and the *Autism Spectrum Disorder Battery – Child Version – Comorbidity* (*ASD-CC;* Matson & González, 2007). While the *CBCL* and the *BASC-2* were not specifically intended for use within the ASD population, both are frequently used in autism evaluation. The *ASD-CC* is unique in that it is one of the few measures designed to target comorbid psychopathology specifically within children with ASD. Like the *ASD-CC*, the *Baby and Infant Screen for Children with aUtIsm Traits - Part 2 (BISCUIT-Part 2*) also assesses for comorbid symptoms in infants and toddlers with ASD and will be addressed further within the Method section.

Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000). The *Child Behavior Checklist* (*CBCL*) is a part of a larger assessment procedure called the *Achenbach System of Empirically Based Assessment* (*ASEBA;* Achenbach & Rescorla, 2000). The *ASEBA* consists of the *CBCL*, to be completed by a parent or caregiver, the *Teacher Report Form (TRF*) or the *Caregiver-Teacher Report Form (C-TRF*) for children not yet in school, and the *Youth Self-Report (YSR*) which is a self-rating form for children aged 11 to 18 years. The *CBCL* comes in 2 versions: one for assessing children aged 1.5 to 5 years and one intended for use with children 6 to 18 years old (Achenbach & Rescorla, 2000).

The *CBCL* has an administration time of about 20 minutes and consists of 120 items that are rated on a 3-point Likert scale. A rating of 0 suggests "not true;" a rating of 1 indicates "somewhat or sometimes true;" and a rating of 2 indicates "very true or often true" (Achenbach & Rescorla, 2000). The *CBCL* assesses for internalizing and externalizing problems. The eight behavioral subscales of the *CBCL 6 – 18* consist of Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, and Aggressive Behavior (Achenbach & Rescorla, 2000). The subscales of the *CBCL 1.5 – 5* include Emotionally Reactive, Anxious/Depressed, Somatic Complaints, Withdrawn, Attention Problems, Aggressive Behavior, and Sleep Problems (Achenbach & Rescorla, 2000). Unique to the *CBCL 1.5 – 5* is the Language Development Survey (LDS) designed to assess children less than 3 years of age suspected of having language delays (Achenbach & Rescorla, 2000).

The psychometric properties of the *CBCL* have been extensively researched. The *CBCL 1.5 – 5* as well as the *CBCL 6 – 18* have been found to be reliable with test-retest reliability coefficients above .90 (Achenbach & Rescorla, 2000). Validity estimates have been found adequate for the *CBCL 1.5 – 5,* the *CBCL 6 – 18*, and the *LDC* specifically (Achenbach & Rescorla, 2000; Rescorla, 2005).

Behavioral Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004). The *Behavior Assessment System for Children, Second Edition (BASC-2*) is a multidimensional assessment system designed to identify emotional and behavioral disorders in children and adults aged 2 to 25 years. The *BASC-2* includes five distinct forms combined to achieve a well-rounded perception of the functioning of the individual being assessed. The five components consist of: a Teacher Rating Scale (TRS), a Parent Rating Scale

(PRS), a student Self-Report of Personality (SRP), a Structured Developmental History (SDH), and a Student Observation System (SOS). The TRS and PRS are available in three separate versions: the Preschool version (intended for use with children 2 to 5 years old), the Child version (intended for use with children aged 6 to 11 years), and the Adolescent version (intended for use with adolescents aged 12 to 21 years). Three versions of the SRP are also available; they are the Child (ages 8 to 11), Adolescent (ages 12 to 21), and College (ages 18 to 25 years attending postsecondary school) versions.

Items on the *BASC-2* are rated according to a 4-point scale indicating the frequency of the behavior described. A rating of "N" indicates the item never occurs, "S" indicates sometimes, "O" indicates often, and "A" indicates almost always (Reynolds & Kamphaus, 2004). For the preschool versions, the TRS is made up of 100 items and the PRS consists of 134 items. Scores are reported in the three domains of Externalizing Problems, Internalizing Problems, and Adaptive Skills. The domain of Externalizing Problems consists of items assessing for symptoms like hyperactivity and aggression; while, the Internalizing Problems domain includes symptoms like anxiety, depression, and somatization (Reynolds & Kamphaus, 2004). The Behavioral Symptoms Index or BSI is a composite score of overall problem behaviors (Tan, 2007).

The *BASC-2* has been found reliable with internal consistency estimates above .90 for both the TRS and PRS (Tan, 2007). Inter-rater reliability and test-retest reliability estimates have also been found adequate for the *BASC-2* (Tan, 2007). Criterion-related validity has been found adequate when compared to the *ASEBA* (Achenbach & Rescorla, 2000) and the *Conners' Teacher Rating Scale–Revised (CTRS-R*; Conners, 1997; Tan, 2007). However, a significant

disadvantage of utilizing the *BASC-2* is its age range. Children younger than 2 years cannot be assessed with this measure (Tan, 2007).

Autism Spectrum Disorder Battery, Child Version, Comorbidity (ASD-CC; Matson & González, 2007). The *Autism Spectrum Disorder Battery – Child Version Comorbidity* (*ASD-CC)* is one scale of the three-part *Autism Spectrum Disorder Battery – Child Version (ASD-C)*. The *ASD-CC* was designed to assess for comorbid symptoms that commonly occur within children with ASD. The subscales of the *ASD-CC* include Tantrum Behaviors, Repetitive Behaviors, Worry/Depression, Avoidant Behaviors, Conduct, Over-Eating and Under-Eating. The measure consists of 39 items rated by a caregiver on a 3-point Likert scale. The informant is told to rate the child being assessed, compared to other children his or her age and to the extent that the item is a recent problem. A score of a 0 indicates "not a problem or impairment; not at all;" a score of 1 indicates "mild problem or impairment;" and, a score of 2 indicates "severe problem or impairment." Examples of items on the *ASD-CC* include, "fear of being around others in school, at home, or in social situations," "blames others for his/her misdeeds," "feelings of worthlessness or excessive guilt," and "eats things that are not meant to be eaten."

The *ASD-CC* is unique from the *CBCL* and *BASC* because it is designed to assess for comorbid symptoms specifically in the ASD population. The *ASD-CC* has been found reliable with an internal consistency coefficient of .91, an inter-rater reliability coefficient of .46, and a test-retest reliability coefficient of .51 (Matson & Wilkins, 2008). The *ASD-CC* has been found to correlate with the subscales of the *BASC-2* evidencing its validity (Matson, LoVullo, Rivet, & Boisjoli, 2009).

PURPOSE

Socialization impairment is among the most prominent core deficits in individuals with ASD regardless of the presentation of comorbid psychopathology (Kanner, 1943; Rutter, 1978; Volkmar et al., 2005). However, when the conditions that frequently co-occur with ASD occur alone, social impairment is also evident (Beidel et al., 1999; Coy et al., 2001; Miller et al., 2012). For example, children with ADHD often talk excessively and intrude in on the conversations of others (Miller et al., 2012), children experiencing symptoms of anxiety often actively avoid social situations or experience significant distress when interacting (Beidel et al., 1999), and children with oppositional behaviors experience high rates of interpersonal conflict (Coy et al., 2001). Children with both ASD and a comorbid disorder present a complicated clinical picture (Dover & Le Couteur, 2007). Because symptoms have been found to manifest differently when an individual presents with ASD and comorbid psychopathology, assessment and treatment can be difficult (Morgan et al., 2003). Children with ASD and a co-occurring disorder require individualized treatment plans to target behavioral factors associated with ASD symptomology, with comorbid psychopathology, and with the interaction of the two (Morgan et al., 2003).

Researchers have found that the severity of these comorbid symptoms are related to the severity of core ASD symptoms; when an individual's core symptoms of ASD are more severe, he or she will demonstrate more severe comorbid symptomology (Matson, Hess, & Boisjoli, 2010). Further, Matson, Worley, Neal, Mahan and Fodstad (2010) found that very young children with ASD displaying moderate to severe clinical impairment related to inattention and impulsivity demonstrated significantly more social impairments than children with ASD displaying no to minimal inattention/impulsivity impairment. Taken together, these results

suggest that more severe symptoms of ASD as well as more severe comorbid psychopathology put individuals, even very young children, at greater risk for social deficiencies.

Therefore, the current study aimed to explore the effect of comorbid symptoms on social skills beyond that accounted for by the effect of ASD severity. The current study utilized the *Baby and Infant Screen for Children with aUtIsm Traits - Part 1 (BISCUIT-Part 1*), the *BISCUIT-Part 2*, and the *Battelle Developmental Inventory, Second Edition (BDI-2*) to assess ASD symptom severity, comorbid symptomology, and socialization deficits respectively among infants and toddlers with ASD. Comorbid symptoms consistent with frequently co-occurring disorders (e.g., ADHD, anxiety disorders, ODD) were examined. The purpose of the current study was to explore the interaction between ASD and comorbidity and establish if comorbid symptoms predict socialization impairment beyond what can be predicted by ASD severity in very young children with ASD. The current study sought to enhance the understanding of the role of comorbid psychopathology in the deficits experienced by young children with ASD. Results hold clinical implications for both the assessment and treatment of ASD at early stages of development.

Based on existing literature, it is hypothesized that first, ASD severity will be significantly and negatively correlated with overall social skills as well as skills related to adult interaction, peer interaction, and self-concept and social role. Additionally, ASD severity is expected to be a significant predictor of overall social skills as well as skills related to adult interaction and peer interaction, and self-concept and social role.

Next, it is expected that beyond the influence of ASD severity, the co-occurring symptoms of Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior will be significantly and negatively correlated with social skills in

infants and toddlers with ASD. These comorbid behaviors are also hypothesized to significantly predict some of the variance in overall social skills as well as in skills specific to adult interaction, peer interaction, and self-concept and social role in infants and toddlers with ASD. Each of the four comorbid symptoms examined are not expected to contribute equally to social skill impairment; however, based on previous research, behaviors consistent with *DSM* diagnoses of ODD, ADHD, and anxiety disorders should impact the socialization of these children in some significant way.

METHOD

Participants

The sample consisted of 249 infants and toddlers with ASD, aged 24 to 36 months (M = 28.86, SD = 3.32). All participants in the sample were already recruited through EarlySteps, which is Louisiana's Early Intervention System under the Individuals with Disabilities Education Act, Part C. EarlySteps provides services to infants and toddlers from birth to 36 months of age. To qualify for EarlySteps services, a child must have a developmental delay or have a medical condition likely to result in a developmental delay like cerebral palsy, epilepsy, deafness, blindness, and premature birth. The diagnoses of ASD were assigned by a licensed doctoral level psychologist with over 30 years of experience in the field. Diagnoses were assigned using an algorithm based upon *DSM-5* (APA, 2013) ASD criteria.

Participants were recruited from a pre-existing dataset of demographic information and results from assessment measures for children enrolled in EarlySteps. For inclusion in this study, participants were required to have an ASD diagnosis and be between 24 and 37 months of age at the time of assessment. Further, participants were included in the study if scores from the *BISCUIT-Part 1*, *BISCUIT-Part 2*, and *BDI-2* were available and free of missing or incorrectly coded data. Participants were excluded if they did not meet for a diagnosis of ASD, did not meet age requirements, or were missing data points for any of the measures. The total sample meeting inclusionary criteria (n = 249) consisted of 75.90% males and 24.10% females. Of the total participants, 47.00% were Caucasian (n = 117), 41.36% were African American (n = 103), 1.20% were Hispanic (n = 3) and 10.44% were of other or unspecified ethnicity (n = 26). Complete demographic information can be found in Table 1.

Table 1
Demographic Information

	(*n* = 249)
Age in months	
Range	24-36
M	28.86
SD	3.32
Gender (%)	
Male	75.90%
Female	24.10%
Ethnicity (%)	
Caucasian	47.00%
African American	41.36%
Hispanic	1.20%
Other/Unspecified	10.44%

Measures

Baby and Infant Screen for Children with aUtIsm Traits-Part 1 (BISCUIT-Part 1; Matson, Boisjoli, & Wilkins, 2007). The *Baby and Infant Screen for Children with aUtIsm Traits* (*BISCUIT*) is a three-part, informant-based assessment intended to help in the early detection of ASD in infants and toddlers aged 17 to 37 months (Matson, Wilkins, Sevin et al., 2009). The three parts of the *BISCUIT* assess for ASD symptomology, comorbidities, and challenging behaviors, respectively. The *BISCUIT- Part 1* is the diagnostic section of the measure. The *BISCUIT-Part 1* consists of 62 items administered to the parent/caregiver of the child being assessed. Each item is rated on a 3-point Likert scale by comparing the child being assessed to other children his/her age. A rating of 0 suggests "not different; no impairment." A rating of 1 suggests "somewhat different; mild impairment." A rating of 2 suggests "very different; severe impairment." Examples of items on the *BISCUIT-Part 1* include "ability to recognize the emotions of others," "response to others' social cues," "use of language to communicate," and "engages in repetitive motor movements for no reason."

Cut-off scores included in the *BISCUIT-Part 1* indicate that total scores below 17 are categorized in the "No ASD/Atypical Development" range. Scores between 18 and 34 suggest "Possible ASD/PDD-NOS;" and scores at or above 35 falls into the "Probable ASD/Autistic Disorder" range (Matson, Wilkins, Sharp, et al., 2009). Factor analysis indicated three distinct factors including socialization/nonverbal communication, repetitive behaviors/restricted interest, and communication (Matson, Boisjoli, Hess, & Wilkins, 2010).

The *BISCUIT-Part 1* has been found to have robust psychometric properties. The measure holds an overall correct classification rate of .89 and an internal reliability of .97 (Matson Wilkins, Sevin, et al., 2009). Sensitivity and specificity rates when distinguishing ASD (PDD-NOS and autism) from atypically developing children without ASD have been estimated at 93.4 and 86.6, respectively (Matson, Wilkins, Sharp, et al., 2009). The *BISCUIT-Part 1* will be utilized in the current study as a measure of ASD severity.

Baby and Infant Scale for Children with aUtIsm Traits, Part 2 - Comorbidity (BISCUIT-Part 2; Matson, Boisjoli et al., 2007). The *BISCUIT-Part 2* is designed to assess comorbid symptoms commonly co-occurring in infants and toddlers with ASD. Similar to the *BISCUIT-Part 1*, parent/caregiver informants are read 57 items and instructed to rate their child on a scale of 0 to 2. Each item is rated to the extent it has been a recent problem for the child being assessed. A score of 0 indicates "not a problem or impairment;" a score of 1 indicates "mild problem or impairment;" and a score of 2 indicates "severe problem or impairment." The *BISCUIT-Part 2* is made up of five subscales: Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eat/Sleep Problems.

For the present study, the Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior subscales were utilized. These four subscales were chosen for two reasons. The first is due to their correspondence with the symptoms of common comorbid psychopathologies in individuals with ASD (e.g., ODD, ADHD, various anxiety disorders, OCD). Additionally, when symptoms of these disorders occur alone, socialization is impaired. The Tantrum/Conduct Behavior subscale is made up of 19 items; examples include "bullies, threatens, or intimidates others," "irritable mood," and "deliberately annoys others." The Inattention/Impulsivity subscale includes 16 items such as "distracted by objects or people in the environment," "fidgets or squirms," and "listens when spoken to directly." The Avoidance Behavior subscale consists of 9 items like "unreasonable fear of approaching or touching specific objects, people, or animals," "withdraws him/herself from social situations," and "exposure to specific objects or situations provoke immediate distress that is not age appropriate." Finally, the Anxiety/Repetitive Behavior factor is composed of 11 items such as "repetition of actions or words to reduce stress," "checking on play objects excessively," and "trembles or shakes in the presence of specific objects or situations." Age-based cut-off scores indicating level of impairment specific to each subscale are available (Horovitz & Matson, in press). Of note, diagnosable comorbid disorders were not investigated in this study; rather, behaviors consistent with these common comorbidities were examined. *The BISCUIT-Part 2* has been found reliable with an internal consistency of .96 (Matson, Wilkins, Sevin, et al., 2009).

Battelle Developmental Inventory, Second Edition (BDI-2; Newborg, 2005). The *Battelle Developmental Inventory, Second Edition* (*BDI-2*; Newborg, 2005) consists of both structured observation and interview components and is intended to assess the development of a

child from birth to 7 years 11 months. Five domains of development are evaluated: Adaptive (ADP), Personal-Social (P-S), Communication (COM), Motor (MOT), and Cognitive (COG). The 450 items are scored on a 3-point Likert scale. A rating of 0 indicates that the child being assessed has no ability in this skill. A rating of 1 indicates that the child may have an emerging ability in the area, and a score of 2 indicates that the child has ability with the skill. The item ratings combine to yield scores for each domain, respective subdomains (e.g., the Expressive Language and Receptive Language subdomains comprise the Communication domain), as well as a total developmental quotient (DQ). The domain scores as well as the overall DQ have a mean of 100 and a standard deviation of 15. The subdomain scores have a mean of 10 and a standard deviation of 3.

For the present study, only the scores from the P-S domain were utilized. The P-S domain consists of 100 items that assess social skills (Newborg, 2005). The P-S domain contains 3 subdomains: Adult Interaction (AI), Peer Interaction (PI), and Self-Concept and Social Role (SR). The AI subdomain is administered to only children less than 6 years old and consists of 30 items. The AI subdomain assesses attachment behaviors during infancy, ability to interact with adults during infancy, ability to initiate and maintain social contact, and the use of adults to help with problem solving. The PI subdomain assesses children between the ages of 2 and 6 years old. The PI subdomain includes 25 items evaluating social skills like forming friendships, responding to and initiating social interaction with peers, and playing well in a small group. The SR subdomain is administered to children of all ages being assessed with the *BDI-2*. The 45 items that make up this subdomain assess self-awareness, empathy, and coping skills.

Results from psychometric studies have indicated that test-retest reliability is above .90 for all domain scores and the total DQ (Newborg, 2005). Internal consistency coefficients were

found to be .99 for overall DQ and .96 for P-S domain score. All three P-S subdomains were also found to have adequate reliability (Newborg, 2005). Finally, convergent validity was established using several different measures of child development (e.g., the Bayley Scales of Infant Development, Second Edition [BSID-II], the Preschool Language Scales [PLS-4]; Newborg, 2005).

Procedure

Before the administration of measures, the study was approved by the Louisiana State University Institutional Review Board as well as the Office for Citizens with Developmental Disabilities (OCDD) of the State of Louisiana. Informed consent was obtained from the guardians before the initiation of study protocol. The assessments were conducted in the participant's home or daycare and consisted of both child observations and caregiver interviews. The assessments were carried out by the EarlySteps service providers. Eligibility requirements for EarlySteps providers include an appropriate degree and certification or licensure. EarlySteps providers are certified or licensed in fields like speech and language pathology, occupational therapy, physical therapy, psychology, social work, or special education. Degrees attained by the providers range from bachelor's degrees to doctoral degrees. There are roughly 175 professionals providing EarlySteps services in Louisiana, all of whom were trained and experienced in the evaluation and treatment of young children and in administering the study's measures.

STATISTICAL ANALYSES

G*Power 3.1, a power analysis software, was utilized to determine an appropriate sample size. For the power analysis, a power of .80, an alpha of .05, and an effect size of .15 (medium effect size for multiple regression; Cohen, 1988) were used to compute sample size. These values are consistent with what is accepted in the field of psychology research (Hinkle, Wiersma, & Jurs, 2003). The power analysis for the multiple regressions identified a sample size of 92 participants as adequate. The EarlySteps dataset exceeds 92 participants; therefore, all participants meeting inclusionary criteria were included in the analyses.

A series of correlation and multiple regression analyses were performed to address four separate research questions: Beyond the effect of ASD severity, (1) Do comorbid symptoms significantly predict social skill deficits? (2) Do comorbid symptoms significantly predict adult interaction skill deficits? (3) Do comorbid symptoms significantly predict peer interaction skill deficits? And, (4) do comorbid symptoms significantly predict self-concept and social role skill deficits? Standard multiple regression analyses were used as each of the four comorbid symptoms have been shown to effect social skills in individuals without ASD; however, the order of importance in predicting social skill deficit in children with ASD is unknown.

ASD severity was used as a predictor in all analyses and was calculated by summing the responses to items of the *BISCUIT-Part 1*. Comorbid symptoms were used as predictors as well and were computed by summing the item ratings for each of the four factors examined on the *BISCUIT-Part 2* (i.e., Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior).

For each analysis conducted, multiple regression assumptions were checked several ways. The assumption of multicollinearity was examined through computed tolerance and

variance inflation factors (VIF) as well as examining correlations between predictor variables. Tolerance greater than .1, VIFs less than 10, and correlation coefficients between IVs less than .9 indicate that the assumption of multicollinearity is met (Field, 2009). For all analyses, no correlation coefficients above .9 were found between any of the predictors (range: 0.30 – 0.82; see Table 2). Tolerance was greater than 0.1 for all IVs (range: 0.25 – 0.74) and VIFs were less than 10 (range: 1.35 – 4.04) indicating that multicollinearity was not an issue. Normality was assessed through an examination of the normal probability plot as well as computed skewedness and kurtosis statistics for each variable used. For sample sizes between 50 and 300 participants, absolute values for skewedness or kurtosis over 3.29 suggests variation from normality (Kim, 2013). Examination of the normal probability plot showed little deviation from normality as the observed residuals fell close to or on the line for each analysis conducted. All variables assessed fell within the acceptable range for skewedness and kurtosis (range: -0.70 – 0.91); therefore, normality could be assumed. Examination of casewise diagnostics, including Mahalanobis and Cook's distance, was used to identify outliers that may be demonstrating undue influence on the models; no such cases were found. Additionally, the plots of standardized residuals against standardized predicted values showed a random, evenly dispersed array of points indicating the assumptions of linearity and homoscedasticity had been met for each analysis. The assumption of independent errors was examined through computing the Durbin-Watson statistic; the Durbin-Watson statistic ranged from 1.98 to 2.06 indicating that there were no serial correlations among residuals for any multiple regression analysis conducted.

Table 2
Correlations Among Predictors

	ASD Severity	Tantrum/Conduct Behavior	Inattention/ Impulsivity	Avoidance Behavior	Anxiety/ Repetitive Behavior
ASD Severity	---	0.36**	0.49**	0.30**	0.43**
Tantrum/Conduct Behavior	0.36**	---	0.82**	0.42**	0.73**
Inattention/ Impulsivity	0.49**	0.82**	---	0.56**	0.74**
Avoidance Behavior	0.30**	0.42**	0.56**	---	0.64**
Anxiety/Repetitive Behavior	0.43**	0.73**	0.74**	0.64**	---

Note: * = $p<.05$; ** = $p<.0$

Research Question 1: Overall Social Skills

Correlation and multiple regression analyses were conducted to examine the relationship between overall social skills and the five predictors (ASD severity as well as the four comorbid symptom subscales). P-S domain scores served as the outcome or dependent variable (DV) while *BISCUIT-Part 1* total scores and scores from the Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior subscales on the *BISCUIT-Part 2* served as the predictors or independent variables (IVs).

Research Question 2: Adult Interaction Skills

To examine the relationship between adult interaction skills, ASD severity, and comorbid symptomology, correlations and standard multiple regression analyses were conducted. The same IVs as in Research Question 1 were used. The DV was AI subdomain scores from the *BDI-2*.

Research Question 3: Peer Interaction Skills

Correlations and standard multiple regression analyses were conducted to explore the relationship between peer interaction skills, ASD severity, and comorbid symptomology. The same IVs as addressed in the previous research questions were used; and, the DV was PI subdomain scores from the *BDI-2*.

Research Question 4: Self-Concept and Social Role Skills

To assess the relationship between self-concept and social role skills, ASD severity, and comorbid symptomology, correlations and standard multiple regression analyses were conducted. The IVs used to address the prior research questions were utilized. The DV was SR subdomain scores from the *BDI-2*.

RESULTS

First, descriptive statistics for each measure used were computed to examine the distribution of scores across the sample. The total score of the *BISCUIT-Part 1* measuring ASD severity ranged from 8 – 113 in the sample (M = 53.16*;* SD = 20.38). A significant amount of variability was seen in the range of comorbid symptoms across participants. Tantrum/Conduct Behavior subscale scores ranged from 0 – 31 (M = 10.96; SD = 7.78). According to age-based impairment cut-off scores for the ASD population (Horovitz & Matson, in press), 83.93% of the sample fell within the no/minimal impairment range, 14.06% fell within the moderate impairment range, and 2.01% fell within the severe impairment range for tantrum and conduct related symptoms. The Inattention/Impulsivity subscale scores ranged from 0 – 26 *(*M = 8.88; SD = 5.77), with 87.95% of the sample falling in the no/minimal impairment range, 10.04% falling within the moderate impairment range, and 2.01% evincing severe impairment related to inattention and impulsivity. Avoidance Behavior subscale scores ranged from 0 – 13 *(*M = 2.83; SD = 3.20). 85.94% of the participants had no/minimal impairment, 11.25% has moderate impairment, and 2.81% had severe impairment regarding avoidance behaviors. Anxiety/Repetitive Behavior subscale scores ranged from 0 – 15 across participants *(*M = 4.26; SD = 3.42), with 81.93% falling in the no/minimal impairment range, 10.44% falling in the moderate impairment range, and 7.63% of participants falling in the severe impairment range of anxiety behaviors.

The P-S domain score of the *BDI-2* ranged from 55 – 104 across participants with a mean between 1 and 2 SDs below the typically developing population (M = 78.60; SD = 10.61). Scores on the AI subdomain ranged from 1 – 16 (M = 4.37; SD =3.06). The PI subdomain scores ranged from 1 – 13 (M = 5.15; SD = 2.47). SR subdomain scores ranged from 1 – 15 across

participants (M = 4.72; SD = 2.61). All means computed from subdomain scores across participants fell approximately 2 SDs below the typically developing population. Descriptive statistics and impairment ranges are presented on Table 3 and 4.

Table 3
Descriptive Statistics

(n = 249)	***M***	***SD***	***Range***
BISCUIT-Part 1			
Total Score	53.16	20.38	8-113
BISCUIT-Part 2			
Tantrum/Conduct Behavior Score	10.96	7.78	0-31
Inattention/Impulsivity Score	8.88	5.77	0-26
Avoidance Behavior Score	2.83	3.20	0-13
Anxiety/Repetitive Behavior Score	4.26	3.42	0-15
BDI-2			
P-S Domain Score	78.60	10.61	55-104
AI Subdomain Score	4.37	3.06	1-16
PI Subdomain Score	5.15	2.47	1-13
SR Subdomain Score	4.72	2.61	1-15

Table 4
Degree of Impairment Related to Comorbid Psychopathology Across Participants

BISCUIT-Part 2 Comorbid Symptoms	***No/minimal Impairment % (n)***	***Moderate Impairment % (n)***	***Severe Impairment % (n)***
Tantrum/Conduct Behavior	83.93% (209)	14.06% (35)	2.01% (5)
Inattention/Impulsivity	87.95% (219)	10.04% (25)	2.01% (5)
Avoidance Behavior	85.94% (214)	11.25% (28)	2.81% (7)
Anxiety/Repetitive Behavior	81.93% (204)	10.44% (26)	7.63% (19)

Research Question 1: Overall Social Skills

Correlation and multiple regression analyses were conducted to assess the relationship between overall social skills, and ASD and comorbid symptomology. Results indicated that

ASD severity as measured by *BISCUIT-Part 1* total score is significantly, negatively correlated with overall social skills as measured by the P-S domain of the *BDI-2,* $r = -0.44$, $p < .01$. Inattention/Impulsivity was significantly correlated with social skills as well, $r = -0.14$, $p < .05$. Correlations among the Tantrum/Conduct Behavior, Avoidance Behavior, and Anxiety/Repetitive Behavior and the P-S domain were negative however not significant (all $p > .05$). Results from correlation analyses are presented in Table 5.

Table 5
Correlations for Comorbid Psychopathology and P-S Domain Scores

	Correlation with P-S Domain on *BDI-2*
ASD Severity	-0.44**
Tantrum/Conduct Behavior	-0.08
Inattention/Impulsivity	-0.14*
Avoidance Behavior	-0.04
Anxiety/Repetitive Behavior	-0.07

Note: * = $p<.05$; ** = $p<.01$

A standard multiple linear regression was conducted to examine the predictive ability of ASD and comorbid symptomology (i.e., *BISCUIT-Part 1* total and totals from four *BISCUIT-Part 2* subscales) for overall social skills (i.e., P-S domain score). Results indicated that a significant portion of the total variation in social skills was predicted by ASD severity as well as comorbid symptomology, $R^2 = 0.213$, $F(5, 243) = 13.14$, $p < .001$. However, upon further evaluation, ASD severity was the only significant predictor, $\beta = -0.50$, $t(243) = -7.54$, $p < .01$. Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior did not serve as significant predictors (all $p > .05$). Beta values, standard errors, and standardized beta values for the predictors are presented in Table 6.

Table 6
Regression Analysis Predicting P-S Domain Scores

	B	***SE***	β	***t***	**Sig**
ASD Severity	-0.26	1.72	-0.50	-7.54	.000
Tantrum/Conduct Behavior	0.04	0.15	0.03	0.26	.799
Inattention/Impulsivity	-0.08	0.21	-0.04	-0.36	.716
Avoidance Behavior	0.14	0.26	0.04	0.56	.578
Anxiety/Repetitive Behavior	0.40	0.32	0.13	1.27	.204

Research Question 2: Adult Interaction Skills

Similarly, to assess the relationship between adult interaction skills and ASD and comorbid symptomology, correlation and multiple regression analyses were conducted. Results indicated that ASD severity as measured by *BISCUIT-Part 1* total score is significantly, negatively correlated with adult interaction skills as measured by the AI subdomain of the *BDI-2,* $r = -0.39$, $p < .01$. Inattention/Impulsivity was also significantly correlated with adult interaction skills, $r = -0.13$, $p < .05$. Correlations among the Tantrum/Conduct Behavior and Anxiety/Repetitive Behavior with the AI subdomain were negative however not significant. Avoidance Behavior was positively correlated with the AI subdomain but not significant (all $p > .05$). Results from correlation analyses are presented in Table 7.

Table 7
Correlations for Comorbid Psychopathology and AI Subdomain Scores

	Correlation with AI Subdomain on *BDI-2*
ASD Severity	-0.39**
Tantrum/Conduct Behavior	-0.06
Inattention/Impulsivity	-0.13*
Avoidance Behavior	0.02
Anxiety/Repetitive Behavior	-0.01

Note: * = $p<.05$; ** = $p<.01$

A standard multiple linear regression was conducted to examine the predictive ability of ASD and comorbid symptomology (i.e., *BISCUIT-Part 1* total and totals from four *BISCUIT-Part 2* subscales) for adult interaction skills (i.e., AI subdomain score). Results demonstrated that a significant portion of the total variation in social skills was predicted by the model, R^2 = 0.189, F (5, 243) = 11.34, $p < .001$. However, ASD severity was the only significant predictor, β = -0.45, t (243) = -6.76, $p < .01$. Tantrum/Conduct Behavior, Inattention/Impulsivity, and Avoidance Behavior did not serve as significant predictors (all $p > .05$). Anxiety/Repetitive Behavior approached significance in predicting adult interaction skills ($p = .05$). Beta values, standard errors, and standardized beta values for the predictors are presented in Table 8.

Table 8
Regression Analysis Predicting AI Subdomain Scores

	B	***SE***	β	***t***	**Sig**
ASD Severity	-0.07	0.01	-0.45	-6.76	.000
Tantrum/Conduct Behavior	0.01	0.04	0.02	0.15	.879
Inattention/Impulsivity	-0.06	0.06	-0.11	-0.96	.340
Avoidance Behavior	0.08	0.08	0.08	1.03	.305
Anxiety/Repetitive Behavior	0.17	0.09	0.20	1.97	.050

Research Question 3: Peer Interaction Skills

To explore the relationship between peer interaction skills, ASD, and comorbid symptomology, correlation and multiple regression analyses were conducted. Results indicated that peer interaction skills as measured by the PI subdomain on the *BDI-2* significantly and negatively correlated with ASD severity (r = -0.33, $p < .01$), Tantrum/Conduct Behavior (r = -0.17, $p < .01$), Inattention/Impulsivity (r = -0.20, $p < .01$), and Anxiety/Repetitive Behavior (r = -0.19, $p < .01$). Avoidance Behavior was negatively correlated with peer interaction skills;

however, the correlation was not significant ($p > .05$). Results from correlation analyses are presented in Table 9.

Table 9
Correlations for Comorbid Psychopathology and PI Subdomain Scores

	Correlation with PI Subdomain on *BDI-2*
ASD Severity	-0.33**
Tantrum/Conduct Behavior	-0.17**
Inattention/Impulsivity	-0.20**
Avoidance Behavior	-0.10
Anxiety/Repetitive Behavior	-0.19**

Note: * = $p<.05$; ** = $p<.01$

A standard multiple linear regression was conducted to examine the predictive ability of ASD and comorbid symptomology (i.e., *BISCUIT-Part 1* total and totals from four *BISCUIT-Part 2* subscales) for peer interaction skills (i.e., PI subdomain score). Results from the multiple regression indicated that a significant portion of the total variation in social skills was predicted by ASD severity as well as comorbid symptomology, $R^2 = 0.113$, F (5, 243) = 6.17, $p < .001$. As in previous analyses, ASD severity was the only significant predictor, $\beta = -0.31$, t (243) = -4.35, $p < .01$. Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior did not serve as significant predictors (all $p > .05$). Beta values, standard errors, and standardized beta values for the predictors are presented in Table 10.

Table 10
Regression Analysis Predicting PI Subdomain Scores

	B	***SE***	β	***t***	**Sig**
ASD Severity	-0.04	0.01	-0.31	-4.35	.000
Tantrum/Conduct Behavior	-0.01	0.04	-0.02	-0.13	.896
Inattention/Impulsivity	-0.004	0.05	-0.01	-0.08	.939
Avoidance Behavior	0.03	0.06	0.05	0.54	.588
Anxiety/Repetitive Behavior	-0.05	0.08	-0.07	-0.60	.550

Research Question 4: Self-Concept and Social Role Skills

Correlation and multiple regression analyses were conducted to examine the relationship between self-concept and social role skills, ASD, and comorbid symptomology. Results indicated that ASD severity as measured by *BISCUIT-Part 1* total score is significantly, negatively correlated with the SR subdomain of the *BDI-2* measuring skills in self-concept and social role, $r = -0.37$, $p < .01$. Correlations among the Tantrum/Conduct Behavior, Inattention/Impulsivity, and Anxiety/Repetitive Behavior and the AI subdomain were negative but not significant. Avoidance Behavior was positively correlated with the SR subdomain however non-significant (all $p > .05$). Results from correlation analyses are presented in Table 11.

Table 11
Correlations for Comorbid Psychopathology SR Subdomain Scores

	Correlation with SR Subdomain on *BDI-2*
ASD Severity	-0.37**
Tantrum/Conduct Behavior	-0.03
Inattention/Impulsivity	-0.09
Avoidance Behavior	0.04
Anxiety/Repetitive Behavior	-0.01

Note: * = $p<.05$; ** = $p<.01$

A standard multiple linear regression was conducted to examine the predictive ability of ASD and comorbid symptomology (i.e., *BISCUIT-Part 1* total and totals from four *BISCUIT-Part 2* subscales) for skills in self-concept and social role (i.e., SR subdomain score). Results from the multiple regression indicated that a significant portion of the total variation in self-concept and social role skills was predicted by ASD severity as well as comorbid symptomology, $R^2 = 0.169$, $F(5, 243) = 9.85$, $p < .001$. However, as in previous analyses, ASD severity was the

only significant predictor, $\beta = -0.44$, $t\ (243) = -6.46$, $p < .01$. Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior did not serve as significant predictors (all $p > .05$). Beta values, standard errors, and standardized beta values for the predictors are presented in Table 12.

Table 12
Regression Analysis Predicting SR Subdomain Scores

	B	***SE***	β	***t***	**Sig**
ASD Severity	-0.06	0.01	-0.44	-6.46	.000
Tantrum/Conduct Behavior	0.02	0.04	0.07	0.60	.548
Inattention/Impulsivity	-0.04	0.05	-0.08	-0.67	.506
Avoidance Behavior	0.07	0.07	0.09	1.14	.255
Anxiety/Repetitive Behavior	0.10	0.08	0.13	1.27	.206

DISCUSSION

Social behavior is present in all aspects of life, and deficits can prevent inclusion; hinder familial, community and work activities; and keep an individual from reaching his or her potential. Accordingly, it is imperative to address the social skill repertoire of children on the autism spectrum early in life. However, the assessment and treatment of social impairments can become complex when individuals present with both ASD and comorbid psychopathology (Dover & Le Couteur, 2007; Morgan et al., 2003). Individualized treatment plans to target behaviors associated with ASD, with co-occurring psychopathology, and with the interaction of the multiple disorders are warranted in such cases (Morgan et al., 2003). Because social skills are often among the first impairments to be targeted in intervention, understanding the interaction between ASD and comorbid symptomology in social deficits is essential. Therefore, the purpose of this study was to examine the contribution of comorbid symptomology to the social skill deficits seen in young children with ASD. Social skills were examined as a whole as well as with a particular interest in adult interaction (e.g., attachment behaviors, initiating and maintaining social contact, the use of adults to aid in problem solving), peer interaction (e.g., forming friendships, responding to and initiating interactions), and self-concept and social role skills (e.g., self-awareness, coping skills, empathy).

The results of the current study suggest that ASD severity and comorbid symptomology together predicted 11.3 to 21.3% of the variance in the participant's overall social skills as well as in adult and peer interaction, and self-concept and social role skills. However, further evaluation revealed that ASD severity served as the only significant predictor of socialization deficit in each model. Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, and Anxiety/Repetitive Behavior symptoms were not predictive of adult or peer interaction, self-

concept and social role, or overall social skills in this sample. Therefore, the hypotheses regarding the contribution of comorbid symptomology to socialization impairment were not supported.

There were several significant correlations between comorbid symptoms and peer interaction, adult interaction, self-concept and social role, and overall social skills; however, based on the results of the regression analyses, these findings were most likely due to the high correlations between predictors (i.e., ASD severity and comorbid symptoms; see Table 2). The correlations observed between ASD severity and the comorbid psychopathologies studied were not surprising. Researchers have indicated that as ASD severity increases, the likelihood of experiencing comorbid psychopathology also increases (Mannion & Leader, 2014; Matson, Hess, & Boisjoli, 2010; Tureck, Matson, Cervantes, & Turygin, in press; Yerys et al., 2009). Further, if an individual with ASD exhibits one comorbidity, he or she is more likely to demonstrate symptoms of multiple co-occurring psychopathologies (Jang et al., 2013). Due to this trend, several researchers have posed a possible shared etiology between ASD and common comorbid disorders. For example, evidence of shared genetic risk factors for neurodevelopmental and psychiatric disorders has been growing in the literature (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Matson, Rieske, & Williams, 2013). Additionally, prenatal and perinatal complications alone or in conjunction with genetic predispositions may lend to comorbidity in the context of ASD (Mannion & Leader, 2014).

Although results did not indicate any significant contributions of comorbidity in the social skill deficits of infants and toddlers with ASD, it is possible that this may change later in life. Although signs and symptoms of psychopathology are present at a very young age (Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Fodstad, Rojahn, & Matson, 2010), the

symptomology related to comorbidity may not become fully prominent until later in childhood. In typically developing children, psychopathology appears stable from infant and toddlerhood into childhood and adolescence. In fact, researchers have demonstrated that internalizing and externalizing problems at age 2 to 3 years serves as a significant predictor of similar problems later in life (Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006; Cherkasova, Sulla, Dalena, Pondé, & Hechtman, 2013; Fodstad et al., 2010; Mesman, Bongers, & Koot, 2001; Mesman & Koot, 2001). However, researchers have found that not only do individuals with ASD have higher rates of psychopathology than the general population, but the developmental trajectory of comorbid psychopathology differs within the ASD population (Davis III et al., 2011; Fodstad et al., 2010; Konst & Matson, 2014; Midouhas, Yogaratnam, Flouri, & Charman, 2013). While psychopathology often remains stable in typically developing individuals, symptoms of comorbid conditions in individuals with ASD emerge early in life and show a trend of increasing severity and prevalence over time (Fodstad et al., 2010; Konst & Matson, 2014; Midouhas et al., 2013). Based on this pattern, the comorbid symptoms of the participants included in this study may be expected to escalate, with more children falling in the moderate and severe impairment ranges later in life. Therefore, it is possible that a contribution of comorbidity to social skill deficits will be more apparent as these children age or in an older sample.

Interestingly, the sample demonstrated the least impairment in the PI subdomain, and ASD and comorbid symptoms had the least predictive value for peer interaction skills. The model accounted for just 11.3% of the variance compared to 16.9% of self-concept and social role skills, 18.9% of adult interaction skills, and 21.3% of overall social skills. It is possible that these results reflect the complexity of peer interaction skills expected at this age. At age 2 to 3

years old, typically developing children begin the process of transitioning from parallel play (i.e., playing beside other children) to interactive play patterns such as including other children in games (CDC, 2014b). While children with ASD and/or psychopathology may appear fluent in parallel play, skills like social problem solving, cooperative play, and conversation are more complex. When same-aged peers begin to meet these complex peer interaction milestones later in childhood, the differentiation between typical and atypical social development will become more pronounced. Further, greater skill impairment in children with ASD and co-occurring psychopathology compared to children with ASD alone may become more evident and has been demonstrated in the literature at later ages in childhood (Rao & Landa, 2014; Sprenger et al., 2013). Therefore, the effects of comorbidity and ASD severity to socialization deficit as well as the variance accounted for in peer interaction deficits may become more pronounced with increased social demands over time (Larsson, Dilshad, Lichtenstein, & Barker, 2011; Mannion & Leader, 2014; Mesman et al., 2001). A bidirectional relationship between social skill deficit and comorbid psychopathology should also be evaluated in older children with ASD as researchers have found that frequent social failures (e.g., negative peer relationships, peer rejection in school) may lead to low self-esteem, anxiety, and externalizing problems (Mesman et al., 2001).

The current study holds important implications for assessment and treatment. For instance, if the presence of comorbidity does not yet contribute to the social skill deficits in infants and toddlers with ASD, early childhood would prove an ideal time to intervene as a preventative measure. This may help preclude the increase in prevalence and severity of comorbid symptoms over time noted in the ASD population (Davis III et al., 2011; Fodstad et al., 2010; Konst & Matson, 2014; Midouhas et al., 2013). Early intervention incorporating treatment of comorbid symptoms could also better prepare children with ASD and co-occurring

psychopathology for the emerging social demands of childhood. However, in order to better provide services targeting these symptoms, accurate and systematic assessment of comorbid psychopathologies is imperative. One of the challenges faced by clinicians, and particularly those with less experience, involves the difficulty in differentiating core symptoms of ASD from symptoms of comorbid psychopathology (Matson & Cervantes, 2014). As previously stated, the differences in presentation and exacerbation of symptoms associated with the co-occurrence of ASD and psychopathology present a complex clinical picture (Dover & Le Couteur, 2007; Morgan et al., 2003). This highlights the importance of further developing and utilizing psychopathology assessment tools specifically designed or normed for use with the ASD population. Additionally, frequent follow-up assessments are warranted due to the instability of comorbid symptoms over time in individuals with ASD (Konst & Matson, 2014).

Concerning assessment procedures, the field of psychology appears to be moving towards developing transdiagnostic models for psychopathology (Nolen-Hoeksema & Watkins, 2011). Transdiagnostic approaches to assessment focus on fundamental processes shared by frequently co-occurring disorders that may contribute etiologically. Essential to formulating transdiagnostic models is identifying environmental and biological factors that contribute to presenting with a certain set of comorbidities (Nolen-Hoeksema & Watkins, 2011). This is an area that calls for additional research, particularly within the ASD field. Better diagnosis and intervention may result from identifying underlying biological, behavioral, environmental, and socio-emotional factors contributing to psychopathology specifically in the ASD population (Fodstad et al., 2010; Nolen-Hoeksema & Watkins, 2011).

Diagnosing mental health conditions in very young children is unlikely; however, identifying precursors to common comorbidities (e.g., anxiety, ADHD, conduct problems) may

help play a protective role by working to remediate these comorbid symptoms within early intensive behavioral intervention (EIBI) programs before symptoms and associated deficits become more severe (Fodstad et al., 2010; Matson, Boisjoli, Hess, & Wilkins, 2011). Comorbidities in individuals with ASD have been associated with increased challenging behaviors, stereotypies, sleep problems, and adaptive deficits. Therefore, treatment targeting comorbidities may not only be integral for preventing additive social deficits, but is important for improving the individual's quality of life and lessening caregiver distress (Konst & Matson, 2014; Mannion & Leader, 2014). EIBI has been repeatedly shown effective for addressing the core deficits of ASD, including social skills (Matson & Goldin, 2014). However, the integration of treatment for comorbid psychopathology into EIBI programs requires individualization and research on this topic is limited. Though there are evidence-based treatments available for the psychopathologies that co-occur with ASD (e.g., exposure therapy for anxiety, stimulant medications for ADHD), the efficacy of these interventions within the ASD population specifically warrants further research. For example, Reichow, Volkmar, and Bloch (2013) found that although pharmacological treatment of ADHD in ASD was effective, individuals with ASD were at a heightened risk for side effects. Adding to the complexity of treating comorbidities in ASD, the intensity of intervention as well as the number of different interventions necessary increases with the number of co-occurring disorders an individual with ASD experiences (Matson & Goldin, 2014).

One potential limitation to the current findings may involve the accuracy of caregiver report on the comorbidity measure. Due to the developmental characteristics of early childhood when young children often experience rapid change, parents may view behavior or socio-emotional problems as part of typical development that children will grow out of with age. Due

to a common social perception that challenging behaviors are to be expected in a child's "terrible twos," caregivers may have been less likely to endorse comorbid psychopathology on the *BISCUIT-Part 2* (Briggs-Gowan et al., 2006). However, elevated rates of comorbid symptoms were indicated throughout the sample demonstrating that caregivers of young children with ASD were able to discern typical versus atypical symptoms of psychopathology. Additionally, because this study was particularly interested in the interaction between ASD and comorbidity, no control group was used to determine if early psychopathology causes socialization impairment in the general population. Future studies should compare the impact of early psychopathology to social skills in infants and toddlers with typical development and ASD. Findings would help establish if the lack of early comorbid influence on social skills found in this study is consistent across infants and toddlers with and without ASD.

Though results of the current study do not indicate that comorbid psychopathology lends to the social skill deficits of infants and toddlers with ASD, research is warranted addressing the impact of comorbidities on socialization impairment in older children and adults with ASD. Further, the developmental course of common co-occurring disorders should be explored over time particularly in regards to their interaction with core symptoms of ASD. Though researchers have identified several factors associated with persisting psychopathology in typically developing children (e.g., poverty, IQ, poor academic and social functioning, parental psychopathology; Briggs-Gowan et al., 2001; Cherkasova et al., 2013), further research addressing risk factors for the persistence or worsening of psychopathology specific to the ASD population would be beneficial. Identifying risk factors for developing clinically significant comorbid diagnoses from comorbid symptomology seen in early childhood would help inform intervention efforts and target those children with ASD who require more intensive and

individualized services. Finally, future research should consider exploring the preventative value of integrating treatment for comorbid symptomology into EIBI programs for children with ASD. This research would supply greater knowledge regarding the interaction between ASD and common comorbidities as well as provide clinicians with greater ability to individualize interventions for people with ASD and co-occurring psychopathology.

REFERENCES

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA preschool forms and profiles*. Burlington: University of Vermont; Research Center for Children, Youth, and Families.

American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed.)*. Washington, DC: Author.

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed. - Revised)*. Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders, (4th ed.)*. Washington, DC: Author.

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders-Text Revision, (4th ed., text rev.)*. Washington, DC: Author.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders, (5th ed.)*. Washington, DC: Author.

Asperger, H. (1991). 'Autistic psychopathy' in childhood (U. Frith, Trans.). In U. Frith (Ed.), *Autism and Asperger syndrome*. New York: Cambridge University Press.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"?. *Cognition*, *21*(1), 37-46.

Beidel, D. C., Turner, S. M., & Morris, T. L. (1999). Psychopathology of childhood social phobia. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 643-650.

Beighley, J. S., Matson, J. L., Rieske, R. D., Jang, J., Cervantes, P., & Goldin, R. (2013). Comparing challenging behavior in children diagnosed with autism spectrum disorders according to the *DSM-IV-TR* and the proposed *DSM-5*. *Developmental Neurorehabilitation, 16,* 375-381.

Bender, L. (1959). Autism in children with mental deficiency. *American Journal of Mental Deficiency*, *64*, 81-86.

Bleuler, E. (1913). Autistic thinking. *American Journal of Insanity, 69*, 873-886.

Briggs-Gowan, M. J., Carter, A. S., Bosson-Heenan, J., Guyer, A. E., & Horwitz, S. M. (2006). Are infant-toddler social-emotional and behavioral problems transient? *Journal of the American Academy of Child and Adolescent Psychiatry, 45*, 849-858.

Briggs-Gowan, M. J., Carter, A. S., Skuban, E. M., & Horwitz, S. M. (2001). Prevalence of social-emotional and behavioral problems in a community sample of 1-and 2-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 811-819.

Centers for Disease Control and Prevention (2007). *Prevalence of Autism Spectrum Disorders (ASDs).* Retrieved August 23, 2013, from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5601a2.htm

Centers for Disease Control and Prevention (2012). *Prevalence of Autism Spectrum Disorders (ASDs).* Retrieved August 23, 2013, from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6103a1.htm

Centers for Disease Control and Prevention (2014a). *Prevalence of Autism Spectrum Disorders (ASDs).* Retrieved August 5, 2014, from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6302a1.htm

Centers for Disease Control and Prevention (2014b). *Developmental Milestones.* Retrieved August 6, 2014, from http://www.cdc.gov/ncbddd/actearly/milestones/index.html

Cervantes, P. E., Matson, J. L., Adams, H. L., Williams, L. W., Goldin, R. L., & Jang, J. (2013). Comparing social skills profiles of children with autism spectrum disorders versus children with attention deficit hyperactivity disorder: Where the deficits lie. *Research in Autism Spectrum Disorders, 7*, 1004-1110.

Cherkasova, M., Sulla, E. M., Dalena, K. L., Pondé, M. P., & Hechtman, L. (2013). Developmental course of attention deficit hyperactivity disorder and its predictors. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *22*, 47.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cohen, D. J., Paul, R., & Volkmar, F. R. (1986). Issues in the classification of pervasive and other developmental disorders: Toward DSM-IV. *Journal of the American Academy of Child Psychiatry*, *25*, 213-220.

Conners, C. K. (1997). *Conners' Rating Scales–Revised*. North Towanda, NY: Multi-Health Systems.

Coy, K., Speltz, M. L., DeKlyen, M., & Jones, K. (2001). Social-cognitive processes in preschool boys with and without oppositional defiant disorder. *Journal of Abnormal Child Psychology, 29*, 107-119.

Cross-Disorder Group of the Psychiatric Genomics Consortium. (2013). Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *Lancet*, *381*, 1371-1379.

Davis III, T. E., Fodstad, J. C., Jenkins, W. S., Hess, J. A., Moree, B. W., Dempsey, T., & Matson, J. L. (2010). Anxiety and avoidance in infants and toddlers with autism spectrum disorders: Evidence for differing symptom of severity and presentation. *Research in Autism Spectrum Disorders, 5*, 305-313.

Davis III, T. E., Hess, J. A., Moree, B. N., Fodstad, J. C., Dempsey, T., Jenkins, W. S., & Matson, J. L. (2011). Anxiety symptoms across the lifespan in people diagnosed with Autistic Disorder. *Research in Autism Spectrum Disorders*, *5*, 112-118.

Davis III, T. E., Moree, B. N., Dempsey, T., Hess, J. A., Jenkins, W. S., Fodstad, J. C., & Matson, J. L. (2012). The effect of communication deficits on anxiety symptoms in infants and toddlers with autism spectrum disorders. *Behavior Therapy, 43*, 142-152.

de Bruin, E. I., Ferdinand, R. F., Meester, S., de Nijs, P. F., & Verheij, F. (2007). High rates of psychiatric co-morbidity in PDD-NOS. *Journal of Autism and Developmental Disorders, 37*, 877–886.

Dover, C. J., & Le Couteur, A. (2007). How to diagnose autism. *Archives of Disease in Childhood*, *92*, 540-545.

Eveloff, H. H. (1960). The autistic child. *Archives of General Psychiatry, 3*, 66-81.

Field, A. (2009). *Discovering statistics using SPSS*. London: Sage Publications Limited.

Fodstad, J. C., Rojahn, J., & Matson, J. L. (2010). Emergent comorbidity in at risk children with and without ASD: A cross sectional study. *Journal of Developmental and Physical Disabilities, 22,* 381-400.

Fombonne, E. (2009). Epidemiology of Pervasive Developmental Disorders. *Pediatric Research, 65*(6), 591-598.

Frazier, T. W., Youngstrom, E. A., Speer, L., Embacher, R., Law, P., Constantino, J., ... & Eng, C. (2012). Validation of proposed DSM-5 criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(1), 28-40.

Gadow, K. D., DeVincent, C. J., & Drabick, D. A. (2008). Oppositional defiant disorder as a clinical phenotype in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *38*, 1302-1310.

Gadow, K. D., & Drabick, D. A. (2012). Symptoms of autism and schizophrenia spectrum disorders in clinically referred youth with oppositional defiant disorder. *Research in Developmental Disabilities*, *33*, 1157-1168.

Gibbs, V., Aldridge, F., Chandler, F., Witzlsperger, E., & Smith, K. (2012). An exploratory study comparing diagnostic outcomes for autism spectrum disorders under DSM-IV-TR with the proposed DSM-5 revision. *Journal of Autism and Developmental Disorders, 4*, 1750-1756.

Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The Autism Diagnostic Observation Schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, 37, 613-627.

Greene, R. W., Biederman, J., Zerwas, S., Monuteaux, M. C., Goring, J. C., & Faraone, S. V. (2002). Psychiatric comorbidity, family dysfunction, and social impairment in referred youth with oppositional defiant disorder. *American Journal of Psychiatry*, *159*, 1214-1224.

Grossman, J. B., Carter, A., & Volkmar, F. R. (1997). Social behavior in autism. *Annals of the New York Academy of Sciences*, *807*, 440-454.

Grossman, J.B., Klin, A., Carter, A.S., & Volkmar, F.R. (2000). Verbal bias in recognition of facial emotions in children with asperger syndrome. *Journal of Child Psychology and Psychiatry, 41,* 369-379.

Hinkle, D. E., Wiersma, W., & Jurs, S. G. (2003). *Applied statistics for the behavioral sciences.* Boston, MA: Houghton Mifflin Company.

Holtmann, M., Bolte, S., & Poustka, F. (2007). Attention deficit hyperactivity disorder symptoms in pervasive developmental disorders: Association with autistic behavior domains and coexisting psychopathology. *Psychopathology, 40*, 172–177.

Horovitz, M. & Matson, J. L. (in press). The Baby and Infant Screen for Children with aUtIsm Traits-Part 2: The development of age-based cut offs. *Developmental Neurorehabilitation.*

Howlin, P. (2006). Autism spectrum disorders. *Psychiatry*, *5*, 320-324.

Hoza, B., Mrug, S., Gerdes, A. C., Hinshaw, S. P., Bukowski, W. M., Gold, J. A., ... Arnold, L. (2005). What aspects of peer relationships are impaired in children with attention-deficit/hyperactivity disorder? *Journal of Consulting and Clinical Psychology*, *73*, 411-423.

Inglese, M. D., & Elder, J. H. (2009). Caring for children with Autism Spectrum Disorder, part I: Prevalence, etiology, and core features. *Journal of Pediatric Nursing, 24,* 41 – 48.

Jang, J., Matson, J. L., Williams, L. W., Tureck, K., Goldin, R. L., & Cervantes, P. E. (2013). Rates of comorbid symptoms in children with ASD, ADHD and comorbid ASD and ADHD. *Research in Developmental Disabilities, 34,* 2369-2378.

Kamp-Becker, I., Ghahreman, M., Heinzel-Gutenbrunner, M., Peters, M., Remschmidt, H., & Becker, K. (2013). Evaluation of the revised algorithm of Autism Diagnostic Observation Schedule (ADOS) in the diagnostic investigation of high-functioning children and adolescents with autism spectrum disorders. *Autism*, 17, 87-102.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2*, 217-230.

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

Kanner, L. (1951). The conception of wholes and parts in early infantile autism. *American Journal of Psychiatry, 1*, 23-26.

Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioral Science, 4*, 412-420.

Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia, 1,* 119 – 145.

Kanner, L. & Eisenberg, L. (1957). Early infantile autism, 1943-1955. *Psychiatric Research Reports, 7,* 55-65.

Kim, H. Y. (2013). Statistical notes for clinical researchers: Assessing normal distribution (2) using skewness and kurtosis. *Restorative Dentistry and Endodontics*, *38*, 52-54.

Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individual with autism. *Archives of General Psychiatry, 59,* 809-816.

Klin, A., Lang, J., Cicchetti, D. V., & Volkmar, F. R. (2000). Brief report: Interrater reliability of clinical diagnosis and DSM-IV criteria for autistic disorder: Results of the DSM-IV Autism Field Trial. *Journal of Autism and Developmental Disorders, 30*, 163-167.

Koenig, K., Rubin, E., Klin, A. & Volkmar, F. (2000). Autism and the pervasive developmental disorders. In C. Zeanah (Ed.), *Handbook of Infant Mental Health* (2nd ed., pp. 298-310). New York, NY: Guilford Press.

Konst, M. J., & Matson, J. L. (2014). Temporal and diagnostic influences on the expression of comorbid psychopathology symptoms in infants and toddlers with autism spectrum disorder. *Research in Autism Spectrum Disorders*, *8*, 200-208.

Larsson, H., Dilshad, R., Lichtenstein, P., & Barker, E. D. (2011). Developmental trajectories of DSM-IV symptoms of attention-deficit/hyperactivity disorder: Genetic effects, family risk and associated psychopathology. *Journal of Child Psychology and Psychiatry*, *52*, 954-963.

Leonard, H., Dixon, G., Whitehouse, A. J.O., Bourke, J., Aiberti, K., Nassar, N.,… & Glasson, E. J. (2010). Unpacking the complex nature of the autism epidemic. *Research in Autism Spectrum Disorders, 4,* 548 – 554.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., … & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders, 36,* 849–861.

Lockyer, L., & Rutter, M. (1970). A Five-to Fifteen-Year Follow-up Study of Infantile Psychosis: IV. Patterns of Cognitive Ability. *British Journal of Social and Clinical Psychology, 9*, 152-163.

Lord, C., Rutter, M. L., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., & Schopler, E. (1989). Autism Diagnostic Observation Schedule: A standardized observation of communicative and social behavior. *Journal of Autism and Developmental Disorders, 19*(2), 185-212.

Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social Psychiatry*, *1*, 124-135.

Mannion, A., & Leader, G. (2014). Attention-deficit/hyperactivity disorder (AD/HD) in autism spectrum disorder. *Research in Autism Spectrum Disorders*, *8*, 432-439.

Matson, J. L., Belva, B. C., Horovitz, M., Kozlowski, A. M., & Bamburg, J. W. (2012). Comparing symptoms of autism spectrum disorders in a developmentally disabled adult population using the current DSM-IV-TR diagnostic criteria and the proposed DSM-5 diagnostic criteria. *Journal of Developmental and Physical Disabilities, 24*(4), 403-414.

Matson, J. L., Boisjoli, J. A., Hess, J. A., & Wilkins, J. (2011). Comorbid psychopathology factor structure on the Baby and Infant Screen for Children with aUtIsm Traits-Part 2 (BISCUIT-Part 2). *Research in Autism Spectrum Disorders, 5,* 426-432.

Matson, J. L., Boisjoli, J. A., Hess, J., & Wilkins, J. (2010). Factor structure and diagnostic fidelity of the Baby and Infant Screen for Children with aUtIsm Traits - Part 1 (BISCUIT Part - 1). *Developmental Neurorehabilitation, 13,* 72-79.

Matson, J. L., Boisjoli, J., & Wilkins, J. (2007). The Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT). *Baton Rouge, LA: Disability Consultants, LLC*.

Matson, J. L., & Cervantes, P. E. (2014). Commonly studied comorbid psychopathologies among persons with autism spectrum disorder. *Research in Developmental Disabilities*, *35*, 952-962.

Matson, J. L., & Goldin, R. L. (2014). Early intensive behavioral interventions: Selecting behaviors for treatment and assessing treatment effectiveness. *Research in Autism Spectrum Disorders, 8,* 138-142.

Matson, J. L., & González, M. L. (2007). *Autism Spectrum Disorders – Comorbidity – Child Version.* Baton Rouge, La. Disability Consultants, LLC.

Matson, J. L., & González, M. L. (2007). *Autism spectrum disorders – diagnosis – child version*. Baton Rouge, LA: Disability Consultants, LLC.

Matson, J. L., Gonzalez, M., & Wilkins, J. (2009). Validity study of the Autism Spectrum Disorders-Diagnostic for Children (ASD-DC). *Research in Autism Spectrum Disorders, 3*, 196-206.

Matson, J. L., Hattier, M. A., & Williams, L. W. (2012). How does relaxing the algorithm for autism affect DSM-V prevalence rates? *Journal of Autism and Developmental Disorders, 42*, 1549-1556.

Matson, J. L., Hess, J., & Boisjoli, J. A. (2010). Comorbid psychopathology in infants and toddlers with autism and pervasive developmental disorders-not otherwise specified (PDD-NOS). *Research in Autism Spectrum Disorders, 4*, 300-304.

Matson, J. L., Hess, J. A., Neal, D., Mahan, S., & Fodstad, J. C. (2010). The effects inattention/impulsivity and ASD symptom severity on social skills in infants and toddlers. *Developmental Neurorehabilitation, 13,* 408-412.

Matson, J. L., Hess, J. A., Mahan, S., & Fodstad, J. C. (2010). Convergent validity of the Autism Spectrum Disorder-Diagnostic for Children (ASD-DC) and Autism Diagnostic Interview-Revised (ADI-R). *Research in Autism Spectrum Disorders, 4*, 741-745.

Matson, J. L., & Kozlowski, A. M. (2011). The increasing prevalence of autism spectrum disorders. *Research in Autism Spectrum Disorders, 5,* 418-425.

Matson, J. L., LoVullo, S. V., Rivet, T. T., & Boisjoli, J. A. (2009). Validity of the Autism Spectrum Disorder-Comorbid for Children (ASD-CC). *Research in Autism Spectrum Disorders*, 3, 345-357.

Matson, J. L., Mahan, S., Sipes, M., & Kozlowski, A. M. (2010). Effects of symptoms of comorbid psychopathology on challenging behaviors among atypically developing infants and toddlers as assessed with the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT). *Journal of Mental Health Research in Intellectual Disabilities, 3,* 164-176.

Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorders in children: An overview. *Research in Developmental Disabilities, 28,* 341-352.

Matson, J. L., Rieske, R. D., & Williams, L. W. (2013). The relationship between autism spectrum disorders and attention-deficit, hyperactivity disorder: An overview. *Research in Developmental Disabilities, 34*, 2475-2484.

Matson, J. L., & Wilkins, J. (2007). A critical review of assessment targets and methods for social skills excesses and deficits for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *1*, 28-37.

Matson, J. L., & Wilkins, J. (2008). Reliability of the Autism Spectrum Disorders-Comorbid for Children (ASD-CC). *Journal of Developmental and Physical Disabilities, 20*, 327-336.

Matson, J. L., Wilkins, J., Sevin, J. A., Knight, C., Boisjoli, J. A., & Sharp, B. (2009). Reliability and item content of the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT): Parts 1, 2, and 3. *Research in Autism Spectrum Disorders, 3*, 336-344.

Matson, J. L., Wilkins, J., Sharp, B., Knight, K., Sevin, J. A., & Boisjoli, J. A. (2009). Sensitivity and Specificity of the *Baby and Infant Screen for Children with aUtism Traits* (*BISCUIT*): Validity and cut-off scores for autism and PDD-NOS in toddlers. *Research in Autism Spectrum Disorders, 3*, 924-30.

Matson, J. L., Worley, J. A., Neal, D., Mahan, S., & Fodstad, J. C. (2010). The effects of inattention/impulsivity and ASD symptom severity on social skills in toddlers. *Developmental Neurorehabilitation, 13*, 413-422.

Mattila, M., Hurtig, T., Haapsamo, H., Jussila, K., Kuusikko-Gauffin, S., Kielinen, M., & ... Moilanen, I. (2010). Comorbid psychiatric disorders associated with Asperger syndrome/high-functioning autism: A community- and clinic-based study. *Journal of Autism and Developmental Disorders*, *40*, 1080-1093.

Mattila, M., Kielinen, M., Linna, S., Jussila, K., Ebeling, H., Bloigu, R., . . . Moilanen, I. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*, 583-592.

Mayes, S. D., Black, A., & Tierney, C. D. (2013). DSM-5 under-identifies PDDNOS: Diagnostic agreement between the DSM-5, DSM-IV, and Checklist for Autism Spectrum Disorder. *Research in Autism Spectrum Disorders*, *7*, 298-306.

McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*, 368-383.

Mesman, J., Bongers, I. L., & Koot, H. M. (2001). Preschool developmental pathways to preadolescent internalizing and externalizing Problems. *Journal of Child Psychology and Psychiatry, 42*, 679-689.

Mesman, J., & Koot, H. M. (2001). Early preschool predictors of preadolescent internalizing and externalizing DSM-IV diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 1029-1036.

Midouhas, E., Yogaratnam, A., Flouri, E., & Charman, T. (2013). Psychopathology trajectories of children with autism spectrum disorder: the role of family poverty and parenting. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*, 1057-1065.

Miller, L. J., Nielson, D. M., & Schoen, S. A. (2012). Attention deficit hyperactivity disorder and sensory modulation disorder: A comparison of behavior and physiology. *Research in Developmental Disabilities, 33*, 804-818.

Morgan, C. N., Roy, M., & Chance, P. (2003). Psychiatric comorbidity and medication use in autism: A community survey. *Psychiatric Bulletin, 27*, 378-381.

Mosse, H. L. (1958). The misuse of the diagnosis childhood schizophrenia. *The American Journal of Psychiatry, 114*, 791-794.

Newborg, J. (2005). *Battelle Developmental Inventory (2nd ed.).* Itasca, NY: Riverside.

Nijmeijer, J. S., Minderaa, R. B., Buitelaar, J. K., Mulligan, A., Hartman, C. A., & Hoekstra, P. J. (2008). Attention-deficit/hyperactivity disorder and social dysfunctioning. *Clinical Psychology Review*, *28*, 692-708.

Nolen-Hoeksema, S., & Watkins, E. R. (2011). A heuristic for developing transdiagnostic models of psychopathology explaining multifinality and divergent trajectories. *Perspectives on Psychological Science*, *6*, 589-609.

Rao, P. A., & Landa, R. J. (2014). Association between severity of behavioral phenotype and comorbid attention deficit hyperactivity disorder symptoms in children with autism spectrum disorders. *Autism*, *18*, 272-280.

Reichow, B., Volkmar, F. R., & Bloch, M. H. (2013). Systematic review and meta-analysis of pharmacological treatment of the symptoms of attention-deficit/hyperactivity disorder in children with pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *43*, 2435-2441.

Rescorla, L. A. (2005). Assessment of young children using the Achenbach System of Empirically Based Assessment (ASEBA). *Mental Retardation & Developmental Disabilities Research Reviews, 11*, 226-237.

Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior Assessment System for Children (2nd ed.).* Circle Pine, MN: American Guidance Service.

Rieske, R. D., Matson, J. L., Beighley, J. S., Cervantes, P. E., Goldin, R. L., & Jang, J. (in press). Comorbid psychopathology rates in children diagnosed with autism spectrum disorders according to the DSM-IV-TR and the proposed DSM-5. *Developmental Neurorehabilitation.*

Russell, G., Rodgers, L. R., Ukoumunne, O. C., & Ford, T. (2014). Prevalence of parent-reported ASD and ADHD in the UK: Findings from the Millennium Cohort Study. *Journal of Autism and Developmental Disorders*, *44*, 31-40.

Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry, 9*, 1-25.

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

Rutter, M. (2000). Genetic studies of autism: From the 1970s into the millennium. *Journal of Abnormal Child Psychology*, *28*, 3-14.

Rutter, M., & Bartak, L. (1971). Causes of infantile autism: Some considerations from recent research. *Journal of Autism and Childhood Schizophrenia, 1*(1), 20-32.

Rutter, M. & Schopler, E. (1988). Autism and pervasive developmental disorders. In E. Schopler & G. B. Mesibov (Eds.), *Diagnosis and Assessment in Autism* (pp. 15 – 36). NY: Springer Us.

Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *Childhood Autism Rating Scale, Second Edition*. Torrance, CA: Western Psychological Services.

Simonoff, E., Pickles, A., Charman, T., Chandler, T. L., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*, 921–929.

Spitzer, R. L., & Siegel, B. (1990). The DSM-III-R Field Trial of Pervasive Developmental Disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 855-862.

Sprenger, L., Bühler, E., Poustka, L., Bach, C., Heinzel-Gutenbrunner, M., Kamp-Becker, I., & Bachmann, C. (2013). Impact of ADHD symptoms on autism spectrum disorder symptom severity. *Research in Developmental Disabilities*, *34*, 3545-3552.

Strauss, C. C., Lahey, B. B., Frick, P., Frame, C. L., & Hynd, G. W. (1988). Peer social status of children with anxiety disorders. *Journal of Consulting and Clinical Psychology, 56*, 137-141.

Strauss, C. C., Lease, C. A., Kazdin, A. E., Dulcan, M. K., & Last, C. G. (1989). Multimethod assessment of the social competence of children with anxiety disorders. *Journal of Clinical Child Psychology, 18,* 184-189.

Tan, C. (2007). Test Review Behavior assessment system for children (2nd ed.). *Assessment for Effective Intervention*, 32, 121-124.

Tureck, K., Matson, J. L., Cervantes, P. E., & Turygin, N. (in press). Autism severity as a predictor of inattention and impulsivity in toddlers. *Developmental Neurorehabilitation.*

Tureck, K., Matson, J. L., May, A., & Turygin, N. (2013). Externalizing and tantrum behaviours in children with ASD and ADHD compared to children with ADHD. *Developmental Neurorehabilitation, 16*, 52-57.

Turygin, N., Matson, J. L., Beighley, J., & Adams, H. (2013). The effect of DSM-5 criteria on the developmental quotient in toddlers diagnosed with autism spectrum disorder. *Developmental Neurorehabilitation, 16*, 38-43.

Van Houten, R. (1990). Emotional Problems II. *Handbook of Behavior Modification with the Mentally Retarded*, 421-438.

Vaughan, C. A. (2011). Test Review: Childhood Autism Rating Scale (2nd ed.). *Journal of Psychoeducational Assessment, 25,* 489-493.

Volkmar, F., Chawarska, K., & Klin, A. (2005). Autism in Infancy and Early Childhood. *Annual Review of Psychology*, *56,* 315-336.

Volkmar, F. R., & Cohen, D. J. (1991). Comorbid association of autism and schizophrenia. *The American Journal of Psychiatry, 148*, 1705-1707.

White, S., Keonig, K., & Scahill, L. (2007). Social skills development in children with autism spectrum disorders: A review of the intervention research. *Journal of Autism and Developmental Disorders*, *37*, 1858-1868.

White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review, 29*, 216-229.

White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 39*, 1006-1013.

Wing, L., Gould, J., & Gillberg, C. (2011). Autism spectrum disorders in the DSM-V: Better or worse than the DSM-IV? *Research in Developmental Disabilities, 32*, 768-773.

Witwer, A. N., & Lecavalier, L. (2010). Validity of comorbid psychiatric disorders in youngsters with autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, *22*, 367-380.

Wood, J. J., & Gadow, K. D. (2010). Exploring the nature and function of anxiety in youth with autism spectrum disorders. *Clinical Psychology: Science and Practice*, *17*, 281-292.

World Health Organization (1992). *International Classification of Diseases (10th ed.)*. Geneva, Switzerland: World Health Organization.

Worley, J. A., & Matson, J. L. (2012). Comparing symptoms of autism spectrum disorders using the current DSM-IV-TR diagnostic criteria and the proposed DSM-V diagnostic criteria. *Research in Autism Spectrum Disorders, 6*, 965-970.

Yerys, B. E., Wallace, G. L., Sokoloff, J. L., Shook, D. A., James, J. D., & Kenworthy, L. (2009). Attention deficit/hyperactivity disorder symptoms moderate cognition and behavior in children with autism spectrum disorders. *Autism Research, 2*, 322-333.

APPENDIX

Bobby Jindal
GOVERNOR

Kathy Kliebert
SECRETARY

State of Louisiana

Department of Health and Hospitals

December 4, 2013

Dr. Johnny L. Matson
Department of Psychology
Louisiana State University
324 Audubon Hall
Baton Rouge, LA 70803

Via email: johnmatson@aol.com

Re: Autism in Early Childhood

Dear Dr. Matson:

Thank you for submitting the above-referenced proposal. We have taken into advisement information provided in the proposal package. We find that all areas of concerns were clarified and the project has been approved by Expedited Review.

The IRB approves the project for the purposes of investigating developmental patterns and differences in atypically developing children with and without autism spectrum disorders. If you should desire to conduct additional research using the data collected under this project, that proposal much be submitted separately to the IRB for review.

I am requesting that the Principal investigator report to the DHH IRB any emergent problems, serious adverse reactions, or changes to protocol that may affect the status of the investigation and that no such changes be instituted prior to DHH IRB review, except where necessary in order to eliminate immediate hazards. The investigator also agrees to periodic review of this project by the DHH IRB at intervals appropriate to the degree of risk to assure that the project is being conducted in compliance with the DHH IRB's understanding and recommendations.

If I can be of any further assistance to you, please feel free to contact me.

Sincerely,

Nell W. Allbritton, MPA
Director, Institutional Review Board
Department of Health and Hospitals
628 North 4th Street, Third Floor
Baton Rouge, Louisiana 70802
(225) 342-4169
nell.allbritton@la.gov

Application for Exemption from Institutional Oversight

Institutional Review Board
Dr. Robert Mathews, Chair
131 David Boyd Hall
Baton Rouge, LA 70803
P: 225.578.8692
F: 225.578.5983
irb@lsu.edu
lsu.edu/irb

Unless qualified as meeting the specific criteria for exemption from Institutional Review Board (IRB) oversight, ALL LSU research/ projects using living humans as subjects, or samples, or data obtained from humans, directly or indirectly, with or without their consent, must be approved or exempted in advance by the LSU IRB. This Form helps the PI determine if a project may be exempted, and is used to request an exemption.

– Applicant,Please fill out the application in its entirety and include the completed application as well as parts A-F, listed below, when submitting to the IRB. Once the application is completed, please submit two copies of the completed application to the IRB Office or to a member of the Human Subjects Screening Committee. Members of this committee can be found at http://research.lsu.edu/CompliancePoliciesProcedures/InstitutionalReviewBoard%28IRB%29/item24737.html

– A Complete Application Includes All of the Following:
(A) Two copies of this completed form and two copies of parts B thru F.
(B) A brief project description (adequate to evaluate risks to subjects and to explain your responses to Parts 1&2)
(C) Copies of all instruments to be used.
*If this proposal is part of a grant proposal, include a copy of the proposal and all recruitment material.
(D) The consent form that you will use in the study (see part 3 for more information.)
(E) Certificate of Completion of Human Subjects Protection Training for all personnel involved in the project, including students who are involved with testing or handling data, unless already on file with the IRB. Training link: (http://phrp.nihtraining.com/users/login.php)
(F) IRB Security of Data Agreement: (http://research.lsu.edu/files/item26774.pdf)

1) Principal Investigator: Dr. Johnny L. Matson **Rank:** Clinical Director

Dept: Clinical Psychology **Ph:** (225) 578-8745 **E-mail:** johnmatson@aol.com

2) Co Investigator(s): please include department, rank, phone and e-mail for each
*If student, please identify and name supervising professor in this space

IRB# E8292 LSU Proposal #
✓ Complete Application
✓ Human Subjects Training

3) Project Title: Autism in Early Childhood

Study Exempted By:
Dr. Robert C. Mathews, Chairman
Institutional Review Board
Louisiana State University
203 B-1 David Boyd Hall
225-578-8692 | www.lsu.edu/irb
Exemption Expires: 4/30/2016

4) Proposal? (yes or no) no **If Yes, LSU Proposal Number**

Also, if YES, either ◯ **This application completely matches the scope of work in the grant**
OR ◯ **More IRB Applications will be filed later**

5) Subject pool (e.g. Psychology students) Young children assessed for developmental delay via EarlySteps

*Circle any **"vulnerable populations" to be used**: (children <18; the mentally impaired, pregnant women, the ages, other). Projects with incarcerated persons cannot be exempted.

6) PI Signature J Matson **Date** 4-19-13 (no per signatures)

**** I certify my responses are accurate and complete.** If the project scope or design is later changes, I will resubmit for review. I will obtain written approval from the Authorized Representative of all non-LSU institutions in which the study is conducted. I also understand that it is my responsibility to maintain copies of all consent forms at LSU for three years after completion of the study. If I leave LSU before that time the consent forms should be preserved in the Departmental Office.

Screening Committee Action: Exempted ✓ Not Exempted ____ Category/Paragraph 4

Signed Consent Waived?: Yes / No

Reviewer Mathews **Signature** Robert C Mathews **Date** 5/1/13

VITA

Paige Elizabeth Cervantes was born in Long Island, New York, in 1990. She received her Bachelor of Arts degree at Binghamton University in 2012 with a major in psychology and an emphasis in applied behavior analysis. Thereafter, she entered the doctoral program in clinical psychology at Louisiana State University. Her research interests include the assessment and treatment of autism spectrum disorder.