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EXPLORATION OF COMORBID PSYCHOPATHOLOGY SYMPTOMS IN INFANTS AND TODDLERS WITH AUTISM SPECTRUM DISORDERS

A Thesis

Submitted to the Graduate Faculty of the
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Agricultural and Mechanical College
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Master of Arts

in

The Department of Psychology

by
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ABSTRACT

One area of research in the field of Autism Spectrum Disorders (ASDs) involves efforts to better understand symptom manifestation at earlier points in an individual's life. Researchers have consistently emphasized the importance of early intervention for children with an ASD, but the determination of the most efficacious treatment approach is often established on a case-by-case basis by taking into consideration an individual's specific needs. The need for such individualized treatment approaches is accentuated by the high prevalence of comorbid psychopathology within the ASD population. The study of comorbid disorders among young children with ASDs has been hindered by a lack of measures normed for an ASD population, until recently. The development of the *Baby and Infant Screen for Children with aUtIsm Traits-Part 2* (*BISCUIT-Part 2*) assessment battery has provided clinicians with an empirically derived scale designed to assess comorbid psychopathology in young children with ASDs or atypical development. The aim of the present study was to examine the diagnostic and temporal influences on comorbid psychopathology symptoms in infants and toddlers with ASD. Participants were separated into one of three groups based on their diagnoses (i.e., Autistic Disorder [AD], Pervasive Developmental Disorder-Not Otherwise Specified [PDD-NOS], and atypical development without an ASD). Children were assessed with the *BISCUIT-Part 2* twice, with the initial and follow up assessment occurring within one of two time intervals; 4 to 8 months, or 9 to 13 months. Results from the current study indicate that children diagnosed with AD exhibit significantly less stable symptoms of comorbid psychopathology. Further, the time between initial and follow up assessments is a significant factor influencing symptom expression. Implications of these findings are discussed.

INTRODUCTION

Autism Spectrum Disorders (ASDs) are a severe and lifelong set of neurodevelopmental disorders (Rivet & Matson, 2011; Weinkauf, Zeug, Anderson, & Ala'i-Rosales, 2011). In support of the neurodevelopmental origins, McPartland, Coffman, and Pelphrey (2011) have presented research showing that irregular activity in the brain regions associated with social interactions (i.e., mirror neurons) may act as a possible genetic marker for ASDs. The presence of ASD is often recognized based upon abnormalities in three core areas: social interaction, communication skills, and restricted and repetitive behaviors (Rivet & Matson, 2011; Weinkauf, Zeug, Anderson, & Ala'i-Rosales, 2011; Xianchen, Hubbard, Fabes, & Adam, 2006). In addition to these core symptoms, concomitant psychopathology and challenging behaviors are often present (Bakken et al., 2010; Davis et al., 2011; Funabiki, Kawagishi, Uwatoko, Yoshimura, & Murai, 2011; Horovitz et al., 2011).

Traditionally, diagnosticians have disregarded formulations of comorbid psychiatric disorders for individuals with ASD (Lainhart, 1999). This diagnostic indifference has been attributed to many factors. The unique manifestation of psychopathological symptoms when an ASD is present makes differential diagnosis increasingly more difficult (Gillberg, 2010). Rutter (1968) also highlighted the relative lack of agreed upon diagnostic criteria for an ASD diagnosis as a contributing factor. However, due to the mounting evidence investigators have found supporting the presence of co-occurring conditions and their subsequent impact (Bakken et al., 2010; Gillberg, 2010; LoVullo & Matson, 2009; Matson & Minshawi, 2006; Matson & Nebel-Schwalm, 2007), the controversy regarding comorbid psychopathologies within ASDs has largely been quelled. Challenging behaviors and psychopathology have consistently been identified as occurring at high rates with ASD and are often targeted for intervention

(Barthélémy et al., 1992; Dawson, Matson, & Cherry, 1998; Holden & Gitlesen, 2007; Matson & Nebel-Schwalm, 2007; Paclawskyj, Matson, Bamburg, & Baglio 1997).

The addition of a comorbid psychiatric condition to individuals with an ASD diagnosis necessitates the need for highly individualized interventions (LoVullo & Matson, 2009). For example, while a behavioral intervention may be used to address symptoms of ASD, alternative intervention techniques (e.g., psychotropic medication) may be necessary for some comorbid disorders (LoVullo & Matson, 2009; Self, Hale, & Crumrine, 2010). Comorbid conditions can often further complicate the diagnostic process and treatment plan, which often adversely affects not only the individual, but those involved in the provision of care as well (Gray, Ansell, Baird, & Parr 2011; Matson & Minshawi, 2007; Matson & Nebel-Schwalm, 2007).

While the need for early intervention has been recognized, there are currently few existing comorbid psychopathology assessments normed for the ASD population. This dearth of appropriate assessment tools contributes to the lack of understanding researchers, clinicians, and parents have regarding co-occurring conditions in children with ASDs (Matson et al., 2010). The use of traditional assessments (i.e., assessments used in the typically developing population) to investigate and diagnose the presence of comorbid psychopathologies in individuals with ASDs is insufficient due to the additive complexity in behavioral presentation (Matson et al., 2007). Fortunately, the recent development of the *Baby and Infant Screen for Children with aUtIsm Traits-Part 2* (*BISCUIT-Part* 2) has provided an empirically validated assessment of comorbid psychopathology in young children with ASD and atypical development (Matson, Boisjoli, Hess, & Wilkins, 2011). In the current study the researcher utilized the *BISCUIT-Part 2* to examine comorbid conditions in toddlers with and without an ASD by analyzing the

diagnostic and temporal influences on symptom expression. Symptoms will be considered "stable" if they do not differ significantly between the initial and follow up assessment periods.

Symptoms of psychopathology were assessed across two administrations of the *BISCUIT-Part* 2. The same parents/caregivers of the infants and toddlers served as informants each time as part of their participation in Louisiana's EarlySteps Program. Symptom stability was compared across participants falling into one of three diagnostic categories (i.e., Autistic Disorder [AD], Pervasive Developmental Disorder – Not Otherwise Specified [PDD-NOS], and atypically developing without an ASD) in order to determine if diagnosis significantly predicts concomitant psychopathological expression. Additionally, temporal influences were examined for each diagnostic category by inspecting the time interval between initial and follow up assessment (i.e., 4 to 8 months, and 9 to 13 months). The history of ASDs, research on comorbid psychopathology, and assessment techniques for this population are reviewed, followed by details of the current study.

HISTORY OF AUTISM SPECTRUM DISORDERS

In 1943, Leo Kanner published the seminal article "Autistic Disturbances of Affective Contact." He introduced a new type of developmental disorder, which is now commonly referred to as autism. Kanner's (1943) original article contained the descriptions of 11 children (i.e., eight males and three females) ranging from 2 to 8 years of age. Each child presented with similar symptomology which failed to explicitly meet any current diagnostic criteria. Though some variation in appearance and severity was evident, the children all exhibited three core deficits, discrepancies in language use and acquisition, insistence on sameness and stereotypic behavior, and failure to relate to others and form proper social relationships. Van Krevelen (1971) and Rutter (1978) later published research replicating these three core deficits, which were popularized by Wing and Gould (1979) who referred to these deficits as the "autism triad."

The term "autism," itself, was not new to the field of psychology and was actually coined in 1908, by Swiss psychiatrist, Eugene Bleuler (as cited in Fusar-Poli & Politi, 2008). Notably, Bleuler did not introduce the term as nomenclature for a new neurodevelopmental disorder (as cited in Fusar-Poli & Politi, 2008). Instead, he used the term to label, what he considered a core feature of schizophrenia: "turning away from reality, sees life in fantastic pictures, and is founded precisely upon autistic thinking." (Bleuler, 1913, p. 874). Kanner, however, utilized the term "autism" to specifically describe an individual's inability to relate to themselves and others, which he described as "extreme autistic aloneness" (Kanner, 1943, 1944). This overlap in terminology created persistent diagnostic problems and turmoil amongst clinicians in the area of differential diagnosis.

Kanner noted in his observations that clinicians most often described the children expressing autism symptomology as being "feebleminded" or "schizophrenic." The observed

diagnostic overlap can be attributed to the childhood schizophrenia definition proposed by Despert (1938), " a disease process in which the loss of affective contact with reality is coincident with or determined by the appearance of autistic thinking and accompanied by specific phenomena of regression and dissociation." (p. 366). Kanner, however, disagreed with the diagnostic confusion and emphasized that "these characteristics form a unique syndrome, not heretofore reported, which seems to be rare enough, yet is probably more frequent than is indicated due to the paucity of observed cases" (Kanner, 1943, P. 242). In an attempt to distinguish this new neurodevelopmental disorder from childhood schizophrenia, Kanner (1944) proposed to rename it "early infantile autism."

Kanner's utilization of the term "autism" undoubtedly contributed to the general lack of diagnostic differentiation, and consequently served as the topic of much discussion in the literature of the period (Rutter, 1972a, 1978; Volkmar & Klin, 2005). During this period, clinicians freely interchanged diagnoses of childhood schizophrenia, autism, and childhood psychosis (Laufer & Gair, 1969; Rutter, 1978). Eisenburg (1966) noted the direct negative impact that terminology confusion had on the ASD population and associated research, as many clinicians and researchers struggled to distinguish autism and childhood schizophrenia (Matson & Minshawi, 2006). The lack of differential diagnosis may be further attributed to the diagnostic measures developed during this period which only sought to identify the presence of psychosis, not a particular syndrome (Prior & Bence, 1975).

Similarly, early diagnostic manuals failed to differentiate ASD from childhood schizophrenia. Childhood schizophrenia was the only official term available in the first and second editions of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association [APA], 1952, 1968) to describe a child exhibiting the newly discovered

autistic features. When AD first debuted in the World Health Organization's (WHO) *International Classification of Diseases, 8th Revision* (*ICD-8*; 1967), it appeared as "infantile autism," a sub-type of schizophrenia, and lacked any specified criteria. In the *ICD-8 Glossary and Guide*, autism was classified under "behaviour disorders of childhood" (WHO, 1974). Later, autism was later re-categorized as "psychoses with origin specific to childhood" within the *ICD-9* and subsequent *Glossary and Guide* (WHO, 1977, 1978).

The changing terminology was not without influence upon researchers. Diagnostic confusion led some researchers to suggest terminology that pointed to potential causes of the disorder. For example, Rutter and colleagues (1969) suggested the use of the provisional terminology "disintegrative psychosis" to describe the period of profound regression in children around the age of 4 that was considered distinct from both schizophrenia and infantile autism. Van Krevelen (1971) sought to utilize the term "autismus infantum," suggesting cerebral disorders as the causal agent of infantile autism.

Diagnostic Development

Following Kanner and Asperger's original works, research interests expanded exponentially and created a divide amongst researchers. Some researchers sought to differentiate ASDs from childhood schizophrenia, while others produced studies attempting to highlight their similarities. Significant research contributions by Rutter (1968, 1972b, 1978), highlighted the differences between autism and childhood schizophrenia. For example, Rutter (1978) differentiated ASD from childhood schizophrenia by suggesting ASD had a 4:1 (male to female) gender ratio, while suggesting the gender ratio in childhood schizophrenia is generally recognized as being equal. Rutter (1972b, 1978) also showed that in direct contrast with childhood schizophrenia symptoms, ASDs do not include hallucinations and delusions, but do

include impaired intellectual function when compared to typically developing peers, stability of presenting symptomology, and earlier onset. In conjunction with his efforts to distinguish ASDs from childhood schizophrenia, Rutter and colleagues (1969) also sought to identify clear diagnostic criteria to diagnose autism.

Two important distinctions would ultimately differentiate ASD and childhood schizophrenia. The identification of a period of marked "regression" and the age criteria (i.e., 13) identified by Despert (1938) in childhood schizophrenia directly contended with the clear and evident deficit from birth seen in autism (Kanner, 1943; Rutter, 1978). However, in actuality, it would be more than thirty years from the original observations before childhood schizophrenia and autism were diagnostically distinguished (Volkmar, Klin, & Cohen, 1997). This feat only occurred with the introduction of the "Pervasive Developmental Disorders" (PDDs) category, which was included in the 1980 release of *Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition* (*DSM-III*; APA). The terms "PDD" and "ASD" are used interchangeably in current research articles; however, the current paper will utilize only "ASD" henceforth (Baird, Cass, & Slonims, 2003).

The ASD category is an umbrella term encompassing the varied degrees of symptomology observed across the lifespan. Upon its introduction, ASD included five separate disorders: infantile autism, residual infantile autism, childhood onset pervasive developmental disorders, residual child-onset PDD, and atypical pervasive developmental disorder (Volkmar & Klin, 2005). For the first time, the diagnostic criteria for each ASD presented in the *DSM-III* were based on empirical research (Volkmar & Klin, 2005). *DSM-III* also employed a multi-axial diagnostic approach, simultaneously accounting for symptom severity by identifying specific criteria to correspond with each disorder (Matson & Minshawi, 2007; Volkmar & Klin, 2005).

The introduction of ASDs in the *DSM-III* encouraged researchers to explore these disorders further, and as data and knowledge grew, so did the discrepancies surrounding diagnostic criteria. Based on supporting research, the APA sought to clarify the diagnostic criteria along with other issues in the 1987 release of the *Diagnostic and Statistical Manual of Mental Disorders, 3rd edition-revised* (*DSM-III-R*). The *DSM-III-R* reduced the number of ASD diagnoses by completely removing infantile autism, residual infantile autism, and childhood onset pervasive developmental disorders, replacing them with AD and PDD-NOS (Waterhouse, Wing, Spitzer, & Siegel, 1989). A second significant change was the removal of the specified age of onset for AD (i.e., onset prior to 30 months; Waterhouse et al., 1989). Waterhouse, Wing, Spitzer, and Siegel (1989) also noted the additional establishment of diagnostic boundaries for considering comorbid diagnoses.

Despite the progression and changes mentioned, it is notable that no version of the *DSM* had yet sought to include the syndrome that Hans Asperger discovered. Only a year after Kanner's seminal 1943 article, Asperger wrote his thesis "Autistic Psychopathy in Childhood" (Asperger & Frith, 1991). Written in German, Asperger's thesis remained largely undiscovered by the scientific community until it was eventually translated into English forty-seven years later by Uta Frith. The thesis outlined the profiles of four children who expressed symptoms similar to those seen in autism, but with a later age of onset, and "pedantric patterns of speech" (Asperger & Frith, 1991). Asperger's Syndrome (AS) did not appear in a diagnostic manual until its inclusion in the *International Classification of Diseases, 10th Edition* (*ICD-10*), which was released by the WHO in 1992. In addition to AS, the *ICD-10* incorporated multiple ASDs; atypical autism, disintegrative disorder, Rhett's syndrome, and overactive disorder with mental retardation and stereotypies (Rutter, 1989). Szatmari (1992) cited the overwhelming number of

diverse clinical features and varied degrees of severity as reasoning to include such a large number of diagnoses within the ASD umbrella. The diversified presentation of clinical features within the autism spectrum was also acknowledged in the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (*DSM-IV*; APA, 1994) and *DSM-IV-Text Revision* (*DSM-IV-TR*; APA, 2000).

Presently, five disorders comprise the ASD category, AD, AS, Rhett's Disorder, Childhood Disintegrative Disorder, and PDD-NOS (APA, 1994, 2000; Lecavalier, Gadow, DeVincent, Houts, & Edwards, 2009). In addition to categorical changes, the *DSM-IV* also implemented criteria changes such as the reestablishment of an age of onset criteria for AD of 36 months (APA, 1994; Volkmar & Klin, 2005). To address the variance of symptom presentation, the *DSM-IV* task force established separate criteria for each ASD category, with the exception of PDD-NOS. The ASD diagnoses were arranged hierarchically so that AD required a minimum of six criteria for diagnosis, and an AS diagnosis was considered only after AD was ruled out and three criteria were met (Mandy, Charman, Gilmour, & Skuse, 2011). The PDD-NOS diagnosis resides at the bottom of the hierarchal model, and was included to capture individuals that failed to meet the specified criterion for a specific ASD, but still warranted an ASD diagnosis (e.g., sub-threshold symptoms, late onset; Mandy et al., 2011). The WHO (1992) collaborated with the APA's *DSM-IV* task force to ensure that the criteria they established would be in close alignment with those set forth by their release of the *ICD-10*.

Although ASDs encompass five distinct disorders, the present study will only examine AD and PDD-NOS due to limited data and insufficient diagnostic reliability within the toddler age group for the latter three ASDs (i.e., AS, Childhood Disintegrative Disorder, and Rhett's Disorder; Lord, Luyster, Guthrie, & Pickles 2012; Matson, 2007; Matson et al., 2010; Matson,

González, & Wilkins, 2009; Schopler, Reichler, Devellis, & Daly, 1980). Therefore, only AD and PDD-NOS will constitute the ASD category when mentioned henceforth (Matson et al., 2008; Worley, Matson, Mahan, Kozlowski, & Neal, 2011).

Differential Diagnosis

Defining and diagnosing ASDs has advanced considerably over time leading to a better understanding of the presenting symptoms and features. However, Baird, Slonims, and Cass (2003) suggested that differential diagnosis remains problematic due to the variance of presenting symptomology along multiple dimensions, as implied by the ASD nomenclature. The authors postulated that the variable presentation seen in ASDs is inherent and serves as a hallmark of the disorders (Baird et al. 2003). Currently clinicians mainly consult two manuals in the diagnosis of mental disorders, the *DSM-IV-TR* (APA, 2000), and the *ICD-10* (WHO, 1992). Volkmar and Klin (2005) demonstrated the similarity of the criteria levels between the two respective manuals concerning the ASD categories. In a field trial of the DSM-IV conducted by Klin, Lang, Cicchetti, and Volkmar (2000) examining the distinction between AD and non-PDD diagnoses, the researchers observed an excellent inter-rater reliability ($k = 0.95$) across all raters examined (i.e., experienced, inexperienced, psychologists, and psychiatrist). Due to the identified similarity and the popularity of use surrounding the diagnosis of ASDs in the United States, the present study will focus exclusively upon the *DSM-IV-TR*.

The *DSM-IV-TR* identifies the importance of differentiation within the ASD category, to ensure the individual's symptoms are not better accounted for by another ASD within the spectrum (APA, 2000; Ghaziuddin & Mountain-Kimchi, 2004). Researchers have shown that special considerations must be used when differentially diagnosing a younger population due to an identified overlap of symptoms with several other early childhood disorders (e.g., general developmental delays related to intellectual disability [ID] and severely delayed language; Baird

et al., 2003; Charman & Baird 2002; Lord, 1995; Van Daalen et al., 2009). As identified by Willemsen-Swinkels and Buitelaar (2002), the importance of differential diagnosis transcends the avoidance of misdiagnosis to include the identification of other imperatives such as efficacious and ethical treatments, eligibility for services, rendering of appropriate services, and prognosis. The difficulty of differential diagnosis is only exacerbated by the presence of co-occurring disorders which are frequently identified in ASDs (Baird et al., 2003; Gillberg & Billstedt, 2000; Matson et al., 2010; Schreck, Williams, & Smith, 2004).

Autistic Disorder. Within the context of the five current developmental disorders of ASD, AD includes criteria that most closely coincide with the symptomatology originally documented in Kanner's (1943) observations. A diagnosis of AD is made only if an individual is exhibiting "markedly abnormal or impaired development" in each of the three principal symptom domains as defined by the *DSM-IV-TR*: social interaction, communication, and a marked restriction in interests or activities (APA, 2000, Cheng et al., 2009; Gutierrez et al., 2009; Ketelaars et al., 2009; Lacroix et al., 2009; Matson & Sipes, 2010). The use of three distinct categories of criteria for AD remained constant in the text revisions of both the DSM-III (APA, 1987), and DSM-IV (APA, 1994). Sevin, Knight, and Braud (2007) expressed that AD is the most agreed upon and consistent disorder within the ASDs category.

Core symptoms expressed at 14 months in toddlers with AD remained relatively stable when re-assessed at 36 months of age (Landa, Holman, & Garrett-Mayer, 2007). In related research, Werner, Dawson, Munson, and Osterling (2005) reported that symptoms of AD observed by parents between 3-6 months became more obvious deficits in social behavior at 12–15 months of age. When the same children were re-assessed between the ages of 3 and 4, it was found that the symptoms had persisted (Werner, Dawson, Munson, & Osterling, 2005). Matson

and Horovitz (2010) suggested that identification of symptoms at earlier ages (i.e., 20 months) was easier when symptoms were more severe (i.e., AD versus PDD-NOS).The diagnostic clarity provided by the *DSM-IV-TR* (APA, 2000) likely contributes to the high stability observed in the early diagnosis of AD (i.e., 24 months). The finding of high stability rates for early diagnosis has been replicated across several studies (Charman et al., 2005; Cox et al. 1999; Lord 1995; Matson et al., 2008; Moore & Goodson 2003; Worley et al., 2011).

To warrant a diagnosis of AD, an individual must present with two deficits corresponding with impaired social interaction, at least one communication deficit, and at least one behavior, interest, or activity presented in a stereotypic or repetitive nature (Taheri & Perry, 2012). Additionally, atypical or delayed functioning in at least one of three areas must be observed prior to 36 months of age: "(1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play." (APA, 2000, p.75; Buitelaar, Van der Gaag, Klin, & Volkmar, 1999). A third and final criterion requires that the presenting features not be more appropriately captured by either a diagnosis of Rhett's Disorder, or Childhood Disintegrative Disorder (APA, 2000; Wulffaert, van Berckelaer-Onnes, & Scholte, 2009). Examples of the items provided for observable impairment of social interactions include but are not limited to: a) gross impairment of multiple nonverbal behaviors (e.g., eye contact), b) deficiency in emotional reciprocity, c) inability to acquire developmental or age appropriate relationships, and d) retarded spontaneity in the sharing of social interests (APA, 2000; Buitelaar et al., 1999; Hoffman, 2009; Taheri & Perry, 2012). Identified communication impairments include a) a delayed or total absence of a developed spoken language, b) inability to initiate or maintain conversations, c) the use of repetitive or stereotypic language, and d) the observed absence of developmentally appropriate spontaneous play (APA, 2000; Buitelaar et al., 1999). At

least one repetitive and stereotypic behavior, interest, or activity pattern must also be present, such symptoms include a) an abnormal focus or intense preoccupation with at least one stereotypic or repetitive interest pattern, b) insistence of adherence to fixed, nonfunctional rituals or routines, and d) a fixation with object components (APA, 2000; Buitelaar et al., 1999; Taheri & Perry, 2012).

Utilizing the criterion outlined above, Fombonne (2005) estimated a prevalence rate of AD diagnosis at 13 per 10,000. Reviews of the criterion proposed to appear in the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (*DSM-5*; proposed for publication in 2013) has indicated a combination of the impaired social interaction domain with the communication deficit domain into a singular impaired social/communicative behaviors category (APA, 2010; Kuenssberg, McKenzie, & Jones, 2011). This combination of criteria is a source of controversy, as some researchers claim that such changes will greatly reduce ASD prevalence rates (Matson et al., 2012; Taheri & Perry, 2012).

Pervasive Developmental Disorder – Not Otherwise Specified. In the absence of the specified criterion, a more sweeping parameter is provided by the *DSM-IV-TR* for the diagnosis of PDD-NOS and reads as follows: ‘‘should be used when there is a severe and pervasive impairment in the development of reciprocal social interaction associated with impairment in either verbal and nonverbal communication skills, or with the presence of stereotyped behavior, interests, and activities’’ (APA 2000, p.84). Additionally, a PDD-NOS diagnosis should only be given if the presenting symptoms are not better accounted for by other diagnoses (i.e., developmental disorder, schizophrenia, schizotypal personality disorder, or avoidant personality disorder; APA, 2000; Buitelaar et al., 1999; Karabekiroğlu & Akbaş, 2011).

A PDD-NOS diagnosis is often made in light of the atypical or sub threshold presentation of symptoms, or the failure to meet the age of onset criterion (Buitelaar et al., 1999). The PDD-NOS diagnosis is situated at the base of the hierarchical arrangement of ASDs as provided by the *DSM-IV-TR* (APA, 2000; Karabekiroğlu & Akbaş, 2011). Researchers have shown that clinicians often assign a PDD-NOS diagnosis when impairment is observed, but the child's presentation fails to meet the criterion for another diagnosis on the spectrum (APA, 2000; Buitelaar et al., 1999; Lord & Risi, 1998; Matson & Boisjoli, 2007; Tidmarsh & Volkmar, 2003).

The PDD-NOS category and especially early diagnosis is a point of much contention. Towbin (2005) mentioned specific concern surrounding the ambiguity of the current criteria and current diagnostic utilization of PDD-NOS. A recent meta-analysis investigating the stability of a PDD-NOS diagnosis utilizing the *DSM-IV-TR* criterion further highlighted researchers' concerns. Rondeau and colleagues (2011) investigated the findings of eight longitudinal studies, which included a diagnosis made prior to 36 months of age and found that only 35% of those individuals initially diagnosed retained the PDD-NOS diagnosis at a subsequent 3-year follow-up. Stone and colleagues (1999) followed 65 children who received an initial AD or PDD-NOS diagnosis at age 2. When the same children were subsequently re-evaluated at age 3, a greater diagnostic instability (i.e., no diagnosis, or change to AD diagnosis) was observed in those with PDD-NOS than in children who were diagnosed with AD (Stone et al., 1999). Similarly, Worley, Matson, Mahan, Kozlowski, and Neal (2011) examined the stability of early diagnosis in 114 toddlers and reported that 15.8% of toddler's diagnoses changed from either PDD-NOS to atypical development or vice versa.

The indistinct nature of the criteria provided for PDD-NOS has been suggested as a contributing factor to the difficulty of discerning PDD-NOS from atypical development

(Buitelaar et al., 1999; Chawarska, Klin, Paul, & Volkmar, 2007; Nebel-Schwalm & Matson, 2008; Tidmarsh & Volkmar, 2003). Stone and colleagues (1999) suggested that the greater stability observed in AD when compared to PDD-NOS may be reflective of "a more coherent or well-defined symptom cluster" (p. 224). The authors further suggested that the stability of AD is bolstered by the severity of symptoms required for diagnosis (Stone et al., 1999). In addition to relatively vague criteria, other concerns have been voiced. Siegel (1991) expressed the concern that depending on the age at which they are assessed, some deficits may not yet be clearly evident or applicable (e.g., the children may not have peer friends, or the opportunity to develop atypical speech patterns).

In a recent study of PDD-NOS using *DSM-IV-TR* criteria, Fombonne (2005) identified an incidence rate of 20.8 per 10,000. Consequently, the incidence for PDD-NOS is higher than either AD or AS. Despite the observed increase in prevalence rate, PDD-NOS is identified as being studied significantly less than any other ASD (Chakrabarti & Fombonne, 2005; Matson & Boisjoli, 2007). Recent reviews of the proposed changes in *DSM-5* have reported that the changes in criteria will increase diagnostic sensitivity and specificity. Huerta, Bishop, Duncan, Hus, and Lord, (2012) reported that the *DSM-5* is working to create a single diagnostic category of autism spectrum disorder (ASD). Under the proposed changes, the current three-domain model will be replaced by a two-domain model, the age of onset criteria will be relaxed, and additional criteria will include symptoms not previously included in *DSM-IV-TR* (e.g., sensory interests and aversions; Huerta et al., 2012).

COMORBID PSYCHOPATHOLOGY IN ASD

De Graaf, Bijl, Smith, Vollebergy, and Spijker, (2002) described the comorbidity of psychiatric disorders as "the presence, simultaneously or in sequence, of two or more disorders in a person within a certain period of time." Researchers are in agreement that "pure" disorders are relatively infrequent and that most cases involve a client meeting criteria for two or more disorders simultaneously (Goldstein, Lopez, & Puente, 2011). Within the context of the current paper, the term "comorbid" refers to the presence of an ASD and one or more concurrent psychiatric disorders.

Since the initial documentation of children with an ASD, behaviors other than the core symptoms were observed, such as inattentiveness, food selectivity, tantrums, and abnormal attention to detail (Brereton, Tonge, & Einfeld, 2006; Kanner, 1943; LoVullo & Matson, 2012). In comparison to research investigating comorbid psychopathology in other disorders, Matson and Nebel-Schwalm (2007) suggested that the same research in the ASD population is relatively absent. In addition to a general deficit of researchers investigating comorbid psychopathology in the ASD population, Angold and Egger (2004) reported that such studies are extremely rare in young children. Researchers have expressed that the variation in presentation of some disorders may be more easily identified as distinct from an ASD (e.g., depression), while others are in-line with ASD symptomology (e.g., obsessive-compulsive disorder; Matson & Nebel-Schwalm, 2007). Given this information, Matson and Nebel-Schwalm (2007) suggested a cautionary approach to the diagnosis of comorbid psychopathology.

Current Conceptualization

Until recently researchers expressed disagreement concerning the presence of comorbid psychopathology, and the validity of comorbid diagnoses in ASDs. The American Academy of

Child and Adolescent Psychiatry (1999) suggested that the presence of emotional problems and challenging behaviors within the ASD population may not warrant additional diagnosis, instead being better expressed as symptom clusters related to ASD. Hess, Matson, and Dixon (2010) indicated that much of the disagreement surrounding comorbid diagnosis has stemmed from a dearth of assessments available for the identification of comorbid disorders. While measures of comorbid psychopathology are relatively common for the general population, few are normed for an ASD population (Matson et al., 2010). The variation of both severity and functional abilities within the ASD population impacts the presentation of psychiatric symptoms, thus increasing the difficulty of accurate assessment and subsequent differential diagnosis (Gillberg & Billstedt, 2000; Matson & Rivet, 2008; Tsai, 1996).

Researchers targeting children with ASDs have consistently replicated results showing higher occurrence rates of comorbid disorders when compared to their typically developing peers (Gillberg & Billstedt, 2000; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Schreck et al., 2004). For ASD, estimates of the psychiatric comorbidity rate have ranged from 35% (Morgan, Roy, & Chance, 2003) to 70% (Simonoff et al., 2008). A review of current literature reveals multiple studies highlighting the symptomology overlap between ASDs and comorbid disorders (Attention-Deficit/Hyperactivity Disorder; ADHD), as well as psychiatric symptoms such as depression or anxiety (Ghaziuddin, Ghaziuddin, & Greden,, 2002; Hallett, Ronald, & Happe, 2009; Simonoff et al., 2008). Despite the growing interest in comorbid psychopathology in ASD, much of the research is limited to older children, adolescents, and adults. Matson and colleagues (2010) recognized the sparse knowledge of comorbid psychopathology symptoms within the infantile ASD population, while citing the lack of valid assessments as the contributing factor.

The observed high prevalence rate of comorbid symptoms has been suggested to be further influenced by the presence of another disorder found to commonly co-occur with ASD, intellectual disability (Matson & Shoemaker, 2009). Researchers have shown that 50-75% of the ASD population also meet criteria for a concurrent diagnosis of intellectual disability (Matson & Shoemaker, 2009; Rutter & Schopler, 1987; Wing, 1981; Wing & Gould 1979). The overlap between ASD and intellectual disability is critical as both populations are associated with increased risk for comorbid disorders (Borthwick-Duffy & Eyman, 1990; Bregman, 1991; LoVullo & Matson, 2009; Matson & Shoemaker, 2009). Trillingsgaard, Sørensen, Nemec, and Jørgensen (2005) identified that the difficulty of early ASD diagnosis is compounded by the high co-occurrence of intellectual disability and the symptomology overlap with other developmental disorders (e.g., impaired communication and social interaction). Attempts to identify additional markers that may aid in the differentiation of developmental disorders have not been fruitful. Researchers attempting to identify factors such as age, sex, or intelligence quotient as significant predisposing factors for developing a comorbid disorder in children with ASD have been unsuccessful (Brereton et al., 2006; Worley & Matson, 2011).

The development of interventions for ASD has advanced despite the remaining difficulty of diagnostic differentiation. While no identified treatment is unanimously considered the most effective intervention for ASDs, there has been a steady increase in the popularity and perceived benefit of early intervention for ASDs (Hwang, Hughes, 2002; Rogers, 1996; Tanguay, 2000). A growing body of research has linked the early identification and diagnosis of ASDs and comorbid psychopathologies with increased rates of success surrounding the implementation of early intensive intervention programs (Ben-Itzchak, Lahat, Burgin, & Zachor, 2008; Eaves & Ho, 2004; Matson & Smith, 2008; McEachin, Smith, & Lovaas, 1993). Specifically, the use of

early intensive behavioral intervention (EIBI) for the improvement of various functioning domains within toddlers with ASD has been suggested (Granpeesheh, Tarbox, & Dixon, 2009; Reichow, 2012; Worley et al., 2011).

EIBI has been touted as critical for the improvement of an individuals' long-term prognosis following replication in multiple studies (Lord, 1995; Matson & Smith, 2008; Mays & Gillon, 1993; Prizant & Wetherby, 1988; Reichow, 2012). In an initial study investigating the impact of EIBI, Lovaas (1987) reported a success rate of 47% for those children who received EIBI (i.e., intelligence quotient >85 and unassisted placement in a general education setting). Since Lovaas's (1987) exploratory study, a growing body of research supporting EIBI's efficacy has accumulated. In a review of five separate meta-analyses investigating the efficacy of EIBI, Reichow (2012) found that on average, the effects produced were strong and robust. However, the research also highlighted the fact that no treatment to date has been effective for every child with ASD (Reichow, 2012). The variance seen in treatment effectiveness can no doubt be attributed to a multitude of factors. Wallace and Rogers (2010) identified the individualization of treatment as a key factor in the development of effective interventions. Additionally, the presence of comorbid psychopathological conditions has been identified as a significant factor that would necessitate an individualized treatment regimen (Bakken et al., 2010; Gillberg, 2010; LoVullo & Matson, 2009).

Despite such emphasis and interest in early intervention, there has been little research attempting to advance our understanding of ASDs and comorbid psychopathology symptom manifestation in infants (DeGiacomo & Fombonne, 1998; Howlin & Moore, 1997; Matson et al., 2010; Siperstein & Volkmar, 2004). The importance of the early and accurate identification of comorbid psychopathologies and ASDs is inherent if early intervention programs are to be

successful ventures (Hess, Matson, & Dixon, 2010). The necessity of accurate diagnosis becomes especially important when comorbid disorders are present, as this often creates a need for highly specialized intervention centered upon the individual's unique presentation (Joshi et al., 2010). Researchers have highlighted the necessity of identifying psychopathologies in children with ASDs based upon the perceived benefits of the multiple treatment options available to include: educational, pharmacological, social interventions, and psychological treatments (Birmaher, Quintana, & Greenville, 1988; Ghaziuddin, 2002; Howlin, 1998).

Common Comorbid Symptoms in ASD

Researchers examining comorbid psychopathology in children with ASD have shown increased occurrence rates of both internal and external symptoms associated with multiple psychopathological disorders (e.g., ADHD, anxiety disorder, conduct disorder; Hallett, Ronald, & Happe, 2009; Hayashida, Anderson, Paparella, Freeman, & Forness, 2010; Simonoff et al., 2008; Skuse et al., 2009). The increased occurrence of anxiety symptoms in ASD populations in comparison to both typically and atypically developing populations is well founded (Farrugia & Hudson, 2006; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Russell & Sofronoff, 2005). Evans, Canavera, Kleinpetere, Maccubbin, and Taga (2005) determined that children with ASDs exhibit significantly higher levels of fears and phobias than those with Down syndrome (DS), mental age peers, and chronological peers. The researchers also identified a positive correlation between the presence of fears and phobias and increased hyperactivity, impulsivity, and conduct behaviors in children with ASD (Evans, Canavera, Kleinpetere, Maccubbin, & Taga, 2005). Muris, Steerneman, Merckelbach, Holdrinet, and Meesters (1998) expressed the importance of identifying and treating anxiety symptoms due to the impairment of daily functioning and distress these symptoms cause. While researching anxiety symptoms in children with ASD, Chang, Quan, and Wood (2012) observed a strong positive correlation between level of social

functioning impairment, and the severity of symptoms commonly associated with social anxiety disorder. Matson, Mahan, Sipes, and Kozlowski (2010) suggested that the extreme variation in comorbid symptoms can be generally combined into five major categories: tantrum/conduct behavior, inattention/impulsivity, anxiety/repetitive behavior, avoidance behavior, and eating/sleeping problems.

Tantrum/Conduct Behavior. The comorbid incidence of tantrum and conduct behaviors within the ASD population is acknowledged as a contributing factor associated with a poor prognosis due to its hindrance of intervention strategies (Matson et al., 2010). Researchers comparing rates of conduct behavior in typically developing individuals and those with an ASD have identified significantly higher rates of aggression and tantrum behaviors within the ASD population (Horner, Carr, Strain, Todd, & Reed, 2002). Dominick, Davis, Lainhart, Tager-Flusberg, and Folstein (2006) reported that 70% of the children with autism included in their study had experienced severe tantrums. Additionally, the authors reported that onset of tantrums occurred before 3 years of age in half of the participants. Elsewhere, researchers have found tantrum behaviors to occur more frequently in children with ASD than their peers diagnosed with intellectual disability (Ando & Yoshimura, 1979). Utilizing the *BISCUIT-Part* 3 Matson and colleagues (2010) observed an increase in the endorsement for all challenging behaviors (e.g., Aggressive and Destructive Behavior, Stereotypies, and SIB) when symptoms of comorbid psychopathology were also present. Robb (2010) suggested that symptoms linked to irritability such as temper tantrums and aggression are major predisposing factors leading to problems in multiple environments (e.g., school, home), and that these behaviors often result in referrals for evaluation and treatment.

Conduct disorder and challenging behaviors have been identified as behavior which violates societal norms or the basic rights of others, or are a grave violation of established rules such as aggression, property destruction, and theft (APA, 2000; Essau & Anastassiou-Hadjicharalambous, 2011; Lahey & Waldman, 2012; Singh, Lancioni, Winton, & Singh, 2011). Tantrums are often identified as challenging behaviors that encompass a wide-ranging continuum of severity and form. Daniels, Mandleco, and Luthy (2012) defined temper tantrums as extreme and episodic frustration or anger. Tantrum behaviors often include screaming, crying, hitting, and throwing items (Davidson, 2006; Potegal & Davidson, 2003). Matson and Neal (2009) identified aggression, non-adherence, property destruction, and self-injury as challenging behaviors commonly observed in individuals with ASD. These behaviors often become a clear deviation from the norm when the child first enters a scholastic setting (Pringle, Colpe, Blumberg, Avila, & Kogan, 2012). This context allows for the direct comparison to peers, highlighting the differentiation and is often the reasoning for treatment referrals, and subsequent screening (Wiggins, Baio, & Rice, 2006). Prior to any diagnosis, it is important to differentiate the hypothesized cause surrounding the presentation of tantrum and conduct behaviors (Didden et al., 2012). For example, behaviors such as head banging could be elicited due to the experienced discomfort stemming from an undiagnosed medical condition, instead of an ASD. Similarly, in a study examining the presentation of conduct behavior, Evans and colleagues (2005) found a strong positive correlation between the presence of fears and phobias and conduct behaviors.

Inattention/Impulsivity. Several studies have revealed that in comparison to their typically developing peers, children diagnosed with ASDs exhibit symptoms associated with Attention-Deficit/Hyperactivity Disorder (ADHD) at a much higher rate (Brereton et al., 2006;

Kim et al., 2000; Lee & Ousley 2006; Leyfer et al., 2006; Simonoff et al., 2008). Lee and Ousley (2006) identified significantly higher hyperactivity-impulsivity scores in children and adolescents with autism than their peers regardless of ASD type. The presence of ADHD is commonly associated with impaired social and communication skills, both of which are symptoms commonly seen in children with ASDs (Holden & Gitlesen, 2006; Matson & Wilkins, 2008; McConaughy, Volpe, Antshel, Gordon, & Eiraldi, 2011; Schreck &Williams, 2006; Selfe, 2002; Sibley, Evans, & Serpell, 2010). Despite supportive research, a comorbid diagnosis of ADHD when an ASD is present is currently prohibited by the *DSM-IV-TR* (APA, 2000; Willcutt et al., 2012). Regardless, intervention remains crucial due to the observed impairment of individuals interactions in multiple settings (e.g., home and school) associated with symptoms of hyperactivity and impulsivity (Loveland & Tunali-Kotoski, 2005).

Willcutt and colleagues (2012) identified three different diagnoses associated with inattention and impulsivity; ADHD, predominantly inattentive type, ADHD, predominantly hyperactive-impulsive type, and ADHD, combined type. In order to meet criteria for any of the above ADHD types, a child must present with symptoms that are considered to be maladaptive and different from their peers for a period of at least six months (Willcutt et al., 2012). This qualifier is also accompanied by an age criterion indicating that some impairment be present prior to the child's seventh birthday (APA, 2000; Sibley et al., 2012, Willcutt et al., 2012). Additional criteria requires that the behaviors occur in more than one setting (e.g., at home and at school), cause clinically significant impairment in social, academic, or occupational functioning, and they must not occur in-conjunction with a psychotic disorder (e.g., Schizophrenia), or be better accounted for by another mental disorder (e.g., mood disorder, anxiety disorder; APA, 2000; Sibley et al., 2012).

Avoidance Behavior. The active avoidance of social situations, events, or places, is a broad description of the behavior commonly observed within the ASD population. Schleismann and Gillis (2011) identified that avoidance behaviors in children with ASD can be both physical (e.g., hiding, running away) and verbal (e.g., negative verbalizations). Escape behaviors such as the examples given above impede both intervention and caregiving. Similarly, escape maintained behaviors driven by the social aversion commonly witnessed in the ASD population negatively impact the areas of education, and employment (Kemp & Carr 1995; Moore, Yufang, McGrath, & Powell, 2005; Reese, Richman, Belmont, & Morse, 2005).

Multiple factors have been identified as influencing avoidant behaviors. One comparative analysis suggested that disruptive/avoidant behaviors by individuals with an ASD are maintained by factors of both attention and escape (Reese, Richman, Belmont, & Morse, 2005). When discussing their results, the authors postulated that gender may operate as an influencing factor upon behavior function (Reese et al., 2005). Matson and Hess (2010) found that when compared to a PDD-NOS and a control group, children with AD evinced significantly greater avoidance behaviors. The increased prevalence of avoidant behaviors in children with ASD is compounded by an observed increase in the severity of these behaviors. Utilizing the *BISCUIT-Part 2* to assess the severity of avoidance behavior in a sample of 309 infants and toddlers with an ASD diagnosis, Matson, Boisjoli, and Wilkins (2007) found 8.7% exhibiting moderate impairment and 6.1% exhibiting severe impairment. The presence of avoidance behaviors has also been linked to the presence of other comorbid symptoms. Schleismann and Gillis (2011) found an increase in avoidance behaviors when both ASD and symptoms of an anxiety disorder were present. Other researchers have found that when compared to both typically developing and developmentally delayed peers, children with ASDs experience

increased rates of fears and phobias. These researchers postulated that the elevated experience of fear and phobias may be directly related to the presentation of avoidance behaviors (Evans, Canavera, Kleinpeter, Maccubbin, & Taga, 2005; Matson & Love, 1990).

Anxiety/Repetitive Behavior. The presence of restricted and repetitive behaviors has been consistently recognized across the development and evolution of the ASD category. Kanner (1943) described the behaviors he observed as repetitious, high in frequency, and seemingly oriented towards preservation of environment. Despite being a core symptom in ASDs, Lewis and Bodfish (1998) acknowledged the relative lack of research being conducted on restricted and repetitive behaviors. Bodfish, Symons, Parker, and Lewis (2000) hypothesized that the relative ambiguity of terminology being utilized as descriptors for restricted and repetitive behaviors has acted as a source of hindrance upon further development and advancement within the area. The present categorization of restricted and repetitive behaviors provided by the *DSM-IV-TR* has been identified as being a source of further hindrance upon the research due to the broad range of behaviors it encompasses. To address this broad range of behaviors, multiple researchers have proposed a mixed categorization approach (Bodfish et al., 2000; Cuccaro et al., 2003; Papageorgiou, Georgiades, & Mayreas, 2008; Szatmari et al., 2006). Such a mixed categorization has been suggested to be implemented in a mock hierarchical fashion in an effort to distinguish between what researchers identified as "high order" behaviors (i.e., compulsive/impulsive behaviors) and "low-order" behaviors (e.g., repetitive speech, and sensory/motor behaviors; Cuccaro et al,.2003; Szatmari et al., 2006).

Hand flapping, body rocking, and the repetitive use of words or phrases are examples of observed stereotypies in children with ASDs (O'Reilly et al., 2010). These challenging behaviors often become identified as priorities for treatment due to the negative impact they have on the

individual's social encounters (Lam & Aman, 2007), familial life (Gordon, 2000), and education (Sigafoos, Arthur, & O'Reilly, 2003; Varni, Lovaas, Koegel, & Everett, 1979).When compared to other atypically developing populations, there is an significant increase in the prevalence of stereotypic restricted and repetitive behaviors within the ASD population (Green et al., 2006; Lord & Pickles, 1996; Szatmari, Bartolucci, & Bremner, 1989; Turner, 1999). Researchers have expressed the importance of investigating symptoms of restricted and repetitive behaviors due to the negative impact they have upon a child's prognosis due to their inherent complication of their behavioral repertoire and social stigmatization (Howlin, 1998; Howlin & Moss, 2012).

The presence of anxious behavior has been observed continuously since the original discovery of ASD. Kanner (1943) described it as "anxiously obsessive desire for the maintenance of sameness." (p.245). Of the ten different anxiety disorders recognized by the *DSM-IV-TR* (APA, 2000), researchers highlight the common occurrence of symptoms associated with panic disorders, obsessive-compulsive disorder, and social anxiety disorder within the ASD population (Tantum, 2000). Research by Gillott, Furniss, and Walter (2001), highlighted specific endorsement increases on the subscales of symptoms associated with separation anxiety and obsessive-compulsive disorder when an ASD was present. The presence of anxiety symptoms has also been linked to the individual's level of intellectual functioning. Adults with high functioning autism have been identified as exhibiting rates of co-morbid obsessive-compulsive disorder as high as 25% based upon *ICD-10* criteria (Russell, Mataix-Cols, Anson, & Murphy, 2005). The influence of these comorbid symptoms transcends their immediate impact and has been shown to influence other behaviors. For example, the presence of repetitive behaviors and anxiety has been shown to have a strong correlation with increased levels of aggressive and destructive behaviors (Matson, Mahan, Sipes, & Kozlowski, 2010).

Eating Problems. Among the eating disorders defined by the *DSM-IV-TR*, the current study will focus only upon symptoms associated with three due to their elevated co-occurrence in ASDs. Symptoms of Pica, rumination, and feeding disorder of infancy or early childhood have all been observed at increased rates in individuals with ASD (Matson, Hattier, & Turygin, 2012; Råstam, 2008). A diagnosis of pica is centered upon the appetite for and purposeful ingestion of non-nutritive substances (Matson et al., 2012; Tewari et al., 1995). Due to the potential influences of cultural beliefs, chemical imbalances, and social skills, researchers caution that a formal diagnosis should only be ascribed after the investigation of extraneous influences (Matson et al., 2012; Seiverling, Williams, & Sturmey, 2010). Rumination involves the regurgitation of food after a meal with the intent of re-experiencing the previously eaten items. Involuntary occurrences due to gastrointestinal illness or a presenting medical condition must be ruled out before an individual may be diagnosed with rumination (Darling, Otto, & Buckner, 2011; Lang et al., 2011; Råstam, 2008). The diagnosis of a feeding disorder during infancy or early childhood occurs specifically before six years of age and involves the failure to consume enough food across no less than one month to allow for normal growth and weight gain (Chatoor, 2002; Bruns & Thompson, 2010). In addition to symptoms associated with the above disorders, children with ASDs often exhibit behaviors that inhibit or complicate meal consumption. However, these behaviors are not diagnosable disorders, such as food selectivity (e.g., temperature, texture, liquid avoidance, and color), or food refusal (Seiverling, Williams, & Sturmey, 2010).

Across time, a trend of increasing eating and sleeping complications has been documented within the ASD population (Kanner, 1943; Richdale, 1999; Shreck et al., 2004). Pooni, Ninteman, Bryant-Waugh, Nicholls, and Mandy (2012) failed to find a significant

correlation between a group of children diagnosed with an ASD and early onset eating disorder when compared to their typically developing peers. However, the researchers observed that those with an early onset eating disorder consistently expressed elevated levels of ASD traits. Williams and Seiverling (2010) showed that of the available data and reports on feeding problems, food selectivity was the most prevalent in children with ASDs. An examination of feeding program referrals, found that 46% of children with an ASD diagnosis presented with symptoms of food selectivity (Bowers, 2002). While researchers have investigated the presence of eating disorders in children and adults, there is only minimal research investigating occurrence in an infantile ASD population. While commenting on the paucity of research of eating disorders in infants with ASDs, Pooni and colleagues (2012) urged the use of caution in attempting to supplement the knowledge gap with generalized research from an adult population.

Sleeping Problems. Another comorbid disturbance afflicting the ASD population involves sleep. Currently researchers suggest that younger children with ASD most often experience symptoms associated with bedtime resistance, sleep anxiety, parasomnias, and night waking's (Goldman, 2012; Park et al., 2012; Xianchen et al., 2006). In direct agreement with this finding, researchers analyzing parental reports have shown that the rates of reported sleep problems in individuals with an ASD (50%-80%) is significantly more prevalent then comparison group rates (9% to 50%; Allik, Larsson, & Smedje, 2006; Couturier, et al., 2005; Malow et al., 2006; Polimeni, Richdale, & Fancis, 2005; Xianchen et al., 2006). Richdale (2001) suggested that two-thirds of children in the ASD population will experience a sleep problem.

Gillberg and Coleman (1992) proposed the autistic population is one in which etiology varies tremendously based upon a multitude of factors (e.g., genetic, metabolic, and physiological influences). Researchers have highlighted multiple factors influencing sleep

disturbances. The presence of a sleep problem has been shown to correlate with the presence of other comorbid psychopathology symptoms, such as: internalizing and externalizing problems, aggression, somatization problems, and withdrawal (Park et al., 2012; Xianchen et al., 2006). Additionaly, Park and colleagues (2012) identified the significant influence of child-rearing on sleep behaviors. The authors exemplified the factorial influence by identifying an elevated risk of major depression when symptoms of depression (e.g., sleep and appetite disturbance) are combined with the presences of histrionic familial affective disorders. Sleep disturbances have been demonstrated to not only affect the child themselves negatively, but researchers have suggested that the influence also impacts parental quality of life (Lam, Hiscock, & Wake, 2003; Meltzer & Mindell, 2007).)

COMORBID PSYCHOPATHOLOGY IN NON-ASD

ASDs aside, other developmental disorders are included within the *DSM-IV-TR*, a number of which are also positively correlated with increased rates of comorbid psychopathology. When compared to their typically developing peers, children with atypical development without an ASD also encounter increased rates of comorbid psychopathology (Matson, Fodstad, Mahan, & Sevin, 2009; Tervo, 2007) and challenging behaviors (Baker, Blacher, Crnic, & Edelbrock, 2002; Dixon, Kurtz, & Chin, 2008; Matson, Neal, Fodstad, & Hess, 2010; Murphy et al., 2005).

Egger and Angold (2006) identified five groups of psychiatric disorders that are commonly found in atypically developing children: ADHD, oppositional defiant and conduct disorders, anxiety disorders, and depressive disorders. Deficiencies in communication and motor skills have similarly been identified (Matson, Mahan, Kozlowski, & Shoemaker, 2010). Of great importance is the fact that these impairments are not isolated incidents linked to a singular disability and have been identified in children with a variety of developmental diagnoses such as; intellectual disability, premature birth, epilepsy, Down Syndrome, and seizure disorder (APA, 2002; Coe et al., 1999; Perry, Flanagan, Geier, & Freeman, 2009; Ghaziuddin, Tsai, & Ghaziuddin, 1992; Matson et al., 2010; Oliver & Buckley, 1994; Stephenson & Dowrick, 2005).

Matson, Mahan, Kozlowski, and Shoemaker (2010) found that in general, when the ASD population was compared with other diagnoses within the atypically developing population, the latter generally reached each developmental milestone earlier than individuals with an ASD. While the occurrence of comorbid psychopathology and challenging behaviors is exhibited by the atypically developing population, recently researchers have shown that the frequency is significantly less than rates seen in those where an ASD is present (Davis et al., 2010; Matson et

al., 2010). However, in order for proper intervention and services to be rendered, the early identification and proper diagnosis remains paramount regardless of diagnosis or prevalence rate.

Common Comorbid Symptoms in non-ASD

Researchers investigating the atypically developing population indicate that the individual's primary diagnosis may serve as a predisposing factor for the development of concomitant psychopathology. For instance, when compared to peers without Cerebral Palsy, children with Cerebral Palsy were found to exhibit increased rates of symptoms associated with hyperactivity, conduct, and emotional problems (Parkes & McCusker, 2008). Comorbid symptoms such as ADHD, or conduct problems have been frequently observed in those diagnosed with Down syndrome (Myers & Pueschel, 1991; Roizen & Patterson, 2003). Within the intellectually disabled population, researchers have commonly observed symptoms of depression, anxiety disorders, and ADHD (Deb, Thomas, & Bright, 2001; Dekker & Koot, 2003; Hastings, Beck, Daley, & Hill 2005). Of those diagnosed with a Tic Disorder studies have shown that between 35-50% exhibit symptoms sufficient to meet diagnostic criterion for ADHD (Freeman, 2007; Kurlan et al., 2002; Wang & Kuo, 2003). Further, Dimitropoulos, Blackwell, Walden, and Thompson (2006) observed a significant increase in the frequency of ritualistic behaviors in children with Prader-Willi syndrome (PWS) then in children with a developmental delay, but failed to find a significant difference in severity.

The prevalence of concomitant psychopathology within the intellectually disabled population has been estimated to occur within 4-40% of the population (Deb et al., 2001; Dekker & Koot, 2003; Rojahn, Borthwick-Duffy, & Jacobson, 1993). An increase in challenging behaviors (e.g., aggressive and destructive behaviors) was positively correlated with the presence of concomitant mental health symptoms in atypically developing children without an ASD

(Matson et al., 2010). Despite the surge of research surrounding the ASD population, it is important to note that the largest group of atypically developing infants and toddlers are those with developmental and intellectual disabilities.

ASD ASSESSMENT

A dearth in identified organic markers has led to an increased reliance upon observations and the use of diagnostic measures for diagnosing ASDs (Baird et al., 2003; Matson & Neal, 2009). As knowledge of ASDs has increased, so has the availability of assessment measures and screeners to identify autistic symptoms. The *Modified Checklist for Autism in Toddlers* (*M-CHAT*) is a 23-item parent report checklist with answers being provided in a "yes/no" format (Robins, Fein, Barton, & Green, 2001). It was developed specifically to screen toddlers (16-30 months) for the presence of ASDs (Robins et al., 2001). The *Battelle Developmental Inventory, Second Edition* (*BDI-2*) is a comprehensive assessment battery of functional abilities across five developmental domains: (a) personal-social, (b) adaptive, (c) motor, (d) communication, and (e) cognitive (Alfonso, Rentz, & Suehee, 2010). The *BDI-2* is often used to identify current developmental level, assess for children "at-risk" for developmental delays, and identify strengths and weaknesses of typically and atypically developing children (Newborg, 2005). The screening portion of the *BDI-2* requires 10 to 30 minutes for administration (Newborg, 2005).

Additional diagnostic measures for ASDs include but are not limited to the *Autism Diagnostic Interview-Revised* (*ADI-R*; Lord, Rutter, & Le Couteur, 1994), *Baby and Infant Screen for Children with aUtIsm Traits* (*BISCUIT*; Matson et al., 2007) the C*hildhood Autism Rating Scale* (*CARS*; Schopler, Reichler, & Renner, 1986). As discussed previously, multiple behaviors have been identified as commonly occurring in toddlers with ASDs (Baranek, 1999; Matson & Wilkins, 2008; Osterling & Dawson, 1994; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998). Therefore clinicians often utilize direct observation measures to complement informant based measures when diagnosing ASDs (Matson & Wilkens, 2009). The *Autism*

Diagnostic Observation Schedule-Generic (*ADOS-G*) is an example of a standardized observation measure (Lord et al., 2000).

However, researchers have noted the inability to utilize observation measures when evaluating older children and when direct observation is not an option (Charman et al., 2003; Matson & Wilkins, 2008). Different groups of researchers have examined home videos of ASD children and compared them to videos of typically developing peers to identify behaviors unique to ASD (Matson & Wilkins, 2008). The inability to standardize home video observation is an identified flaw in such an approach, but it remains a valuable resource in the evaluation of older children (Charman et al., 2003; Matson & Wilkins, 2008). The observation of poor visual orientation and attention, object mouthing, unusual body posture, and aversion to social touch were suggested by multiple researchers as unique in toddlers with ASD (Baranek, 1999; Osterling & Dawson, 1994; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998). Researchers also noted the common occurrence of certain "autistic behaviors" (e.g., covering ears, stereotypies), social behavior, and joint attention as additional differences in toddlers with ASD during home video analysis (Matson & Wilkins, 2008).

Assessment of Comorbid Psychopathology

The expansion of researchers investigating ASD and comorbid psychopathologies has led to an increase in knowledge of the symptoms and features of ASDs. Tsai (2000) acknowledged the existence of psychiatric symptoms as separate from ASD symptomology, a matter which was previously the source of much debate. Howlin and Moss (2012) also demonstrated that if symptoms of comorbid psychopathology are left untreated they continue to plague those with ASD even as they mature into adulthood. Matson and Nebel-Schwalm (2007) suggested that clinicians evaluating the presence of comorbid psychopathology within the infantile ASD

population should assess for them multiple times due to the often cyclical presentation associated with psychiatric disorders. This distinction made necessary the development of assessments to properly detect the presence of concomitant psychopathologies in children. As agreement concerning the presence of comorbid symptoms and the benefit of early intervention (e.g., EIBI) has increased, researchers have explored the validity of assessing comorbidity in an infantile populace (Dawson, 2008; Lovaas, Schreibman, & Koegel, 1974; Remington, et al., 2007; Smith, Groen, & Wynn, 2004). However, until recently, an acknowledged gap in the research of infantile ASD has been the lack of assessment measures analyzing psychopathology with ASD norms (Matson et al., 2010).

The results gathered from early assessment of comorbid psychopathology help to identify problems unique to an individual child which would allow for the customization of an intervention strategy. Further, the compilation of research mapping the presence of comorbid psychopathology in an infantile ASD population would aid in advancing understanding of the presenting features and symptoms commonly observed in ASDs. The early recognition of comorbid psychopathology may also aid in the identification of patterns or characteristics which may help educate the individual's family and caretakers by drawing their attention to the complex nature of the disorders' interactions they are being confronted with and affording them the opportunity to educate themselves (Williams & Brayne, 2006). Increased knowledge would also allow parents to explore other services available to them such as genetic counseling which would include recurrence rates and inform reproductive decisions (McMahon, Baty, & Botkin, 2006; Simonoff, 1998).

Worley and Matson (2011) stressed the importance of caution and thorough assessment to ensure the suspected psychopathological symptomology is not better accounted for by the

ASD diagnosis. Assessment and interpretation is complicated due to the large variation in range of functional abilities inherent to the hierarchical orientation of the ASD category. These factors combined with the necessity of accurate diagnosis for efficacious intervention, make accurate differential diagnosis crucial (Gillberg & Billstedt, 2000; Tsai, 1996).

Researchers have suggested that the manifestation and expression of comorbid psychopathological symptoms within ASDs are unique and different from the presentation seen in other disorders (Matson et al., 2007). While assessment measures such as the *Behavioral Assessment System for Children-Second Edition* (*BASC-2)* and *Child Behavior Checklist for ages 1.5-5* (*CBCL*) screen for the presence of comorbid psychopathology, these measures do not include the norms necessary to account for the unique presentation witnessed in an ASD population. The development of the *BISCUIT-Part 2* has filled this gap (Matson, Wilkins, Knight, Boisjoli, & Sharp, 2009).

Behavioral Assessment System for Children-Second Edition

The *BASC-2* assessment battery is designed to assess psychopathology in toddlers as young as 24 months of age and spanning up to adults 25 years of age (Reynolds & Kamphaus, 1992). The assessment battery contains a comprehensive set of forms whose inclusion is dependent upon the assessed person's age such as *Teacher rating scale* (*TRS*), *Student observation system* (SOS), and a *parent rating scale* (*PRS*). With an average administration time of 20 minutes, the *Preschool TRS* consists of 100 items. Item responses are rated based upon a four-point scale reflecting the frequency of occurrence: N (never), S (sometimes), O (often), and A (almost always; Reynolds & Kamphaus, 2004). The Preschool *PRS* utilizes a similar four-point response scale for the 134 items presented. Both scales assess for the presence of internalizing and externalizing behavior problems and provide separate subscale scores such as

anxiety, depression, and inattention (Reynolds & Kamphaus, 2004). This measure is limited by a major factor relative to the interests of the present study; its normative age range, which begins at two years of age, which would prevent it from being useful in early identification and intervention approaches (Reynolds & Kamphaus, 2004).

Researchers are consistently placing emphasis on the early identification and intervention in toddlers with ASD. The American Academy of Pediatrics (AAP) has placed an emphasis on early identification by encouraging pediatricians to carry out ASD screeners at a child's 18 and 24 month well-child checkups. Further supporting the role of early assessment, Matson and Smith (2007) suggested participation in intervention programs for children diagnosed with an ASD as early as 18 to 24 months of age. The unique symptoms observed in the ASD population has led researchers to acknowledge the need for a measure that is designed to aid in the identification and classification of symptoms that are indicative of either a concomitant psychopathology, or being better described by the presence of an ASD (Matson, Boisjoli, & Wilkins, 2007).

Child Behavior Checklist for ages 1.5-5

The need for the early assessment of psychopathology (i.e., 18 months of age) was addressed by the development of a parent-report measure known as the *CBCL* (Achenbach & Rescorla, 2000). The *CBCL* is a part of the Achenbach System of Empirically Based Assessment (ASEBA), and is accompanied by the *Caregiver–teacher rating form* (*C-TRF*; Achenbach, 2009). The *CBCL* is a standardized measure that can be administered in 20 minutes. It assesses a child's psychopathology by examining both internalizing (e.g., anxiety, withdrawn, and depressed), and externalizing (e.g., aggression, attention) behaviors as well as language acquisition via the *Language development survey* (*LDS*). The *CBCL* and *C-TRF* forms are

comprised of 99 items total, as well as additional descriptor sections allowing informants to report any additional concerns or behaviors observed.

Separate factor analyses for both the *CBCL* and the *C-TRF* have identified a total of seven factors for the *CBCL* (i.e., Emotionally Reactive, Anxious/Depressed, Aggressive Behavior, Attention Problems, Somatic Complaints, Withdrawn, and Sleep problems) while the latter excludes the "sleep problems syndrome" for a total of six factors (Liu, Cheng, & Leung, 2011). Also included in the scoring of the *CBCL* and *C-TRF* are five DSM-oriented scales: Affective Problems, Anxiety Problems, Pervasive Developmental Problems, Attention Deficit/Hyperactivity Problems, and Oppositional Defiant Problems (Achenbach & Rescorla, 2000). Statistical analysis of test-retest reliability identified an average correlation of .85 for the *CBCL* and an average correlation of .81 for *C-TRF* (Rescorla, 2005). However, a limitation of their paper was the failure to offer specialized norms on the *CBCL* for the ASD population.

Baby and Infant Screen for Children with aUtIsm Traits

To address both the need for early identification, and better account for the unique presentation occurring in the ASD population an informant-based measure, the *BISCUIT* was created (Matson et al., 2007). The *BISCUIT* assessment battery is composed of three parts and is designed to assess infants and toddlers aged 17 to 37 months. Item selection was based upon the methodology of scale development outlined by Crocker and Aligna (1986), and DeVellis (1991). *BISCUIT-Part 1* was created as a diagnostic tool for assessing infants and toddlers for either AD or PDD-NOS. The *BISCUIT-Part 2* is the primary focus of the present article, and measures symptoms associated with concomitant mental health disorders (e.g., ADHD, obsessive-compulsive disorder, tic disorders, and specific phobias). Lastly, *BISCUIT-Part 3* assesses for the incidence of those challenging behaviors commonly observed in the ASD population.

BISCUIT-Part 2 contains 65 items that are rated based upon the extent to which they have been the source of recent impairment as follows: 0, “not a problem or impairment”; 1, “mild problem or impairment”; 2, “severe problem or impairment”; or X, “does not apply or don’t know” (Matson et al., 2008). Ratings of impairment/occurrence are accumulated and then mapped onto five different subscales: ADHD, tic disorder, obsessive-compulsive disorder, specific phobia, and eating/feeding difficulties. Matson, Boisjoli, Hess, and Wilkins (2011) found the *BISCUIT-Part 2* to have excellent internal consistency with alpha levels exceeding .80, which is considered ideal (Clark & Watson, 1995).

PURPOSE

Significant research has been published highlighting the increased occurrence of comorbid psychopathological conditions in toddlers with ASDs when compared to both typically and atypically developing peers (Barthélémy et al., 1992; Dawson, Matson, & Cherry, 1998; Gadow, DeVincent, Pomeroy, & Azizian, 2004; Holden & Gitlesen, 2007; Horner, Carr, Strain, Todd, & Reed, 2002; Matson et al., 2010; Matson & Nebel-Schwalm, 2007; Matson & Rivet, 2008; Paclawskyj, Matson, Bamburg, & Baglio 1997). Comorbid psychopathology in toddlers with ASD complicates not only differential diagnosis and intervention approaches, but also negatively impacts caregivers and the individual's prognosis (Gray, Ansell, Baird, & Parr 2011; Matson & Minshawi, 2007; Matson & Nebel-Schwalm, 2007). However, research has failed to extend beyond prevalence rates to investigate the topography of comorbid symptoms in toddlers. An increased knowledge concerning the topography of comorbid psychopathologies in toddlers with ASDs is imperative to increase the knowledge of symptom manifestation, common behavioral presentations within this population, and the trajectory and stability of symptoms. This knowledge can inform multiple domains, including assessment, treatment, and etiology (Matson et al., 1996; Matson & Bamburg, 1998; Matson, Hess, & Boisjoli, 2010).

The importance and success of early intervention approaches in the ASD population has been recognized by many researchers (Peters-Scheffer, Didden, Korzilius, & Matson, 2012; Peters-Scheffer, Didden, Korzilius, & Sturmey, 2011; Poustka, Rothermel, Banaschewski, & Kamp-Becker, 2012; Reichow, 2012). Additionally, LoVullo and Matson (2009) identified the need for highly individualized interventions when comorbid symptoms are present in toddlers with ASD. However, these intervention techniques can only be beneficial when behavioral and psychopathological factors are identified and targeted by intervention. It is also necessary that

these interventions occur before the implementation of other interventions (e.g., educational) can be expected to be fruitful. However, there is a scarcity of comorbid psychopathology research available in toddlers, who have been identified as the benefactors of early interventions (Matson et al., 2010).

Researchers have only just recently begun to extend the investigation of comorbid symptoms to infants and toddlers. The current paper served as a means of further analysis of developmental psychopathology. In order to assess for any comorbid psychopathology symptoms the *BISCUIT-Part 2* was administered to parents of infants and toddlers (17-37 months of age) with diagnoses of AD, PDD-NOS, or atypical development. The purpose of the current study was primarily to investigate the expression of comorbid psychopathological symptoms in an infant ASD population when compared to their atypically developing peers without an ASD. Temporal influences on the expression of comorbid psychopathology symptoms were also evaluated. The current research is important for enhancing the understanding of the relationship between ASDs and comorbid psychopathology manifestation during the early stages of development.

The stability of core symptoms in individuals with AD has been replicated in multiple studies (Landa et al., 2007; Werner et al., 2005). Additionally, reliable early identification of ASD has been linked to the presence of more severe symptoms found in toddlers with AD (Matson & Horovitz, 2010). However, research investigating the manifestation of comorbid psychopathology in infants is sparse. Investigation of developmental psychopathology in an infantile and toddler populace with an ASD diagnosis is to the extent of this researcher's knowledge, very limited.

It is hypothesized that comorbid psychopathology symptoms will be moderately stable in all participants belonging to the AD and atypical development categories. A perfect replication of scores from the first administration to the second cannot be anticipated due to the informant-based nature of the measure and testing familiarity associated with multiple administrations of the same assessment. A significant amount of instability, however, is expected for the PDD-NOS group due to the lack of specified criteria provided by the *DSM-IV-TR* for this diagnosis. Furthermore, researchers have suggested concern surrounding the instability of the PDD-NOS diagnosis itself in an infantile population (Stone et al., 1999, Worley et al., 2011). The identification of symptom instability should not deter the assessment of individuals with a PDD-NOS diagnosis. Rather, it should educate and prepare clinicians by identifying the need for periodic reassessment to evaluate the intervention's accuracy. Investigation of the temporal influences are exploratory in nature and do not include a formal hypothesized outcome

METHODS

Participants

Participants in the current study consisted of 315 infants and toddlers who had received services provided by the state of Louisiana's EarlySteps program. Louisiana's Individuals with Disabilities Education Act, Part C (2004) created EarlySteps, a program within Louisiana's Early Intervention System providing services to children and their families. In order to qualify for services, children must have been identified as having a developmental delay or a medical condition that places them as at risk for a developmental delay and fall within a specified age range (i.e., birth to 36 months).

Following their participation in the EarlySteps program, individuals were separated into one of three groups dependent upon their clinical diagnosis (i.e., AD, PDD-NOS, or atypical development without an ASD). Diagnoses for an ASD were made in accordance with the criteria outlined by the *DSM-IV-TR* by a licensed clinical psychologist. Diagnostic decisions were made by reviewing the descriptors provided in the *DSM-IV-TR* for PDD-NOS (APA, 2000), the *DSM-IV-TR* algorithm for AD, the individual's scores on the *BDI-2* (Newborg, 2005), and *M-CHAT* scores (Robins et al., 2001). Participants were identified as atypically developing based upon a failure to meet developmental milestones or the presence of genetic disorders (e.g., Down's syndrome) or physical disabilities. The clinician assigning the diagnoses was a licensed psychologist with more than 30 years of experience within the developmental disabilities field. During the reviewing and subsequent diagnosis of cases, the clinician was blind to results from the *BISCUIT* assessment. Initially, a total of 315 individuals were identified from a large dataset who received two administrations of the *BISCUIT-Part 2* and were within the identified age range were considered for inclusion in the study. Scores for each of these individuals, diagnosis,

and length of time between initial and follow-up administration were examined. Participants were excluded from the study if adequate information was not included in the database (e.g., incomplete measures), if they did not retain their initial diagnosis, or if they received a follow-up assessment in fewer than four months, or after 13 months. The final sample consisted of 205 toddlers (see Table 1 for demographic information). The sample was divided into three groups based upon diagnosis: AD (*n*=35); PDD-NOS (*n*=32); or atypically developing (*n*=138).

Participants in each diagnostic category were also separated into two temporal groups for further analysis, Time1 (AD *n*=20; PDD-NOS *n*=17; atypical development; *n*=72) or Time2 (AD *n*=15; PDD-NOS *n*=15; or atypical development *n*=66). Additional information was also gathered from the database (i.e., demographics [ethnicity and gender] and assessment information [developmental quotient]). A preliminary Chi-square analysis was then used to determine if significant differences in demographic variables (i.e., ethnicity or gender) and assessment information between the diagnostic groups was evident. Demographic characteristics are presented in Table 1.

Table 1
Participant Demographics

	AD	PDD-NOS	Atypical	Total Sample
Age in months (SD)	23.51 (4.1)	23.09 (3.84)	23.24 (3.84)	23.26 (3.86)
Gender				
Male	77.1%	78.1%	78.3%	78%
Female	22.9%	21.9%	21.7%	22%
Ethnicity				
Caucasian	45.7%	43.8%	41.3%	48.3%
African-American	45.7%	43.8%	50.0%	42.4%
Hispanic	5.7%	0%	5.8%	4.9%
Other/Unspecified	2.9%	12.4%	2.9%	4.4%
Diagnostic percentage	17.1%	15.6%	67.3%	
Developmental Quotient	72 (13.38)	80.56 (14.23)	86.77 (11.44)	83.67 (13.22)

Note: SD = Standard Deviation, AD = Autistic Disorder, PDD-NOS = Pervasive Developmental Disorder – Not Otherwise Specified.

Measure

Baby and Infant Screen for Children with aUtIsm Traits-Part 2 (BISCUIT-Part 2; Matson et al., 2007). Researchers have suggested that the manifestation and expression of comorbid psychopathological symptoms within ASDs are unique and different from the presentation seen in other disorders (Matson et al., 2007). While assessment measures such as the *BASC-2* and *CBCL* screen for the presence of comorbid psychopathology, these measures are not designed to account for the unique presentation witnessed in an ASD population. The development of the *BISCUIT-Part 2* to assess for comorbid psychopathologies has filled this gap (Matson et al., 2009).

The *BISCUIT-Part 2* is a singular component of a triad of informant based measures (i.e., *BISCUIT-Parts 1-3*) falling under the general battery developed to specifically screen for the presence of ASD symptomology, comorbid psychopathology, and challenging behaviors in infants and toddlers within the 17 to 37 month old age range (Matson et al., 2009). The *BISCUIT-Part 2* contains 65 items that respondents endorse on a 4-point Likert scale based upon the extent that the item has been a recent problem as follows: 0, "not a problem or impairment, not at all"; 1, "mild problem or impairment"; 2, "severe problem or impairment"; or X, "does not apply or don't know" (Matson et al., 2008). The *BISCUIT-Part 2* is designed to assess for symptoms associated with the following comorbid psychopathological disorders: ADHD, tic disorder, obsessive-compulsive disorder, specific phobia, and eating/feeding difficulties (Matson et al., 2009). An exploratory factor analysis identified five factors and led to the creation of the following subscales: Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eat/Sleep Problems (Matson et al., 2011). When assessing reliability, LoVullo and Matson (2012) found the *BISCUIT-Part 2* to have excellent internal

reliability with an alpha level of .96, which is indicated as ideal in the literature (Clark & Watson, 1995). The cutoffs for the *BISCUIT-Part 2* are different and based upon the toddlers diagnosis (i.e., ASD or atypically developing) and are further separated by score into one of three sections, No/Minimal Impairment, Moderate Impairment, and Severe Impairment.

See Table 2 for an indication of how the *BISCUIT-Part 2* scores correspond with their respective levels of impairment unique to each subscale in the ASD population. See Table 3 to determine how the *BISCUIT-Part 2* scores correspond with their respective levels of impairment unique to each subscale in the atypically developing population.

Table 2
Severity levels in ASDs

	No/Minimal Impairment	Moderate Impairment	Severe Impairment
Tantrum/Conduct Behavior	0 to 16	17 to 24	25 and up
Inattention/Impulsivity	0 to 15	16 to 22	23 and up
Avoidance Behavior	0 to 6	7 to 10	11 and up
Anxiety/Repetitive Behavior	0 to 6	7 to 9	10 and up
Eat/Sleep Problems	0 to 3	4 to 5	6 and up

Table 3
Severity levels in atypical development without an ASD

	No/Minimal Impairment	Moderate Impairment	Severe Impairment
Tantrum/Conduct Behavior	0 to 6	7 to 10	11 and up
Inattention/Impulsivity	0 to 5	6 to 9	10 and up
Avoidance Behavior	0 to 1	1 to 2	3 and up
Anxiety/Repetitive Behavior	0 to 1	1 to 2	3 and up
Eat/Sleep Problems	0 to 1	2 to 3	4 and up

Procedure

Following referral to Louisiana's EarlySteps program, the *BISCUIT* assessment battery was administered to a parent/caregiver in addition to a comprehensive package of assessments to gain relevant demographic and diagnostic information. The assessment battery was administered

to the same informant in the presence of the at-risk toddler twice with no less than three months, but no more than one year separating the initial and follow-up administration. Assessments were administered by evaluators employed by the EarlySteps program with a minimum of a bachelor's degree, but ranging up to a doctoral degree. The variance of educational attainment is further complemented by assorted certifications and licenses representative of their respective disciplines (e.g., psychology, education, social work, speech/language pathology, and early childhood development). In addition to their educational background, each evaluator received training in standardized assessment administration methods, and training specific to the measures being administered. Approval of both the state of Louisiana's Office for Citizens with Developmental Disabilities and the Louisiana State University Institutional Review Board was obtained prior to the utilization of the demographic and diagnostic information. Parental consent was given at the time of assessment. The data obtained from these interviews was coded and entered into an electronic database for analysis purposes.

STATISTICAL ANALYSES

An a priori power analysis was carried out utilizing a statistical software package identified as GPower*3 (Faul, Erdfelder, & Buchner, 2007) to determine the sensitivity of the factorial multivariate analysis of variance (MANOVA) with a conservative harmonic mean. In an effort to satisfy the standards provided by Cohen (1992) a desired power level of .80 was selected along with an alpha (α) set at a significance level of .05 to determine the effect size given the conservative harmonic mean sample size included. The parameters outlined above are considered by Hinkle, Wiersma, and Jurs (2003) to be ideal in behavioral science research. Given a conservative harmonic mean sample size (n = 45) an effect size of .20 would have an achieved power of .80, an effect size of .25 would equate to an achieved power of .90, and an effect size of .50 would have an achieved power of .99.

Prior to statistical analyses, both of the administrations of *BISCUIT Part-2* were reviewed to ensure that there were no missing item responses and that item values were valid (i.e., 0, 1, or 2). Participants found to be missing more than 10% of responses for any subscale for each administration of the *BISCUIT Part-2* were excluded from the study. For the remaining data set, Little's Missing Completely At Random test (MCAR; Little, 1988) was run to ensure that the missing data was missing at random and not predictable from other variables or related to the dependent variables or informant omission. The MCAR was not significant, indicating that the items were missing completely at random (Tabachnick & Fidell, 2007; Van Ness, Murphy, Araujo, Pisani, & Allore, 2007).

In order to estimate values for the missing items, the multiple imputation procedure was used. The multiple imputation procedure involves multiple steps for estimating missing item values (Tabachnick & Fidell, 2007). First, a logistic regression was carried out utilizing

diagnostic category, time category, developmental quotient, and age as predictor variables. Based upon these variables, the logistic regression creates an equation for estimating missing item values for each diagnostic category (Tabachnick & Fidell, 2007). In order to identify the distribution of variables with missing data, a random sample with replacement is then drawn from the cases without missing variables. The identified variable distribution for each missing item was then used to provide an estimate for the missing items in five random samples (with replacement). The average imputed variable across all five samples for each missing item was then included in a sixth dataset for analysis (Mehrotra, Li, Liu, & Lu, 2012; Rubin, 1987). Statistical Solutions, Ltd. (1997) suggested that multiple imputations are advantageous for within-subjects designs because the procedure retains the sampling variability.

Researchers have found the MANOVA to be robust to violations of non-normality, when ten or more participants are included in each group (Seo, Kanda, & Fujikoshi, 1995). Tabachnick and Fidell (2007) suggested that when sample sizes are unequal, a significant result from Box's M test for homogeneity of variance-covariance matrices should be followed by additional analyses. For the current analysis, the ratio for any dependent variable for each diagnostic group from smallest to largest variance does not exceed the 10:1 (Actual current $F_{max} = 3.71$; 4:1) ratio suggested by Tabachnick and Fidell (2007) to invalidate the use of a MANOVA. Further, the discrepancy in sample size between diagnostic categories in the current sample is more representative of the population distribution ($F_{max} = 4:1$). All statistical analyses were run utilizing the SPSS 21.0 software package.

Preliminary Statistics

A priori analyses were conducted to investigate potential differences between the three diagnostic groups with respect to demographic variables including gender, ethnicity,

developmental quotient, and age (see Table 1). Results of the chi-square analysis indicated that there were no differences in gender ($\chi 2$ [2, N = 205] = .02, p = .99) or ethnicity ($\chi 2$ [6, N = 205] = 7.94, p = .24) among the three groups. Although there was an observed difference in male to female ratios in the ASD groups, the gender difference (approximately four times as common in males) has been recognized by multiple researchers (Fombonne, 2005; Kanner, 1971). An analysis of variance (ANOVA) was then conducted to test for any statistically significant differences in age between diagnostic groups. Age was examined using Levene's test, which showed that homogeneity of variance was upheld (p = .90). Results of the ANOVA indicated no significant age differences among the three diagnostic groups F (2, 201) = .11, p = .90.

An additional ANOVA was computed to determine if a statistically significant difference existed between groups for the developmental quotient. Only a portion of the participants in the current study had a developmental quotient score and could be included in this analysis (AD = 22, PDD-NOS = 25, Atypical = 108). The ANOVA revealed a significant difference among the three groups for developmental quotient, F (2, 152) = 14.36, p < .01; however, due to the unequal sample sizes, Hochberg's GT2 corrected *post hoc* comparisons were computed (Field, 2009). Results of this analysis revealed that the difference resulted from significant differences between the atypically developing group and the AD group (p < .01), there was not a significant difference between the atypically developing and the PDD-NOS groups (p = .07) or the PDD-NOS and AD groups (p = .06). Due to the observed differences in developmental quotient among diagnostic categories this variable will not be entered as a covariate (Field, 2009).

A Kolmogorov-Smirnov test was then conducted for each factor (i.e., diagnostic category and time) to test if the dataset was normally distributed. The result of this test of normality was significant (p < .05) for Time and Diagnosis for each dependent variable. Histograms and Q-Q

plots were also reviewed during tests of normality, and a positively skewed distribution was observed. However, Field (2009) identified that when large samples are used, the Kolmogorov-Smirnov test can be significant even when scores vary slightly from a normal distribution. Field (2009) additionally identified that due to the problem of small standard errors when a large sample ($n > 200$) is used, no criterion for skewness and kurtosis should be applied.

In order to investigate the stability of comorbid psychopathological symptoms across diagnostic groups, participants were first separated into their respective diagnostic categories dependent upon diagnosis received following their participation in the EarlySteps program (i.e., AD, PDD-NOS, or atypically developing). The diagnosis being utilized was based upon the methodology outlined in the contents of the participant section above. The difference between each individual's achieved score across both administrations of the *BISCUIT-Part 2* for each of the five factors was then computed. An absolute difference score between initial and follow-up assessment was then computed for each factor and represents the amount of change for each factor among the three diagnostic groups. Due to the desire to measure any change in symptoms, the absolute difference score ignored directionality (i.e., increased item endorsement, or decreased item endorsement). Utilizing this classification system, the stability of symptom expression was then examined based upon observed changes in item endorsement between two assessments.

The time lapse between successive administrations was also examined. In order to thoroughly investigate the influence of temporal factors on symptom stability, a two-part classification system was created. Participants were segregated into two groups dependent upon the time span separating their initial and follow-up evaluation. Participants with a span of 4 to 8 months between their initial and follow-up assessments composed one group (i.e., Time1), while

the second group consisted of participants whose follow-up assessments were conducted 9 to 13 months (i.e., Time2) after initial evaluation. The ranges expressed here are centered upon the EarlySteps programs attempt to reassess individuals at six month intervals (e.g., 6 months, and 12 months).

Study

A 3 X 2 factorial between-subjects MANOVA was computed to assess whether there are significant changes in item endorsement based upon diagnosis (i.e., AD, PDD-NOS, and atypical development) and/or time (i.e., time spans between the initial and follow up evaluation) across two administrations of the *BISCUIT-Part 2*. The current study contained two independent variables. The identified between-subjects factors were, diagnosis (i.e., AD, PDD-NOS, and atypical development), and the length of time between the assessments (i.e., 4 to 8 months or 9 to 13 months). The dependent variables between-subjects factor were the absolute difference's derived from each participant's respective *BISCUIT-Part 2* scores across both administrations for each subscale. Descriptive statistics were run in order to identify the respective mean for each temporal group. A series of univariate ANOVAs were computed for each dependent variable with Bonferroni corrections to control the inflation of Type I error. Following a significant main effect for diagnosis, pairwise comparisons were computed between each respective diagnostic category which included all individuals regardless of the time factor. Due to the observed differences in sample sizes, Field (2009) recommends that a Gabriel's procedure will be too liberal and instead both Hochberg's GT2 and Games-Howell procedures be selected for post hoc analysis. Finally, the significant MANOVA was followed with a discriminant functional analysis for the diagnostic categories to analyze the underlying relationships between dependent variables.

RESULTS

Initially, descriptive statistics were computed to look at the mean and standard deviation for the absolute difference for each of the five factors of the *BISCUIT-Part 2* based upon diagnostic category (Table 4).

Table 4
Means and standard deviations of the absolute difference factor scores on the BISCUIT Part-2 for all three levels of the independent variables

	AD M (SD)	**PDD-NOS M (SD)**	**Atypical M (SD)**
	***n* = 35**	***n* = 32**	***n* = 138**
Factor 1			
Sleep	2.11 (1.43)	1.00 (1.24)	0.84 (1.10)
Factor II			
ADHD	5.63 (4.94)	3.22 (3.53)	2.3 (2.88)
Factor III			
Avoidant	2.51 (2.65)	1.78 (2.56)	0.78 (1.40)
Factor IV			
Anxiety	3.80 (2.95)	1.84 (2.30)	1.18 (1.64)
Factor V			
Tantrum	6.60 (6.07)	4.78 (5.10)	2.62 (3.11)

Note: SD = Standard Deviation, AD = Autistic Disorder, PDD-NOS = Pervasive Developmental Disorder – Not Otherwise Specified. The absolute difference between the initial and follow-up assessment was used to calculate the above values.

A MANOVA was performed on two independent variables: diagnostic group (i.e., AD, PDD-NOS, and Atypical) and time lapse (i.e., Time1 and Time2). There were five dependent variables based upon the absolute difference between the two subscale totals of the five factors of the *BISCUIT-Part 2*. A significant Box's test suggested that homogeneity of variance-covariance was violated. However, due to the vulnerability of having a significant Box's test when there is a discrepancy in sample size is present, additional analyses were conducted, revealing that the assumption was tenable (F*max* = 4:1). Field (2009) suggested the use of Pillai's trace for interpreting the results of a MANOVA when group sizes are different and groups differ along more than one dimension. Using Pillai's trace, a significant main effect of

diagnosis on the stability of comorbid symptoms was observed, $V = .29$, F (10, 392) = 6.54, $p < .01$, partial $\eta 2 = .14$. Additionally, a significant main effect of Time was also observed, $V = .08$, F (5, 195) = 3.33, $p < .05$, partial $\eta 2 = .08$. The interaction between diagnostic category and the time between original and follow-up assessment was not found to be significant, $V = .08$, F (10, 392) = 1.63, $p = .1$, partial $\eta 2 = .04$. The observed interaction between diagnosis and time was not significant. The results of the significant MANOVA were indicative of differences among the diagnostic categories. The significant main effect for Time indicated that three factors of the *BISCUIT-Part 2* were significantly different when assessed at a later time period (i.e., Time2; Table 5). Specifically, significant differences between Time1 and Time2 were observed for the following factors: ADHD F (1, 203) = 4.61, $p < .05$, partial $\eta 2 = .02$, Avoidant F (1, 203) = 6.1, $p < .01$, partial $\eta 2 = .03$, and Tantrum F (1, 203) = 6.19, $p < .01$, partial $\eta 2 = .03$.

Table 5
Means and standard deviations of factor scores on the BISCUIT Part-2 based upon time between initial and follow-up assessment

	Time1 M (SD)	**Time2 M (SD)**
	***n* = 109**	***n* = 96**
Factor 1		
Sleep	0.10 (1.75)	-0.26 (1.56)
Factor II		
ADHD	-0.22 (4.17)	-2.15 (4.81)
Factor III		
Avoidant	-0.06 (1.88)	-0.64 (2.71)
Factor IV		
Anxiety	-0.03 (2.64)	-0.52 (2.99)
Factor V		
Tantrum	0.01 (4.88)	-1.70 (6.26)

Note: SD = Standard Deviation. The absolute difference between the initial and follow-up assessment was used to calculate the above values.

The significant main effect for diagnosis observed in the MANOVA was followed up with ANOVAs for each of the five factors of the *BISCUIT Part-2*. The factor score for each

ANOVA will be discussed separately. Factor I, Sleep, did not violate any of the assumptions of the ANOVA and a significant main effect of diagnosis was observed F (2,202) =16.20, $p < .01$, partial $\eta^2 = .13$. Each of the remaining four factors did violate the homogeneity of variance assumption for an ANOVA, and therefore the more robust Welch's test is reported. Factor II, ADHD, indicated a main effect of diagnosis Fw (2, 52.84) = 7.69, $p < .01$, partial $\eta 2 = .13$. A Welch's test for Factor III, Avoidant, indicated a main effect of diagnosis Fw (2, 49) = 8.69, $p < .01$, partial $\eta 2 = .13$. A Welch's test Factor IV, Anxiety, indicated a main effect of diagnosis Fw (2, 51.15) = 13.24, $p < .01$, partial $\eta 2 = .20$. Finally, a Welch's test for Factor V, Tantrum, also indicated a main effect of diagnosis Fw (2, 49.50) = 9.04, $p < .01$, partial $\eta 2 = .14$.

Due to the difference in sample sizes and difference in population variances (Fmax = 4:1), the Games-Howell procedure was used to control for the inflation of Type I errors when running multiple comparisons. The Games-Howell procedure has been identified as the most powerful and accurate when sample sizes are not equal (Field, 2009). The post hoc comparisons for each of the five factors of the *BISCUIT Part-2* were examined separately. Post hoc comparisons using the Games-Howell procedure indicated that the mean difference between the AD and Atypical groups were significant for each factor ($p < .05$; Table 6). Additionally, the average difference between ASD groups was significant for two factors, Sleep and Anxiety ($p < .05$; Table 6). No significant differences in developmental psychopathology were observed between the PDD-NOS and Atypical groups (Table 6).

The MANOVA was also followed by descriptive statistics to analyze the observed trend of change in comorbid symptom expression for each diagnostic category across administrations and in what direction the change occurred (Table 7).

Table 6
Mean difference between diagnostic groups following Post hoc comparisons using the Games-Howell procedure

	AD/PDD-NOS M (SD)	PDD-NOS/Atypical M (SD)	AD/Atypical M (SD)
	***n* = 35**	***n* = 32**	***n* = 138**
Factor I			
Sleep	1.11**	0.16	1.27**
Factor II			
ADHD	2.41	0.91	3.32**
Factor III			
Avoidant	0.73	1.00	1.73**
Factor IV			
Anxiety	1.96*	0.66	2.62**
Factor V			
Tantrum	1.82	2.17	3.98*

Note: SD = Standard Deviation, AD = Autistic Disorder, PDD-NOS = Pervasive Developmental Disorder – Not Otherwise Specified. The absolute difference between the initial and follow-up assessment was used to calculate the above values. *significant at the .05 level, **significant at the .01

Table 7
Mean difference for factor scores on the BISCUIT Part-2 for all three levels of the independent variable

	AD M (SD)	PDD-NOS M (SD)	Atypical M (SD)
	***n* = 35**	***n* = 32**	***n* = 138**
Factor I			
Sleep	0.57 (2.51)	-0.25 (1.58)	-0.19 (1.38)
Factor II			
ADHD	-0.94 (7.49)	-0.78 (4.74)	-1.25 (3.47)
Factor III			
Avoidant	-0.46 (3.65)	-0.97 (2.98)	-0.14 (1.60)
Factor IV			
Anxiety	-0.09 (4.85)	-0.47 (2.93)	-0.25 (2.00)
Factor V			
Tantrum	-0.77 (9.00)	-1.28 (6.92)	-0.68 (4.00)

Note: SD = Standard Deviation, AD = Autistic Disorder, PDD-NOS = Pervasive Developmental Disorder – Not Otherwise Specified.

Additional post hoc analyses included a discriminant analysis to investigate the relationship between the dependent variables and how the dependent variables discriminate the diagnostic categories in the current study (Field, 2009). In the current model discriminant analysis revealed two discriminant functions. The first function explained 87.7% of the variance

(canonical R2 = .23), whereas the second function explained 12.3% of the variance (canonical R2 = .04). In combination these discriminant functions significantly differentiated the diagnostic groups, $\Lambda = .76$, $\chi 2\ (10) = 61.50$, $p < .01$. However, removing the first function revealed that the second function did not significantly differentiate the diagnostic groups, $\Lambda = .96$, $\chi 2\ (4) = 8.34$, $p = .08$. The correlations between outcomes and the identified discriminant functions revealed that Anxiety loaded disproportionately higher on the first function and not the second ($r = .87$ and $r = -.10$ respectively). Similar patterns were observed for the factors Sleep ($r = .72$ and $r = -.33$) and ADHD ($r = .66$ and $r = -.04$). Correlations between the first and second discriminate function remained disproportionate but positive for the remaining two dependent variables, Tantrum ($r = .67$ and $r = .43$) and Avoidant ($r = .64$ and $r = .46$). The discriminant function plot (Figure 1.) showed that the first function discriminated the AD and PDD-NOS groups from the Atypical group, while the second function differentiated the AD and Atypical groups from the PDD-NOS group.

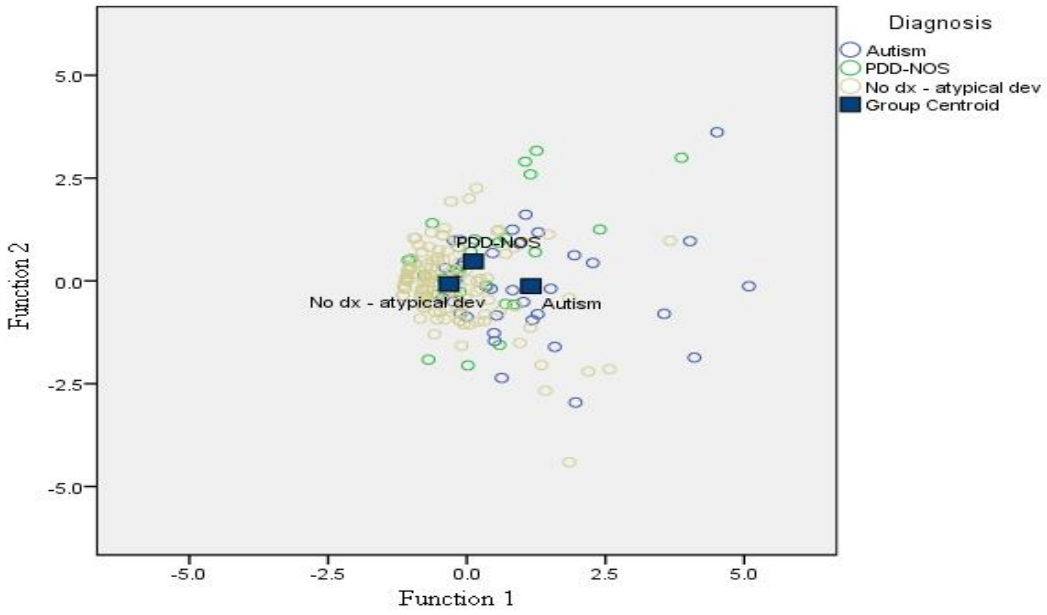


Figure 1 Discriminant function plot. This figure depicts the two functions underlying the relationships between the dependent and independent variables.

DISCUSSION

Treating an ASD during infancy has been proven beneficial (Ben-Itzchak et al., 2008; Eaves & Ho, 2004; Matson & Smith, 2008; McEachin et al., 1993). Additionally, core symptom stability in individuals with AD has been replicated in multiple studies (Landa et al., 2007; Werner et al., 2005). However, researchers have not yet expanded this research to include symptoms of developmental psychopathology. Researchers have repeatedly reported the higher occurrence of comorbid symptoms in the ASD population when compared to their typically and atypically developing peers (Gillberg & Billstedt, 2000; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Matson, Hess, & Boisjoli, 2010; Park et al., 2012; Schreck et al., 2004). Despite the observed increase in frequency of occurrence in individuals with an ASD, this topic has received minimal empirical research to date (Matson et al., 2010; Matson & Nebel-Schwalm, 2007). Even less frequent have been investigations of the expression of comorbid symptoms in an infantile ASD populace (Angold & Egger, 2004; Matson et al., 2010). The current study sought to explore symptoms of developmental psychopathology in an infantile ASD population based upon the *BISCUIT-Part 2*. Results from the current study indicated that not only are symptoms of developmental psychopathology less stable in infants with ASD, but also that the time between assessments is a significant factor influencing stability.

The diagnostic group had more comorbid symptoms than controls. This finding indicates that not only are comorbid symptoms more prevalent in infants with ASD (Gillberg & Billstedt, 2000; Kim et al., 2000; Park et al., 2012; Schreck et al., 2004), but that the prevalence of these symptoms increase with age. The current findings are commensurate with researchers who have also reported that juveniles and adults with ASDs exhibit significantly increased rates of comorbid conduct behaviors (i.e., tantrums and aggression) and ADHD when compared to

typically developing peers (Horner, Carr, Strain, Todd, & Reed, 2002; Matson et al., 2010; Matson & Rivet, 2008). Contrary to these results, some researchers have not reported significant differences in comorbidity when comparing ASD and non-ASD populations. Utilizing the *Autism Spectrum Quotient* (*AQ*; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) to identify participants with ASD, Ketelaars and colleagues (2008) did not observe significant differences in the expression of comorbid symptoms when compared to non-ASD peers. However, The *AQ* was not developed as a diagnostic measure, and was originally developed to screen for autistic symptoms in individuals with normal intellectual functioning (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001).

A significant main effect for time in the current study revealed that the length of time between follow up assessments had a medium effect on the expression of comorbid symptoms. Post hoc analyses indicated that time had a small, yet significant, effect on the stability of three factors (i.e., ADHD, Avoidant, and Tantrum behaviors), but not the remaining two factors (i.e., Sleep and Anxiety). Descriptive statistics revealed an increase in behavior exhibition for each factor for the Time2 group. The same pattern was not observed in individuals in the Time1 group. Contrarily, item endorsements for the factors Sleep and Tantrum indicate that there was a decrease in these behaviors when follow-up assessment occurred within six to eight months.

The significant effect of time on developmental psychopathology and the observed increase for each of the five factors assessed suggests that the exhibition of comorbid symptoms is not static and may increase with time. Symptoms of comorbid psychopathology increased across time regardless of primary diagnoses. While two factors (i.e., Sleep and Tantrum) actually decreased in Time1, all five factors of the *BISCUIT-Part 2* increased in prevalence when assessed between nine and twelve months. Statistically significant differences were observed for

three factors in the Time2 group, ADHD, Anxiety, and Tantrum (Table 5). While these factors were significantly different, only a small time effect was observed.

Contrary to my hypothesis, the expression of comorbid symptoms in the PDD-NOS group was not significantly different than that seen in the Atypical group for any factor on the *BISCUIT-Part 2*. Individual analyses of the absolute differences between item responses for each factor indicated that although symptom expression for infants with PDD-NOS was not significantly different, it did reveal an overall increase in symptom expression (Table 4). A large effect of diagnosis on developmental psychopathology was observed for each of the five *BISCUIT-Part 2* factors. These findings are commensurate with previous research by Matson and colleagues (2010) who found a significantly higher prevalence of comorbid symptoms in ASD toddlers when compared to controls. Multiple comparisons for each of the five factors were conducted following significant ANOVAs. Post hoc analyses revealed that symptom stability for the factors significantly differed amongst the ASD categories. Specifically, item responses for the AD group when compared to the PDD-NOS group significantly differed for two factors (i.e., Anxiety and Sleep). Researchers have previously observed significantly higher endorsement of anxiety symptoms in AD populations when compared to a PDD-NOS group and control group using the *BISCUIT-Part 2* (Matson et al., 2010). Similarly, researchers have suggested that AD populations exhibit fears and phobias at higher rates than both typical and atypically developing peers (Evans et al., 2005; Matson & Love, 1990). Increased rates of anxiety and repetitive behaviors (i.e., tics), have also been observed in ASD populations (Attwood, 1998; Ringman & Jankovic, 2000). Post hoc analysis of item responses revealed a decrease in the expression of comorbid sleep symptoms for the AD group (Table 7).

Sleep was the only factor in the current analysis for which the observed difference between the initial and follow up assessment was positive. However, as indicated in Table 7, the average difference in item responses for the Anxiety factor within the AD group was nearly positive. The difference in expression of anxiety and sleep symptoms among the AD and PDD-NOS groups cannot be fully understood with this initial analysis. In the future researchers should explore the differences amongst these factors in depth. The remaining factors (i.e., Tantrum, Avoidant, and ADHD), did not significantly differ among ASD diagnoses. Ignoring directionality, analysis of item responses for each of the five factors of the *BISCUIT-Part 2* across two administrations were significantly less stable for the AD group when compared to the atypical group.

The observed findings suggest that the expression of comorbid symptoms by infants with AD is not static and can change across time when compared to atypically developing peers. The instability in the AD group identifies the need for reassessment/evaluation across time for this group in particular when developing and implementing intervention programs. Regardless of diagnosis, an overall increase in developmental psychopathology was observed for each factor of the *BISCUIT-Part 2* with the exception of Sleep for the AD group. Aside from this specific factor, the overall trend observed in the data indicates that the assessment and continuous monitoring of developmental psychopathology is important regardless of primary diagnosis. Research regarding the manifestation of comorbid symptoms in infants with ASD is paramount in developing and implementing proper interventions (Kim et al., 2000).

The observed outcome of the discriminant analysis indicates that one underlying dimension best captures group separation. As exemplified by a discriminant function plot (Figure 1), the presence of an ASD is likely to be the underlying dimension producing significant

differences among diagnostic groups. This observation directly supports the results of multiple post hoc comparisons which revealed significant differences between the AD and Atypical groups for each of the five factors and insignificant differences between the ASD groups for three of the five factors. The first variate has a strong positive relationship with each of the five DV's, but most notably with Anxiety and Sleep. These two factors were also the only two factors that significantly distinguished the AD group from the PDD-NOS group during post-hoc multiple comparisons. In direct contrast, the second variate affects Anxiety, Sleep, and ADHD differently from Tantrums and Avoidant symptoms. When the canonical variate correlations of DV's are high for one variate and low for another, the high correlations contribute the most to group separation (Bargman, 1970). This information suggests that the first variate affects each of the *BISCUIT-Part 2* subscales in the same manner, whereas the second variate differentiates groups on some dimension that affects Anxiety, Sleep, and ADHD differently. When the two discriminate functions were compared, only the first function significantly discriminated diagnostic group membership.

In contrast, with the stability of the core ASD symptoms observed in children with AD by multiple researchers (Landa et al., 2007; Werner et al., 2005), the current research suggests that symptoms of comorbid psychopathology are not stable, and actually increase across time regardless of diagnosis. Previous researchers have identified the negative impact of comorbid symptoms on individuals with ASDs (Gray, Ansell, Baird, & Parr 2011; Matson & Minshawi, 2007; Matson & Nebel-Schwalm, 2007). The current findings highlight the importance of the continuous monitoring of developmental psychopathology and the need for the development and implementation of specific interventions for infants and toddlers.

While behavioral interventions (i.e., EIBI) have been shown to be effective for children with an ASD, the presence of other comorbid developmental psychopathology problems requires the development of highly individualized interventions (LoVullo & Matson, 2009). Additional interventions, such as the use of psychotropic medication in combination with EIBI, may be necessary for children with ASD when comorbid disorders are present (LoVullo & Matson, 2009; Self, Hale, & Crumrine, 2010). Research surrounding the manifestation of comorbid symptoms in an infantile ASD population is critical. The presence of comorbid symptoms influence diagnostic formulation and the development of treatment plans, which may negatively impact not only the individual, but also parental stress (Gray, Ansell, Baird, & Parr 2011; Matson & Minshawi, 2007; Matson & Nebel-Schwalm, 2007). While it is not efficacious to withhold treatment from those that may benefit from it, it is equally important to understand how symptoms, especially comorbid symptoms manifest independent of treatment in order for researchers to reliably determine the efficacy of additional interventions. Outcomes of the present study suggest that the presence of comorbid symptoms do not simply dissipate as children age. Instead, these symptoms become increasingly prevalent and severe. Comparisons between the AD, PDD-NOS, and Atypical groups indicated that this increase in or worsening of comorbid symptomatology was significantly greater in those infants with AD. These findings suggest that intervention for comorbid symptoms in an infantile ASD population is a necessary venture, especially infants with AD. While differences between both time groups analyzed where only significant for the Time2, an overall positive trend in comorbid symptom expression was observed for both groups (Table 5). The findings of the current study also highlight the need for consideration of the time between initial and follow-up assessments. They suggest that the assessment of comorbid symptoms may need to occur at a later date. Dominick and colleagues

(2006) reported that half of their children with autism in their study expressed severe tantrums after the age of 3. The current findings should be considered in light of the current legislative approaches to early autism intervention. The current EarlySteps program only pertains to individuals up to 36 months. If participants are identified within the 24-36 month age range (73 of the participants in the current study) they would likely age out of the program before their presentation of comorbid symptoms was fully assessed and certainly before adequate interventions for comorbid symptoms were developed and introduced. Additionally, these factors likely interfere with the administration of other interventions (I.e., OT, PT), especially if challenging behaviors are present.

The results of this study should be confirmed by research which monitors any change in developmental psychopathology over the course of the implementation of highly individualized intervention programs. Such research may help to inform the development of future treatment approaches. The current findings may also inform the development of diagnostic and assessment measures to assist in the early detection and treatment of both ASDs and comorbid psychopathologies in toddlers. The results of the current research offer new information concerning the manifestation of psychopathology symptoms in toddlers with an ASD. Such knowledge contributes to the ongoing efforts to better understand the features and symptoms of ASDs, provide valuable information for the assessment of psychopathology in toddlerhood, and lead to the development of more efficacious and streamlined treatment approaches for early intervention in ASDs. Enhancing our understanding and ability to identify comorbid symptoms in infants with ASD remains important. Matson and colleagues (2010) suggested that the course of symptoms associated with conduct problems and behavioral challenges may be altered with early identification and intervention implementation. In an effort to include as many participants

as possible, no effort was made to control group sample size. Future research should utilize equal sample sizes and include a typically developing control group. This direct comparison may highlight the benefit of interventions for comorbid symptomology in both the ASD and Atypical groups. Researchers should continue to identify and include additional factors that may influence the expression of comorbid symptoms or predispose children with ASD to developing comorbid symptoms.

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APPENDIX: IRB APPROVAL FORMS

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

Bienville Building • 628 N. 4th Street • P.O. Box 629 • Baton Rouge, Louisiana 70821-0629
Phone # : 225/342-9500 • Fax # : 225/342-5568• WWW.DHH.LA.GOV
"An Equal Opportunity Employer"

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.
Address: 324 Audubon Hall
Department of Psychology
Louisiana State University
Baton Rouge, LA 70803
Phone: (225)578-4104
Affiliations and Education/Qualifications (attach vita if applicable): Ph.D.; Professor; Psychologist; CV attached.

Co-Investigator: Brenda Barron Sharp, M.A.
Address: Office for Citizens with Developmental Disabilities
628 North 4th St.
PO Box 3117 – Bin#21
Baton Rouge, LA 70821
Phone: (225)342-8853
Affiliations and Education/Qualifications (attach vita if applicable): M.A.; CCC-SLP; CV attached

Co-Investigator: Brandi Smiroldo, Ph.D.
Address: Office for Citizens with Developmental Disabilities
628 North 4th St.
PO Box 3117 – Bin#21
Baton Rouge, LA 70821
Phone: (225)342-0095
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.

Signature of Principal Investigator — Date: 05.26.10
Cheryl L. Knight, Ph.D.

Signature of Co-Investigator — Date: May 12, 2010
Johnny L. Matson, Ph.D.

Signature of Co-Investigator — Date: 5/26/10
Brenda Barron Sharp, M.A.

Signature of Co-Investigator — Date: 5/26/10
Brandi Smiroldo, Ph.D.

Attachment 4
UNIVERSITY FACULTY SPONSORSHIP

Project Report and Continuation Application

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

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

IRB#: 2609 Current Approval Expires On: 09/14/2012

Review Type: Expedited Risk Factor: small

PI: Johnny Matson Dept: Psychology Phone: 225-578-8745

Student/Co-Investigator:

Project Title: Developing the Autism Spectrum Disorder

Number of Subjects Authorized: 2000

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

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

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

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

III. PROTOCOL: (Check one).

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

IV. UNEXPECTED PROBLEMS: (did anything occur that increased risks to participants):
--State number of events since study inception: 0 since last report: 0
--If such events occured, describe them and how they affect risks in your study, in an attached report
--Have there been any **previously unreported** events? Yes/No: n

V. CONSENT FORM AND RISK/BENEFIT RATIO:
Do new knowledge or adverse events change the risk/benefit ratio? Yes/No: n
Is a corresponding change in the consent form needed? Yes/No: n

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

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

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

Signature of Principle Investigator: J. Matson **Date:** Sept. 4, 2012

IRB Action:
- √ Continuation approved; **Approval Expires:** 9/9/13
- ____ Disapproved
- ____ File Closed

Signed Robert Mathews Date 9/10/12

Print Form

ASD STUDY Consent Form

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

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

3. **Contacts:** Johnny L. Matson, Ph.D. (225) 578-8745 Mon-Fri

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

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

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

7. **Benefits:** Participants under the age of 18 years may benefit from this study by taking advantage of reduced price assessment services at the Psychological Services Clinic in Baton Rouge, Louisiana. If participants decide to take advantage of this offered benefit, participants will be required to come into the clinic to complete a parent interview and child observation session. If further assessment services are recommended, the participant may receive these services at half of the normal fee. All treatment services will be full price. Further, participants may benefit from professionals developing more reliable and valid assessment measures, suggesting improved diagnostic capabilities and more effective treatment interventions.

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

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

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

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

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

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

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

ASD Consent Form—Detach this page, Complete, and Return

The study has been described to me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators by contacting Megan Hattier at 225-578-1494 or asdlsu@gmail.com.

If I have questions about subjects' rights or other concerns, I can contact Robert C. Mathews, Chairman, LSU Institutional Review Board, (225) 578-8692. I agree to participate in the study described above and acknowledge the researchers' obligation to provide me with a copy of this consent form if signed by me.

_____ Parent/Guardian/Informant Signature _____ Date

_____ (Please Print Name of Parent/Guardian/Informant)

_____ Signature of Adult Participant (if applicable) _____ Date

_____ (Please Print Name of Adult Participant if applicable)

The participant has indicated to me that he/she is unable to read. I certify that I have read this consent form to the participant and explained that by completing the signature line above he/she has given permission to participate in the study.

_____ Signature of Reader _____ Date

****

PLEASE FILL OUT THE FOLLOWING CONTACT INFORMATION:
(A research assistant will contact you to obtain additional information and answer any questions you may have before mailing questionnaires or sending email link to survey)

Telephone number(s) where informant can be reached: _____

Best time of day to be reached: _____

Mailing Address: _____

Email Address: _____

Circle to indicate your preference for the question below:

INTERNET (electronic)	**MAIL (paper)**	Would you prefer to be mailed the questionnaires in paper with a prepaid envelope included OR receive an Internet link via email to the questionnaires to complete the questionnaires electronically on the Internet.

If you answered MAIL (paper), please answer the following additional questions:

YES	NO	1. Would you be willing to complete a shorter set of similar questions approximately 2 weeks after completing the first?
YES	NO	2. Is there a second adult who knows your child well (other parent,

grandparent, etc.) who would be willing to complete the questionnaires for your child independently from yourself?

YES NO 3. Do you consent to your child's teacher completing a similar set of questionnaires for your child?

Study Approved By:
Dr. Robert C. Mathews, Chairman
Institutional Review Board
Louisiana State University
203 B-1 David Boyd Hall
225-578-8692 | www.lsu.edu/irb
Approval Expires: 9/9/2013

VITA

Matthew J. Konst was born in Morganton, North Carolina, in 1987. He has worked with adults with developmental disabilities as well as adolescents with severe mental illness in residential treatment settings. He received his Bachelor of Science degree in psychology from Appalachian State University in 2011. He enrolled in Louisiana State University's Clinical Psychology Doctoral Program in 2011. 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 developmental psychopathology.