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The moderating effect of autism symptomology on the relationship of cognitive and adaptive functioning with anxiety symptoms in infants and toddlers

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THE MODERATING EFFECT OF AUTISM SYMPTOMOLOGY ON THE RELATIONSHIP OF COGNITIVE AND ADAPTIVE FUNCTIONING WITH ANXIETY SYMPTOMS IN INFANTS AND TODDLERS

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Dedication

This work is dedicated to all of the individuals with developmental disabilities and their families as well as everyone that works with these extraordinary individuals. I have learned so much from my interactions with all of them and continue to be amazed by their growth and strength. I also dedicate this to all of the important people in my life that have supported me to this point, especially my mother, my children, and my ever-supportive wife Lisa. I could not have done any of this without you all. Finally, a special dedication to my inspiration and motivation for all of my work is given to my youngest brother, Brent.

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Abstract

Anxiety disorders have been shown to have a high prevalence rate in the general population and the prevalence in those with Autism Spectrum Disorders (ASD) is even higher. The detection, diagnosis, and implementation of an early intervention program for these disorders are crucial to the developmental outcome for such individuals. Researchers have shown how cognitive and adaptive functioning are related and affect anxiety symptoms in children as well as the high comorbidity with ASD. The aim of this study was to confirm those relationships, using scores from the BISCUIT-Part 2 (anxiety symptomology) and the BDI-II (Cognitive and Adaptive Developmental Quotient), and to show the moderating effect of autism symptomology, as measured by the BISCUIT-Part 1, in infants and toddlers. A sample of 2,366 infants and toddlers between the ages of 17 -36 months of age was utilized in a hierarchical moderation analysis and follow-up post-hoc analyses were also completed to determine the source of the interaction within subdomains of cognitive and adaptive functioning. The relationship between autism symptomology and anxiety was confirmed as well as the relationship between Cognitive DQ and anxiety. Adaptive DQ was found to be positively correlated with anxiety but in the opposite direction as expected. The moderating effect of autism symptomology in the interaction terms between Cognitive and Adaptive DQ individually with anxiety was statistically significant but with a small effect size. Similar results were found for the full regression model including the 3-way interaction between Cognitive DQ, Adaptive DQ, and autism symptomology with a negligible effect size.

Introduction

Autism Spectrum Disorders (ASDs) are a set of neurodevelopmental disorders that are typically diagnosed within the first few years of life and include Autistic Disorder (Autism), Asperger's Disorder (Asperger's syndrome), Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), Rett's Disorder (Rett's syndrome), and Childhood Disintegrative Disorder (Matson, 2007; Nebel-Schwalm & Matson, 2008; Tidmarsh & Volkmar, 2003). The core features of these disorders include marked deficiencies in socialization and communication skills in addition to the presence of restricted interests and repetitive behaviors (Brereton, Tonge, & Einfeld, 2006; Leekam, Prior, & Uljarevic, 2011; Tidmarsh & Volkmar, 2003). Under the current Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) this set of disorders are labeled Pervasive Developmental Disorders; however, since the introduction of the term ASD (Wing, 1996) its use has been common in research and similar settings to convey the similarities, core features, and level of severity of symptoms within the spectrum (Akshoomoff, 2006) and will be used throughout this paper.

One of the most prevalent disorders in children, and more specifically in children with ASDs, are anxiety disorders (Kessler et al., 2005). The comorbidity between autism and anxiety disorders leads to increased challenges for the individual and contributes to a poor prognosis (Remington et al., 2007). The early detection of ASDs as well as comorbid psychopathologies, such as anxiety disorders, is key in early and successful treatment and intervention. Research regarding the development and treatment of anxiety disorders in those with ASDs has been deficient. The purpose of the current study was to examine the relationship between cognitive and adaptive functioning with symptoms of anxiety and the moderating effect of autism symptomology. The history, differential diagnosis, prevalence, early detection, and assessment

of ASDs will be discussed as well as the assessment of anxiety and cognitive/adaptive functioning in children. In addition, common discussions and arguments presented throughout the history of ASDs will be presented. The current study aims to broaden the literature and knowledge of how autism symptomology affects cognitive and adaptive functioning in relation to symptoms of anxiety.

Autism Spectrum Disorders

Early History

The term "autism" was originally introduced by Eugen Bleuler (1913) as a trait of Schizophrenia, rather than the disorder that we have come to know, and is also why autism has had such close ties to the schizophrenias throughout its history. "Autistic thinking" was described by Bleuler (1913) as a fairy tale or fantasy state of thinking that is a common feature in adults with Schizophrenia. Bleuler noted that this type of thinking can also be seen in children or adults without Schizophrenia in the form of daydreaming or imaginative play, however, the difference lies in that the individual with Schizophrenia becomes so lost in their fantasy that the lines between reality and fantasy are blurred. This blurring of reality occurs in a process of isolation in which the schizophrenic is able to immerse himself in the fantasy to an extent that he begins to believe the fantasy is reality. Bleuler noted that as long as the fantasy or fairy tale that the schizophrenic individual believes is not interfered with he will continue to be rational and reasonable. However, once an outside individual attempts to hinder his progress or dilute his fantasy the irrational and illogical manifestations of the disease are apparent (Bleuler, 1913). The term "autism" has since come to mean something very different over several decades from the initial research of young children thought to be showing symptoms of Schizophrenia.

In 1943, Leo Kanner published what has become the seminal article on autism entitled "Autistic Disturbances of Affective Contact." Beginning in 1938, Kanner began documenting eleven children, all under the age of 12, in which he noticed similar traits that were markedly different from any other syndrome or disorder currently categorized. Kanner noticed that the common thread between all of these children was a set of symptoms including a lack of eye contact, abnormalities in speech and communication, excellent rote memory, extreme social

impairments, a strong desire for the preservation of sameness, and an "extreme autistic aloneness" as well as other commonalities (Kanner, 1943, 1944). Kanner observed that this group of individuals had certain core features that, in combination, were distinct from any other currently classified disorder and termed it "early infantile autism" (Kanner, 1944, 1954).

Kanner continued to follow these children and their families and made several observations regarding the presentation and common factors amongst the case studies. In addition to the core features Kanner noted that the majority of cases involved males, the symptoms began almost from birth, the children came from backgrounds of high parental intelligence and achievement, and he suspected that at least some of the symptoms of autism were maintained, or even fostered, through a common pattern of parental indifference toward the child with little attention paid to the emotional needs of the child, but rather attending only to physical needs (Kanner, 1949).

Initial Core Features. Even from the first article that Kanner published on the cases of early infantile autism he was able to observe, identify, and succinctly describe many of the core features of autism which are still a part of our diagnostic criteria today (Kanner, 1943). Kanner's first major observation that was common amongst all cases was an overall inability for these children to form appropriate social connections with the people around them. He referred to this as an "extreme autistic aloneness" that can be observed from the beginning of life. Parents of these children described them as "self-sufficient" in their play and appeared to be "in a shell" with no attention paid to the outside world and "oblivious to things around them." These children had a lack of social awareness and any outside interference was either ignored to the best of their ability or they would become upset, engage in tantrum behavior, or be visibly distraught until the interference ceased and things returned back to their static state.

A second observation that Kanner (1943) made was in regards to the acquisition and use of language. Of Kanner's initial 11 cases, 8 cases had acquired speech although some with delay. He observed that, of those that had acquired speech, they had very strong vocabularies and excellent rote memory for repeating previously learned nursery rhymes, prayers, lists, phrases, etc. However, Kanner pointed out that, although these children had excellent memory and vocabulary skills, their language skills were not used for communicative purposes. The peculiarities in their use of language could be seen in the literalness of their use of prepositions and other phrases, echolalia (both immediate and delayed), pronoun reversals (as the pronouns are repeated just as initially heard), and general speech delays or abnormal patterns of speech development (e.g., speaking full sentences after years with no speech).

A third important core feature of these children that Kanner (1943) observed was that the behavior of the autistic children was governed by an extreme desire and persistence of sameness and consistency that nobody but the child could disrupt including changes in routine, patterns, and order of objects. This perseveration to keep the autistic child's environment in a static state became one of the main focuses of his daily life and behavior. Even minute changes in the environment would be easily perceived by the child and would cause severe distress, anxiety, or lead to tantrums. According to Kanner, the strong desire for the "preservation of sameness" that was so apparent in these children eventually lead to repetitive behaviors and a restricted range of interests and activities.

Other observations made by Kanner (1943) in the initial cases he presented still are major signs and symptoms of current diagnostic criteria. Kanner noticed a lack of eye contact and social reciprocity but a good "relation with objects." In fact, many of the children would treat other individuals in the environment as if they were just another piece of furniture in the room

and would only respond if the individual posed as a threat to interfere in the intense desire for sameness or isolation. Kanner also observed that all of the children seemed to have an impression of high cognitive abilities with a stoic type affect and as well as apprehension around other individuals. Other noted commonalities included early feeding problems, excessive tantrums, and several instances of children being judged to be either deaf or feeble-minded (Kanner, 1944). He also stated that they were generally normal physically and had excellent fine motor abilities.

Gender Ratio. Although not as apparent in the first study of early infantile autism presented by Kanner, the disparity between the numbers of males versus females in the population of autistic children quickly exhibited a higher prevalence rate in males. By the time Kanner had over 100 cases diagnosed with early infantile autism there was a clear gender ratio of approximately 4:1 (Kanner, 1951; Kanner & Eisenberg, 1957). The higher prevalence rate in males eventually became an important aspect in the search for etiological factors and also in the nosology and differentiation from childhood Schizophrenia (Kanner, 1971b; Rutter, 1968). Kanner also notes that males were generally referred to clinics for evaluation between 2 and 6 years of age, whereas females were generally referred between 6 and 8 years of age, possibly suggesting a different trajectory and severity of symptoms and development of the disorder between genders (Kanner, 1971b).

First symptoms. Kanner stated that some of the initial signs of autism occur during the first 2 years of life, and one of the initial observations that emerged in preliminary research was a lack of anticipatory reaction to being picked up as reported by a majority of parents (Kanner, 1954). Gesell, as cited by Kanner (1943) in his original article, states that the "average child at 4 months makes an anticipatory motor adjustment by facial tension and shrugging attitude of the

shoulders when lifted from a table or placed on a table" (p. 242). However, this anticipatory motor adjustment appeared to be either missing or delayed in the children with autism. In addition, many of the core symptoms may have been present from birth but remained unrecognized by parents until the use of such skills were essential to their integration into other environments (e.g. school, church, social gathering, etc.). Most parents described their children as being self-sufficient, happiest when left alone, and relatively quiet from infancy (Kanner, 1943). It was also noted that many of the children had feeding problems as young children and even problems with nursing from birth.

Parental backgrounds. Some of the first observations that Kanner made regarding early infantile autism were several commonalities regarding the backgrounds of these children's parents. He noted that, of the original 11 cases, all fathers were fairly successful and intelligent holding advanced degrees and most of the mothers were college graduates holding a wide range of prestigious careers (Kanner, 1943). Even over a decade later, with exactly 100 cases of early infantile autism being examined, Kanner reports similar findings and stated that "to this day, we have not encountered any one autistic child who came of unintelligent parents" (Kanner, 1954). This issue was addressed shortly thereafter in a censuring response by Bender (1959) in which she reports that many cases of autism also come from parents with "defective" or "mediocre" intelligence; however, the correlations between highly successful and intelligent parents continued to be reported throughout research in support of Kanner's initial observation (Rutter, 1968). Even into the 1970's and 80's, researchers continued to cite the highly affluent and intellectual backgrounds of children with autism (Dor-Shav & Horowitz, 1984; McAdoo & DeMyer, 1977).

Emotional deprivation. In regards to the causes of early infantile autism, Kanner noted that there were no generalizations that could be made regarding the individual's physical condition, circumstances of birth, or a general pattern of heredity (noting that almost none of the family members had a history of Schizophrenia) (Kanner, 1954). Throughout Kanner's work, however, there is a general theme that suggested that nearly all cases of early infantile autism came from families in which the parents of the autistic child presented with a distinct pattern of obsessiveness regarding their child's development and rearing and a general lack of affection toward their children. He even went as far as to call many of these parents "successfully autistic adults" (Kanner, 1954).

This idea of parental emotional deprivation as the cause of autistic-like traits was seen in other areas and gave support to Kanner's claims. Goldfarb (1945) presented an article regarding the effects of psychological deprivation and the consequences of emotional deprivation in infants. In the article, Goldfarb reported that such emotional deprivation can cause isolation, aggression, "affective impoverishment," anxiety, and language deficiencies in infants. Bakwin (1949) reported that many cases of "hospitalism," which was a failure to thrive in infants placed in hospitals before the age of one, were due to emotional deprivation. The characteristics of these children were similar to those of autistic children, including no interest in the environment, no smiling in response to others, feeding problems, and rarely crying. These symptoms were first thought to be due to malnutrition or infection and so nutrition was increased and boxes were built to decrease the amount of human contact that each child had. It was later determined that more handling, attention, affection, and presence of mothers decreased the rate of "hospitalism" without increasing the rate of infections. Within this context of knowledge, it is understandable that Kanner would make such conclusion regarding the symptoms and causes of infantile autism.

A general consensus was beginning to develop amongst researchers regarding these "frigid" mothers and their contribution to the development, or lack thereof, of their children showing signs of infantile autism. This eventually led to the term "refrigerator mothers" which blamed mothers' cold indifference towards their children for their autistic symptoms. A major proponent and one of the biggest propagators of the term was a child psychologist named Bruno Bettelheim (1967) who presented and discussed the theory in his book *The Empty Fortress*. Additionally, Despert (1951) provided an account of a mother that fits this model described above. The mother had a son who was given the diagnosis of infantile autism and eventually was sent to live somewhere else to receive the care that doctors and psychiatrists insinuated she could not provide. The account became more concerning when the mother returned to relay that she was pregnant with another child and was worried that her and her husband would not be able to provide for the child's needs and feared that the second child would eventually develop another case of infantile autism. The child was born and after several years of normal development the child began to show similar signs of infantile autism and the parents were then directed to hire someone to take care of the child in the home. Even though the child never met criteria for infantile autism, this was attributed by Despert to be the effectiveness of the individual hired to care for the child and is further evidence for the "refrigerator mother" concept, and to a lesser degree, included fathers as well.

The research supporting the "refrigerator mother" concept continued to be perpetuated by numerous reports (Eveloff, 1960; Kanner, 1958; Kanner & Eisenberg, 1957) until about the mid 1960's. At this time, even Kanner himself began to question this assumption. In an article Kanner (1965) stated that four viewpoints existed regarding parental interaction and its role in the cause of autism. First was the view that parental behaviors that are typical (according to

Kanner) of autistic children could be a reaction to the child's peculiarities; but he also points out that in many cases these behavior traits were apparent in the parents before the child was born and therefore discounted this view. The second view was that parents (especially mothers) were the basic cause of infantile autism and that healthier maternal attitudes would have precluded the disorder. The third view was that the autistic child has an innate disability to relate to people which is further exacerbated by parent's emotional detachment. The final view presented by Kanner was that the core features of infantile autism stem from a common biological factor and he stated that many of the parents of autistic children have traits of autism themselves. These opposing views to the well-established idea that parental emotion deprivation was the cause of autism began to be supported by other researchers throughout the rest of the decade and beyond (DeMyer, Hingtgen, & Jackson, 1981; McAdoo & DeMyer, 1977; Rutter, 1968; Rutter & Bartak, 1971). Kanner eventually even clarified his position by citing his original work in 1943, in which he states that the autistic aloneness that he observed could be observed from the beginning of life and that he only could not preclude the parent-child relationship as a possible factor in the development of the disorder (Kanner, 1971b). Kanner also supported research and a book proposing additional theories by Bernard Rimland (1964) which were in direct opposition to the "refrigerator mother" theory. Although it was learned that some of Kanner's suppositions regarding the causes of autism were misguided, his work proved to be important in the discovery and nosology of autism.

Much of the early history of ASDs is attributed to the discoveries of Kanner. However, at about the same time an Austrian doctoral student published a work in German called "Autistic Psychopathy in Childhood." Hans Asperger's discovery was relatively unnoticed until it was translated into English in 1991 by Uta Frith (Asperger & Frith, 1991). Asperger's work did not

go completely unnoticed prior to 1991. Van Krevelen (1971) published a paper discussing the differences between Autistic Disorder and Autistic Psychopathy, claiming that they were erroneously thought to be the same disorder. He posited that these were two separate disorders and described the essential characteristics and differences of each disorder. Asperger's work on Autistic Psychopathy has naturally led to the diagnostic category of Asperger's Disorder. Asperger's descriptions of these children were similar to those of Kanner in 1943 including special interests, odd eye gaze, behavioral problems, language abnormalities, and other symptoms. Probably the most striking similarity was both Kanner's and Asperger's choice to describe these children as "autistic."

Historical Nosology and Diagnostic Criteria

After receiving numerous reports of similar cases around the country, and having over 55 case histories of his own, Kanner (1949) attempted to provide a classification of early infantile autism as its own separate diagnosis, *sui generis*. Kanner compared and contrasted the symptomology of early infantile autism with Heller's disease (now known as Childhood Disintegrative Disorder), congenital word deafness, and the schizophrenias. He ultimately provided support for including autism as a form of, but yet still different from, the earliest onset of childhood Schizophrenia (Kanner, 1949). Kanner supported this separation by describing the differences in self-isolation between the autistic and schizophrenic individual. He stated that in the schizophrenic individual there is a social withdrawal that ultimately results in marked isolation from the outside world. The autistic individual, Kanner postulated, has marked deficiencies in socialization and attachment to others from the beginning of life and therefore does not meet the same criteria or pattern of schizophrenic withdrawal (Kanner, 1954).

By the mid-1950's Kanner, along with Eisenberg, narrowed the core features of early infantile autism to two main symptoms: an extreme self-isolation or autistic aloneness and an obsessive desire for the preservation of sameness that commonly results in the restriction of interests and activities (Kanner, 1954, 1958; Kanner & Eisenberg, 1957). Autism had, by this time, been described and diagnosed by researchers in the U.S., Canada, England, France, Holland, and other areas and most considered autism as either the earliest onset of Schizophrenia (yet still separate from childhood Schizophrenia) or as a distinct diagnosis separate from the schizophrenias (Kanner, 1958). However, some researchers still failed to distinguish individuals with early infantile autism from those diagnosed with childhood Schizophrenia (Bender & Grugett, 1956).

As the symptoms and presentation of early infantile autism became more popular the classification, presentation, and misuse of the diagnosis became a growing problem. The sensitivity and specificity of diagnostic criteria for early infantile autism became a debate amongst researchers. Early infantile autism was the default diagnosis for many cases of the earlier described hospitalism and for separation anxiety, organic disorders, analytic depression, and childhood Schizophrenia (Eveloff, 1960; Kanner, 1958). At the same time, many individuals with autism were labeled as deaf, mentally defective, or with childhood Schizophrenia (Mosse, 1958; Ritvo & Provence, 1954). Mosse (1958) addressed the misuse of childhood Schizophrenia, citing the "enormous increase" in the diagnosis and discussing the consequences of misdiagnosis and the generalization of treatments for adults with Schizophrenia to children. Mosse noted that many adults with Schizophrenia were "model children," leading him to the belief that Schizophrenia is not a disease of childhood and that the cases described as originating in childhood were actually not Schizophrenia at all, but something else. He

mentioned several cases in which children with behavior problems or juvenile delinquents were labeled as schizophrenic and in turn received multiple treatments of electroconvulsive therapy, as that was the popular treatment for adults with Schizophrenia at the time. He stated that childhood Schizophrenia, and in relation early infantile autism, was a fashionable and misused diagnosis in the United States (Mosse, 1958).

The differentiation of similar but distinct disorders continued to be further clouded by several researchers and clinicians. Rank (1949) proposed a treatment for children who were developmentally delayed due to emotional deprivation, which categorized the "atypical child" as one considered to be psychotic, feebleminded, or having any abnormal characteristics of development. Bender (1959) believed that the differentiation of autism from mental deficiency was an unnecessary distinction. The deep ties that autism had to Schizophrenia from its inception were very hard to break, and the diagnosis of childhood Schizophrenia became, according to Kanner, a "pseudo diagnostic waste basket" (Kanner, 1971a). It was clear that the classification of infantile autism, childhood Schizophrenia, and other disorders diagnosed in childhood needed more distinct criteria for differentiation and classification.

As the need for a clinical differentiation increased, researchers began to delineate different classifications of childhood disorders. Eveloff (1960) stated that the autistic child was still commonly confused with hospitalism and analytic depression, as well as the broader categories of childhood Schizophrenia and mental defectiveness. In an attempt to differentiate autism and childhood Schizophrenia, Eveloff cited evidence that schizophrenic children have an abnormal EEG whereas autistic children generally have a normal EEG. Eveloff also cited the low incidence of Schizophrenia in family members of autistic children, which has also been seen in other studies throughout the history of autism research (Bender & Grugett, 1956; Kanner &

Eisenberg, 1957). Rutter (1968) provided additional characteristics to differentiate between the two disorders. In addition to the lack of family history of Schizophrenia in autism, Rutter also cited the higher sex ratio for males, the incidence of comorbid mental retardation, lack of delusions and hallucinations, the distinct patterns in IQ subtests, and the general steady course of autism development as compared to childhood Schizophrenia.

In an attempt to elucidate the murky distinction between disorders of childhood, Rutter (1968) provided a classification system of psychotic disorders in childhood. The individual classifications of this system were generally demarcated by the age of onset. The first classification was psychotic disorders that are first apparent in early adolescents. Rutter explained that this classification was most like adult Schizophrenia and should include childhood Schizophrenia. The second classification was an onset of autistic-like features between the ages of three and five years of age after a period of normal development. This could include disorders such as Heller's disease or Childhood Disintegrative Disorder. The last classification is for children who have an onset of symptoms from birth up to three years of age and would include Kanner's autism.

At this time many clinicians still struggled with the distinction between those with Autistic Disorder and those with intellectual disability (ID), as these two disorders commonly overlapped. This eventually led to a tri-axial model of classification which viewed a child's intellectual functioning as separate from a clinical psychiatric syndrome as well as etiological factors (Rutter et al., 1969). This changed Rutter's previous classification slightly, as infantile autism would now be included under the broader term of infantile psychosis which has an onset of symptoms within the first 36 months. This was differentiated from disintegrative psychosis, which presented with normal development for a period and severe disintegration after 36 months

of age; Schizophrenia, which were childhood cases similar to adult cases normally developing in adolescence; and other psychoses.

The model of classification was further refined to include 4 axes which included: Axis I, psychiatric syndrome; Axis II, intellectual level; Axis III, associated or etiological biological factors; and Axis IV, associated or etiological psychosocial factors (Rutter, 1972) and is similar to the current classification model (APA, 2000). Rutter also revised the cutoff age for onset of autism from 36 months to 30 months and noted that, as most children within the infantile autism classification show symptoms or developmental abnormalities from infancy, approximately 20% of these children may show a period of normal development followed by a regression period. This period of regression has been referred to as an autistic regression (Tidmarsh & Volkmar, 2003). As an additional note, Rutter (1972), citing the similarities between true cases of childhood Schizophrenia and adult Schizophrenia, called for an end of the term "childhood Schizophrenia" and instead proposed that it should be included as Schizophrenia, as it usually presents in adolescence and has the same features as adult onset Schizophrenia.

During this period of time individuals with autism were still diagnosed with childhood Schizophrenia due to the lack of an autism diagnosis in the first and second editions of the APA's *Diagnostic and Statistical Manual of Mental Disorders* (DSM & DSM-II; APA 1952, 1968). It was not until 1980 that infantile autism was entered into the third edition of the DSM (DSM-III; APA, 1980). In this edition infantile autism was listed under a newly created category called Pervasive Developmental Disorders (PDD) along with Childhood Onset Pervasive Developmental Disorder and Atypical Pervasive Developmental Disorder. In order to receive a diagnosis of infantile autism an individual needed to meet all six of the following criteria: age of onset prior to 30 months; pervasive lack of response to other people; language development

deficits; peculiar speech patterns, metaphorical language, and pronoun reversal (if speech is present); resistance to change, peculiar interests or attachments to objects; and an absence of schizophrenic features such as delusions, hallucinations, loose associations, and incoherence.

The name and diagnostic criteria for infantile autism was changed in the 1987 revision of the DSM (DSM-III-R; APA, 1987). It was then referred to as Autistic Disorder and the criteria were completely changed and more clearly resemble the current diagnostic criteria. To meet the criteria at least 8 of the 16 items had to be present including at least two impairments in the area of reciprocal social interaction and at least one each in the areas of communication impairment and restricted activities/interests. Another important difference from DSM-III is that the revised edition no longer required onset of symptoms prior to 30 months of age. The clinician was only asked to specify if onset was after 36 months of age but could still give the diagnosis with onset after that period. In addition to these changes the DSM-III-R also removed the Childhood Onset and Atypical Pervasive Developmental Disorders. These were replaced by the now infamous Pervasive Developmental Disorder, Not Otherwise Specified (PDD-NOS). The age of onset was eventually reinstated for Autistic Disorder with the DSM-IV requiring onset prior to age 3 in the areas of social interaction, social communication, or symbolic play.

Diagnostic Criteria and Differential Diagnosis

Diagnostic criteria of mental disorders have become necessary to help differentiate between certain groups of symptoms, and to increase treatment validity, appropriate use of medications, cognitive and behavioral treatments, and intervention programs. Careful and accurate diagnosis followed by appropriately applied treatments is important to help improve prognosis and to facilitate research on a global stage. Reliability of mental disorder categories is crucial to the collaboration of research across the world as it is imperative that an individual

diagnosed with an Autistic Disorder in the United States is very similar and meets the same diagnostic criteria as an individual with Autistic Disorder in England, Norway, China, or Spain. The two most used manuals for diagnosis and classification throughout the world include the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; APA, 2000) and the World Health Organization's *International Classification of Diseases* (ICD-10; WHO, 1993). These two diagnostic manuals have somewhat different criteria for mental disorders but have continued to grow closer in their similarities overtime (Tidmarsh & Volkmar, 2003). Due to the similarities between the two diagnostic manuals in relation to pervasive developmental disabilities, this paper will focus on the criteria of the DSM-IV-TR.

Autistic Disorder. The current diagnostic criteria for autism maintain many of the core features and criteria that Kanner observed in his initial cases of early infantile autism. According to the DSM-IV-TR (APA, 2000) to meet a diagnosis of autism an individual must display deficits in social interaction and communication as well as restricted or repetitive interests, movements, and activities. At least six of the 12 criteria must be met to warrant an autism diagnosis. The diagnostic logarithm for autistic disorder places a greater emphasis on the impairments of socialization, requiring at least two of the following criteria to be met: (a) impairments in nonverbal social behaviors such as sustained eye contact, facial expressions, and gestures; (b) lack of appropriate peer relationships; (c) lack of spontaneous sharing; and (d) lack of social or emotional reciprocity. At least one of the following criteria must also be met for impairments in communication including: (a) lack or delay in verbal language; (b) impairment in ability to initiate or sustain conversation; (c) stereotyped or idiosyncratic language; and (d) lack of social imitative or make-believe play. In addition at least one of the following criteria must be met for restrictive/repetitive behaviors, interests, or activities including: (a) abnormally

restrictive interests and preoccupation; (b) inflexible adherence to nonfunctional routines or rituals; (c) stereotyped and repetitive motor movements; and (d) preoccupation with parts of objects. In addition to meeting at least six of the above criteria, following the diagnostic algorithm, the individual must display delays or abnormal functioning in social interaction, use of language in social communication, or symbolic or imaginative play with an onset prior to 3 years of age. Finally, the impairments noted must not be better accounted for by other mental disorders, specifically Rett's Disorder or Childhood Disintegrative Disorder.

The differential diagnosis of Autistic Disorder from other disorders is an important and highly researched area of interest. It is important for treatment and diagnostic purposes to consider other disorders that may better account for the pattern of deficits seen in an individual. The most obvious disorders to consider in the differential diagnosis of Autistic Disorder are other Pervasive Developmental Disorders. The differential diagnosis of these separate disorders from Autistic Disorder will be discussed within their respective sections below. Other diagnoses that must be considered, according to the current *DSM* (APA, 2000), include Schizophrenia, Selective Mutism, Expressive and Mixed Receptive-Expressive Language Disorders, and Mental Retardation.

Because of the similarities and long history of association between Autistic Disorder and Schizophrenia it is important to differentiate between the two. The main distinguishing feature of Schizophrenia is the age of onset. Schizophrenia rarely occurs before 7 or 8 years of age, whereas autism symptoms can appear as early as birth but at least by 3 years of age (Green et al., 1984). In addition to age of onset, those with autism generally have a higher incidence of intellectual disability and no presence of hallucinations or delusion, symptoms often seen in Schizophrenia (Green et al., 1984; Mash & Barkley, 2003). Although there have been a few

cases of reported co-occurrence of Autistic Disorder and Schizophrenia, this combination is very rare and those with Autistic Disorder appear to have rates of Schizophrenia similar to the general population (Volkmar & Cohen, 1991).

Selective mutism is a disorder in which a child refuses or is phobic of speaking in everyday social situations, such as at school, while showing no deficits in speech and communication in other situations, generally at home with family members (Scott & Beidel, 2011). The onset of the disorder is usually before the age of 5, when it begins to interfere in educational settings as children begin schooling (Reuther, Davis, Moree, & Matson, 2011). This can be differentiated from autism most noticeably by the presence of normal communication skills in some settings and also an absence of both social impairments and repetitive and restricted behaviors or interests. Similarly, developmental language disorders, such as expressive and mixed receptive-expressive language disorder, can also be differentiated by a lack of social impairments and repetitive and restricted behaviors or interests seen in autism. In addition, children with autism generally show more severe impairments compared to those with developmental language disorders, as evidenced by echolalia, pronoun reversals, and metaphorical language (Bartak, Rutter, & Cox, 1975; Mash & Barkley, 2003).

Finally, because of the high incidence of intellectual disability in those with autism it is important, and many times difficult, to differentiate between autism and intellectual disability. This is especially difficult in individuals with severe and profound mental retardation which is present in over half of those with an Autistic Disorder (Fombonne, 1999). Social and communication deficits are common in persons with intellectual disabilities; however, the presence of social and communication deficits above and beyond what is attributable to intellectual disability is a sign of a comorbid Autistic Disorder. Additionally, other disorders

such as Obsessive Compulsive Disorder and Attention-Deficit/Hyperactivity Disorder share several symptoms with ASDs, and can thus potentially complicated differential diagnosis in young children.

Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). When an individual displays characteristics of autism or other PDDs but does not meet all of the criteria for such disorders, a diagnosis of PDD-NOS may be given (Tidmarsh & Volkmar, 2003). There are no specific diagnostic criteria for PDD-NOS, which makes diagnosis complicated. According to the *DSM-IV-TR* (APA, 2000), a diagnosis of PDD-NOS is given when there is a severe and pervasive impairment in reciprocal social interactions, verbal or nonverbal communication, or presence of stereotyped behaviors, interest, or activities. However, diagnostic criteria of other disorders should be considered first, including all other PDDs, Schizophrenia, Schizotypal Personality Disorder, and Avoidant Personality Disorder. PDD-NOS, therefore, is a diagnosis intended to include "subthreshold autism" or atypical autism (Mesibov, 1997) and has become a diagnostic category "with enormous clinical variation" (Tidmarsh & Volkmar, 2003).

Several researchers have argued that the diagnostic criteria for PDD-NOS are too inclusive, leading to several issues in the diagnosis, research, and treatment of PDD-NOS. Volkmar, Shaffer, and First (2000) suggested requiring an impairment in social interaction, which is considered a hallmark symptom of ASDs, along with impairments in either communication or restricted interests in order to meet the criteria of PDD-NOS. This would in turn narrow the scope of the diagnosis and increase its validity as well as its utility in research and treatment.

Asperger's Disorder. Those with a diagnosis of Asperger's Disorder have impairments in social interaction, restricted interests, and are generally described as having normal language development but may have some peculiarities in communication (Tidmarsh & Volkmar, 2003). The validity of an Asperger's Disorder versus a high-functioning form of Autistic Disorder has been in question throughout the research. Some researchers claim that Asperger's Disorder and high-functioning autism are difficult to differentiate due to overlapping symptoms and should essentially be considered one disorder (Howlin, 2003). Other researchers have provided evidence for a differentiation between the two disorders (Matson & Boisjoli, 2008; Matson & Wilkins, 2008; Szatmari, 1992), which are currently separate diagnoses in the *DSM-IV-TR* (APA, 2000). In spite of this research the newly proposed criteria for Autism Spectrum Disorders in the *DSM-V* excludes Asperger's Disorder as a differentiation from a high-functioning form of Autistic Disorder (Ghaziuddin, 2010). Regardless, the current diagnostic criteria in *the DSM-IV-TR* (APA, 2000) require that six criteria be met, with an emphasis on impairments in social interaction, before a diagnosis of Asperger's Disorder can be given.

First, the individual must exhibit at least two of the following social impairments: (1) marked impairment in eye gaze, facial expressions, body postures, or gestures of social interaction; (2) a failure to develop appropriate peer relationships; (3) lack of spontaneous sharing of enjoyment, interests, or achievements; and (4) lack of social or emotional reciprocity. Second, the individual must exhibit at least one of the following restricted repetitive and stereotyped patterns of behavior, interests, and activities: (1) extreme preoccupation with a specific interest; (2) inflexible adherence to rituals or routines; (3) stereotyped and repetitive motor movements; and (4) preoccupation with parts of objects. These disturbances must also cause significant impairment in important areas of functioning with no general delay in language,

cognitive development, self-help skills, or adaptive behavior. The final criterion requires that the individual does not meet criteria for another specific PDD or Schizophrenia (APA, 2000).

The differential diagnosis of Asperger's Disorder from autism, especially high functioning autism, can be difficult and the validity of a distinction has been brought into question as discussed above. Matson and Wilkins (2008) provide a collection of evidence from numerous studies helping to differentiate Asperger's Disorder from high functioning autism. From this research Matson and Wilkins show that those with Asperger's syndrome are less likely to be diagnosed before the age of 10, and have less severe early symptoms, less social impairment, superior language comprehension, higher adaptive and cognitive functioning, and fewer symptoms of autism. Those with Asperger's have also been found to have a milder developmental course and better prognosis (Ozonoff, South, & Miller, 2000). According to current diagnostic criteria, children with Asperger's Disorder have no delay in language or overall cognitive abilities. Those with Asperger's generally show a difference in restricted, repetitive, and stereotyped interests and behaviors, with less abnormal motor mannerisms and more restricted interests and preoccupations than those with Autistic Disorder. However, repetitive behavior profiles of individuals with Asperger's Disorder do not significantly differ from those with high functioning autism, as reported by South, Ozonoff, and McMahon (2005).

Childhood Disintegrative Disorder (CDD). Being one of the less common forms of ASDs, CDD was the first to be described amongst those now classified as Pervasive Developmental Disorders. Theodore Heller reported on five boys and one girl in 1908 that presented with a severe regression in development after a period of normal development up to three or four years of age. This regression generally leads to a profound intellectual disability with a poor prognosis for recovery (Kurita, 2011; Volkmar, Koenig, & State, 2005). The

disorder was originally termed “dementia infantilis” by Heller and is commonly referred to as Heller’s disease or Heller’s syndrome throughout the history of ASD. The current diagnostic criteria (APA, 2000) state that in order to meet the diagnostic criteria for CDD an individual must first have an apparently normal development for the first two years of life including appropriate verbal and non-verbal communication, social relationships, and adaptive behavior. Second, the individual must have a significant loss of previously acquired skills in at least two areas including: expressive or receptive language, social skills or adaptive behavior, bowel or bladder control, play, or motor skills. This loss of skills must also occur before the age of 10. Third, the individual must have abnormal functioning in two of the following: social interaction, communication, or restricted, repetitive, and stereotyped patterns of behavior, interests, or activities. Finally, the individual must also not meet criteria for another specific PDD or Schizophrenia.

Researchers point out the many overlaps and difficulty in differential diagnosis of CDD versus Autistic Disorder, stating that further research is needed to increase the differentiation between the two (Kurita, 2011). The differential diagnosis of CDD can be difficult according to current diagnostic criteria. A diagnosis of CDD includes impaired functioning in the same three core areas as Autistic Disorder. The only differentiation between the two is that CDD is prefaced by at least 2 years of normal development and Autistic Disorder is defined as having an age of onset prior to 3 years of age. This leaves a 1-year gap in which the differentiation between CDD and Autistic Disorder is unclear. Although some preliminary research has shown some differentiation based on age of onset, intelligence, and muteness (Hendry, 2000) the differences have not yet been validated. This is mainly due to the fact that CDD is rarely diagnosed and only about 100 cases were reported in the literature by Klin and Volkmar (1997).

Because of the lack of differentiation between the two disorders some researchers have called into question the validity of a CDD diagnosis (Hendry, 2000) while others continue to support its clinical and diagnostic validity (Malhotra & Gupta, 2002; Mouridsen, 2003; Volkmar, 1992; Volkmar & Rutter, 1995).

Rett's Disorder. First described by Andreas Rett in 1966, Rett's Disorder is the rarest of all ASDs and is found almost exclusively in females. Rett's discovery of 22 females who showed repetitive hand-wringing and other symptoms went relatively unnoticed until other researchers revived his research in 1983 by describing 35 females with traits similar to those described by Rett, subsequently crediting Rett for the discovery (Hagberg, Aicardi, Dias, & Ramos, 1983). Although Rett's Disorder is one of the rarest ASDs, it has been cited as one of the most common causes of intellectual disability in females (Amir et al., 1999). Amir and colleagues state that individuals with Rett's Disorder generally have a period of normal development followed by a severe regression after 6-18 months. They cite the cause of many cases of Rett's Disorder as a mutation in the X-linked MECP2 gene which then fails to encode for the MeCP2 binding protein. Because the gene is found on the X chromosome, mutations of the gene in males are generally lethal or lead to severe disability.

The current diagnostic criteria (APA, 2000) for Rett's Disorder require that the individual display an apparently normal prenatal and perinatal development, normal psychomotor development for the first 5 months of life, and normal head circumference at birth. After the period of normal development the individual must meet all of the following criteria: (1) a slowing of head growth between 5 and 48 months; (2) loss of previously acquired purposeful hand movements between 5 and 30 months and replaced by stereotyped hand movements; (3)

loss of social engagement; (4) poor coordination in gait or trunk movements; and (5) impaired expressive and receptive language as well as impairments in psychomotor abilities.

Differential diagnosis of Rett's Disorder from other Pervasive Developmental Disorders can be differentiated by sex ratio, onset, and specific deficits. Because of the lethality of Rett's Disorder in males, the disorder is almost exclusively limited to females. It can also be differentiated by its characteristic deceleration of head growth and earlier onset than in Childhood Disintegrative Disorder or Asperger's Disorder. In addition, the presence of poorly coordinated gait or trunk movements and loss of purposeful hand movements are characteristic of those with Rett's Disorder.

Prevalence

The prevalence rates of ASDs have been a topic of research and contention for several years. Since the discovery of autism and initial studies of its prevalence the rate of ASDs has grown almost exponentially. This rapid increase in the rate of ASDs has continued over the last several decades and the reason for the increase has been debated throughout the literature (Fombonne, Quirke, & Hagen, 2009; Matson & Kozlowski, 2011). Rice (2009) reported an increase of approximately 57% in 10 of 11 sites included in a CDC study between the years of 2002 to 2006. Estimates of the prevalence of ASDs have varied significantly throughout time as well as between studies within the same relative time period. The current estimates of ASDs appear to be between approximately 1 in 150 children (Fombonne et al., 2009; Matson & Shoemaker, 2009; Nicholas et al., 2008) and 1 in 110 children (Lord & Bishop, 2010; Rice, 2009). ASDs are now considered the second most frequently occurring developmental disorder, with intellectual disability as the first and cerebral palsy as the third most common (Nicholas et al., 2008).

Even more complicating is the apportioning of rates between the different diagnostic categories within ASD. Fombonne et al. (2009) examined studies between 1966 and 2009 and selected studies based on their methodological soundness and proper use of diagnostic categories. According to Fombonne and colleagues, the best estimate for the prevalence of Autistic Disorder is approximately 22 per 10,000 or 1 in every 455 individuals. Rates for other diagnostic groups have had much less reliable and consistent results as well as fewer overall studies. In the same study, Fombonne et al. reported rates of CDD to be extremely rare with 1 in every 55,556 individuals. The rates for Asperger's Disorder, however, have been extremely discrepant and unreliable with rates ranging from 0.3 to 48.4 per 10,000 individuals. Therefore, the current rates of the other diagnostic categories are much less reliable due to methodological issues, changing diagnostic criteria, and lack of studies focusing on the individual diagnoses.

Although the rates of ASDs are somewhat unreliable at this point, there are few that would argue against the trend that the overall rates of such disorders have increased significantly over the past several decades. The debate, however, is in the reasons for the increase in prevalence. Several researchers have cited numerous factors involved in the rise in prevalence rates. One of the more common arguments is that the increase is due to the changes and disparities between the DSM and ICD and within different versions of the DSM. Changes within the DSM, as discussed earlier, have directly affected the rates of ASDs between editions and have led to diagnostic substitution (Fombonne et al., 2009; Matson & Kozlowski, 2011; Shattuck, 2006). The theory of diagnostic substitution is that when the diagnostic criteria for disorders change, even slightly, many individuals are moved from one diagnostic category to another leading to apparent increases and decreases in prevalence rates. This is evidenced by the increase in ASD rates and the decrease in ID rates over the same time periods. As the

diagnostic criteria change, the specificity and sensitivity of the diagnostic categories become more exclusive or inclusive; however, this cannot account for the entire increase in the rate of ASDs.

Matson and Kozlowski (2011) provided an examination and review of other causes of the increase in prevalence rates discussed throughout the literature. In addition to disparities and changes to diagnostic criteria and diagnostic substitution, several other factors play a role in the dynamic changes to prevalence rates. These include inaccurate diagnoses, differences in research methodology, environmental factors, cultural differences, and increased awareness. It is likely that each of these plays a factor in the prevalence rates of ASDs but most researchers cite the increased awareness amongst researchers, clinicians, primary care physicians, and parents as a likely source of the increase. In addition, the constant advancement in the diagnosis of ASDs at progressively earlier ages is likely a significant factor in the increase in rates as well (Rice, 2009).

Early Detection

The early detection and diagnosis of ASDs is an essential precursor to early intervention and treatment. Many treatment programs and state intervention programs, such as Louisiana's EarlySteps program, are aimed at providing treatment and related services to children under the age of 3 with developmental delays and disorders such as ASD. The focus of early intervention has in turn led to a focus on early detection (Gutierrez et al., 2009; Hayward, Gale, & Eikeseth, 2009). Due primarily to the research and development of scales designed for the early detection of autism, and a general awareness amongst parents and professionals, the age of diagnosis is decreasing (Charman & Baird, 2002). Parents of autistic children tend to notice problems in their child's development well before 3 years of age. In a study completed by Chakrabarti

(2009) it was reported that the age that parents began to recognize problems in their autistic child was 23.4 month on average. Parents, on average, would seek professional help approximately 4 months later but the mean time to diagnosis was approximately 32 months after the problem was first recognized by parents. That means that on average more than 2.5 years were lost between parental detection and diagnosis. An additional study showed that 50% of parents of autistic children reported concerns before 12 months of age (Kishore & Basu, 2011). Furthermore, Planche (2010), not unlike Kanner (1943), posits that many symptoms are present from birth but just go undetected until the child is older. Researchers have shown that earlier detection by professionals leads to earlier intervention and improved overall prognosis (Matson, Wilkins, & Gonzalez, 2008). However, the current gap between detection and intervention is too large and researchers and clinicians alike need to focus on earlier detection.

Early detection of ASD relies on development of assessments that are designed specifically for the pattern of symptoms seen in young infants and toddlers that are unique to that population. Charman and Baird (2002) stated that assessments should focus on impairments in "social orienting, joint attention, imitation, play, and reciprocal affective behavior." They also point out that the pattern of symptoms evinced by a 2-year-old child is different than the pattern of symptoms exhibited by a 4- or 5-year-old child. The following common assessments used in clinical practice for ASD are focused on early detection and diagnosis to facilitate early intervention and treatment.

Assessment

Assessment of ASDs has been a major point of focus in autism research in the past decade. A complete assessment of a child believed to have autism should include a pregnancy, birth, family, and developmental history which should include age of first concern, eating

difficulties, food selectivity, as well as current and past behaviors (Tidmarsh & Volkmar, 2003). Assessment may also include a test of hearing to rule out any aural problems, a measure of adaptive functioning, assessment of motor skills and sensory problems, and a behavioral assessment. However, all of this testing must begin with a diagnostic measure to assess for the core features and criteria for an ASD as well as a broad assessment of comorbid psychopathology common in this population (Matson, Rieske et al., 2011). There are many assessments that have been in use for several years and also many recently published assessments. A broad sampling of measures common to the literature will be discussed. Many of these measures have limited psychometric research while others have received more empirical support.

Two of the more commonly used assessments in the diagnosis of ASD, the Autism Diagnostic Interview-Revised (ADI-R; Lord & Rutter, 1994) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al., 2000) are frequently used in tandem. The ADOS-G is a widely used semi-structured observation assessment, whereas the ADI-R is a semi-structured interview. Researchers have shown that the combination of these two diagnostic tools is effective in the clinical classification ASD and has sufficient reliability and validity (de Bildt et al., 2004); however, this research study included children older than 4 years of age. The ADOS-G was not designed for early detection and diagnosis and most of the research regarding the psychometric properties is utilizing samples with the mean age of approximately 4 years or above (Lord et al., 2000). In addition, the ADOS-G takes much longer than other diagnostic assessment tools, especially when used in combination with the ADI-R, and the resource investment in administration time, training, and overall cost may not be efficient in clinical settings.

The Childhood Autism Rating Scale (CARS; Schopler, Reichler, DeVellis, & Daly, 1980) is one of the older assessment tools still used today in the diagnosis of autism. The CARS is an observational assessment to be completed by the clinician after observations of the child's behavior or review of developmental history. The measure in composed of 15 items rated on a scales ranging from 1 (within normal limits) to 4 (severely abnormal). Authors report acceptable psychometric properties overall with an internal consistency coefficient of .94 and interrater reliability value of .71. The CARS places a child, based on their score, on a continuum ranging from non-autistic, to mild/moderate autism, and finally severe autism. The CARS is used for a wide range of ages in childhood and is based on comparisons to same-aged typically developing children. This assumes that the administrator of the CARS is familiar with the age-appropriate behaviors displayed in each of the 15 items. The CARS is still used in research and clinical settings today (Chlebowski, Green, Barton, & Fein, 2010; Matson, Mahan, Hess, Fodstad, & Neal, 2010; Mayes et al., 2009) and has been translated into several languages for use in other countries (Kurita, Miyake, & Katsuno, 1989; Pereira, Riesgo, & Wagner, 2008).

Other scales were developed more specifically for early screening and detection of autism symptoms. The Pervasive Developmental Disorders Screening Test, Second Edition (PDDST-II; Siegel, 2004) is a screener used to assess for ASDs in children between the ages of 12-48 months of age. It is a parent/caregiver report in a yes/no format that is broken up into three different stages. The Primary Care Screener (stage 1) is for use with children 12-18 months of age and is the initial screener. The Developmental Clinic Screener (stage 2) is to be used in clinics that generally screen for developmental delays. The final screener is the Autism Clinic Severity Screener to be used when completing an assessment for children with an ASD. The sensitivity and specificity of the initial screener is strong with values of .92 and .91 respectively. Research

regarding the other psychometric properties of the PDDST-II, however, appear to be either lacking or altogether non-existent. In addition, preliminary data from clinical use of the PDDST-II have shown an approximate false positive and false negative rate of about 30% each (McQuistin & Zieren, 2006).

The Screening Tool for Autism in Two-year-olds (STAT; Stone, Coonrod, & Ousley, 2000) was developed as a stage two screening assessment for use in clinics to distinguish between those with an ASD and those with another developmental disability between the ages of 24-35 months of age. The STAT is a structured observation which includes 12 items scored as "pass" or "fail" during a play-like interaction between the administrator and the child. It is initially reported to have a sensitivity and specificity of .83 and .86, respectively. A more recent study, using a new scoring algorithm, increased the sensitivity to .95, but with a subsequent decrease in specificity to .73 (Stone, McMahon, & Henderson, 2008). Research regarding other psychometric properties of the STAT has been lacking. Stone, Coonrod, Turner, and Pozdol (2004) report an interobserver agreement value of 1.00 and test-retest value of .90 in distinguishing between high and low risk categories. However, the overall sample size was small and has not been replicated in further studies.

Some more recent assessments have been studied but with limited psychometrics. The First Year Inventory (FYI; Reznick, Baranek, Reavis, Watson, & Crais, 2007; Watson et al., 2007) was developed to assess behaviors in infants at their 12-month birthday. Higher scores on the scale would suggest a higher risk of an autism diagnosis. The FYI is a parent-report measure with a total of 63 items including several open-ended questions. Psychometric properties pertaining to the FYI have yet to be assessed or reported. The Early Screening of Autistic Traits Questionnaire (ESAT; Dietz, Swinkels, van Daalen, van Engeland, & Buitelaar, 2006; Swinkels

et al., 2006) is a screening assessment for children between 14 to 15 months of age. The ESAT has both a 4-item pre-screener for use by primary care physicians and the 14-item ESAT which is to be used by a trained psychologist during an in home visit which generally lasts about 1.5 hours. The ESAT was tested on a large population of over 30,000 infants; however psychometric properties were not calculated and seemed to have a high false positive rate for those with learning disabilities and mental retardation. Although the ESAT appears to have some promise in utility in clinical practice (Oosterling et al., 2010) further studies of the psychometric properties of this assessment should be completed.

Other assessment tools have been developed specifically for early detection and have more psychometric studies supporting their use. The Modified Checklist for Autism in Toddlers (M-CHAT; Robins, Fein, Barton, & Green, 2001) is another assessment tool that was specifically designed to assess children at 24 months of age for ASDs. The M-CHAT was developed from the earlier Checklist for Autism in Toddlers (CHAT; Baron-Cohen, Allen, & Gillberg, 1992), follows the same format, and has some identical items. The measure is a 23 item parent-report measure examining developmental milestones. Initial reliability estimates were strong with internal reliability coefficients of .85 for both the entire checklist as well as for a subset of six items found to be critical to the discrimination between children diagnosed with ASDs versus those that are not. Initial sensitivity and specificity values were also high with values of .87 and .99 respectively. However, more recent studies have shown the sensitivity and specificity to be lower as the initial study was conducted in a population that was already determined to be "at-risk". The M-CHAT, much like the CARS, has also been translated and used in several different languages including: Chinese (Wong et al., 2004), Arabic (Seif Eldin et

al., 2008), Portuguese (Losapio & Pondé, 2008), Sinhala (Perera, Wijewardena, & Aluthwelage, 2009), Spanish (Canal-Bedia et al., 2011) and Japanese (Inada, Kamio, & Koyama, 2010).

Additionally, the Baby and Infant Screen for Children with aUtism Traits: Part 1 (BISCUIT-Part 1; Matson, Boisjoli, & Wilkins, 2007) is a 62-item informant based measure used for diagnosis of ASDs in infants and toddlers between 17 and 37 months of age. Items on the measure are rated as 0 (not different; no impairment), 1 (different; mild impairment) and 2 (very different; severe impairment). This measure is part of a larger battery of assessments which focus on diagnosis (Part-1), comorbid psychopathology (Part-2), and challenging behaviors (Part-3).

The *BISCUIT-Part 1* has been shown to have excellent internal reliability with a reported coefficient alpha of .91 (Matson, Wilkins, Sevin, et al., 2009). The sensitivity and specificity of the measure has been shown to be strong with reported values of 93.4 and 86.6 respectively (Matson, Wilkins, Sharp, et al., 2009). The measure also had an overall correct classification rate of 88.8. The *BISCUIT-Part 1* has been shown to have strong convergent validity with the M-CHAT and other measures and appropriate discriminant validity with non-related measures (Matson, Wilkins, & Fodstad, 2011). The *BISCUIT* measures a wide range of ASD symptomology with a maximum possible score of 124. Matson, Wilkins, Sharp et al. (2009) found that those with an Autistic Disorder diagnosis average approximately 59 points on the diagnostic measure of the *BISCUIT* while those with PDD-NOS or no diagnosis averaging approximately 28 and 10 points, respectively. Those with scores of 17 or higher on the measure are considered to be "at-risk." The measure is relatively quick and easy to administer and requires less time, training, and financial resources than many of the other available assessments and is therefore more efficient for use in both clinical and research settings.

Anxiety Symptoms in Children

Anxiety is a common part of childhood development as children begin, through different stages of their lives, to experience and make sense of the world around them while developing and using newly acquired skills. In many children, however, those levels of anxiety can raise to levels that can hinder their developmental progress in areas such as communication and language development, socialization, and several other areas of life. When the levels of anxiety reach a point of clinical significance and meet criteria for an anxiety disorder, the level of interference with development can increase and early treatment methods should be considered to prevent further developmental interference (Kessler et al., 2005). The causes of anxiety have been researched showing links to genetics factors (Stevenson, Batten, & Cherner, 1992; Tambs et al., 2012; Trzaskowski, Zavos, Haworth, Plomin, & Eley, 2012), familial interactions and experiences (Ollendick & Benoit, 2012; Verhoeven, Bögels, & Bruggen, 2012), and peer relationships (Scharfstein, Alfano, Beidel, & Wong, 2011; Zalk, Zalk, & Kerr, 2011). Several types of anxiety disorders have been categorized and according to the most recent publication of the DSM (DSM IV-TR; APA, 2000) include specific phobia, social phobia, obsessive-compulsive disorder, post-traumatic stress disorder, acute stress disorder, generalized anxiety disorder, separation anxiety disorder, agoraphobia, panic disorder (with or without agoraphobia), and anxiety disorders due to substances, general medical conditions, and anxiety disorders not otherwise specified.

The lifetime prevalence rate of an anxiety disorder is amongst the highest of all DSM-IV disorders with estimates ranging from 2.6% to 41.2% (Cartwright-Hatton, McNicol & Doubleday, 2006) with an average of about 28.8%, which is more prevalent than mood disorders, impulse-control disorders, and substance abuse disorders (Davis, Munson, & Tarcza, 2009; Kessler et al., 2005). Albano, Chorpita, and Barlow (1996) reported that anxiety disorders were

the most prevalent disorder in children and adolescents. In a national prevalence study, Kessler et al. (2005) found that the age of onset was also much earlier for anxiety and impulse control disorders (about 11 years of age) compared to substance or mood disorders. The early detection and treatment of an anxiety disorder is key to the prevention of developmental delays and worsening of anxiety-related behavior and symptoms (Kendall, 1994; Kessler et al., 2005); however, it is still unclear exactly how anxiety manifests in young children under the age of three.

Separation anxiety is the most common symptom seen in young children and is the most supported by empirical evidence in toddlers (Alman, Sommer, & McGoey, 2009). Research differentiating other anxiety symptoms in infants and toddlers is sparse; however, Mian, Godoy, Briggs-Gowan, and Carter (2011) found that through confirmatory and exploratory factor analysis among children ages 2-3 years, symptoms appeared to group in categories consistent with generalized anxiety, obsessive-compulsive symptoms, separation anxiety, and social phobia. This suggests that symptoms of anxiety disorders can begin to manifest themselves in children under the age of 3 years. The majority of available instruments for measuring anxiety have a minimum age of 6-8 years but several instruments have been created for use specifically with younger children including the Infant-Toddler Social and Emotional Assessment (ITSEA; Briggs-Gowan & Carter, 2006), the Fear Survey Schedule for Infants and Preschoolers and the Infant-Preschool Scale for Inhibited Behaviors (Warren, 2004), and the Child Behavior Checklist (CBCL; Achenbach & Rescrola, 1992).

Anxiety and ASD

We have seen that anxiety is amongst the most prevalent psychiatric disorders in typically developing children. The rate of anxiety disorders in children with ASDs has been

found to be much higher with reported prevalence rates averaging around 40-50% with reported rates as high as 84% (de Bruin et al., 2007; Gjevik et al., 2011; Morgan et al., 2003; Simonoff et al., 2008; White, Oswald et al., 2009). This high rate of comorbid anxiety symptoms has been reported in those diagnosed with autism regardless of level of intellectual functioning (Mayes, Calhoun, Murray, Ahuja, & Smith, 2011) and regardless of whether anxiety is measured as categorical or dimensional (White, Oswald et al., 2009). The high prevalence rate of anxiety in ASD has even led some researchers to characterize anxiety as a common feature of ASD (Bellini, 2004) and can be utilized as a diagnostic sign as well as a predictor of treatment outcome for Autistic Disorder (Remington et al., 2007). Several other researchers have questioned whether anxiety and ASD can truly be comorbid disorders or if the co-occurrence is illusory and accounted for by ASD itself (Caron & Rutter, 1991; White, Bray, & Ollendick, 2012; White, Oswald et al., 2009).

In further examination of the possible true comorbidity between ASD and anxiety, Wood and Gadow (2010) discussed the pathogenesis of anxiety in ASD and stated that anxiety could be a consequence of ASD symptoms, a moderator of ASD severity, or a representation of core ASD symptoms. To determine if anxiety and ASD are truly comorbid disorders Wood and Gadow stated that researchers need to learn more about the two disorders when they do co-occur to determine if 1) the etiology and phenotype are the same in ASD versus non-ASD populations; 2) the symptoms are true anxiety symptoms that are phenotypically altered by ASD and is therefore an ASD-specific variant of an anxiety disorder; 3) an aspect of a unique subtype of ASD; or 4) simply an artifactual comorbidity. Several studies have begun to research these questions but more work is still needed to delineate between the two disorders when they do co-occur.

Several anxiety disorders have overlapping symptoms with ASD which adds to the difficulty of determining when an anxiety disorder is truly present in a child with an ASD. Social Anxiety Disorder is one of the most prevalent anxiety disorders in children and adults with ASD, especially in those without an intellectual disability (Bellini, 2004; White, Bray, & Ollendick, 2012) and both have overlapping symptoms in terms of socialization and communication. Additionally, Obsessive-Compulsive Disorder and ASD have overlapping symptoms in terms of repetitive behaviors that many times are hard to distinguish, especially with measures that do not distinguish the qualitative differences between the behaviors (Lewin, Wood, Gunderson, Murphy, & Storch, 2011; Wood & Gadow, 2010). Further overlap exists in the higher rates of overall anxiety disorders in individuals and families with ASD and research which shows that high levels of anxiety in those with ASD covaries with more social maladjustment and core symptoms of ASD (Chang, Quan, & Wood, 2012; Wood & Gadow, 2010).

Anxiety symptoms in children with ASDs have been shown by several researchers to increase in relation to the severity of autism symptoms (Sukhodolsky et al., 2008). In a study conducted with 177 children with ASDs and their siblings, Kanne, Abbacchi, and Constantino (2009) found that anxiety symptoms increased as a function of autism symptom severity. They also reported that autism severity scores, as rated by teachers and parents, had moderate correlations with general psychopathology. Mayes, Calhoun, Murray, and Zahid (2011) recently reported that autism severity, verbal IQ, and age were the strongest predictors of anxiety and combined explained 25% of the variance in their sample of 627 children with autism. However, other researchers have not found such relationships between autism severity and anxiety (Simonoff et al., 2008). Anxiety has also been found to increase in adolescence in those with

ASD, especially without co-occurring intellectual disability, likely due to emerging awareness of differences and increases in social demand (White, Oswald et al., 2009).

Few researchers have examined the development and treatment of anxiety disorders as a comorbid disorder with ASD. Davis, Hess, Moree et al. (2011) described the patterns of anxiety development throughout the lifespan in individuals with autism. They state that anxiety usually builds throughout childhood and begins to level off and decrease in adolescence and young adulthood. The levels of anxiety then increase again later in life. These patterns of results could be hypothesized to be correlated with the pattern of treatment of such symptoms throughout the lifespan. Little attention is currently given in the treatment of anxiety in very young children with ASD as well as the treatment of ASD and anxiety in adults. Most of the current research and treatment focus has been on older children and adolescents with ASD. This pattern is supported by research in infants and toddlers with ASD which shows that, when compared to those with atypical development without an ASD, young children with ASD have higher rates of avoidance behavior, anxiety symptoms, and repetitive behaviors (Matson, Hess, & Boisjoli, 2010). In addition, Lovullo and Matson (2009) found that adults with ASD and comorbid intellectual disability have higher rates of anxiety as well as repetitive behavior, inattention, hyperactivity, and impulsivity when compared to adults with an intellectual disability without an ASD diagnosis.

A limited number of treatments designed for use in an ASD population in treating symptoms of anxiety have been developed and are currently being researched. Cognitive-behavioral treatments have been studied for treatment of anxiety in ASD amongst those without an intellectual disability. Wood et al. (2009) studied the effectiveness of cognitive behavioral therapy for anxiety in children with ASD and also examined its effects on daily living skills in

those with high-functioning autism (Drahota, Wood, Sze, & Dyke, 2011). Additionally, the Multimodal Anxiety and Social Skills Intervention (MASSI; White et al., 2010) is a cognitive behavioral intervention developed to treat both anxiety and social deficits simultaneously in high-functioning teens with an ASD (White, Ollendick, Scahill, Oswald, & Albano, 2009) in order to address both symptoms which have been shown to have a reciprocal relationship between anxiety and the social deficits of ASD (White, Oswald et al., 2009).

Early Identification and Assessment

Early assessment and identification of anxiety symptoms in children is key to reducing developmental effects and worsening of anxiety symptoms. Several researchers have become aware of the need for assessment in children and have created several different types of measures including structured or semi-structured interviews, self-report rating scales, as well as parent and teacher report rating scales.

Structured and semi-structured interviews have become an important part of childhood diagnostic assessment for anxiety disorders. Common examples of such interviews include the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Silverman, Saavedra, & Pina, 2001), the National Institute of Mental Health Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), and the Schedule for Affective Disorders and Schizophrenia for school-age children (K-SADS; Ambrosini, 2000). All of these interviews include both a parent and child interview form, assess anxiety as well as other disorders of childhood, and are used with children generally between the ages of 6 and 18 years of age (9 to 17 years for the NIMH DISC-IV).

Self-report rating scales are also common in anxiety assessment and research in children. These assessments are generally in questionnaire form and are used with children and

adolescents to measure levels of anxiety by asking the child several questions regarding different aspects of anxiety. These measure can range anywhere from 11 items to 80 or more items depending on the scale and age range and generally ask children to rate their fears or anxiety on a Likert-type scale. Examples of such measures include the Child Anxiety Sensitivity Index (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991), Fear Survey Schedule for Children-Revised (FSSC-R; Ollendick, 1983), Penn State Worry Questionnaire for Children (PSWQ-C; Chorpita, Tracey, Brown, Collica, & Barlow, 1997), Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000), and the Revised Children's Manifest Anxiety Scale: Second Edition (RCMAS-II; Reynolds & Richmond, 2008). All of these interviews assess, either broadly or specifically, some aspect of anxiety through self-reports. Much like the interview schedules discussed previously, the nature of these scales renders them unable to assess children under the age of 6.

Finally, parent and teacher report rating scales are also used in the assessment and treatment of anxiety symptoms in children. These measures ask parents and teachers a series of questions regarding their child's development, behavior, and anxiety symptoms. The measures are generally in questionnaire format using a Likert-type scale and range anywhere from approximately 20 items to well over 100 items. Examples of commonly used measures include the Child Behavior Checklist (CBCL; Achenbach, 2000), Conner's Rating Scales- Revised (CTRS-R/CPRS-R; Conners, Sitarenios, Parker, & Epstein, 1998a, 1998b), and the Devereux Behavior Rating Scale- School Form (Naglieri, LeBuffe, & Pfeiffer, 1993). Unlike the structured interview and self-report measures discussed earlier some of the parent and teacher rating scales, such as the CBCL, can be used with children as young as 3 years of age to measure symptoms of anxiety and other common behavioral problems of childhood.

Even with researchers demonstrating the effects of anxiety symptoms on early development very few assessments of anxiety in childhood are developed and focused on the early symptoms in infants and toddlers. Conners (2009) has recently developed an Early Childhood version (Conners EC) of his behavior rating scale which is focused on pre-school aged children ranging from 2 to 6 years of age. The measure includes several different forms for multiple informants and includes a behavior scale of anxiety symptoms. Another similar measure is the Infant Toddler Social Emotional Assessment which comes in a full and brief form (ITSEA/BITSEA; Briggs Gowan & Carter, 2006). This measure can be used with children as young as 12 months of age and is a nationally normed screening test that includes a broad subscale of general anxiety. The ITSEA is also a parent/caregiver report measure and provides information in several domains including externalizing and internalizing behaviors, dysregulation, and competence. However, these types of assessments are very few and relatively new. Although these measures can be used with children as young as 12 and 24 months of age, they are not specifically designed for use with an ASD population.

Given the high incidence of anxiety symptoms in young children with ASD it is important to assess and provide treatment for such symptoms as early as possible. These comorbid disorders also face comorbid obstacles in assessment. There are many assessments which measure anxiety symptoms in children and many assessments that assist in the diagnosis of autism as discussed previously. However, there are very few assessments that assess both autism and anxiety as comorbid disorders, and very few anxiety or autism scales that measure symptoms before the age of 3. Combined, it is exceptionally difficult to find assessments that have been designed to measure comorbid anxiety symptoms in children with ASD before the age

of 3. The Baby and Infant Screen for Children with aUtism Traits- Part 2 (BISCUIT-Part 2) (Matson et al., 2007) is one such measure.

***Baby and Infant Screen for Children with aUtIsm Traits-Part 2 (BISCUIT-Part 2;* Matson et al., 2007).** The *BISCUIT-Part 2* is a portion of a larger battery including a diagnostic scale and a measure of challenging behaviors common in children with ASDs. The *BISCUIT-Part 2* is a 57-item informant based measure used to assess comorbid psychopathologies in children 17 through 37 months of age with ASD or atypical development. Items on the measure are rated as 0 (not a problem or impairment; not at all), 1 (mild problem or impairment) or 2 (severe problem or impairment). The measure has five subscales derived through exploratory factor analysis including Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eating/Sleep Problems (Matson, Boisjoli, Hess, & Wilkins, 2011). The BISCUIT-Part 2 has been shown to have excellent reliability with a reported internal consistency coefficient of .96 (Matson, Wilkins, Sevin, et al., 2009).

Cognitive/Adaptive Functioning

The assessment of cognitive and adaptive functioning is an important part of screening for ASDs. The relationship between cognitive and adaptive functioning with anxiety, especially in a group considered "at risk" for ASDs or other developmental disabilities, show some interesting trends. In regards to cognitive functioning in children, researchers have found that higher levels of anxiety are associated with higher IQ in children with ASDs (Sukhodolsky et al., 2008). In one study researchers reported that a higher percentage of mothers of children with high functioning autism (HFA; 79%) reported symptoms of anxiety than mothers of children with low functioning autism (LFA; 67%), although both had high rates overall (Mayes et al., 2011). In a study by Gadow, DeVincent, and Schneider (2008), researchers found that IQ was positively correlated with Generalized Anxiety Disorder (GAD; r=.23) and Specific Phobia (r=.13) in children with ASDs, both significant at the .05 level. The developmental level trend has also been reported by several other researchers. Weisbrot, Gadow, DeVincent, and Pomeroy (2005) reported that increases in age and IQ were associated with higher levels of anxiety. More recently, Mayes, Calhoun, Murray, and Zahid (2011) conducted a study with 627 children with ASDs between the ages of 1 and 17 with IQ's ranging from 16-146. They found that maternal ratings of anxiety and depression increased with age and IQ. They reported that verbal IQ was a stronger predictor of anxiety than non-verbal IQ; however, other researchers have reported no such significant association between IQ and anxiety.

Zimet, Zimet, Farley, Adler, and Zimmerman (1994) found that non-ASD children with anxiety did not score significantly different on tests of intelligence; however, Davis, Ollendick, and Nebel-Schwalm (2008) found that non-ASD children diagnosed with anxiety disorders scored significantly lower on tests of intellectual ability than children without psychopathology. As Davis, Ollendick, and Nebel-Schwalm (2008) indicate, these differences were likely due to

differences in methodology and categorizing of groups according to primary and comorbid disorders. Additionally, those with anxiety symptoms have also been shown to perform more poorly on tests of achievement than peers without anxiety (Preckel, Holling, & Vock, 2006; Rapport, Denney, Chung, & Hustace, 2001).

In relation to adaptive skills, Sparrow and Cicchetti (1987) found that in typically developing children those with anxiety disorders had significantly lower adaptive scores when compared to normal controls matched on age and IQ. Within the same study, children with an ASD diagnosis had even lower adaptive scores when compared to both those with anxiety disorders and normal controls, also matched on age and IQ. Gadow, DeVincent, and Schneider (2008) also found correlations between adaptive skills and generalized anxiety when controlling for autism severity according to teacher ratings. However, the correlations were not significant when examining maternal ratings. Further research regarding adaptive skills and anxiety in young children with ASDs is extremely lacking.

Assessment

Battelle Developmental Inventory-Second Edition (BDI-2*; Newborg, 2005*). The BDI-2 is a 450-item measure which utilizes parent/caregiver interview, structured assessment, and clinical observations to determine a complete picture of a child's overall development. The measure is for use with children from birth to 7 years 11 months of age and used to screen and diagnose children considered to be at risk for developmental delays. The items are rated as 0 (no ability in this skill), 1 (emerging ability in this skill), or 2 (ability in this skill) and comprise the five separate domains including: adaptive, personal-social, communication, motor, and cognitive. The score from the five separate domains can then be calculated and each represents a domain Developmental Quotient (e.g., Motor Domain Developmental Quotient). The five domain scores

can then be utilized to attain a total Developmental Quotient. The Total and domain Developmental Quotients each have a mean of 100 and a standard deviation of 15. The entire assessment takes approximately 60-90 minutes to complete and provides a developmental profile of children's strengths and weaknesses as compared to same aged peers (Newborg, 2005).

The Cognitive domain of the BDI-2 is a measure of mental abilities observable in young children including the subdomains of Attention and Memory, Reasoning and Academic Skills, and Perception and Concepts. It is also important to note that the Cognitive domain of the BDI-2 does not include abilities related to language or communication as these are measured by the Communication domain. The Attention and Memory subdomain consists of 30 items that measures a child's ability to attend to environmental stimuli and retrieve information from short term and long term memory. The Reasoning and Academic Skill subdomain includes 35 items used to measure a child's ability to use critical thinking, problem solving, and mathematical skills. The Perception and Concepts subdomain is a 40 item scale used to assess interactions and discrimination abilities such as comparing and sorting objects or putting together pieces of a puzzle (Newborg, 2005).

The Adaptive domain of the BDI-2 is a measure of a child's ability to generalize information and skills previously acquired to other situations including the subdomains of Self-Care and Personal Responsibility. The Self-Care subdomain tracks milestones beginning at birth in the development of self-sufficiency from dependence on a caregiver or parent. The scale contains 35 items evaluating milestones in eating, dressing, toileting, grooming, and preparing for sleep. The Personal Responsibility subdomain tracks milestones beginning at age 2, such as a child's ability to assume responsibility for actions and to move around their environment

safely. The 25 items scale evaluates ability to initiate play, carry out tasks, avoid danger, and demonstrate care and caution (Newborg, 2005).

The psychometric properties have been evaluated in several studies and incorporate changes from the original inventory published in 1984. Reliability estimates of internal consistency have yielded coefficients of .99 for the Total score, .90 to .96 for domain scores, and .86 to .89 for individual subdomains. Test-retest values were also high with .93 to .94 for the Total score, .88 to .92 for domain scores, and .74 to .91 for individual subdomains. In addition, inter-rater reliability was shown to be excellent with values between .97 and .99. The BDI-2 has also been shown to have correlations estimated at .78 with the original BDI (Newborg, Stock, Wnek, Guidubaldi, & Svinicki, 1984) and has been shown to have convergent and divergent validity with several other tests including the *Bayley Scales of Infant Development, Second Edition* (BSID-II; Bayley, 1993), the *Denver Developmental Screening Test-II* (DDST-II; Frankenburg, Dodds, Archer, Shapiro, & Bresnick, 1992), the *Preschool Language Scale, Fourth Edition* (PLS-4; Zimmerman, Steiner, & Pond, 2002), the *Vineland Social-Emotional Early Childhood Scales* (Vineland SEEC; Sparrow, Balla, & Cicchetti, 1998), and others (Newborg, 2005).

Finally, the BDI-2 has also been shown to have good discriminating power between children with autism and their typically developing peers. Those with autism were shown to have a mean difference of 43.01 points less than typically developing peers on the Total Developmental Quotient of the BDI-2 with an estimated effect size of 2.87. In a sample of 88 children the BDI-2 Total Developmental Quotient was able to discriminate between children with autism and children without with sensitivity and specificity coefficients of .86 and .91, respectively (Newborg, 2005).

Purpose

Although researchers have reported high prevalence rates of comorbid anxiety amongst individuals with ASD (Kessler et al., 2005), research examining this relationship has been sparse especially in relation to infants and toddlers. Early detection and treatment has been a key goal in the recent research on autism (Matson, Rieske, & Tureck, 2011) and should include detection and treatment of comorbid disorders as well. Anxiety disorders are amongst the most common comorbid diagnoses in individuals with ASD (de Bruin, Ferdinand, Meester, de Nijs, & Verhij, 2007; Simonoff et al., 2008) and given the research regarding the worsening in severity of anxiety without early detection and treatment (Kendall, 1994) the relationship between these comorbid disorders should be more closely examined.

Recent research has focused on the moderating effects of communication deficits on anxiety symptoms in infants and toddlers with an ASD diagnosis (Davis et al., 2012) as well as children and early adolescents (Davis, Moree, et al., 2011). The aim of this study was to examine the relationship between cognitive and adaptive functioning (as measured by the BDI-2) with symptoms of anxiety (as measured by the BISCUIT-Part 2). Then the possible moderating effect of autism symptomology (as measured by the BISCUIT-Part 1) would be examined to determine how it affects the relationships above. The research regarding comorbid psychopathology in individuals with an ASD has received little attention in the history of ASD research and is still considerably new in the current body of research. In addition, very few researchers have viewed autism symptomology as a possible moderating factor in such a relationship. This research is important for broadening the understanding of the relationship between ASDs and comorbid anxiety, and to assist in the early detection and treatment of both ASDs and comorbid psychopathologies and the development of diagnostic and assessment measures.

Several hypotheses have been formed in regards to the results of the current study based on the literature in the area of autism and anxiety research. Initially, it was hypothesized that there would be a clear positive correlation between autism symptomology scores and symptoms of anxiety as has been seen in the general literature investigating prevalence rates of comorbid psychopathologies in ASDs (de Bruin et al., 2007; Gjevik, Eldevik, Fjaeran-Granum, & Sponheim, 2011; Morgan, Roy, & Chance, 2003; Simonoff et al., 2008; White, Oswald, Ollendick, & Scahill, 2009). The current study also looked to confirm ambiguous relationships that have been seen by other researchers. First it was hypothesized that there would be a relationship between cognitive abilities, as measure by the BDI-II and represented by the Cognitive DQ, and anxiety symptoms, as measured by the BISCUIT-Part 2. It was believed that this relationship would be positively correlated such that as cognitive abilities increase, symptoms of anxiety would also increase as seen in previous research (Gadow, Devincent, & Schneider, 2008; Mayes, Calhoun, Murray, Ahuja, & Smith, 2011; Mayes, Calhoun, Murray, & Zahid, 2011; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005).

Second, it was hypothesized that there would be a relationship between adaptive abilities, as measure by the BDI-II and represented by the Adaptive DQ, and anxiety symptoms, as measure by the BISCUIT-Part 2. It was believed that this relationship would be negatively correlated such that as adaptive abilities increase symptoms of anxiety would decrease due to a higher ability to utilize those skills that the individual has attained in multiple different environments and situations and therefore reducing stress and lowering anxiety risk.

Third, it was hypothesized that degree of autism symptomology, as measured by the BISCUIT-Part 1, would moderate the relationship between both cognitive and adaptive abilities with anxiety symptoms. It was believed that more severe autism symptomology may strengthen

the effects of cognitive and adaptive abilities individually on anxiety symptoms in such a way that the following patterns could be observed: 1) higher cognitive abilities and autism symptomology ratings would lead to higher ratings of anxiety symptoms and 2) lower adaptive abilities and higher autism symptomology ratings would lead to higher ratings of anxiety symptoms.

Finally, it was hypothesized that the three-way interaction between cognitive and adaptive abilities with autism symptomology would affect the relationship in such a way that those with high cognitive abilities, low adaptive abilities, and high autism symptomology would evince the highest levels of anxiety symptoms and those with low cognitive abilities, high adaptive abilities, and low autism symptomology would evince the lowest levels of anxiety symptoms.

Method

Participants

The participants for this study consisted of 2,366 children ranging from 17 to 36 months of age (M = 25.70, SD = 4.67) who were recruited through the EarlySteps program in Louisiana. EarlySteps is Louisiana's Early Intervention System under the Individuals with Disabilities Education Act, Part C, which provides services to infants and toddlers and their families from birth to 36 months of age. Children qualify for services if they have a developmental delay or a medical condition likely to result in a developmental delay. The participants were selected from a pre-existing database which contains demographic, diagnostic, and assessment information that is gathered in coordination with the EarlySteps program on an ongoing basis, as will be discussed below. Child participants were predominantly Caucasian (49.1%) and African American (38.9%), but some identified Hispanic (2.2%), or Other/Unidentified (5.4%) with 4.3% unreported. Males made up the majority of the child participants (71.3%).

Diagnostic assignments had been previously established by a licensed doctoral level psychologist, who was blind to BISCUIT scores, based on scores obtained on the M-CHAT (Kleinman et al., 2008; Robins et al., 2001), the DSM-IV-TR criteria (APA, 2000), the developmental profiles of the BDI-2 (Newborg, 2005), and clinical judgment. However, such diagnostic assignments were not used during this study. Rather, autism symptomology was measured using the BISCUIT Part-1 to determine the effect of autism symptomology from more of a dimensional and continuous perspective. Axis I diagnoses assigned by the initial psychologist, for demographic purposes only, were as follows: Autism (12.8%), PDD-NOS (10.6%), no diagnosis/atypical development (67.8%), and other/unreported (8.8%). Demographic characteristics are presented in Table 1.

Table 1
Participant Demographics

	(n = 2366)
Age in years	
Range	17-36 mo.
Mean	25.70
SD	4.67
Gender	
Male	71.3%
Female	28.7%
Race	
Caucasian	49.1%
African-American	38.9%
Hispanic	2.2%
Other/Unspecified	5.4%
Unreported	4.3%
Diagnosis	
Autism	12.8%
PDD-NOS	10.6%
No Diagnosis/Atypical	67.8%
Development	8.8%
Other/Unreported	

Note: SD = Standard Deviation.

Measures

Battelle Developmental Inventory-Second Edition (BDI-2*; Newborg, 2005*). The BDI-2 is a 450-item observational and informant based measure for use with children from birth to 7 years 11 months of age. The measure is used to screen and diagnose children considered to be at risk for developmental delays and to help guide and facilitate treatment planning. The items are rated as 0 (no ability in this skill), 1 (emerging ability in this skill), or 2 (ability in this skill) and comprise the five separate domains including: adaptive, personal-social, communication, motor, and cognitive. The score from the five separate domains can then be calculated and each represents a domain Developmental Quotient (e.g., Motor Domain Developmental Quotient). The five domain score can then be used to attain a total Developmental Quotient. A standard score (M = 100; SD = 15) is used for each domain as well as the total Developmental Quotient (Newborg, 2005). The BDI-II was not developed as a test of intelligence; however, several

studies have shown that the BDI is significantly correlated with measures of intelligence and appear to be the best estimate of intellectual abilities in children under the age of three (Berls & McEwan, 1999; Guidubaldi & Perry, 1984; Saylor, Boyce, Peagler, & Callahan, 2000). For the purposes of this study only the adaptive and cognitive domain scores, and their respective subdomain scores, were subjected for analysis.

***Baby and Infant Screen for Children with aUtIsm Traits-Part 1 (BISCUIT-Part 1;* Matson et al., 2007).** The BUISCUIT-*Part 1* is a 62-item informant based measure used for diagnosis of ASDs in infants and toddlers between 17 and 37 months of age. Items on the measure are rated as 0 (not different; no impairment), 1 (different; mild impairment) and 2 (very different; severe impairment). The measure is a diagnostic measure that is part of a larger battery which includes assessment of comorbidity (Part-2) and challenging behaviors (Part-3). Larger scores on this scale indicate higher levels of autism symptomology and therefore higher probability of an ASD diagnosis.

***Baby and Infant Screen for Children with aUtIsm Traits-Part 2 (BISCUIT-Part 2;* Matson et al., 2007).** The *BISCUIT-Part 2* is a 57-item informant based measure used to assess comorbid psychopathologies in children 17 through 37 months of age with ASD or atypical development. Items on the measure are rated as 0 (not a problem or impairment; not at all), 1 (mild problem or impairment) and 2 (severe problem or impairment). The measure has five subscales derived through exploratory factor analysis including Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eating/Sleep Problems (Matson, Boisjoli, et al., 2011). For the purpose of this study only the Avoidance Behavior scale and the Anxiety/Repetitive Behavior scale were utilized for statistical analyses and had reported alpha values of .83 and .82 respectively. The two scales were combined to

create a Total Anxiety score as done in previous research (Davis et al., 2010; Davis, Hess, Matthews, et al., 2011; Davis et al., 2012).

Procedure

All participants received a comprehensive battery of assessments offered by the EarlySteps program including the *BISCUIT and BDI-2*. All measures were administered to the parent/guardian by EarlySteps staff who were trained interviewers employed by the state of Louisiana. All interviewers had attended training on the measures used, including scoring and standardized administration methods, in addition to receiving education on ASDs and hold a minimum of a bachelor's degree. The parents/guardians of the children participating in this study served as informants on all administered measures and provided informed consent for their participation. The Louisiana State University Institutional Review Board and Louisiana's Office for Citizens with Developmental Disabilities provided prior approval for this study. Although only portions of the BISCUIT and BDI-2 were used during this study, all measures were administered in their entirety with other assessments that are a part of the comprehensive battery. Participants were excluded if they were outside the given age range or were missing more than two items on the described scales.

Statistical Analyses

Prior to computing statistical analyses, the *BISCUIT* and *BDI-2* data were reviewed in order to ensure that item values were present and valid (i.e., within the constraints of the measure's scoring criteria). In the case that a participant was missing data, the missing datum point was replaced with the item's mean score. Participants missing more than two data points were excluded from analyses. Several terms were created for use in subsequent analyses. A Total Anxiety score was calculated by combining the Anxiety/Repetitive Behavior and Avoidance Behavior domains of the BISCUIT Part-2 as done in similar previous research (Davis et al., 2010; Davis et al., 2012; Davis, Hess, Matthews, et al., 2011; Davis, Hess, Moree et al., 2011). Higher scores on this scale represent higher reported symptoms of anxiety. Descriptive statistics were conducted in order to determine the means of all included variables (cognitive, adaptive, autism symptomology, and anxiety scores). Demographics were also calculated for the total sample. All analyses were conducted using SPSS 19.0. Means of included variables are summarized in Table 2.

Table 2
Sample means (n = 2166)

	Mean	SD	Minimum	Maximum
Total Anxiety Score	1.64	3.51	0.00	28.00
Autism Symp. Score	19.48	18.85	0.00	113.00
Cognitive DQ	83.24	12.23	55.00	130.00
Adaptive DQ	87.50	13.93	55.00	140.00

Note: Autism Symptomology Score measured by BISCUIT-Part1;
Total Anxiety Score measured by BISCUIT-Part2

Preliminary Statistics

Preliminary analyses were conducted to test the relationship between cognitive, adaptive, and autism symptomology scores with anxiety scores using a randomized confirmation sample (n = 200) of the total sample. Simple regression models were completed independently to test the relationship between: 1) Autism Symptomology with Total Anxiety; 2) Cognitive abilities (BDI Cognitive DQ) with Total Anxiety score; 3) Adaptive abilities (BDI Adaptive DQ) with Total Anxiety score; and 4) an interaction of Cognitive and Adaptive DQ with Total Anxiety Score.

Study

The remaining sample (n = 2166) was utilized for the following statistical analyses. Pearson's correlations were conducted to determine if age was correlated with any of the predictor or outcome variables for the moderation analysis as this has been found to be a covariate in other similar studies (Davis et al., 2010; Davis et al., 2012; Davis, Hess, Matthews et al., 2011). Variables found to significantly correlate with predictor or outcome variables were included as covariates in the moderation analysis discussed below.

A moderation analysis was conducted based upon the assumptions and steps of the work of Baron and Kenny (1986). Any significant correlations that emerged in preliminary analyses were entered as covariates for the moderation analysis. Hierarchical regression procedures, as guided by Field (2009), were used to examine the Total Anxiety score from the *BISCUIT-Part2*. Covariates were entered into step 1 of the regression model as control variables.

This model assessed the effect of autism symptomology on the relationship between cognitive and adaptive abilities with symptoms of anxiety. Covariates, as discussed above, were entered into step 1 of the hierarchical regression. For step 2 the developmental quotients for the cognitive and adaptive domains of the *BDI-2* were entered along with autism symptomology

score as measured by the *BISCUIT-Part1*. Interaction terms were then created between cognitive and adaptive scores with autism symptomology scores after first standardizing the overall scores to prevent any violation of multicollinearity (Field, 2009). The interaction terms were created by simply multiplying the cognitive and adaptive scores separately with autism symptomology scores and creating a third interaction between cognitive and adaptive scores. These terms were then entered into step 3. Finally, a three-way interaction term was created by multiplying cognitive, adaptive, and autism symptomology scores together. This three-way interaction term was entered into the final step of the regression model.

Post-hoc Analyses

Subsequent moderation analyses were conducted with each of the cognitive subdomains (attention and memory; reasoning and academic skills; and perception and concepts) and adaptive subdomains (self-care and personal responsibility) utilizing the same moderation analysis method to further investigate the source of the effects.

Results

Preliminary analyses testing the individual relationships between cognitive, adaptive, and autism symptomology scores with anxiety scores were completed utilizing the confirmation sample of participants from the total sample. Results of a simple regression show that autism symptomology significantly predicted Total Anxiety scores, $\beta = .71$, t (198) = 14.23, $p < .001$. Autism symptomology also explained a significant proportion of variance in Total Anxiety scores, $R^2 = .51$, F (1,199) = 202.56, $p < .001$. These results are depicted graphically in Figure 1 and show that as autism symptomology increased, symptoms of anxiety were found to be higher. It was also found that Cognitive DQ significantly predicted Total Anxiety scores, $\beta = -.36$, t (198) = -5.51, $p < .001$; and also explained a significant proportion of variance in Total Anxiety scores, $R^2 = .13$, F (1,199) = 30.33, $p < .001$. These results are depicted in Figure 2 and show that as Cognitive DQ increased, symptoms of anxiety were found to be higher. Similarly, Adaptive DQ was also found to significantly predict Total Anxiety scores, $\beta = -.28$, t (198) = -4.10, $p < .001$; and also explained a significant proportion of variance in Total Anxiety scores, $R^2 = .08$, F (1,199) = 16.80, $p < .001$. These results are depicted in Figure 3 and much like Cognitive DQ and autism symptomology, as Adaptive DQ increased, symptoms of anxiety were found to be higher. Finally, the interaction between Cognitive and Adaptive DQ was found to significantly predict Total Anxiety scores, $\beta = -.35$, t (198) = -5.22, $p < .001$; and also explained a significant proportion of variance in Total Anxiety scores, $R^2 = .12$, F (1,199) = 27.20, $p < .001$. These results, shown in Figure 4, show that as the product of Cognitive and Adaptive DQ increases, symptoms of anxiety were found to be higher.

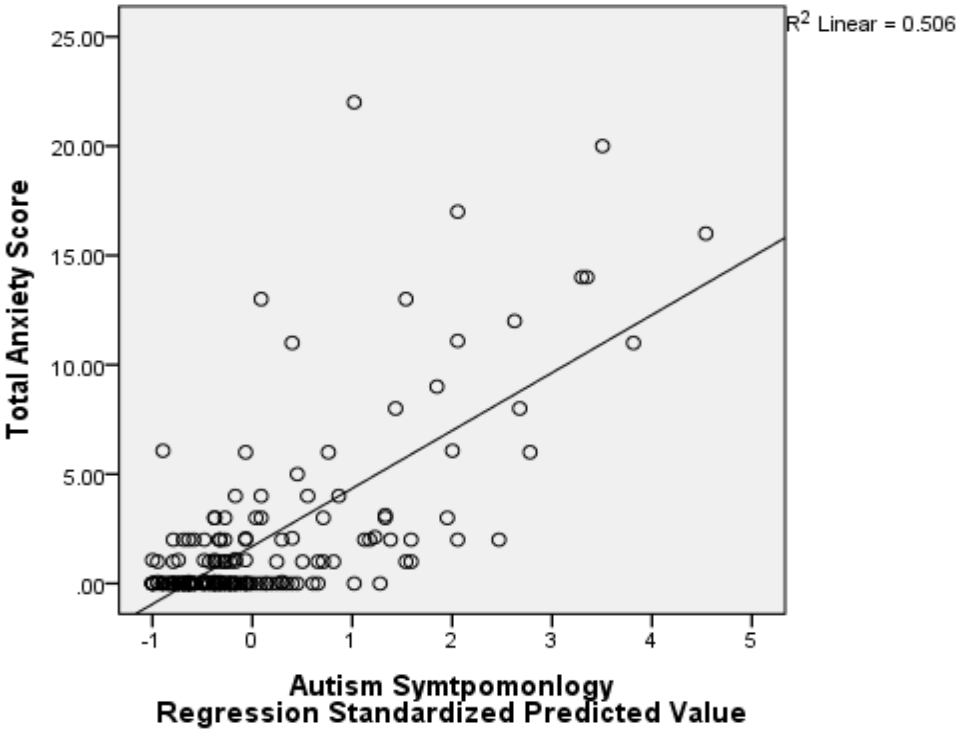

Figure 1 Autism Symptomology by Total Anxiety

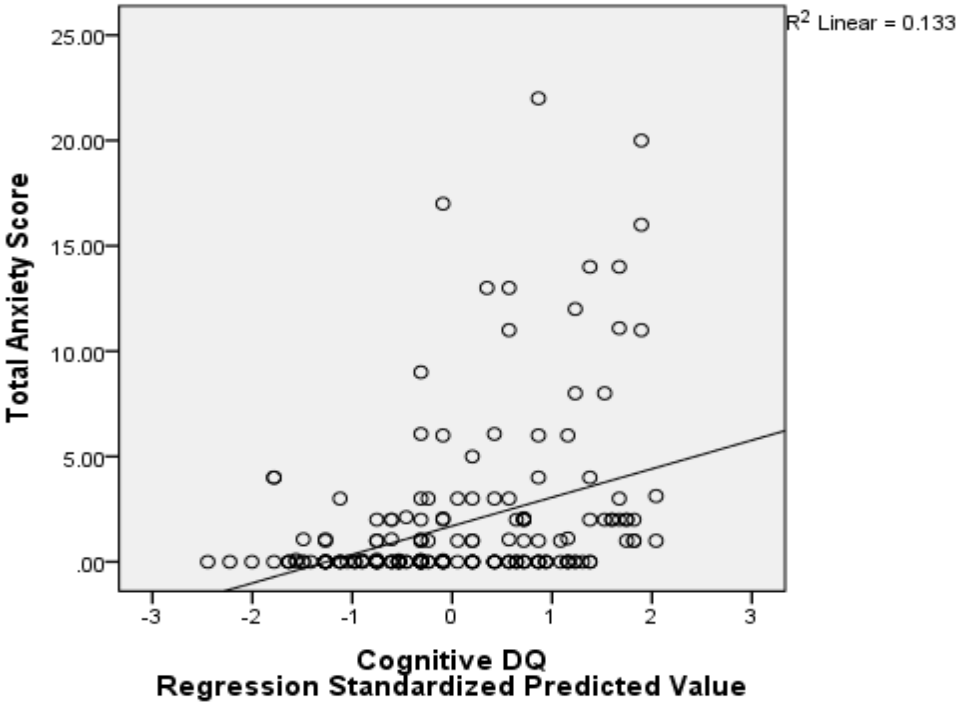

Figure 2 Cognitive DQ by Total Anxiety

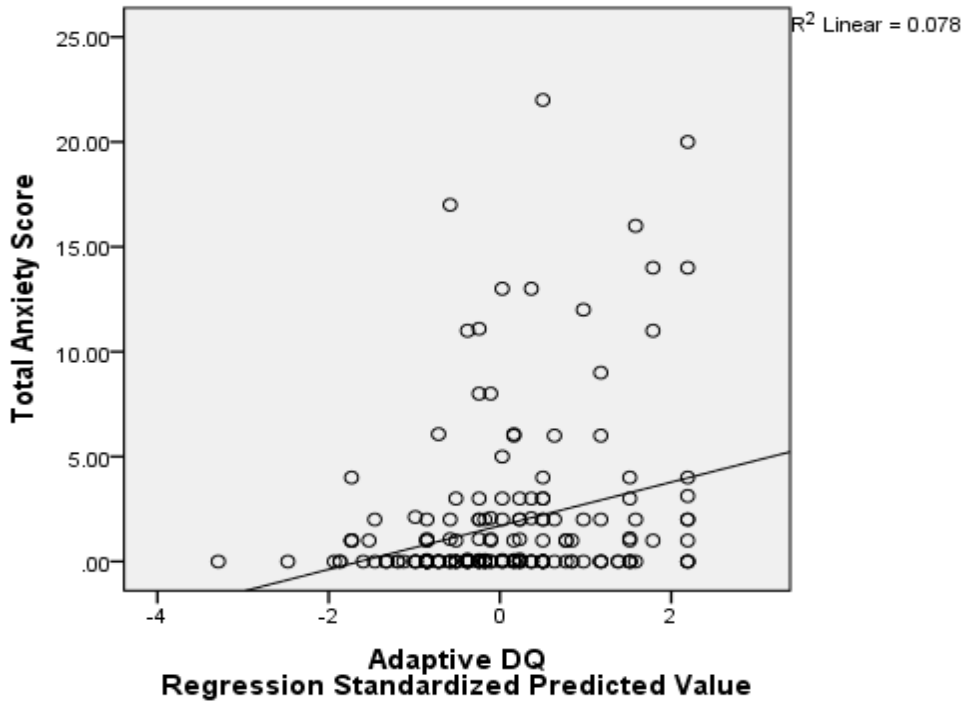

Figure 3 Adaptive DQ by Total Anxiety

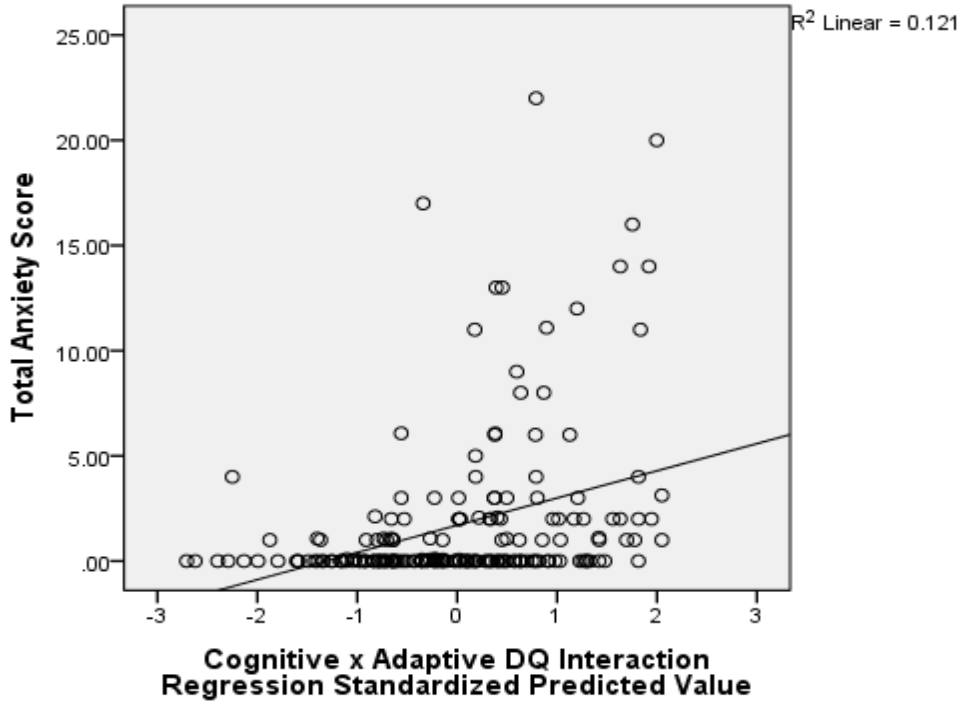

Figure 4 Cognitive/Adaptive DQ by Total Anxiety

Correlational statistics were completed using the full sample to determine variables that must be entered as covariates in the moderation analysis. Pearson's correlations between age and all predictor and outcome variables indicated that age was significantly correlated with Cognitive DQ, r = -.16; Adaptive DQ, r = .07; and Total Anxiety Scores, r = .08 (all ps < .01); but not with autism symptomology, r = .04, p = .06. Due to the significant correlations, age was then entered into the moderation analysis as a covariate.

A moderation analysis was then conducted based upon the assumptions and steps created by Baron and Kenny (1986). The hierarchical regression was completed, as guided by Field (2009), to examine the Total Anxiety score from the *BISCUIT*. The covariate of age, as identified previously, was entered into step 1 of the regression model. For step 2, the predictor variables (Cognitive DQ, Adaptive DQ, and Autism Symptomology) were entered after being standardized. The previously formed two-way interactions were then entered into step 3 of the regressions model and finally the three-way interaction was then entered in the fourth and final step of the regression.

The final model accounted for a significant proportion of the variance in Total Anxiety scores [R^2 = .44, F (8, 2165) = 215.12, p < .001]; however, the final model including the interaction term did not differ much from the previous model [ΔR^2 = .002, ΔF (1, 2157) = 6.29, p < .05]. Although this finding was statistically significant, the test was ultimately overpowered and the change in R^2 exhibited a negligible effect size (f^2 = .004; Cohen, 1988). Step 3 of the model was found to account for a significant increase in the amount of variance in Total Anxiety scores over the previous model [ΔR^2 = .01, ΔF (3, 2158) = 13.48, p < .001]. These results show that as anxiety symptomology increases with cognitive or adaptive DQ, symptoms of anxiety are shown to increase. Additionally, the change in R^2 exhibited a small effect size (f^2 = .02) over

step 2 of the regression. Finally, Step 2 was found to account for a large part of the variance in Total Anxiety scores over the model containing only the covariate of age [ΔR^2 = .43, ΔF (3, 2161) = 538.76, p < .001] exhibiting a large effect size (f^2 = .75) over step 1 of the regression. Examination of these results show that as autism symptomology, cognitive DQ, or Adaptive DQ increase symptoms of anxiety are shown to increase, although at different rates. A summary of the hierarchical regression analyses are available in Table 3.

Table 3
Hierarchical regression analysis (n = 2166)

		ΔR^2	Cohen's f^2	b	$SE\ b$	β
Step 1						
	Constant			0.05	0.42	
	Age			0.06	0.02	0.08***
Step 2		0.43***	.75			
	Constant			-5.87	0.65	
	Age			0.05	0.01	0.07***
	Autism			0.13	0.00	0.71***
	Cognitive DQ			0.03	0.01	0.10***
	Adaptive DQ			0.02	0.01	0.06**
Step 3		0.01***	.02			
	Constant			-6.03	0.65	
	Age			0.05	0.01	0.06***
	Autism			0.15	0.00	0.78***
	Cognitive DQ			0.03	0.01	0.10***
	Adaptive DQ			0.02	0.01	0.06**
	Cog. x Aut.Symp.			0.10	0.07	0.03
	Adap. x Aut.Symp.			0.20	0.07	0.07*
	Cog. x Adap.			-0.11	0.06	-0.04
Step 4		.002*	.004			
	Constant			-5.34	0.70	
	Age			0.05	0.01	0.07***
	Autism			0.15	0.00	0.78***
	Cognitive DQ			0.03	0.01	0.09***
	Adaptive DQ			0.01	0.01	0.04*
	Cog. x Aut. Symp.			0.02	0.08	0.01
	Adap. x Aut. Symp.			0.12	0.08	0.04
	Cog. x Adap.			-0.13	0.06	-0.04*
	3-way Interaction			-0.13	0.05	-0.07*

Note. R^2 =.007 for Step 1. *p<.05, **p<.01, ***p<.001.

Post-hoc analyses of Adaptive and Cognitive DQ by subdomains was utilized for subsequent moderation analyses to determine if different components of adaptive or cognitive skills better predict Total Anxiety scores. The moderation analyses utilized the same methods mentioned above with the respective subdomains being placed in step 3 as an interaction term with autism symptomology. Results of these analyses produced significant results but ultimately with negligible effect sizes and are summarized in Table 4.

Table 4
Follow-up regression analysis (n =2166)

		Step 3			
Domain	Subdomain	ΔR^2	ΔF	p	Cohen's f^2
Cognitive					
	Reasoning & Academic Skills	.002	5.40	.02	.004
	Perceptual Discrimination/ Conceptual Development	.002	7.11	.01	.004
	Attention & Memory	.001	4.133	.04	.002
Adaptive			.		
	Personal Responsibility	.002	4.50	.03	.004
	Self-Care	.005	19.16	< .001	.009

Note. All models have age in step1; cognitive, adaptive, and autism symptomology in step 2; and the subdomain interaction in step 3.

Discussion

This study confirmed the relationship between autism symptomology with anxiety scores on the combined Total Anxiety scale utilized which has been evidenced in other studies by the significantly higher prevalence rate of anxiety in an ASD population when compared to typically developing individuals (de Bruin et al., 2007; Gjevik et al., 2011; Morgan, et al., 2003; Simonoff et al., 2008; White, Oswald, et al., 2009). In the simple regression completed with the confimation sample, autism symptomology was able to account for over 50% of the variance in Total Anxiety scores. Although Cognitive and Adaptive DQ's were also found to be significantly related with Total Anxiety scores, they accounted for a considerably smaller amount of the variance (13% and 8%, respectively), with the interaction between the two variables accounting for 12% of the variance. As can be seen, cognitive abilities were more predictive of Total Anxiety scores alone than the Adaptive DQ or interaction term.

The moderation analysis proved to be interesting and the final model accounted for a large portion of the variance in Total Anxiety scores (44%), although this was smaller than the amount of variance accounted for by autism symptomology alone in the confirmation sample. The 3-way interaction term (cognitive DQ, Adaptive DQ, and autism symptomology), although significant, was not shown to have a large effect on the overall model with an increase in accounted variance of less than 1%. The two way interaction terms entered into step two were also significant and increased the predicitve validity of the model by approximately 1% of the accounted variance in Total Anxiety scores. This increase was shown to have a small effect size according to the standards of Cohen's f^2; however, the total model was shown to have a large effect size, $f^2 = .80$ (Cohen, 1988). Partial regression plots of the three individual variables and the interaction term depict the relative strength of the relationship between autism symptomology

with Total Anxiety (Figure 5) versus Cognitive DQ (Figure 6), Adaptive DQ (Figure 7), and the interaction term (Figure 8) which actually showed a negative trend.

Additional analyses of the Cognitive and Adaptive subdomains of the *BDI-2* to determine the source of accounted variance produced results that were all statistically significant; however, the increase in accounted variance was minimal (all less than 1%) with similarly negligible effect sizes (all f^2 less than .01). Of the five subdomains examined, the Adaptive Self-Care subdomain had the largest effect with .5% of the variance accounted for over the previous step of the model. The moderation analysis indicates that autism symptomology, although statistically significant, does not have a significant moderating effect on the relationship between cognitive and adaptive abilities with anxiety. While the model accounts for a large percentage of the variance, this is mostly due to the correlations between autism symptomology and Total Anxiety scores which appears to account for a larger portion of the variance than Cognitive or Adpative DQ alone.

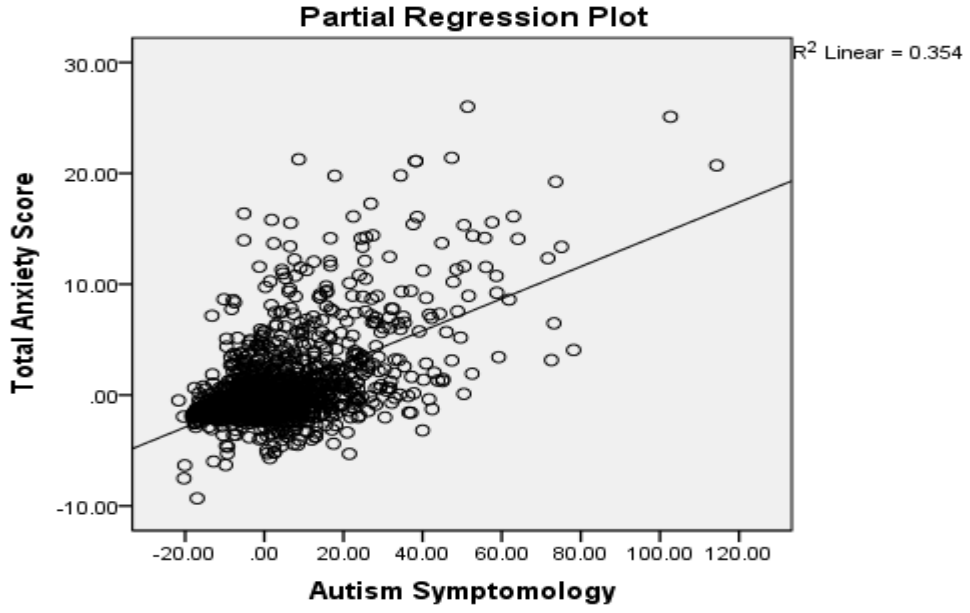

Figure 5 Autism Symptomology Partial Regression

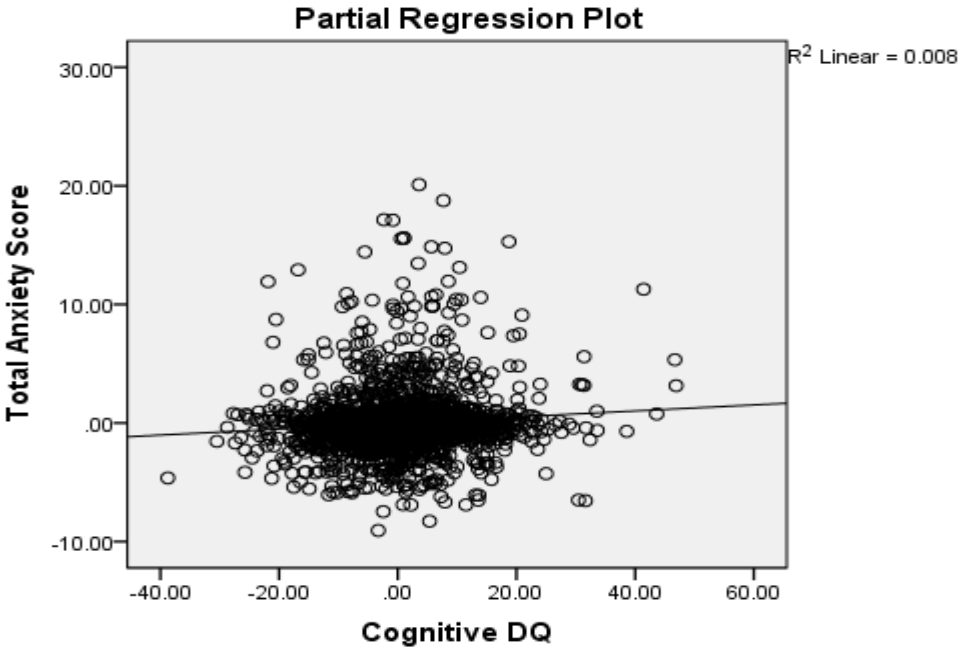

Figure 6 Cognitive DQ Partial Regression

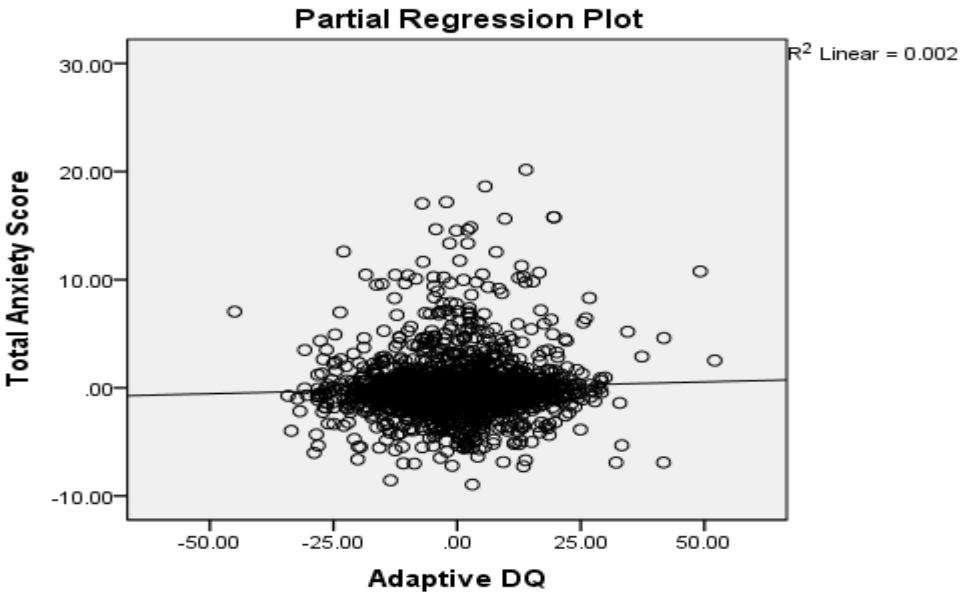

Figure 7 Adaptive DQ Partial Regression

Figure 8 3-Way Interaction Partial Regression

Several hypotheses were formed prior to completing the current study according to previous research findings in the area of autism and anxiety. It was confirmed that there was a significant positive relationship between autism symptomology and Total Anxiety scores as hypothesized showing that as autism symptomology increased, symptoms of anxiety were also found to increase. This finding is consistent with results from previous studies in which researchers showed that anxiety symptoms in those with ASD increased in relation to the severity of autism symptoms (Kanne et al., 2009; Sukhodolsky et al., 2008) and in contrast to other researchers which found no such relation (Simonoff et al., 2008).

Additionally it was confirmed that there was a significant positive relationship between Cognitive DQ and Total Anxiety scores, as hypothesized, confirming that as Cognitive DQ increased symptoms of anxiety were also found to increase. These results confirm findings of previous researchers (Gadow et al., 2008; Mayes, Calhoun, Murray, & Zahid, 2011;

Sukhodolsky et al., 2008; Weisbrot et al., 2005) and shows a different pattern of results than those seen in studies of children without an ASD (Davis, Ollendick, & Nebel-Schwalm, 2008; Zimet et al., 1994). It can be seen from these findings that different patterns of anxiety emerge based on the presence or absence of autism symptoms. Results regarding Adaptive DQ and Total Anxiety, although significant, were positively correlated, therefore disconfirming the original hypothesis. These results show that as adaptive skills increased anxiety also increased and accentuates the importance of continued research in this area.

The moderating effect of autism symptomology on the relationship between Cognitive and Adaptive DQ individually with Total Anxiety, although statistically significant, also had very negligible effect sizes ($f^2 < .02$) showing that although autism symptomology moderated those relationships the effect was insignificant except when combined into step 3 of the model in which case the effect size was shown to be small ($f^2 = .02$). Similar results were found for the 3-way interaction as well with negligible effect sizes. This is likely due to the earlier observations that autism symptomology likely accounts for a large percentage of the variance that Adaptive and Cognitive DQ's accounted for, therefore not increasing the accounted variance by a significant amount.

These findings are not completely unexpected. Researchers have shown that autism symptomology, age, and verbal IQ are strong predictors of anxiety (Mayes, Calhoun, Murray, & Zahid, 2011) in an autistic population; however, such studies have not examined the strong overlap and shared variance between autism symptomology and cognitive abilities. While each is a significant predictor within itself, examining the increase in accounted variance of each variable is important in determining the incremental validity of each as a predictor of anxiety symptoms. This is especially important to consider when developing tools for assessing anxiety

and other comrobid conditions in those with an ASD. Furthermore, it is also important to keep in mind that those with subthreshold symptoms of ASD may fall into the same pattern of results which could contribute to an increased likelihood of anxiety symptoms and severity, although this would be expected to be less of a contributor as those with clinically significant symptoms of ASD.

The current study was not without limitations. First, all of the participants in the study were part of the Early Steps program for assessment of developmental disabilities. It is possible that the sample of those with ASD do not include a representative sample of those with milder behavioral phenotypes for both anxiety and ASD symptoms and due to their age liekly only include those with the most severe behavioral presentations. Those with, for example, Asperger's may not be identified at such a young age as having developmental problems and could be excluded from the current sample. In addition, the use of the combined subscales of the *BISCUIT* (Anxiety/Repetitive and Avoidance Behavior) has not specifically been validated as a measure of anxiety and may be more representative of anxiety symptoms of those with ASDs and not typically developing children. The scale also may show an increase in those with ASDs due to the core features of autism including repetive behaviors as well as social deficits which could include avoidant behavior. This could create higher scores on the Total Anxiety scale for those with higher ASD symptomology which may not be related specifically to the construct of anxiety. Additionally, examination of the sample means show an average Cognitive and Adaptive DQ approximately a standard deviation below the national norms with a wide range. Future studies may look to find a sample that is more representative of the national population, although such trends are not uncommon in a clinical sample including individuals with an ASD.

Future research should include more of a longitudinal approach to determine the developmental trajectories of these comorbid disorders while accounting for the influence of other important variables. Assessments of anxiety specific to those with an ASD diagnosis are scarce and little attention has been paid to this area of research. Validation of the combined scale as a measure of anxiety should be completed in future resesrch showing incremental and construct validity (through convergent and discriminant validity). Delineation of the scale from other related constructs (depression and other internalizing disorders) should be examined to better understand how these construct are related in an ASD population and to assure anxiety is the only construct being measure by the assessment scale. Although large effects were not seen between the interactions included in this study, additional studies should be completed assessing this model in other age groups and between diagnostic groups, investigating ASD from a categorical perspective. It would also be important to complete similar research using a lognitudinal method to examine the development of anxiety and its relationship to autism symptoms. With the relationship between higher cognitive abilities and anxiety, a possible early phenotype may be apparent of those with Asperger's Disorder. Finally, researchers have shown that cognitive abilities and IQ are not stable at such a young age and further examination after the key years of cognitive development may reveal relationships which were unclear during this study.

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Appendix

Bobby Jindal
GOVERNOR

Alan Levine
SECRETARY

State of Louisiana
Department of Health and Hospitals
Office of the Secretary

Louisiana Department of Health and Hospitals
Institutional Review Board
Notice of Review

Project Title: An Early Autism Screening Initiative within a State Early Intervention Program: Description of Results and Comparison of Two Screening Instruments

Principal Investigator: Cheryl L. Knight, Ph.D., BCBA-D
Coordinator of Autism Initiatives
Office for Citizens with Developmental Disabilities

Date: June 10, 2010

X 1. In accordance with Louisiana Department of Health and Hospitals Institutional Review Board Guidelines and Practices the above research has been reviewed and has been APPROVED by DHH IRB on this date. The research is subject to continuing review and any conditions listed in the comments section below.

_ 2. In accordance with Louisiana Department of Health and Hospitals Institutional Review Board Guidelines and Practices the above research has been reviewed and found to be DEFICIENT for reasons listed in comments section below.

_3. In accordance with Louisiana Department of Health and Hospitals Institutional Review Board Guidelines and Practices the above research has been reviewed and APPROVED via Expedited Review procedures.

_4. In accordance with Louisiana Department of Health and Hospitals Institutional Review Board Guideline and Practice the above research has been reviewed and found to be EXEMP from further IRB review.

Comments:
We are continuing to request that any emergent problems or changes to protocol that may affect the status of this project be reported to this office and that no such changes be instituted prior to DHH IRB review, except where necessary in order to eliminate immediate hazards.

Sheila Bridgewater
Interim IRB Chairperson

Audrey Pugh
Program Manager
Bureau of Policy Research and Health System Analysis

Cc: Mary Johnson

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This application must accompany all research proposals submitted for review by the DHH IRB. All items must be either completed or indicated as not applicable.

1. Title of Research Proposal: An Early Autism Screening Initiative within a State Early Intervention Program: Description of Results and Comparison of Two Screening Instruments

2. Principal Investigator: Cheryl L. Knight, Ph.D
Address: Office for Citizens with Developmental Disabilities
628 North 4th St
PO Box 3117 – Bin#21
Baton Rouge, LA 70821
Phone: (225)342-3106
Affiliations: Office for Citizens with Developmental Disabilities
Education/Qualifications (attach vita): Ph.D., Clinical Psychologist; CV attached.

3. Co-Investigator: Johnny L. Matson, Ph.D.
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Louisiana State University
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Affiliations and Education/Qualifications (attach vita if applicable): Ph.D.; Professor; Psychologist; CV attached.

Co-Investigator: Brenda Barron Sharp, M.A.
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Affiliations and Education/Qualifications (attach vita if applicable): M.A.; CCC-SLP; CV attached

Co-Investigator: Brandi Smiroldo, Ph.D.
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PO Box 3117 – Bin#21
Baton Rouge, LA 70821
Phone: (225)342-0095
Affiliations and Education/Qualifications (attach vita if applicable): Ph.D., Clinical Psychologist; CV attached

4. University Faculty Sponsor (complete if researcher is a student): Not Applicable
5. Approximate dates research is to be conducted: (ex. xx/xx/xxx)

*Begin date: 07/28/2008
End date: 06/30/2012

- NOTE: This is a request to extend an IRB with the original Begin Date of 7/28/2008 and End Date of 06/30/2010. The current request is to extend the project until 06/30/2012, and includes some changes in Investigators, an instruments, and data management procedures.

6. DHH Facilities and location where research is to be conducted:
   a. Administrative location for coordinating all research activities, which will consist solely of the extraction and analyses of de-identified information from the records of children served across the State by DHH/OCDD's EarlySteps:

      Office for Citizens with Developmental Disabilities
      628 North 4th St
      Baton Rouge, LA 70821

   b. Additional research analyses, following de-identification of data:

      324 Audubon Hall
      Department of Psychology
      Louisiana State University
      Baton Rouge, LA 70803

7. Requirements of research project from DHH:
   a. number of subjects/time required:

      The proposed research consists of the analysis of information extracted from the records of approximately 6000 children, ages 18-36 months, who receive Initial, Annual or Six-Month Reviews through EarlySteps. This number is an estimate based on enrollment from the fiscal year 07/08. This research project will not require any additional time from the children and families served by EarlySteps.

   b. program support personnel/space/equipment:

      Additional administrative time (e.g., project communication/coordination; procedures; tracking and monitoring; electronic and hardcopy data de-identification and management; etc.), training time, report-writing, dissemination is estimated at 0.5 FTE for an additional two years, to be incorporated within current TO (e.g., no new positions).

      Total amount of program support from administrative assistant personnel is estimated as requiring only occasional time with printing-copying, training material assembly, some assistance with monitoring and tracking; and data de-identification and monitoring, which will not exceed current resources.

      No additional office space is required for completing this research. Administrative space, regional team meeting space, or other space requirements are adequately addressed by existing resources.

      No additional equipment is required for completing this research. Existing computer equipment, software, desk/office space and set-up and related materials are adequate for the needs of this project and are otherwise contained within the scope of current operations.

   c. other needs (specify): None.

8. Attach Abstract of the Research Proposal. Attached.
9. Attach brief description of potential benefits of this research. Attached
10. Attach brief description of potential risks of physical or psychological harm or discomfort to participant (if any). Attached
11. Attach brief description of procedures to be used to establish informed consent of research participants (if applicable). Attach Informed Consent Form immediately after this page. If a waiver of any aspects of informed consent is requested, a statement of justification is required here. Detailed explanation that research consists solely of extraction and analysis of de-identified data from clients' clinical records is attached.
12. Will client personal-identifying information (e.g., name, address, Medicaid recipient number, Social Security Number, phone number) be collected in the course of this research project? NO; If yes, attach explanation why it is necessary to identify the clients.

RESEARCHER'S PLEDGE

I am applying to conduct the research project entitle above at the indicated DHH facilities/programs. I agree to conduct this research in an ethical and responsible manner and as stipulated by the proposal and this application. I agree to secure the approval of the DHH IRB for any modifications to the research protocol. I understand that I have an ethical and legal responsibility not to divulge the identity of any clients or any information about them as identifiable individuals, nor will the final compilation of results of this project contain any client identification information. As soon as the project is complete, all client-identifying information collected will be destroyed. I agree to keep the DHH IRB informed periodically of the progress of the project, and I will submit a report of the final results to the IRB and facilities/programs involved.

05.26.10

Signature of Principal Investigator — Date
Cheryl L. Knight, Ph.D.

May 12, 2010

Signature of Co-Investigator — Date
Johnny L. Matson, Ph.D.

Brenda Barron Sharp 5/26/10

Signature of Co-Investigator — Date
Brenda Barron Sharp, M.A.

5/26/10

Signature of Co-Investigator — Date
Brandi Smiroldo, Ph.D.

Attachment 4
UNIVERSITY FACULTY SPONSORSHIP

Vita

Robert D. Rieske was born in Provo, Utah, in 1981. He is married with three children and has worked with youth with developmental disabilities as well as adolescents with severe mental illness in residential treatment settings. He received his Bachelor of Science degree in behavioral science from Utah Valley University in 2008. He enrolled in Louisiana State University's Clinical Psychology Doctoral Program in 2010. His current clinical and research interests are the assessment and treatment of individuals with Autism Spectrum Disorders and other developmental disabilities, with a particular emphasis in comorbid anxiety.