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The Baby and Infant Screen for Children with Autism Traits: Age-based Scoring Procedures

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THE BABY AND INFANT SCREEN FOR CHILDREN WITH AUTISM TRAITS: AGE-
BASED SCORING PROCEDURES

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
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in

The Department of Psychology

by
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Abstract

As increasing interest and emphasis has been placed on early intervention for children with Autism Spectrum Disorders (ASD), the need for reliable and valid early assessment techniques has grown significantly. The Baby and Infant Screen for Children with aUtism Traits (BISCUIT) is a three-part battery designed to comprehensively assess for ASD in infants and toddlers aged 17 to 37 months. While studies of the measure's psychometric properties have been promising, the measure's scoring procedures do not take the child's age into account. Given the significant amount of development that occurs in the first three years of life, the current paper examined the utility of age-based scoring procedures for each part of the BISCUIT. Study 1 found the BISCUIT-Part 1 to have good to excellent discriminating ability for each age group. As age increased, higher cutoff scores were needed to distinguish toddlers with PDD-NOS from those with atypical development. A different pattern emerged when distinguishing PDD-NOS from autism, with toddlers in the middle age cohort requiring the highest cutoffs. Studies 2 and 3 found that, for toddlers with ASD, as age increased, higher cutoffs were needed to indicate moderate and severe impairments in the areas of comorbidity and challenging behaviors, respectively. Less variation occurred with changes in age in toddlers with non-ASD related atypical development. The implications of these results, as well as possible areas of future research, are discussed.

Introduction

Autism Spectrum Disorders (ASD) are a group of neurodevelopmental disorders characterized by impairments in socialization, communication, and repetitive behaviors or restricted interests (Bodfish, Symons, Parker, & Lewis, 2000; Cederlund, Hagberg, & Gillberg, 2010; Charman, Baron-Cohen et al, 2003; Duffy & Healey, 2011; Fodstad, Matson, Hess, & Neal, 2009; Hauck, Fein, Waterhouse, & Feinstein, 1995; Horovitz & Matson, 2010; Landa, Holman, & Garrett-Mayer, 2007; Lord & Paul, 1997; Macintosh & Dissanayake, 2006; Matson, Dempsey, & Fodstad, 2009; Matson, Dempsey, & LoVullo, 2009; Matson, Mayville, Lott, Bielecki, & Logan, 2003; Militerni, Bravaccio, Falco, Fico, & Palermo, 2002; Mundy, 2003; Rutter, 1968, 1978; Sigman & Mundy, 1989; Wetherby, Watt, Morgan, & Shumway, 2007). As the rates of ASD diagnoses have increased, there has been a surge in public interest in these disorders (Center for Disease Control and Prevention [CDC], 2011; M. Evans et al., 2001; Fombonne, 2003; Wing & Potter, 2002). Increasingly, researchers and practitioners are pushing for treatment to be implemented as early as possible, due to the preliminary findings that such early intervention may lead to improved outcomes (Corsello, 2005; Eikeseth, Smith, Jahr, & Eldevik, 2007; Eldevik, Eikeseth, Jahr, & Smith, 2006; Eldevik et al., 2009; Fenske, Zalenski, Krantz, & McClannahan, 1985; Harris, Handleman, Gordon, Kristoff, & Fuentes, 1991; Howard, Sparkman, Cohen, Green, & Stanislaw, 2005; Matson, 2007; Matson & Smith, 2008; Matson, Wilkins, & González, 2008; Peters-Scheffer, Didden, Korzilius, & Sturmey, 2011; Remington et al., 2007; Shea, 2005; T. Smith, 1999; Werner, Dawson, Osterling, & Dinno, 2000). However, in order for such treatment to be appropriately disseminated, children must be accurately assessed and diagnosed at a young age. Currently, diagnoses of an ASD are not typically given until children are at least three to four years of age (DeGiacomo & Fombonne, 1998; Matson, 2005;

Tager-Flusberg & Anderson, 1992); in order for early intervention to take place, there is a need for psychometrically sound tools capable of assessing ASD before 3 years. The Baby and Infant Screen for Children with aUtism Traits (BISCUIT; Matson, Wilkins, Sevin et al, 2009) is a recently developed assessment battery designed for this purpose. The initial findings on the psychometric properties of this battery have been promising (Matson, Fodstad, & Mahan, 2009; Matson, Fodstad, Mahan, & Rojahn, 2010; Matson, Fodstad, Mahan, & Sevin, 2009; Matson, Wilkins, Sevin et al, 2009; Matson, Wilkins, Sharp et al, 2009; Rojahn et al., 2009); however, any improvements to the psychometric properties would be critical, as they would help to ensure that more children receive appropriate services at a young age. The aim of the current paper was to examine the utility of using age-based scoring procedures for the BISCUIT, given the rapid development and change that occurs in the first few years of life (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991). Age-based cutoffs may improve the psychometric properties of the measure, specifically the sensitivity and specificity, and aid it in providing a more accurate representation of the child being assessed. The diagnostic history, diagnostic criteria, core features, etiology, prevalence, and assessment of ASD are discussed, followed by a review of the research on infants and toddlers with ASD.

Autism Spectrum Disorders

Diagnostic History

Leo Kanner (1943) is typically credited as the first person to identify autism as a unique cluster of symptoms that could not be explained by any known psychiatric disorder at the time. In his seminal 1943 paper, Kanner described 11 children (eight male, three female) that exhibited severe impairments in socialization, communication, and an insistence on sameness in their routines. Although heterogeneity existed among these children, Kanner believed that the similarities suggested that the symptoms were indicative of a common pathology. As such, in a follow-up paper, Kanner (1944) referred to this pathology as “early infantile autism.”

Since Kanner’s (1943) initial description of autism, numerous researchers have put forward their own definitions and descriptions of the disorder (e.g., Creak, 1961; Ritvo, 1978; Rutter, 1968, 1972, 1978; Rutter & Bartak, 1971). Nevertheless, the three core symptoms described by Kanner (1943) have stood the test of time, as they remain the core diagnostic features outlined in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association [APA], 2000)*.

The *DSM-III* was the first of the Diagnostic and Statistical Manuals to formally include a category for autism; it was labeled Pervasive Developmental Disorders (PDDs; APA, 1980). The *DSM-III* based its definition of infantile autism on the criteria put forward by Rutter (1978), which were in turn based on the core features originally outlined by Kanner (1943). Rutter (1978) defined the key features of autism to be impairments in social relationships, delays or abnormalities in language development, and an insistence on sameness. Additionally, Rutter placed a requirement of onset of symptoms before 30 months of age.

Five disorders were initially included under this heading: Infantile autism, Residual infantile autism, Childhood onset Pervasive Developmental Disorder, Residual childhood onset Pervasive Developmental Disorder, and Atypical autism. The revised edition of the *DSM-III* changed the term Infantile autism to Autistic Disorder, in recognition that symptoms continued beyond childhood (Matson & Minshawi, 2006; Tidmarsh & Volkmar, 2003). Additionally, the residual diagnoses were removed, criteria were broadened, and the term Atypical autism was changed to Not Otherwise Specified (Matson & Minshawi, 2006; Tidmarsh & Volkmar, 2003). Of note, researchers have begun using the term ASD, rather than PDD, to reference the fact that these disorders occur across a spectrum or continuum (Boisjoli & Matson, in press; Matson, Wilkins, Boisjoli, & Smith, 2008; Wing, 1997).

Current and Future Diagnostic Criteria

Two classification systems are primarily used today in the diagnosis of mental health disorders: the *Diagnostic and Statistical Manual, Fourth Edition, Text Revision (DSM-IV-TR;* APA, 2000) and the *International Statistical Classification of Diseases and Health Related Problems, 10th edition (ICD-10;* World Health Organization [WHO], 1992). The *DSM-IV-TR* criteria for the PDDs were developed based on a field trial using criteria from the *ICD-10*, *DSM-III* and *DSM-III-R* (Volkmar et al., 1994). As the criteria used by the *DSM-IV-TR* and the *ICD-10* are very similar, and the *DSM-IV-TR* is the more widely cited and used instrument in the United States, the *DSM-IV-TR* will be the focus of this paper (Matson & Minshawi, 2006; Tidmarsh & Volkmar, 2003).

The *DSM-IV-TR*, within the section Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence, includes a section titled Pervasive Developmental Disorders. Five disorders currently fall within this heading: Autistic Disorder, Asperger's Disorder, Rett's

Disorder, Childhood Disintegrative Disorder (CDD), and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). The common bonds linking these disorders are the impairments, or core features, first noted by Kanner (1943) and later emphasized by Rutter (1978): deficits in social interaction, impaired communication, and the presence of repetitive, restricted, and stereotyped behaviors, interests and activities. However, it should be noted that impairments in all three areas are not required for each of the PDDs, as will be discussed further below with respect to PDD-NOS. As the current study will focus on children with diagnoses of Autistic Disorder and PDD-NOS, the current *DSM-IV-TR* criteria for these disorders will be discussed, followed by a discussion of proposed revisions to be included in the *DSM-V*.

Autistic Disorder. Autistic Disorder is often referred to as “early infantile autism,” “childhood autism,” “Kanner’s autism,” or simply “autism” (APA, 2000). These names reflect the antiquated belief that the disorder only occurred in children and the fact that Autistic Disorder is the term currently used to describe the syndrome first described by Kanner (APA, 2000; Kanner, 1943; Matson & Minshawi, 2006). In this paper, the term autism refers specifically to Autistic Disorder, whereas the terms ASD and PDD refer to the broader spectrum of disorders. Current criteria require children with autism to exhibit impairments across all three of the core features (APA, 2000; Fodstad et al., 2009; Horovitz & Matson, 2010; Matson et al., 1996; Matson, Dempsey, & Fodstad, 2009; Matson, Dempsey, & LoVullo, 2009; Matson, Wilkins, & Ancona, 2008; J. A. Sevin et al., 1995; Wilkins & Matson, 2007). The criteria require at least two impairments in social interaction, at least one impairment in communication, and at least one restricted, repetitive, and/or stereotyped pattern of behavior, interest, and/or activities, with a total of at least six impairments noted. Possible impairments in social interaction include: (1) Impairment in the use of multiple nonverbal behaviors; (2) Failure to develop peer

relationships commensurate with developmental level; (3) A lack of spontaneous seeking to share enjoyment, interests, or achievements with others; and (4) A lack of social or emotional reciprocity. Possible impairments in communication include: (1) Delay in, or lack of, the development of spoken language, without attempts to compensate through alternative forms of communication; (2) Impairment in the ability to initiate or sustain conversations with others; (3) Stereotyped and repetitive use of language or idiosyncratic language; and (4) A lack of spontaneous make-believe or social imitative play commensurate with developmental level. Possible restricted, repetitive, and stereotyped patterns of behavior, interests, and activities include: (1) preoccupation with one or more stereotyped and restricted pattern of interest that is abnormal in intensity or focus; (2) Inflexible adherence to specific, nonfunctional routines or rituals; (3) Stereotyped and repetitive motor mannerisms; and (4) Persistent preoccupation with parts of objects. In addition to these criteria, symptoms in at least one area must begin before 3 years of age. Finally, the symptoms cannot be better explained by a diagnosis of Rett's Disorder or Childhood Disintegrative Disorder.

PDD-NOS. While the *DSM-IV-TR* criteria for autism are fairly clear and straightforward, the same cannot be said for PDD-NOS. Despite being the most common of the PDDs (Buitelaar & Van der Gaag, 1998; Chakrabarti & Fombonne, 2005; Matson & Boisjoli, 2007; Mayes, Volkmar, Hooks, & Cicchetti, 1993), no specific rules are given by the *DSM-IV-TR* for diagnosing PDD-NOS (APA, 2000). Instead, the *DSM-IV-TR* (APA, 2000) states that the diagnosis should be given “when there is a severe and pervasive impairment in the development of reciprocal social interaction associated with impairment in either verbal or nonverbal communication skills or with the presence of stereotyped behaviors, interests, and activities...”

(p. 84). It is additionally stipulated that criteria cannot be met for another PDD, Schizophrenia, Schizotypal Personality Disorder, or Avoidant Personality Disorder.

Based on this definition, many authors have described PDD-NOS as a sort of catchall category for children who have symptoms of ASD but do not meet criteria for any of the other four ASD (Matson & Boisjoli, 2007; Matson & Minshawi, 2006; Tidmarsh & Volkmar, 2003). However, this definition has made it difficult to reliably and validly assess for and diagnose the disorder (Lord, Rutter, & Couteur, 1994; Matson & Boisjoli, 2007; Matson, Gonzalez, Wilkins, & Rivet, 2008; Mayes et al., 1993). Given the ambiguity of the criteria and the problems arising from this ambiguity, multiple authors have attempted to more clearly define the disorder and explain its current use. For example, Buitelaar and Van der Gaag (1998) suggested four situations in which a diagnosis of PDD-NOS may currently be given: (1) Age of onset after three years; (2) Presence of atypical symptoms that do not fit with current *DSM-IV-TR* criteria for other ; (3) A presentation just missing threshold for autism, such as having five of the required symptoms rather than six; and (4) Failing to meet the pattern of symptoms required for a diagnosis of autism (e.g., impairments in social interaction and restricted/repetitive behaviors, but no communication impairments). At present, no clear definition of PDD-NOS exists; however, most researchers generally consider PDD-NOS as a lesser variant of autism (Boisjoli & Matson, in press; Buitelaar, Van der Gaag, Klin, & Volkmar, 1999; Horovitz & Matson, 2010; Matson & Boisjoli, 2007).

DSM-V. The release of the fifth edition of the *DSM* in 2013 will bring about further changes in the conceptualization and definition of ASD. Firstly, it has been proposed that the term Pervasive Developmental Disorders be replaced with Autism Spectrum Disorder, reflecting the current trends in the literature (APA, 2011). As currently proposed, only one diagnosis will

fall under this heading, albeit with varying levels of severity. Thus, individual diagnoses, such as PDD-NOS and Asperger's Disorder will no longer be given. To meet criteria for ASD, individuals will be required to manifest each of the following three deficits in social communication and social interaction: (1) Deficits in social-emotional reciprocity; (2) Deficits in nonverbal communicative behaviors used for social interaction; and (3) Deficits in developing and maintaining relationships appropriate to developmental level. Additionally, individuals will be required to evince at least two of the following restricted, repetitive patterns of behavior, interests, or activities: (1) Stereotyped or repetitive speech, motor movements, or use of objects; (2) Excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change; (3) Highly restricted, fixated interests that are abnormal in intensity or focus; or (4) Hyperreactivity or hyporeactivity to sensory input or an unusual interest in sensory aspects of the environment. Additionally, symptoms must be present in early childhood (no exact age is given), and symptoms must cause impairments in everyday functioning. Those meeting criteria will then have their severity level for ASD rated for social communication and restricted interests and repetitive behaviors. Level 3 will indicate, "requiring very substantial support," Level 2 will indicate, "requiring substantial support," and Level 1 will indicate, "requiring support."

A number of things stand out when examining these proposed changes. Firstly, the three core features will be collapsed into two, with communication impairments primarily being understood within the framework of social impairments. Reasons given by the APA for this change include the difficulty in separating deficits in communication from deficits in social behaviors and the fact that delays in verbal language are neither unique to nor universal in ASD (APA, 2011). Additionally, unusual sensory behaviors are now to be included in the formal

diagnostic criteria. While these behaviors were included in the criteria put forth by Ritvo and the NASC (Ritvo, 1978), this will mark the first time they have been included as formal diagnostic criteria in the *DSM*. A final striking feature is the removal of subgroups within the category of ASD. While there has been a great deal of debate in the literature regarding the validity of the current diagnoses that fall under the category of ASD (Boisjoli & Matson, in press; Matson, Nebel-Schwalm, & Matson, 2007; Matson, Wilkins, Boisjoli et al, 2008; Wing, Gould, & Gillberg, 2011; Worley & Matson, 2012), it is possible that the abolition of these diagnoses may create more problems than it solves (Wing et al., 2011). Much debate has already arisen regarding these changes (Kaland, 2011; Wing et al., 2011; Worley & Matson, 2012). For example, under the proposed more stringent criteria, individuals with less severe or atypical symptoms may no longer receive a diagnosis (Wing et al., 2011; Worley & Matson, 2012). Thus, the proposed revisions may reduce the sensitivity of the diagnostic criteria (Worley & Matson, 2012). The debate over the proposed revisions is likely to continue long after the release of the *DSM-V*.

Core Features

Social Skills. Impairments in social skills are generally considered to be the primary feature of ASD (B. M. Sevin, Knight, & Braud, 2007; Volkmar, Cohen, Bregman, Hooks, & Stevenson, 1989; Walters, Barrett, & Feinstein, 1990). While all children with ASD share impairment in this area, these impairments may manifest in a number of ways. Impairments may exist in areas such as initiation of interaction with others (Hauck et al., 1995), peer relationships (Clifford, Young, & Williamson, 2007; Matson, Fodstad, & Dempsey, 2009b; Matson, Wilkins, Sevin et al, 2009; Ventola et al., 2007), facial expressions (Matson, Fodstad, & Dempsey, 2009b; Ventola et al., 2007; Wetherby et al., 2007), use of nonverbal gestures (Matson, Fodstad, &

Dempsey, 2009b; Wetherby et al., 2007), anticipatory posture (Clifford et al., 2007), empathy (Charman et al., 1997), perception of emotions in others (Golan, Baron-Cohen, Hill, & Rutherford, 2007; Travis & Sigman, 1998), joint attention skills (Charman et al., 1997; Clifford et al., 2007; Naber et al., 2008; Ventola et al., 2007; Wetherby et al., 2007), use of eye gaze (Clifford et al., 2007; Matson, Dempsey, & Fodstad, 2009; Ruffman, Garnham, & Rideout, 2001; Rutter, 1978; Wetherby et al., 2007; Wetherby et al., 2004), sharing of interests and activities with others (Matson, Wilkins, Sevin et al, 2009), and imitation (Charman et al., 1997; I.M. Smith & Bryson, 1994). Clearly, a great deal of heterogeneity exists with respect to what constitutes social impairments in individuals with ASD.

These social deficits may have a number of negative consequences, particularly a lack of appropriate peer relationships (Travis & Sigman, 1998). While children with ASD often form attachments with others, particularly caregivers, the quality of these attachments are typically impaired (Naber et al., 2007; Sigman & Mundy, 1989; Travis & Sigman, 1998). Children with ASD therefore often have difficulty playing with others and developing meaningful relationships (Rutter, 1978). While social relationships may improve somewhat over time, impairments in this domain tend to remain stable over time (Matson & Horovitz, 2010; Travis & Sigman, 1998).

Communication Skills. While impairments in communication skills are currently included as one of the core features, they will be combined with social skills and labeled social communication and social interaction in the upcoming release of the *DSM-V* (APA, 2011). As previously discussed, it has been argued that it is difficult to differentiate between the communication impairments found in children with ASD and the other core features, primarily social skills (APA, 2011). Many of the communication impairments found in ASD are described in a social context. While delays in the development of verbal speech, or the complete absence of

verbal speech, are common in children with ASD, this feature is neither unique to ASD nor universal within ASD (APA, 2011; Fodstad et al., 2009; Horovitz & Matson, 2010; Matson, Fodstad, & Dempsey, 2009b; Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978). Despite this, delays in the development of communication and language skills are typically the first concerns noted by parents of children with ASD (DeGiacomo & Fombonne, 1998; Howlin & Moore, 1997; Kozlowski, Matson, Horovitz, Worley, & Neal, 2011). It should, however, be noted that the same is true of children with developmental disorders (DD) that are not an ASD (Kozlowski et al., 2011). Thus, while it is important to understand the communication impairments of individuals with ASD, they cannot be viewed in a vacuum; rather it is imperative that they be interpreted within the context of the other core features of ASD.

It is estimated that between 33 to 50% of children with ASD never develop meaningful, functional speech (Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978). Among those who do develop functional speech, a number of oddities and deficits exist. As first outlined by Kanner (1943), the speech of children with ASD is often characterized by immediate and delayed echolalia, or repetition of stereotyped words and phrases (Eveloff, 1960; Folstein, 1999; Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978). Echolalia can at times be used non-socially, whereas at other times it may serve a meaningful function (Folstein, 1999). In the latter cases, individuals with ASD often fail to use the correct pronouns, another feature common in this population (Folstein, 1999; Kanner, 1943; Rutter, 1978). Even when speech is used meaningfully, it is typically used to express needs, rather than to socialize (Folstein, 1999). When children with ASD do use speech socially, they tend to veer off topic more frequently than children with other DD (Tager-Flusberg & Anderson, 1992). Impairments also exist with respect

to the quality of speech produced by children with ASD. Abnormalities are common with respect to quality of voice, inflection, stress pattern, and syntax (Noens & Van Berckelaer-Onnes, 2005).

In addition to difficulties with verbal communication, children with ASD also exhibit significant impairments in non-verbal communication. Whereas children with speech delays typically compensate for their deficits with non-verbal skills (e.g., pointing, facial expressions, use of eye contact), the same is not typical of children with ASD (Noens & Van Berckelaer-Onnes, 2005; Shumway & Wetherby, 2009; Stone, Ousley, Yoder, Hogan, & Hepburn, 1997; Wetherby et al., 2007). Instead, children with ASD often use atypical forms of nonverbal communication, such as direct manipulation of others' hands or attempting to use others' hands as a tool (Matson, Wilkins, Sevin et al, 2009; Matson, Wilkins, Sharp et al, 2009; Shumway & Wetherby, 2009; Stone et al., 1997). When compared to children with DD, children with ASD make use of fewer social forms of non-verbal communication, such as protodeclarative pointing (Shumway & Wetherby, 2009; Stone et al., 1997; Wetherby et al., 2007).

Finally, the communication impairments of children with ASD also extend to receptive communication (Charman, Drew, Baird, & Baird, 2003; Luyster, Kadlec, Carter, & Tager-Flusberg, 2008; Noens & Van Berckelaer-Onnes, 2005; Ungerer & Sigman, 1987). Responses to common phrases are often impaired, and receptive communication may be even more delayed than expressive communication (Charman, Drew et al, 2003). As with the expressive communication impairments, there is often a social aspect to the receptive communication impairments of children with ASD. For example, children with autism have failed to show the expected preference for their mother's voice over general noises found in their environment (Klin, 1991). Similarly, children with autism often fail to respond or orient when their name is called (Baranek, 1999; Guinchat et al., 2012; Muratori, Apicella, Muratori, & Maestro, 2011)

Restricted/Repetitive Behaviors and Interests. The final core feature of ASD includes restricted or repetitive behaviors or interests, oftentimes referred to as RRBI or stereotypy. This feature may be the most varied, with a multitude of symptoms falling under this category. Common topographies include abnormal sensory responses (e.g., licking, sniffing, self-injurious behavior, unusual response to lights and sounds), stereotypical movements (e.g., rocking, mid-line hand movements, hand flapping), insistence on sameness, need for strict routines, complex motor sequences, repetitive use of words or phrases, compulsions, tics, preoccupation with different topics or objects, and much more (Bodfish et al., 2000; Matson, Dempsey, & Fodstad, 2009; Matson, Wilkins, Sevin et al, 2009; Matson, Wilkins, Sharp et al, 2009; Militerni et al., 2002; Rutter, 1978; Rutter & Bartak, 1971).

RRBI are often separated into two categories: lower-order RRBI and higher-order RRBI (Bishop, Richler, & Lord, 2006; Hattier, Matson, Tureck, & Horovitz, 2011). Lower-order RRBI include behaviors such as sensory responses or stereotypical movements and occur more frequently in younger children with ASD and those with lower IQ levels (Bishop et al., 2006; Hattier et al., 2011; Militerni et al., 2002; Rutter, 1978; Turner, 1999). Conversely, higher-order RRBI include behaviors such as insistence on sameness and preoccupations occur more frequently in older individuals with ASD and those with higher IQ levels (Bishop et al., 2006; Hattier et al., 2011; Militerni et al., 2002; Rutter, 1978; Turner, 1999). As RRBI commonly occur in young typically developing children and individuals with ID, they are not unique to ASD (D.W. Evans et al., 1997; Foster, 1998; Morgan, Wetherby, & Barber, 2008; Richler, Bishop, Kleinke, & Lord, 2007; Thelan, 1998; Tröster, 1994). However, they occur at higher rates and intensities in individuals with ASD (Bodfish et al., 2000; Matson, Dempsey, & Fodstad, 2009; Morgan et al., 2008; Richler et al., 2007). RRBI can significantly interfere with a

child's daily functioning and impair the ability to learn new skills (Morrison & Rosales-Ruiz, 1997). Additionally, a negative relationship has been found between engaging in RRBI and engaging in play activities (Honey, Leekam, Turner, & McConachie, 2007).

Etiology

The etiology of ASD has caused considerable controversy (Matson & Minshawi, 2006). Kanner first suggested the possibility that the personalities of parents were at least partially responsible for their children's development of autism (Kanner, 1943). Kanner noted that few of the parents were warmhearted and that they showed limited social interaction. Bruno Bettelheim furthered this idea, claiming that cold, unloving mothers were the cause of autism (Bettelheim, 1967). He coined the term "refrigerator mothers" to refer to these mothers, and his ideas were widely accepted, due to the prevailing psychodynamic theory of the time period (Bettelheim, 1967; Matson & Minshawi, 2006). While this theory caused significant distress to parents of children with autism, it is not supported by the literature, and instead has been replaced primarily by genetic, neurobiological, and learning theories (Matson & Minshawi, 2006).

One of the first studies to discount the "refrigerator mother" theory was a small sample study conducted by Folstein and Rutter (Folstein & Rutter, 1977a, 1977b). Folstein and Rutter began by countering the evidence that had widely been used to discount genetic theories of autism. They argued that the lack of evidence from family studies was faulty, as few individuals with autism develop romantic relationships, and it is very rare for individuals with autism to have children. Additionally, they argued that the seemingly low rate of ASD found in siblings was actually quite high when compared to rates in the general population. In their study, Folstein and Rutter compared the rates of autism in 11 monozygotic (MZ) and 10 dizygotic (DZ) twins (Folstein & Rutter, 1977b). Concordance for autism was found in four of the MZ pairs, whereas

concordance was found in none of the DZ pairs. Additionally, the increased concordance in MZ pairs extended to broader difficulties, such as cognitive and social deficits. These differences in concordance, both for the actual disorder and for broader symptoms, provided strong preliminary support for a genetic influence.

Similar findings were later found by follow-up studies in Scandinavia (Steffenburg et al., 1989) and Great Britain (Bailey et al., 1995). The British twin study (Bailey et al., 1995) was critical, as it included total population screening and used standardized methods of diagnosis. Like previous studies, the authors found a large discrepancy in concordance for both autism (60% MZ versus 0% DZ) and broader difficulties (92% MZ versus 10% DZ). When combining the rates of concordance in all non-MZ pairs, thereby including non-twin siblings, they found a rate of 5%. Based on their findings, the authors suggested that heritability of autism likely exceeds 90%. Additionally, the pattern of findings suggested that multiple genes, rather than one single gene, contribute to the development of autism, a finding that has been supported by later researchers (Pickles et al., 2000; Risch et al., 1999).

Support for a genetic influence of autism has been further strengthened by family studies. The Maudsley Hospital family study examined 99 families of individuals with autism and compared them to 36 families of individuals with Down syndrome (Bolton et al., 1994). The rate of autism in siblings of individuals with autism was 3% (6% for a broader ASD) compared to 0% in siblings of individuals with Down syndrome. Similar to the findings of the twin studies, symptoms of the broader phenotype of autism were more common in the families of individuals with autism. Szatmari and colleagues (2000) further examined this broader phenotype by comparing biological and non-biological relatives of individuals with autism. They found all aspects of the broader phenotype (i.e., social, communicative, and repetitive behaviors) to be

more common in biological relatives. Taken together, the twin studies and family studies provide compelling evidence for the primary role of genetics in etiology. Additionally, they suggest the contribution of multiple genes, resulting in a great deal of heterogeneity and a broader autism phenotype that is often found in family members who do not have the disorder (Bailey et al., 1995; Bolton et al., 1994; Folstein & Rutter, 1977a, 1977b; Pickles et al., 2000; Risch et al., 1999; Steffenburg et al., 1989; Szatmari et al., 2000).

Although it is clear that genetics play a strong role in the etiology of autism, it cannot be the sole explanation, as evidenced by results from twin and family studies. There have therefore been many attempts to identify non-genetic risk factors for autism. Unfortunately, these attempts have led to a great deal of misguided claims. The proposed risk factor that has been the most controversial has been the Measles Mumps Rubella (MMR) vaccination. Many parents and non-professionals have come to believe that the MMR vaccination can directly cause autism. In a study on parental beliefs about the causes of autism, researchers found that 29% cited immunizations as the cause of their child's autism, compared to 26% citing genetic predisposition (Harrington, Patrick, Edwards, & Brand, 2006). The belief that autism could be caused by vaccinations, particularly the MMR vaccination, originated from a work by Wakefield and colleagues (1998), where 12 children with ASD were examined due to gastrointestinal problems. Based on inadequate evidence, it was suggested that autism could be caused by side effects from the MMR vaccination. This was based on a correlation between the timing of the MMR vaccination and the time at which symptoms of autism first become apparent (Rutter, 2005). Although the findings were based on faulty evidence and a small, biased sample, many media outlets began popularizing the possible dangers of the MMR vaccination, leading many parents to become fearful about giving their children the vaccine (M. Evans et al., 2001). In the

previously cited study on parental beliefs about the etiology of autism, 13% of parents reported that they would refuse some or all vaccinations for their children (Harrington et al., 2006). Multiple empirical studies have discredited the link between the MMR vaccination (and others) and autism, and it is now widely accepted within the scientific community that no such link exists (Honda, Shimzu, & Rutter, 2005; Madsen et al., 2002; Madsen et al., 2003; Rutter, 2005; Smeeth et al., 2004). Additionally, the *Lancet*, which originally published the article, has since retracted the original article by Wakefield et al. (1998).

Aside from vaccinations, other non-genetic risk factors have been proposed. For example, a relationship has been seen between obstetric complications and autism (Rutter, 2005). Although such a relationship does exist, it is now believed that obstetric complications represent either an epiphenomenon or are derived from a shared risk factor, rather than playing an etiological role (Bolton et al., 1997). Other possible environmental factors proposed have included yeast infections, gluten, environmental pollutants, antibiotics, hypothyroidism, and maternal substance use. While it is possible that some of these factors play a partial role in individual cases, these explanations have not been validated empirically (Herbert, Sharp, & Gaudiano, 2002; Rutter, 2005; Wing & Potter, 2002).

Prevalence

As with etiology, the issue of prevalence has been a highly debatable topic, particularly over recent years. Current estimates of the prevalence of ASD in the United States are approximately 1 in every 110 children, according to the CDC (2011). In a review of epidemiological studies, Fombonne estimated the overall rate of ASD to be approximately 60/10,000 (or 1 in ~166) (Fombonne, 2003). Estimates of the prevalence of autism ranged from .7/10,000 to 72.6/10,000 with the best estimate of current prevalence being between

approximately 10 children per 10,000 (Fombonne, 2003). These numbers, and those found by other researchers, represent an increase in reported prevalence (Fombonne, 2003; Wing & Potter, 2002). The meaning of this prevalence increase has been the source of much of the debate. Possible explanations for the rise include environmental factors such as gluten, environmental pollutants, antibiotics, mercury, yeast infections, and as previously discussed, vaccinations; however, none of these hypotheses have been supported in the literature (Herbert et al., 2002; Wing & Potter, 2002).

Researchers have found more plausible explanations for the increasing prevalence. These include a broadening of diagnostic criteria, changes in definitions, different methods used to calculate prevalence, diagnosis of milder forms of ASD, recognition that ASD and ID can occur comorbidly, increasing parental awareness, and the development of autism specialists (Fombonne, 2001; Matson & Minshawi, 2006; K. Williams, Mellis, & Peat, 2005; Wing & Potter, 2002). Additionally, Fombonne (2003) stresses the importance of distinguishing prevalence from incidence. Whereas prevalence refers to the number of cases at a given time, incidence refers to the number of new cases. While researchers have found support for the increasing prevalence of ASD, they have found little support for an increase in incidence (Fombonne, 1996, 2001, 2003). Thus it is inaccurate to say that there is currently an autism epidemic, as is often heard in the mainstream media (Fombonne, 1996, 2001, 2003). It should be noted that a number of methodological shortcomings limit our current understanding of trends in both prevalence and incidence of ASD (Fombonne, 1996, 2001, 2003). Until these can be overcome, much more research will be needed to truly understand what changes, if any, have occurred in rates of ASD over time.

Assessment

Since Kanner's (1943) original description of autism, there have been many attempts to develop psychometrically sound measures capable of assessing for and diagnosing autism and other ASD. A brief overview will be given of the most influential of these measures. One of the first such attempts was by Rimland (Rimland, 1964, 1971), with Diagnostic Forms E-1 and E-2. Form E-1 included 76 questions about symptoms, speech characteristics, age of onset, and more (Rimland, 1964). Form E-2 added questions about social interaction, speech and motor abilities, development of the problems, intelligence, and reactions to sensory stimuli (Rimland, 1971). Critiques of this measure include the lack of objective definitions and the sole reliance on parent report (DeMyer, Churchill, Pontius, & Gilkey, 1971; Masters & Miller, 1970; Matson & Minshawi, 2006). As such, the reliability and validity of this measure have been questioned.

The Autism Behavior Checklist (ABC) was developed to screen for autism in individuals aged 18 months to 35 years and to differentiate autism from other disorders characterized by abnormal behaviors (Krug et al., 1980; Lord & Risi, 1998; Matson & Minshawi, 2006). The ABC includes 57 questions collecting information on five symptom areas: sensory, relating, body and object use, language, and social skills (Krug et al., 1980). Raters endorse items as "Yes" or "No" and then assign weights ranging from 1 to 4, with higher scores indicating greater impairment. Scores greater than 68 fall in the high probability range, while scores from 54 to 67 indicate some behaviors consistent with autism. While the scale's authors found high inter-rater reliability, concurrent validity, and criterion validity, more recent studies have found these rates to be lower, particularly in higher functioning individuals (J. A. Sevin, Matson, Coe, Fee, & Sevin, 1991; Volkmar et al., 1988). It has thus been suggested that the measure be primarily used to screen for autism, rather than to diagnose (Volkmar et al., 1988). As more psychometrically

sound measures have been developed for this purpose, the ABC is no longer commonly used (Matson & Minshawi, 2006).

The Childhood Autism Rating Scale (CARS) is one of the most commonly used scales today. The CARS was developed as a part of the Treatment and Education of Autistic and related Communication handiCapped cHildren (TEACCH) program for children over two years of age with autism and their families (Schopler, Reichler, DeVellis et al, 1980). The measure was developed due to a belief that no other measure adequately assessed for autism (Schopler, Reichler, DeVellis et al, 1980). The CARS incorporates the criteria put forward by Kanner (1943) and Creak (1961), in addition to other symptoms associated with autism. The CARS includes 15 scales independently scored, through observation and parent report, from 1 (*normal*) to 4 (*severely abnormal*): impairment in human relationships; imitation; inappropriate affect; bizarre use of body movement and persistence of stereotypes; peculiarities in relating to nonhuman objects; resistance to environmental change; peculiarities of visual responsiveness; peculiarities of auditory responsiveness; near receptor responsiveness; anxiety reaction; verbal communication; nonverbal communication; activity level; intellectual functioning; and general impressions. Individual scores are added to obtain a total score, with a score above 30 indicating the presence of autism.

Inter-rater reliability and internal consistency were established by the authors at .71 and .94, respectively (Schopler, Reichler, DeVellis et al, 1980). Additionally a correlation of $r = .80$ was obtained between CARS scores and an independent clinical assessment by a psychiatrist or psychologist (Schopler, Reichler, DeVellis et al, 1980). The validity of the CARS was established in multiple settings (Schopler, Reichler, & Renner, 1980). As with the ABC, researchers have more recently suggested that the reliability may be somewhat lower than

initially reported (J. A. Sevin et al., 1991). Strengths of the measure include its psychometric properties, its large research base, and its validation in multiple settings. One important limitation is that the CARS was developed before current *DSM-IV-TR* criteria were established, and thus does not fall in line with current accepted diagnostic criteria (Klinger & Renner, 2000; Lord & Risi, 1998; Matson & Minshawi, 2006). Additionally, the CARS is not able to differentiate among different ASD (Klinger & Renner, 2000). An updated version of the CARS, the CARS2 (Schopler, Van Bourgondien, Wellman, & Love, 2010), has recently been released, although little research on this update has been published.

The Autism Diagnostic Interview – Revised (ADI-R; Lord et al., 1994) is a structured interview designed to establish clinical diagnoses of autism (Cohen, 2003; Constantino et al., 2003; La Malfa et al., 2007; Mazurek, Kanne, & Miles, in press). The ADI-R is a revision to the original Autism Diagnostic Interview (ADI), incorporating more up-to-date research on diagnosing autism. Revisions were intended to improve the measure’s ability to differentially diagnose autism from other conditions, such as ID, and to lower the age range to three years. More recently, a new scoring algorithm was developed for toddlers aged 12 to 47 months, allowing for even younger ages of administration (Kim & Lord, 2012). The ADI-R is a clinical interview comprised of five sections: opening questions; communication; social development and play; repetitive and restricted behaviors; and general behavior problems. Inter-rater reliability for these sections ranged from .62 to .89 (Lord et al., 1994). The ADI-R is one of the most widely used and accepted diagnostic instruments for children with ASD and is often described in the literature as a gold standard (Cohen, 2003; Constantino et al., 2003; La Malfa et al., 2007; Mazurek et al., in press). However, the measure is not without flaws. Firstly, despite attempts to shorten the measure, it remains very lengthy and can require up to three hours to

administer. Additionally, it does not have an observation component, relying solely on parent report. The psychometric properties of the ADI-R have also been called into question (Matson, Nebel-Schwalm et al, 2007). Finally, the ADI-R is not capable of differentiating among ASD, as it was specifically designed for the criteria of autism (Cox et al., 1999). Given these flaws, it has been stressed by many that the gold standard of autism assessment does not rest in one single measure, such as the ADI-R; instead, it should be based on clinical information from multiple informants, via multiple methods, with one component being a psychometrically sound diagnostic measure (Cox et al., 1999; Matson, Nebel-Schwalm et al, 2007; Reilly, 2009; Woolfenden, Sarkozy, Ridley, & Williams, in press).

As previously discussed, one weakness of the ADI-R is the lack of an observation component. The Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000) was created to address this weakness. The ADOS is a standardized observation measure of social behavior, communication, and play in school-aged children suspected of having an ASD (Lord et al., 2000). The most recent revision of the measure, the ADOS-G, is a 30-minute, semi-structure assessment that places individuals in specific situations designed to prompt social and/or communicative responses. Items are scored from 0 (*no evidence of abnormality related to autism*) to 2 (*define evidence*), with a code of 3 indicating abnormalities that interfered with the ability to complete the observation. One advantage of the ADOS-G over previous versions is its validity with individuals with varying levels of expressive language, thereby reducing the overdiagnosis of children with poor expressive language. The authors of the scale found it to have excellent inter-rater reliability, internal consistency, and test-retest reliability (Lord et al., 2000). As the standardization sample included children with PDD-NOS, the ADOS-G is capable of differentiating children with autism or PDD-NOS from individuals who do not have an ASD

(Lord et al., 2000); however, like the ADI-R, the ADOS-G cannot differentiate among the different ASD (Lord et al., 2000). As with the ADI-R, the ADOS-G requires extensive training, experience, and time to administer (Bishop, Luyster, Richler, & Lord, 2008; Lord et al., 2000; Matson, Rieske, & Tureck, in press). Additionally, the ADOS-G only measures current functioning, and thus does not take a lifespan approach.

Of the instruments discussed thus far, none are capable of differentiating between ASD. The Autism Spectrum Disorders-Diagnostic for Adults (ASD-DA) was developed to be capable of achieving this differentiation in adults with intellectual disability (Matson, Wilkins, Boisjoli et al, 2008). The scale assesses for autism, PDD-NOS and Asperger's Disorder and is one of the first scales developed for use with adults, rather than children. The scale is made of 31 items that ask the informant to compare the individual to others living in the community. Items are scored as 0 (*not different, no impairment*) or 1 (*different, some impairment*). The measure is reported to have adequate inter-rater and test-retest reliability, in addition to high internal consistency (Matson, Wilkins, & González, 2007).

After its development, the ASD-DA was adapted for use with children and the new scale was named the Autism Spectrum Disorder-Diagnostic for Children (ASD-DC; Matson, Gonzalez, Wilkins et al, 2008). The ASD-DC was designed for use with children with or without intellectual disability, and like the ASD-DA, assesses for autism, PDD-NOS, and Asperger's Disorder. Forty items are scored by caregivers in comparison to other children in the community as 0 (*not different, no impairment*), 1 (*somewhat different, mild impairment*), or 2 (*very different, severe impairment*). The authors reported internal consistency of .99, inter-rater reliability of .67, and test-retest reliability of .77 (Matson, Gonzalez, Wilkins et al, 2008). When validated against *DSM-IV* and *ICD-10* criteria, the ASD-DC had an overall correct classification rate of 91.3%. In

addition to strong psychometric properties, the ASD-DC and ASD-DA share a number of strengths. Both measures are brief and can be quickly administered to a parent or caregiver (Matson, Gonzalez, Wilkins et al, 2008; Matson, Wilkins, Boisjoli et al, 2008). Additionally, both measures are included as part of a larger battery that assess for comorbid psychopathology and challenging behaviors, important conditions that are common in individuals with ASD but rarely included in diagnostic instruments (Matson, Wilkins, Sevin et al, 2009). Like the ADI-R, a weakness of these measures is their reliance on parent or caregiver report, without an observation component, although such a component is currently under development (Neal, Matson, & Belva, 2013; Neal, Matson, & Hattier, in press).

Infants and Toddlers

Early Development of Psychopathology

Before discussing the specific early development of children with ASD, a general discussion of the development of psychopathology in infants and toddlers is warranted. As mentioned previously, within the *DSM-IV-TR* (APA, 2000), ASD fall under a larger class of disorders labeled Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence. This reflects the fact that ASD are typically first diagnosed when children are three to four years old (DeGiacomo & Fombonne, 1998; Kozlowski et al., 2011; Matson, 2005; Tager-Flusberg & Anderson, 1992) and that symptoms are thought to emerge in the first year of life (DeGiacomo & Fombonne, 1998; Dixon, Granpeesheh, Tarbox, & Smith, 2011; Maestro et al., 2005; Ornitz, Guthrie, & Farley, 1977). Other disorders included under this heading include ID (referred to as mental retardation in the *DSM-IV-TR*); learning disorders; motor skills disorders; communication disorders; attention-deficit and disruptive behavior disorders; feeding disorders; tic disorders; elimination disorders; and other disorders of infancy, childhood, or adolescence (e.g., separation anxiety disorder, stereotypic movement disorder; APA, 2000).

Lyons-Ruth, Zeanah, and Benoit (2003) explain two approaches towards viewing mental health problems in infants and toddlers. The first approach aims to examine intrinsic (e.g., biological) and extrinsic (e.g., social) risk and/or protective factors that increase or decrease the likelihood of later psychopathology. The other approach looks at whether or not infants and toddlers meet criteria for specific psychopathology. Both of these approaches will be briefly discussed.

A number of variables have been studied as potential risk and/or protective factors for the later development of psychopathology. Among these factors, one that has gained increased

attention is the early relationships of the infant, particularly with his or her mother (Lyons-Ruth et al., 2003; Mäntymaa, Puura, Luoma, Salmelin, & Tamminen, 2004; Zeanah, Boris, & Larrieu, 1997). Mäntymaa and colleagues (2004) found that mothers' hostility and/or intrusiveness in the first year of the infant's life predicted the presence of significant externalizing behaviors in toddlers at 24 months. Others have similarly found a significant relationship between early parent-child interactions and attachment and the development of later psychopathology (Lewis, Feiring, McGuffog, & Jaskir, 1984; Lyons-Ruth, 1996; Lyons-Ruth et al., 2003; Rettew, Stanger, McKee, Doyle, & Hudziak, 2006; Schmid et al., 2011; Zeanah et al., 1997). Other potential early risk factors that have been identified have included premature birth, infant temperament, parental psychopathology, parental stress, and socioeconomic status (Goodman & Gotlib, 1999; Lyons-Ruth et al., 2003; Viaux-Savelon et al., 2010; Zeanah et al., 1997). These findings must be taken cautiously, however, due to numerous methodological shortcomings, such as small sample size and correlational data. Additionally, it should be stressed that there is rarely a linear relationship between such risk factors and later outcomes; rather there is a complex interplay between the various intrinsic and extrinsic risk and protective factors (Zeanah et al., 1997). Along these lines, Cicchetti and Rogosch (1996) stress the concepts of equifinality and multifinality. Equifinality refers to the fact that children coming from different pathways (i.e., having different initial risk factors) may develop similar forms of psychopathology. Conversely, multifinality refers to the fact that children coming from similar pathways (i.e., having similar initial risk factors) may develop quite differently.

Less research has been conducted on the actual presence of psychopathology in infants and toddlers. Assessment of children before three years is rare, and therefore little is known about the development of most forms of psychopathology in infancy (Beernink, Swinkels, &

Buitelaar, 2007; Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Elberling & Skovgaard, 2002; Matson, Wilkins, Sevin et al., 2009; Skovgaard et al., 2007; Viaux-Savelon et al., 2010). This issue is further compounded by the fact that classifications systems, such as the *DSM-IV-TR*, use the same criteria for infants and toddlers that are used for older children, thus failing to take into account developmental considerations (Lyons-Ruth et al., 2003). Nevertheless, researchers have attempted to better understand the occurrence of psychopathology in infants and toddlers. For example, Skovgaard and colleagues (2007) examined the prevalence of psychopathology in a random sample of 18-month-old children in Copenhagen. They found that approximately 17% of the toddlers studied met criteria for at least one form of psychopathology. Others have estimated the prevalence of psychopathology in children younger than three years to be approximately 10% (Briggs-Gowan et al., 2001). Additionally, researchers have shown that infants and toddlers are more likely to present with externalizing problems, rather than internalizing problems (Beernink et al., 2007; Briggs-Gowan et al., 2001). The interpretability of the above findings is limited, however, due to varying methodologies and the limitations of current assessment techniques for infants and toddlers. Much more research is clearly needed to better understand the manifestation of psychopathology in infants and toddlers.

Early Development and Milestone Attainment

One area that has received increasing attention in recent years is the early development and manifestation of ASD specifically. The term Pervasive Developmental Disorder reflects the fact that children with ASD do not follow a typical developmental trajectory (Landa et al., 2007; Landa & Garret-Mayer, 2006). It is hypothesized that the brain abnormalities associated with ASD have an onset within the first one to two years after birth (Courchesne et al., 2007; Courchesne, Redcay, & Kennedy, 2004; Hazlett et al., 2005; Landa et al., 2007; Landa & Garret-

Mayer, 2006). As such, delays in development in children with ASD are seen beginning in infancy (Adrien et al., 1993; Baranek, 1999; Charman, Baron-Cohen et al., 2003; Charman et al., 1997; Clifford et al., 2007; Desombre et al., 2006; Fodstad et al., 2009; Horovitz & Matson 2010; Landa & Garrett-Mayer, 2006; Luyster et al., 2008; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b; Mitchell et al., 2006; Stone et al., 1997; Ventola et al., 2007; Werner & Dawson, 2005; Wetherby et al., 2007; Wetherby et al., 2004). To best understand these delays, one must understand the norms and expectations of development in typically developing children, particularly as they relate to the core features of ASD (i.e., social skills, communication skills, and repetitive behaviors). A great amount of variability exists in what is considered typical development and the age at which children should reach certain milestones (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991). Nevertheless, general milestones and expectations of skill acquisition exist for the first few years of development (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991). This section will briefly review these milestones and expectations from birth to three years of age and how infants and toddlers with ASD experience delays in attaining these skills. These milestones and expectations should not be understood as strict rules for typical development; rather, they should be seen as general patterns of development seen in typically developing children. Unless otherwise cited, the discussion of typically developing infants and toddlers will be based on the works of Green and Palfrey (2002) and Shelov and Hannemann (1991), which are the basis for the developmental guidelines put forward by the CDC (2011).

Social Skills. One of the first social skills to emerge in typically developing infants is the smile. Infants display a smile in the first month after birth; while at first the smile may occur non-socially, infants typically begin displaying a social smile in response to caregivers by three

months of age. Additionally, typically developing infants begin showing a preference for human faces in the first month. By six to nine months of age, typically developing infants begin showing an increased interest in others (particularly other children), responding to their spoken name, enjoying simple interaction games, imitating simple movements, and differentiating between people. Around this time basic joint attention skills begin to emerge, such as making eye contact and coordinating eyes with movements. By 12 months, increased social play begins to emerge, in addition to increased social responsiveness to others. An understanding of more subtle social cues begins to develop and infants begin showing an increased preference for certain people and toys. At this time, anxiety and shyness with strangers may begin to emerge as well. By 18 months, typically developing toddlers begin showing affection to familiar people, playing simple pretend games (e.g., feeding a doll), pointing to show others, imitating others' behavior, and enjoying the company of other children.

By 24 months, more complex imitation and pretend play are common in typically developing toddlers. Toddlers begin learning to take turns while playing games and will often spontaneously show affection to familiar people, including other children. Aggression (e.g., pushing other children), defiance, and shyness can all commonly be seen in typically developing children at this age. By 36 months, toddlers have typically begun playing interactively with other children, as opposed to parallel play, including more complex forms of imaginative play. Children begin forming peer relationships and understanding that not everyone thinks as they do. By 36 months, children typically have a wide range of socially appropriate facial expressions and they begin showing concern or sympathy for others. At this age, children are less likely to show separation anxiety and may be more willing to separate from their parents.

The development of social skills in children with ASD does not typically follow this same pattern (Adrien et al., 1993; Landa et al., 2007; Landa & Garret-Mayer, 2006; Osterling & Dawson, 1994; Osterling, Dawson, & Munson, 2002; Werner & Dawson, 2005; Werner et al., 2000). It is difficult, however, to establish similar norms in children with ASD, as first concerns are not typically noted until after the first birthday, and diagnoses are not typically given until children are three to four years old (DeGiacomo & Fombonne, 1998; Kozlowski et al., 2011; Matson, 2005; Tager-Flusberg & Anderson, 1992). Two methods have commonly been used to overcome this problem: (1) retrospective viewing of videotapes of children later diagnosed with ASD and (2) prospective studying of children considered at-risk for later developing an ASD (e.g., children with older siblings already diagnosed with ASD). Researchers employing these methods have shown that, by 12 to 18 months, children with ASD are observed to display fewer social skills, including orienting to name, pointing, making eye contact, smiling socially, attending to social cues, showing objects to adults, initiating and responding to joint attention, interacting socially, playing socially, and looking others in the face (Adrient et al., 1993; Baranek, 1999; Landa et al., 2007; Maestro et al., 2005; Osterling & Dawson, 1994; Osterling et al., 2002; Werner & Dawson, 2005; Werner et al., 2000). By 20 to 24 months, the social skills of children with ASD have been observed to fall even further behind in areas such as pointing, anticipatory posture, functional play, pretend play, imitation, empathy, peer interest, eye gaze, and orienting to name (Charman et al., 1997; Clifford et al., 2007; Werner & Dawson, 2005; Wetherby et al., 2007).

Communication Skills. In the first month, typically developing infants show the first signs of receptive communication by responding and orienting to their parents' voices. By three months cooing and other sounds begin to emerge. Around four months of age, typically

developing infants begin babbling and imitating some simple sounds. By six to nine months infants begin frequently repeating sounds, babbling chains of consonants reciprocally, making sounds to show emotions, responding to sounds with sounds, understanding “no,” and responding to their name. Additionally, nonverbal skills, such as pointing and shaking the head may begin to emerge at this time. By 12 months, children typically will say their first word (e.g., “bye-bye” or “ma-ma”) and may begin to use these words with communicative intent. At this age, typically developing toddlers pay more attention to the speech of others and respond to simple verbal requests. Increases in nonverbal communication, such as head shaking and waving are seen around this age as well. Around 18 months, the toddler’s vocabulary expands to 15 to 20 words, often including the use of simple two-word phrases. Toddlers begin listening to stories, naming body parts and familiar objects, and following simple directions.

By 24 months, toddlers typically have a rapidly expanding vocabulary that includes 50 or more words. Toddlers begin using pronouns (e.g., I, me, we) and speaking in simple sentences. At this age, the toddler’s speech is mostly intelligible to strangers. Receptive vocabulary expands significantly around this age as well, with toddlers beginning to understand most sentences, understand physical relationships (e.g., on, in, under), recognize most common objects, and follow multi-step instructions. At 36 months, toddlers typically have an expansive vocabulary, including up to 1,000 words. Toddlers at this age can name most familiar objects, speak in more complex sentences, tell simple stories, and understand some basic rules of grammar (e.g., simple plurals). While the speech of toddlers should be intelligible to strangers at this age, sound substitutions or mispronunciations are still common at this age. By 36 months, typically developing toddlers’ receptive vocabulary will likely be much greater as well, with greater

understanding of physical relationships (e.g., on, in, under) and concepts such as “same” and “different.”

As with the development of social skills, the development of communication skills in infants and toddlers with ASD does not follow this same trajectory (Adrien et al., 1993; Charman, Baron-Cohen et al., 2003; Landa & Garrett-Mayer, 2006; Luyster et al., 2008; Mitchell et al., 2006; Stone et al., 1997; Werner & Dawson, 2005). As previously discussed, approximately 33 to 50% of children with ASD never develop meaningful speech (Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978). Using similar methodologies to those described in the Social Skills section, researchers have begun to gain an understanding of the early development of communication skills in infants and toddlers with ASD. Researchers have found that the rate and pattern of communication development are different in infants and toddlers with ASD (Charman, Baron-Cohen et al., 2003; Landa & Garret-Mayer, 2006). At six months of age, Landa & Garrett-Mayer (2006) did not detect significant receptive or expressive communication delays in infants and toddlers later diagnosed with ASD. However, by 12 to 18 months, multiple researchers have found children with ASD to show delays in many receptive and expressive communication skills, including use of gestures, understanding of simple phrases, response to simple verbal requests, babbling, rate of communication, understanding, and word production (Adrien et al., 1993; Landa & Garrett-Mayer, 2006; Mitchell et al., 2006; Werner & Dawson, 2005; Wetherby et al., 2007). By 24 months, these delays became more pronounced and easier to detect (Desombre et al., 2006; Landa & Garrett-Mayer, 2006; Mitchell et al., 2006; Werner & Dawson, 2005).

Matson, Mahan, Kozlowski, and Shoemaker (2010) conducted a study looking at developmental milestones in 331 infants and toddlers with ASD. They found that approximately

60% of these infants and toddlers spoke their first word within typical limits, while 17% were delayed and 22% had not yet attained the milestone. With respect to first phrase, they found that approximately 13% of infants and toddlers with ASD met this milestone within typical limits, while 9% were delayed and 78% had not yet attained the milestone. In those that had attained these milestones, they found the average age of first word in infants and toddlers with ASD to be 13.97 months and the average age of first phrase to be 22.76 months. Finally, multiple researchers have shown that marked delays in expressive and receptive communication skills are present in toddlers with ASD by 36 months of age (Charman, Baron-Cohen et al., 2003; Luyster et al., 2008; Stone et al., 1997). These delays include deficits in word production, word comprehension, and phrase comprehension (Charman, Baron-Cohen et al., 2003).

Repetitive and Stereotyped Behaviors and Interests. While restricted and repetitive behaviors and interests (RRBI) are one of the core features of ASD, they are not unique to children with ASD, and they frequently occur in typically developing infants and toddlers (D.W. Evans et al., 1997; Foster, 1998; Morgan et al., 2008; Richler et al., 2007; Thelan, 1998; Tröster, 1994). D.W. Evans and colleagues (1997) conducted a study examining the prevalence and development of behaviors similar to RRBI in typically developing young children. They assessed these behaviors in 1,488 children aged 8 to 72 months using the Childhood Routines Inventory, a 19-item parent-report questionnaire examining compulsive-like behaviors. Items on the questionnaire included: preferring to have things done in a particular order; arranging objects or performing certain behaviors until they seem “Just Right;” lining up objects; preferring the same household schedule or routine; repeating certain actions over and over; and having a strong preference for certain foods. These behaviors were found to occur commonly across the sample, occurring most frequently and intensely in children between 24 and 48 months of age. Richler et

al. (2007) conducted a study examining rates of RRBI in children with ASD less than three years of age, using selected items from the ADI-R. Their study included a comparison group consisting of 65 typically developing toddlers. While they found most of the RRBI to occur more frequently in children with ASD, many of these behaviors were still found to occur frequently in typically developing toddlers. For example, approximately 20 to 30% of typically developing toddlers were reported to exhibit the following behaviors: unusual sensory interests, repetitive use of objects, sensitivity to noise, and self-injurious behaviors. The remaining behaviors were endorsed in approximately 5 to 10% of typically developing toddlers. Morgan et al. (2008) similarly found a broad range of RRBI to be endorsed in typically developing toddlers between 18 and 24 months of age.

While RRBI occur in many typically developing infants and toddlers, a number of factors differentiate the RRBI seen in infants and toddlers with ASD. Firstly, the RRBI seen in typically developing toddlers are likely to occur predictably in specific situations (Foster, 1998), whereas the RRBI seen in infants and toddlers with ASD often occur irrespective of the situation (Rutter, 1978). While the RRBI of typically developing toddlers decrease in frequency with age (D. W. Evans et al., 1997; Thelan, 1979), symptoms tend to persist through life in individuals with ASD (Hattier et al., 2011; Matson & Dempsey, 2008; Matson & Horovitz, 2010). Additionally, infants and toddlers with ASD have been found to engage in a broader range of RRBI at much higher rates and intensities than toddlers that are typically developing or experiencing non-ASD related delays (Matson, Fodstad, & Dempsey, 2009; Morgan et al., 2008; Richler et al., 2007).

Summary. Infants and toddlers undergo significant development in the first few years of life, and while the course of development varies from child to child, general patterns of development and milestone attainment can be drawn. The early development of infants and

toddlers with ASD, however, differs significantly from that of their typically developing peers; many behaviors and skills are delayed and some never emerge (Adrien et al., 1993; Baranek, 1999; Charman, Baron-Cohen et al., 2003; Charman et al., 1997; Clifford et al., 2007; Desombre et al., 2006; Fodstad et al., 2009; Horovitz & Matson 2010; Landa & Garrett-Mayer, 2006; Luyster et al., 2008; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b; Mitchell et al., 2006; Stone et al., 1997; Ventola et al, 2007; Werner & Dawson, 2005; Wetherby et al., 2007; Wetherby et al., 2004). Yet much is still unknown about the early development of this population. While studies using retrospective and prospective data have been informative, methodological flaws inherent in these procedures limit the conclusions that can be drawn. Additionally, many of the aforementioned findings are not specific or unique to infants and toddlers with ASD. Similar findings and impairments are often found with children with other DD (Fodstad et al., 2009; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b). Impairments and features specific to infants and toddlers with ASD are discussed further below, in the Differential Diagnosis section.

Early Assessment

There has been a great deal of debate in the literature as to the age at which ASD can and should be assessed. Some have suggested that the identification of symptoms of ASD is possible as early as 12 months of age (Adrien et al., 1993; DeGiacomo & Fombonne, 1998; Mitchell et al., 2006; Osterling & Dawson, 1994; Osterling et al., 2002; Rogers & DiLalla, 1990; Werner & Dawson, 2005); however, it is generally agreed that the characteristic features of autism stabilize and become reliably detectable closer to 18 to 24 months (Adrien et al., 1993; Baron-Cohen, Allen, & Gillberg, 1992; Matson, Wilkins, Sevin et al, 2009; Mitchell et al., 2006; Osterling & Dawson, 1994; Werner & Dawson, 2005). Still others have argued that not enough is known at

this time to conduct early screenings for autism (J. Williams & Brayne, 2006). While there is unlikely to be a specific cutoff age where early identification is consistently reliable, researchers have shown that reliable assessment of ASD is possible as early as 18 months (Baron-Cohen et al., 1992; Matson, Wilkins, & González, 2008; Matson, Wilkins, Sevin et al, 2009).

Early assessment measures. A number of measures currently exist that may be employed in the assessment and diagnosis of ASD before three years. General assessment measures previously discussed, such as the ADI-R (Lord et al., 1994), the CARS (Schopler, Reichler, & Renner, 1980), and the ADOS-G (Lord et al., 2000) can all be used with children as young as 2 years of age. Despite this, none of these measures were designed specifically for the assessment of infants and toddlers, and the psychometric properties of these measures with children in this younger age range need further testing. Although a revised algorithm has been developed for the ADI-R for children under two years of age, sufficient research on this revision has not yet been carried out (Kim & Lord, 2012). In recent years, a number of measures have been designed for the specific purpose of assessing ASD in children before three years. Of these measures, few have the research needed to determine their psychometric properties and recommend their use with infants and toddlers (Matson et al., in press). As discussed in a review by Matson et al. (in press), two early assessment and screening measures have an extensive research base: the CHAT; Baron-Cohen et al., 1992) and its variations and the Baby and Infant Screen for Children with aUtism Traits (BISCUIT; Matson, Wilkins, Sevin et al, 2009).

The CHAT was originally developed as a screening instrument for children aged 18 months who were considered at risk for an ASD (Baron-Cohen et al., 1992; Baron-Cohen et al., 2000). Two sections make up the original CHAT: A) Nine parent-report questions, emphasizing

pretend play and joint attention; and B) Five items completed via direct observation. Items are answered “Yes” or “No” by caregivers, allowing for easy and quick administration. One downside to the measure is that it was developed for screening, rather than diagnostic, purposes. As a screening measure, its sensitivity is considered to be below acceptable levels (Allison et al., 2008; Robins, Fein, Barton, & Green, 2001).

Since first developed, two variations of the CHAT have been created, in an attempt to improve its psychometric properties: The Modified CHECKlist for Autism in Toddlers (M-CHAT; Robins et al., 2001) and the Quantitative CHECKlist for Autism in Toddlers (Q-CHAT; Allison et al., 2008). The Q-CHAT removed the observational section of the CHAT, choosing to only include parent report. The Q-CHAT contains 25 items scored on a scale from 0 to 4, with higher scores representing greater impairment. The initial test-retest reliability of the Q-CHAT was found to be .82 (Allison et al., 2008). One limitation of the scale is that it is not able to differentiate among ASD (Allison et al., 2008; Matson, Wilkins, Sevin et al, 2009). Additionally, limitations in the original reliability study and a lack of follow-up studies on the psychometrics of the measure hamper its utility at this time (Matson et al., in press).

The M-CHAT is currently the most widely used variation of the CHAT (Matson et al., in press). The M-CHAT retained the original nine parent-report questions of the CHAT and added 21 additional items to identify a broader range of children and compensate for the removal of the direct observation component (Robins et al., 2001). Factor analysis reduced the item total to 23 and the age of screening was moved to 24 months (Robins et al., 2001). In an initial assessment of psychometric properties, internal reliability of the entire checklist and critical items were $\alpha = .85$ and $\alpha = .83$, respectively (Robins et al., 2001). Additionally, high sensitivity and specificity of the measure were reported, with less false positives than the CHAT. Replication of the

findings on internal consistency of the M-CHAT produced similar results (Kleinman et al., 2008). Additionally, the M-CHAT has been successfully translated into a number of languages, with adequate psychometric properties reported (Inada, Koyama, Inokuchi, Kuroda, & Kamio, 2011; Losapio & Pondé, 2008; Perera, Wijewardena, & Aluthwelage, 2009). Despite the sound psychometrics properties of the M-CHAT, researchers have suggested that the M-CHAT be primarily used as a screening tool, questioning its independent utility for diagnosing (Kleinman et al., 2008; Matson et al., in press). Additionally, the M-CHAT was not designed to differentiate among ASD.

The Baby and Infant Screen for Children with aUtism Traits (BISCUIT) is a three-part parent-report measure developed to assess toddlers aged 17 to 37 months who are considered at-risk for developing an ASD due to developmental delays (Matson, Wilkins, Sevin et al., 2009). Whereas the CHAT and its variants were designed as screeners, the BISCUIT was designed as a diagnostic tool for both autism and PDD-NOS. However, like the CHAT, Part 1 of the BISCUIT can be used independently as a screening measure. The measure does not assess for Asperger's Disorder, as Asperger's Disorder is not typically apparent until a later age, and its inclusion was not supported by a study of the measure's reliability (Howlin & Asgharian, 2007; Mandell, Novak, & Zubritsky, 2005; Matson, Wilkins, Sevin et al, 2009). The developers of the ASD Child and Adult Batteries similarly designed the BISCUIT battery to assess diagnostic criteria, comorbidity, and challenging behaviors. Part 1 of the BISCUIT, the diagnostic section, includes 62 items assessing for the core symptoms of ASD. Parents or caregivers rate items on a 3-point Likert-type scale, comparing their child to a typically developing child of the same age. An attached appendix is designed to assist parents in determining what is considered typical and atypical behavior. Cutoffs for Part 1 have been established, placing toddlers into one of three

categories: (1) Probable ASD/Autistic Disorder; (2) Possible ASD/PDD-NOS; and (3) No ASD/Atypical Development (Matson, Wilkins, Sharp et al, 2009). High sensitivity and specificity have been reported for distinguishing between PDD-NOS and atypical development, as well as for distinguishing between autism and PDD-NOS. Sensitivity when comparing ASD to atypical development was found to be higher for the BISCUIT when compared to the M-CHAT (93.4 versus 74.1), while specificity was found to be comparable (86.6 versus 87.5). The internal consistency of the BISCUIT-Part1 was found to be .97 (Matson, Wilkins, Sevin et al, 2009). Strengths of the BISCUIT include its ability to differentiate between autism and PDD-NOS, its use as a diagnostic tool, and its brevity and ease of administration. Like the ASD Child and Adult batteries, one weakness of the measure is its sole use of parent-report.

Differential Diagnosis. Differential diagnosis of children with ASD can be very difficult, particularly at an early age. This is especially difficult given the high overlap between ASD and ID (Bender, 1959; Fombonne, 2003; La Malfa et al., 2004; Matson & Shoemaker, 2009; Rutter, 1968). However, Rutter (1968, 1978) helped to establish that ASD is not simply a form of ID. Firstly, not all children with autism have ID, with current estimates ranging from approximately 50 to 70% (Bender, 1959; Fombonne, 2003; La Malfa et al., 2004; Matson & Shoemaker, 2009; Rutter, 1968). Additionally, Rutter hypothesized that many children with autism may have impairments in specific areas (e.g., language, coding), rather than global intellectual deficits (Rutter, 1968, 1978). Given the high comorbidity of ID, Rutter (1978) stressed the need to consider developmental level when assessing for autism. While ID cannot be reliably identified at the time of early assessment (Bayley, 1949; Hopkins & Bracht, 1975), developmental functioning, while not perfect, is often used as an early indicator of later intellectual functioning (Fodstad, Rojahn, & Matson, 2010; Matson, Mahan, Hess, & Fodstad, 2010).

While researchers have primarily focused on comparisons between children with ASD and typically developing children, there is a greater need for comparisons between children with ASD and children with other, non-ASD related DD, such as speech delays or Down syndrome, as these are the populations for which differential diagnosis is typically needed. Additionally, such research would allow for the identification of early behavioral signs and symptoms that are specific to ASD, rather than indicative of a general DD (Saint-Georges et al., 2010). In addition to the need to differentially diagnose ASD from other, non-ASD related developmental delays, it is also important that differential diagnosis occur within the broader category of ASD. Due to the rarity of CDD and Rett's Disorder (Volkmar, Lord, Bailey, Schultz, & Klin, 2004) and the lack of support for diagnosing Asperger's Disorder at a young age (Matson, Wilkins, Sevin et al, 2009), such differential diagnosis is typically necessary between autism and PDD-NOS. Differential diagnosis is necessary to ensure that the most evidence-based treatment and support can be given to children and their families. For example, some interventions may be more likely to lead to improved outcomes in children with PDD-NOS when compared to children with autism (Rogers & Vismara, 2008). In order for successful differential diagnosis to occur at an early age, an understanding of the early behavioral signs of ASD is necessary.

ASD compared to DD. Children with ASD and children with DD often exhibit similar patterns of behavior, making differential diagnosis between the two populations very difficult (Fodstad et al., 2009; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b). Nevertheless, a number of significant differences have been found between these populations. While there is considerable variability in the findings of individual researchers, likely due in part to variations in methodology, researchers have generally found those with ASD to exhibit more impairments across each of the three core features (Baranek, 1999; Charman et

al., 1997; Clifford et al., 2007; Desombre et al., 2006; Fodstad et al., 2009; Horovitz & Matson, 2010; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b; Ventola et al., 2007; Wetherby et al., 2007; Wetherby et al., 2004). With respect to social and communication impairments, Fodstad et al. (2009) compared the impairments of toddlers with ASD and toddlers with non-ASD related DD. Overall, they found a greater number and severity of impairments to be endorsed for toddlers with ASD. Items that were found to be significant predictors of group membership included: shares enjoyment, interests, or achievement with others; interest in participating in social games, sports and activities; use of too few or too many social gestures; development of social relationships; use of language to communicate; use of language in conversation with others; communicating effectively; and language development (Fodstad et al., 2009). Similar findings have been found by others with respect to peer interest (Clifford et al., 2007; Matson, Fodstad, & Dempsey, 2009b; Ventola et al., 2007), anticipatory posture (Clifford et al., 2007), facial expressions (Matson, Fodstad, & Dempsey, 2009b; Ventola et al., 2007; Wetherby et al., 2004), prosody (Wetherby et al., 2004), empathy (Charman et al., 1997), sharing of enjoyment in interactions (Charman et al., 1997; Matson, Fodstad, & Dempsey, 2009b; Ventola et al., 2007; Wetherby et al., 2004), language development (Horovitz & Matson, 2010; Matson, Fodstad, & Dempsey, 2009b), non-verbal communication (Matson, Fodstad, & Dempsey, 2009b; Wetherby et al., 2007), response to name (Baranek, 1999; Wetherby et al., 2004), eye gaze (Clifford et al., 2007; Wetherby et al., 2007) (Matson, Fodstad, & Dempsey, 2009b; Wetherby et al., 2004), rate of communication (Wetherby et al., 2007), and initiation of and response to joint-attention behaviors (Charman et al., 1997; Clifford et al., 2007; Ventola et al., 2007; Wetherby et al., 2007).

With respect to RRBI, researchers similarly found that toddlers with ASD exhibit significantly greater stereotypic behaviors than toddlers with DD (Matson, Dempsey, & Fodstad, 2009). Differences have been found in a number of areas, with infants and toddlers with ASD exhibiting significantly greater impairments in areas such as resistance to change (Desombre et al., 2006), restricted number of interests (Matson, Dempsey, & Fodstad, 2009), repetitive motor movements (Matson, Dempsey, & Fodstad, 2009; Wetherby et al., 2004), preoccupation with parts of an object (Matson, Dempsey, & Fodstad, 2009), repetitive body movements (Matson, Fodstad, & Dempsey, 2009b; Wetherby et al., 2004), low frustration tolerance (Desombre et al., 2006), maintenance of odd routines (Matson, Fodstad, & Dempsey, 2009b), and preference for foods of specific textures or smells (Matson, Fodstad, & Dempsey, 2009b). As can be seen by these findings, toddlers with DD may exhibit a number of the impairments that are characteristic of an ASD; however, many of these impairments occur at greater frequencies and severities in those with ASD and can thereby be used to aid in differential diagnosis between the two groups. These findings are consistent with the proposed dimensional structure of ASD symptoms among toddlers at-risk for developmental disabilities (Boisjoli & Matson, in press).

Autism compared to PDD-NOS. Most ASD researchers have designed studies employing samples of children with autism or children who fit into the broader category of ASD. Few researchers have compared children with specific ASD diagnoses, particularly those with PDD-NOS to those with autism. This lack of research may in part be due to the inability for many assessment measures to accurately detect PDD-NOS (Cox et al., 1999; Lord et al., 2000; Matson, Wilkins, Boisjoli et al, 2008; Matson, Wilkins, Sevin et al, 2009) and a lack of consensus on how to best define PDD-NOS (Buitelaar & Van der Gaag, 1998; Buitelaar et al., 1999; Matson & Boisjoli, 2007; Tidmarsh & Volkmar, 2003). However, such research is critical, as differential

diagnosis of ASD in infants and toddlers typically involves autism and PDD-NOS. When this type of research has been conducted, the findings have followed a similar pattern to those discussed in the previous section. Similar impairments are seen in both populations, but toddlers with autism evince greater and more severe impairments than toddlers with PDD-NOS (Charman, Baron-Cohen et al, 2003; Fodstad et al., 2009; Horovitz & Matson, 2010; Matson, Dempsey, & Fodstad, 2009; Matson, Fodstad, & Dempsey, 2009b). Matson, Fodstad, and Dempsey (2009b) examined symptoms of ASD in infants and toddlers using the BISCUIT Part-1. They found the following 11 items best predicted group membership for infants and toddlers with autism and PDD-NOS: engages in repetitive motor movements for no reason; use of language in conversation with others; shares enjoyment, interests, or achievements with others; interest in participating in social games, sports and activities; restricted interests and activities; sticking to odd routines or rituals; abnormal preoccupation with parts of an object; reads non-verbal cues of others; use of non-verbal communication; abnormal, repetitive hand or arm movements; and development of social relationships. A model using these 11 items correctly classified 88.90% of the toddlers with an autism diagnosis and 88.20% of those with a PDD-NOS diagnosis. In fact, the same items that differentiated toddlers with ASD from toddlers with non-ASD related developmental delays could be used to differentiate between toddlers with autism and PDD-NOS, lending further support to the dimensional nature of ASD in infants and toddlers (Boisjoli & Matson, in press). Similar findings can be found elsewhere in the literature with respect to joint attention skills (Charman, Baron-Cohen et al, 2003), receptive communication (Charman, Baron-Cohen et al, 2003), and communication skills (Horovitz & Matson, 2010). As mentioned above, one reason for the paucity of research in this area is the disagreement amongst researchers on how to best define PDD-NOS. The findings presented here

lend support to the idea that PDD-NOS is best defined as a lesser variant of autism (Buitelaar & Van der Gaag, 1998; Buitelaar et al., 1999; Matson & Boisjoli, 2007; Matson & Smith, 2008).

Diagnostic Stability. Given the debates over the age at which reliable assessment of ASD is possible, it follows that a number of researchers have attempted to empirically examine the stability of early diagnoses and symptoms. In a sample of children that received an ASD diagnosis before 28 months, Chawarska, Klin, Paul, Macari, and Volkmar (2009) found the short-term stability (with an average time lapse of 25 months) of diagnoses to be 74% for autism, 83% for PDD-NOS, and 81% for non-ASD related DD. Additionally, all children originally diagnosed with ASD retained an ASD diagnosis at follow-up. In a similar study, Worley, Matson, Mahan, Kozlowski, and Neal (2011) found that overall diagnostic classification remained the same in 67.5% of children that were first diagnosed before 36 months. Again, greater stability was found within ASD in general, with more variability found between diagnoses of autism and PDD-NOS. Together, these studies support the stability of ASD diagnoses before three years of age. While some variability exists within diagnoses of ASD, correct classification rates remain relatively high. Similar findings have been found with respect to diagnoses and symptom stability elsewhere in the literature (e.g., Cox et al., 1999; Lord et al., 2006; Werner, Dawson, Munson, & Osterling, 2005). It is generally agreed upon that, although symptom improvement may be seen with proper treatment, ASD symptoms and diagnoses remain relatively chronic across the lifespan (Matson & Horovitz, 2010). Given the research showing that ASD symptoms emerge in the first year of life (DeGiacomo & Fombonne, 1998; Dixon, Granpeesheh, Tarbox, & Smith, 2011; Maestro et al., 2005; Ornitz et al., 1977), it is thus reasonable to expect that diagnoses provided by early assessment will remain stable. However, researchers have suggested that symptoms become more apparent and more accurate diagnoses

of ASD can be given as a child ages (Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; Mitchell et al., 2006; Werner & Dawson, 2005). Additionally, it is unknown what effects the implementation of early intervention has on later symptom presentation and diagnostic classification. Thus, while initial evidence is encouraging, more research is still clearly needed on the long-term stability of early diagnoses.

Additional assessment considerations. The core component of early assessment is the use of a psychometrically sound diagnostic assessment measure, such as the BISCUIT Part-1. However, researchers have stressed the need for assessment to involve multiple sources and multiple methods, rather than using information from one single measure administered to one informant (Chawarska & Bearss, 2008). In addition to a psychometrically sound diagnostic assessment measure, early assessment should include a detailed medical history, a parent interview, an observation of the child, an assessment of comorbid difficulties, an assessment of developmental and adaptive functioning, and a functional assessment of problem behaviors (Bishop et al., 2008; Chawarska & Bearss, 2008; Matson, Wilkins, Sevin et al, 2009). Information from each of these areas must carefully be integrated to best understand symptoms, conceptualize the case, and provide recommendations. The most commonly used measures of developmental level for infants and toddlers with ASD are the Bayley Scales of Infants and Toddler Development, Third Edition (Bayley, 2006) and the Mullen Scales of Early Learning (Mullen, 1995). The most widely used comprehensive measure of adaptive skills with this population is the Vineland Adaptive Behavior Scales, Second Edition (VABS-II) (Sparrow, Cicchetti, & Balla, 2005). Further discussion will be given below on comorbidity and problem behaviors with this population, including how to assess each of these areas.

Comorbid Psychopathology

Comorbidity refers to the occurrence of two or more forms of psychopathology in one person (Matson & Nebel-Schwalm, 2007b). While it has been readily accepted that comorbidity of Axis I disorders is common (Caron & Rutter, 1981), there has historically been debate if this trend applies to individuals with ASD (Sipes, Matson, Horovitz, & Shoemaker, 2011). One likely reason for this debate has been diagnostic overshadowing (Helveschou, Bakken, & Martinsen, 2011; McCarthy et al., 2010; Simonoff et al., 2008). Diagnostic overshadowing occurs when the presence of one disorder masks the presence of others (Helveschou et al., 2011). As was the case with individuals with ID (Reiss & Syzszko, 1983), it is likely that symptoms of comorbid psychopathology have historically been attributed to the primary diagnosis of ASD, and have thus gone under-diagnosed in this population (Helveschou et al., 2011; McCarthy et al., 2010; Simonoff et al., 2008). For example, take a child who has a diagnosis of autism and is experiencing symptoms of social anxiety and attention-deficit/hyperactivity disorder (ADHD). While the child may meet criteria for each of these disorders, a clinician may only give a diagnosis of autism, attributing the symptoms of inattention, hyperactivity and social anxiety to the diagnosis of autism. Given the lack of clinical comorbid diagnoses given to individuals with ASD, little research has been conducted on comorbidity in this population, especially when compared to the large body of research on comorbidity in other childhood disorders (Matson & Nebel-Schwalm, 2007b). Most of the research that has been conducted has focused on the well-established relationship between ASD and ID, as discussed previously (Fombonne, 2003; La Malfa et al., 2004; Matson & Shoemaker, 2009). Research on ASD and other Axis I disorders has been scarce (Ghaziuddin, Tsai, & Ghaziuddin, 1992; Matson & Nebel-Schwalm, 2007b), particularly in infants and toddlers (Matson, Boisjoli, Hess, & Wilkins, 2011; Matson, Fodstad,

Mahan et al, 2009). In the little research that has been conducted, researchers have shown that individuals with ASD are at increased risk for developing comorbid psychopathology, in a similar to fashion to individuals with other Axis I disorders (Brereton, Tonge, & Einfeld, 2006; Gadow, DeVincent, Pomeroy, & Azizian, 2004; Helverschou et al., 2011; Hess, Matson, & Dixon, 2009; Matson, Hess, & Boisjoli, 2010; Matson & Nebel-Schwalm, 2007b). This section will briefly review the current understanding of comorbid psychopathology in individuals with ASD, followed by a discussion of comorbid psychopathology in infants and toddlers with ASD.

Current estimates suggest that up to 70 to 75% of individuals with ASD experience clinically significant comorbid difficulties and over 40% meet criteria for more than one comorbid psychiatric disorder (Brereton et al., 2006; Ghaziuddin & Zafar, 2008; Gillberg & Billstedt, 2000; Simonoff et al., 2008). However, identification and actual diagnosis of comorbid psychopathology is infrequent in this population for a number of reasons (Matson & Nebel-Schwalm, 2007b; Simonoff et al., 2008). These reasons include the aforementioned diagnostic overshadowing, the high overlap between autism and ID, the communication deficits characteristic of ASD, the overlap between symptoms of ASD and symptoms of comorbidity, the often atypical presentation of comorbid symptoms in individuals with ASD, and the wide range of heterogeneity in individuals with ASD (Hutton, Goode, Murphy, Le Couteur, & Rutter, 2008; Matson & Nebel-Schwalm, 2007b; Simonoff et al., 2008; Underwood, McCarthy, & Tsakanikos, 2011). The most frequently reported symptoms of comorbidity include depression, social anxiety, phobias, oppositional behaviors, and attention-deficit/hyperactivity behaviors (Gadow et al., 2004; Ghaziuddin, Ghaziuddin, & Greden, 2002; Matson & Nebel-Schwalm, 2007b; Simonoff et al., 2008). Additionally, researchers have found symptoms of comorbidity to vary as a function of a number of factors, including gender (Hartley & Sikora, 2009; Simonoff et al.,

2008), level of intellectual functioning (Tsakanikos et al., 2006), nationality (Zachor et al., 2011), and the presence of medical conditions (Simonoff et al., 2008). Still, much is not yet understood about comorbidity in individuals with ASD (Matson & Nebel-Schwalm, 2007b; McCarthy et al., 2010).

Infants and toddlers. Very little is known about comorbid psychopathology in infants and toddlers with ASD. As little is known about the early development of core ASD symptoms, it is not surprising then that even less is known about the early development of comorbid difficulties. One reason that comorbidity in individuals with ASD, particularly infants and toddlers, is poorly understood is that few assessment measures have been designed to assess comorbidity in this population (E. Davis, Atezaz Saeed, & Antonacci, 2008; Hutton et al., 2008; Matson & Boisjoli, 2008; Matson, LoVullo, Rivet, & Boisjoli, 2009). The most common method of assessing psychiatric symptoms in individuals with ASD is clinical judgment, despite the numerous disadvantages (e.g., lack of standardization) of this approach (Underwood et al., 2011). Nevertheless, there is a growing understanding that assessment of comorbidity should be based on psychometrically sound, standardized measures (Matson & Boisjoli, 2008; Underwood et al., 2011). Tools for assessing psychopathology come in a variety of forms, including checklists, rating scales, and structured interviews (Underwood et al., 2011). These tools may be broad, assessing for symptoms of multiple disorders, whereas others may assess for symptoms of a specific disorder. While only a handful of measures have been developed specifically for use with ASD, some non-ASD specific measures have been used successfully to assess comorbidity in children with ASD. Such scales have included broadband rating scales, such as the Child Behavior Checklist (Achenbach & Rescorla, 2000) and the Behavior Assessment System for Children, Second Edition (Reynolds & Kamphaus, 2004); structured and semi-structured clinical

interviews, such as the Anxiety Disorders Interview Schedule for DSM-IV (Silverman & Albano, 1996) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (Kaufman et al., 1997); and rating scales, such as the Conners Rating Scales-Revised (Conners, 2000) and the Children's Depression Inventory (Gold, 1993; Holtmann, Bölte, & Poustka, 2007; Kovacs, 1992; Mahan & Matson, 2010; Matson, LoVullo et al, 2009; Mehtar & Mukaddes, 2011; O'Connor & Healey, 2010; Pandolfi, Magyar, & Dill, 2009, 2012). Although these measures are considered psychometrically sound, they were not designed specifically for use with individuals with ASD and therefore cannot be assumed to adequately assess psychopathology in individuals with ASD. While some attempts have been made to investigate the psychometric properties of these scales with individuals with ASD (e.g., Mahan & Matson, 2010; Pandolfi et al., 2009, 2012), there has unfortunately been an overall lack of empirical evidence providing such validation (MacNeil, Lopes, & Minnes, 2009; Matson, LoVullo et al, 2009; Underwood et al., 2011).

In recent years, there have been some attempts to design measures for the specific purpose of assessing comorbid psychopathology in individuals with ASD. A review by Underwood et al (2011) identified six measures designed as such. These measures were the Autism Comorbidity Interview – Present and Lifetime Version (ACI-PL; Leyfer et al., 2006), the Schedule for the Assessment of Psychiatric Problems Associated with Autism (and Other Developmental Disorders) (SAPPA; Bolton & Rutter, 1994), the Psychopathology in Autism Checklist (PAC; Helterschou, Bakken, & Martinsen, 2009), the Autism Spectrum Disorder – Comorbidity for Children (ASD-CC; Matson & Wilkins, 2008), and the Autism Spectrum Disorder – Comorbidity for Adults (ASD-CA; Matson & Boisjoli, 2008). None of these measures, however, were designed for use with infants and toddlers. The Baby and Infant Screen for

Children with Autism Traits, Part-2 (BISCUIT Part-2; Matson, Wilkins, Sevin et al, 2009) was developed from the ASD-CC to fill this gap.

The Baby and Infant Screen for Children with Autism Traits Part 2 (BISCUIT-Part 2) (Matson, Wilkins, Sevin et al, 2009) was developed for use with infants and toddlers aged 17 to 37 months. The BISCUIT Part-2 contains 57 items that are completed by an informant on a 3-point Likert-type scale. Like the ASD-CC, the BISCUIT-Part 2 is administered alongside an assessment of diagnostic symptoms and challenging behaviors, in the larger BISCUIT battery. The initial study of reliability demonstrated internal consistency of the BISCUIT-Part 2 (Matson, Wilkins, Sevin et al, 2009), while follow-up studies established cutoffs and norms for children with ASD and for children with non-ASD related developmental disabilities (Matson, Fodstad, & Mahan, 2009; Matson, Fodstad, Mahan et al, 2009). Convergent and discriminant validity have not yet been established for the scale.

The development of the BISCUIT Part-2 has opened the door for research on comorbidity in infants and toddlers with ASD and has begun to shed some light on this topic. Researchers have shown that infants and toddlers with ASD are at greater risk for comorbid psychopathology, which is consistent with the research showing similar trends in older children and adults (Hess et al., 2009; LoVullo & Matson, 2009; Matson, Hess et al, 2010; Matson & Nebel-Schwalm, 2007b). Researchers have examined possible moderating factors of comorbidity in toddlers with ASD (Sipes et al., 2011), the relationship between comorbid symptoms and challenging behaviors in toddlers (Matson, Mahan et al, 2011), anxiety symptom presentation in toddlers (T. E. Davis et al., 2010) and gender differences in toddlers (Horovitz, Matson, & Sipes, 2011). Still, much is yet to be learned about the emergence of comorbidity in children with ASD.

As greater emphasis is placed on early assessment of ASD, a better understanding of the early development of comorbid psychopathology will begin to surface.

Challenging Behaviors

Challenging behaviors, also commonly referred to as maladaptive or problem behaviors, occur frequently in individuals with ASD (Cohen, Yoo, Goodwin, & Moskowitz, 2011; Matson & Nebel-Schwalm, 2007a). In fact, it is estimated that over half of individuals with ASD engage in at least one challenging behavior (Baghdadli, Pascal, Grisi, & Aussilloux, 2003; Bodfish et al., 2000; Holden & Gitlesen, 2006; Matson & Nebel-Schwalm, 2007a; Matson, Wilkins, & Macken, 2009). The term challenging behaviors is applied to a number of behaviors with different topographies. The most frequently occurring challenging behaviors include aggression, property destruction, tantrums, and self-injurious behavior (SIB) (Cohen et al., 2011; Hattier, Matson, Belva, & Horovitz, 2011; Horovitz, Matson, Rieske, Kozlowski, & Sipes, 2011; Matson & Rivet, 2008a; McCarthy et al., 2010; Weeden, Mahoney, & Poling, 2010). Challenging behaviors are not specific to ASD, as they are exhibited by individuals with ID, individuals with various psychiatric disorders, and individuals from the typically developing population (Dominick, Davis, Lainhart, Tager-Flusberg, & Folstein, 2007; Emerson et al., 2001; Holden & Gitlesen, 2006; Lavigne et al., 2009; McCarthy et al., 2010). However, researchers have shown that individuals with ASD are more likely to engage in challenging behaviors than are individuals from other populations, with individuals with ASD and comorbid ID engaging in the highest rates (Bodfish et al., 2000; Matson & Rivet, 2008a; McCarthy et al., 2010; Rojahn, Wilkins, Matson, & Boisjoli, 2010). Challenging behaviors are typically chronic, occurring throughout the lifespan (Ballaban-Gil, Rapin, Tuchman, & Shinnar, 1996; Billstedt, Gillberg, & Gillberg, 2007; Matson & Boisjoli, 2008; Matson, Cooper, Malone, & Moskow, 2008; Matson, Fodstad, &

Boisjoli, 2008). These behaviors can severely impact an individual's overall development and quality of life. Challenging behaviors can increase the risk of injury to self and others, interfere with skill acquisition, limit an individual's opportunities to socialize appropriately, limit an individual's involvement in community activities, increase stigmatization, lead to monetary repercussions, lead to psychotropic medication use, and, in severe cases, lead to death (Horner, Carr, Strain, Todd, & Reed, 2002; Luiselli, Blew, Keane, Thibadeau, & Holzman, 2000; Luiselli & Slocumb, 1983; Matson & Nebel-Schwalm, 2007a; McLoughlin, 1998; Mukaddes & Topcu, 2006; Paclawskyj, Matson, Rush, Smalls, & Vollmer, 2000, 2001; Peters-Scheffer, Didden, Mulders, & Korzilius, 2010; Sigafos, Arthur, & O'Reilly, 2003).

Given the potential severe effects of challenging behaviors, an understanding of challenging behaviors in individuals with ASD is critical. Thus, it is somewhat surprising that research on the topic has been limited (Horovitz, Matson, Hattier, Tureck, & Bamburg, in press; Horovitz, Matson, Rieske et al, 2011; Rojahn et al., 2009). Researchers have found a number of possible factors that may influence the prevalence and severity of challenging behaviors in individuals with ASD, including cognitive abilities (Bodfish et al., 2000; Dominick et al., 2007; Hartley, Sikora, & McCoy, 2008; Holden & Gitlesen, 2006; Matson & Nebel-Schwalm, 2007a; Matson, Wilkins, & Macken, 2009; McTiernan, Leader, Healy, & Mannion, 2011), severity of ASD symptoms (Baghdadli et al., 2003; Jang, Dixon, Tarbox, & Granpeesheh, 2011; Matson, Wilkins, & Macken, 2009), gender (Kozlowski & Matson, 2012), age (Baghdadli et al., 2003; Hartley et al., 2008), race (Chung et al., in press; Hartley et al., 2008; Horovitz et al., in press; Horovitz, Matson, Rieske et al, 2011), and adaptive abilities (Baghdadli et al., 2003; Dominick et al., 2007; Hartley et al., 2008). However, results have often been inconsistent or contradictory and need replication.

Infants and Toddlers. Like comorbid psychopathology, little is known about the development of challenging behaviors in infants and toddlers with ASD (Rojahn et al., 2009). Yet such knowledge is critical, as researchers have shown that interventions for challenging behaviors are most successful when initiated before five years of age, with continuing treatment being given as necessary (Carr, Severtson, & Lepper, 2009; Eikeseth, 2009; Hartley et al., 2008; Lancioni, Singh, O'Reilly, & Sigafos, 2009; Matson & Smith, 2008). While a number of instruments have been designed to assess for challenging behaviors in individuals with ID, few have been specifically designed for use with individuals with ASD, particularly infants and toddlers (Cohen et al., 2011). Measures of challenging behaviors in the ID population have, however, been frequently used with individuals with ASD. Two of the most commonly used and researched measures that fall into this category are the Aberrant Behavior Checklist (Aman, Singh, Stewart, & Field, 1985) and the Behavior Problem Inventory (BPI; Rojahn, Matson, Lott, Ebensen, & Smalls, 2001). In recent years researchers have begun to develop assessment measures of challenging behaviors designed specifically for use with individuals with ASD. These include the PDD Behavior Inventory (PDDBI; Cohen, Schmidt-Lackner, Romanczyk, & Sudhalter, 2003), the Autism Spectrum Disorders – Behavior Problems for Adults (ASD-BPA) (Matson & Rivet, 2007, 2008b), and the Autism Spectrum Disorders – Behavior Problems for Children (ASD-BPC; Matson, Gonzalez, & Rivet, 2008). As was the case with comorbidity, none of these measures were designed specifically to assess challenging behaviors in infants and toddlers. The BISCUIT-Part 3 was therefore developed for such use.

The Baby and Infant Screen for Children with aUtism Traits Part 3 (BISCUIT-Part 3) was developed from the ASD-CC to identify and assess for challenging behaviors that are typical of infants and toddlers with ASD (Matson, Wilkins, Sevin et al, 2009). The BISCUIT Part-3 is

part of the BISCUIT Battery, a comprehensive assessment battery of ASD in infants and toddlers aged 17 to 37 months. The BISCUIT-Part 3 includes 15 parent or caregiver-rated items that are scored in comparison to other toddlers of the same age. Items are rated on a three-point Likert scale of increasing severity. Factor analysis revealed a three-factor solution:

Aggressive/disruptive behaviors, Stereotypic behaviors, and SIB (Matson, Boisjoli, Rojahn, & Hess, 2009; Rojahn et al., 2009). Cutoffs and norms were established for each factor for children with and without an ASD (Matson, Fodstad et al, 2010; Rojahn et al., 2009). Internal consistency of the BISCUIT-Part 3 was found to be .91 (Matson, Wilkins, Sevin et al, 2009). Validity studies of the BISCUIT-Part 3 have not yet been conducted.

In addition to assessing for the presence, frequency, and severity of challenging behaviors, it is critical to understand why these behaviors are occurring. As such, it is generally agreed that a functional assessment of challenging behaviors is critical for the purposes of assessment and treatment planning (Chawarska & Bearss, 2008; Cohen et al., 2011; Council, 2001; Dawson & Osterling, 1997; Koegel et al., 2008; Matson & Nebel-Schwalm, 2007a). As operant conditioning (e.g., positive and negative reinforcement) is thought to most often explain the development and maintenance of challenging behaviors, functional assessment attempts to identify those variables that maintain challenging behaviors (Cohen et al., 2011; Iwata, Dorsey, Slifer, Bauman, & Richman, 1982; Matson & Minshawi, 2006). A full discussion of functional assessment is beyond the scope of this paper; however, it should be noted that the two primary methods of conducting functional assessment are experimental functional analysis (EFA) and the use of scaling measures. EFA involves experimental manipulation of possible maintaining variables and is often considered by many to be the preferred method of functional assessment (G. P. Hanley, Iwata, & McCord, 2003; Iwata et al., 1982; Matson & Minshawi, 2006).

Conversely, others have argued that scaling measures such as the Questions About Behavioral Function (QABF; Paclawskyj et al., 2000) or the Motivation Assessment Scale (MAS; Durand & Crimmins, 1988) may produce similar results, while posing fewer logistical or ethical difficulties (Hall, 2005; Matson & Minshawi, 2006, 2007; Paclawskyj et al., 2001).

Early Intervention

The advancement in early assessment for young children with ASD has allowed for the widespread implementation of early intervention. Researchers and clinicians have begun pushing for intervention to take place as early as possible, due to the possibility of improved outcomes across a multitude of areas (Corsello, 2005; Eikeseth et al., 2007; Eldevik et al., 2006; Eldevik et al., 2009; Fenske et al., 1985; Harris et al., 1991; Howard et al., 2005; Matson, 2007; Matson & Smith, 2008; Matson, Wilkins, & González, 2008; Peters-Scheffer et al., 2011; Remington et al., 2007; Shea, 2005; T. Smith, 1999; Werner et al., 2000). Such areas include receptive and expressive language (Eikeseth et al., 2007; Eldevik et al., 2006; Howard et al., 2005; Remington et al., 2007), adaptive behavior (Eikeseth et al., 2007; Howard et al., 2005; Remington et al., 2007), intelligence scores (Eldevik et al., 2006; Howard et al., 2005; Remington et al., 2007), challenging behaviors (Eikeseth et al., 2007), appropriate play (Eldevik et al., 2006), and positive social behaviors (Eikeseth et al., 2007; Remington et al., 2007), among others. These findings are not without controversy, as will be discussed further below (Matson, 2007; Matson & Smith, 2008; Peters-Scheffer et al., 2011). The most well-researched types of early intervention for ASD are behaviorally-based and are referred to as early intensive behavioral interventions (EIBI) (Matson & Smith, 2008; Shea, 2005)

Although most researchers agree that EIBI is the most successful approach to early intervention, there is less agreement as to what actually constitutes EIBI (Dawson & Osterling,

1997; Koegel, Koegel, Fredeen, & Gengoux, 2008; Love, Carr, Almason, & Petursdottir, 2008; Matson, 2007; Shea, 2005). This is because no standardized criteria for EIBI exist (Shea, 2005). Seven active ingredients of effective intervention were put forward by the National Research Council in 2001, based on a review of the literature: (1) Implementation of intervention as early as possible; (2) Intensive instruction for a minimum of five hours per day, five days a week; (3) Repeated planned teaching opportunities; (4) Daily, individual attention; (5) Parent and family training; (6) Ongoing assessment; (7) Priority given to functional communication, generalization of social skills, interaction skills with peers, maintenance and generalization of new skills, and functional assessment and positive behavior support for challenging behaviors. Other researchers have similarly outlined the essential elements of EIBI to include a highly supportive environment, a predictable routine, intensive one-on-one instruction up to 40 hours a week, the use of operant conditioning, the use of discrete trial training and incidental teaching, the involvement of family members and others in the environment, and a functional approach to challenging behaviors (Dawson & Osterling, 1997; Koegel et al., 2008). To better understand the components of EIBI that are currently used in clinical practice, Love, Carr, Almason, and Petursdottir (2008) conducted a survey of 211 EIBI programs. They found the following components to be most the common: provision of services in the home, the inclusion of speech and occupational therapy, the use of parents in incidental and naturalistic teaching, the use of choice-based preference assessments, the implementation of generalization procedures, the frequent collection of data, an assessment of reliability on a monthly basis, and a descriptive assessment of challenging behaviors. Despite these commonalities, a great deal of variability remains in EIBI programs (Love et al., 2008).

Early Intervention Outcomes

The variability in EIBI programs has led to difficulty in accurately assessing their effectiveness. Despite this, a number of studies have been conducted in an attempt to determine the effects of EIBI. In an attempt to summarize these findings, using stringent inclusion criteria, Eldevik et al. (2009) conducted a meta-analysis on nine well-designed EIBI studies. The authors found an overall effect size (Hedges' g) of 1.103 for IQ score changes and an effect size of .660 for changes in adaptive behavior composite scores. A more recent meta-analysis was conducted by Peters-Scheffer et al. (2011) on 11 studies meeting similar inclusion criteria. The authors found moderate to large effects for changes in full scale and non-verbal IQ, expressive and receptive language, and adaptive behaviors. Although stringent inclusion criteria were employed, only one of the included studies could truly be classified as a randomized controlled trial. Thus, while these two meta-analyses provide strong evidence in support of EIBI, more research is clearly needed.

Although strong preliminary support has been found for EIBI, researchers have pointed out a number of limitations. Firstly, a number of methodological problems limit the interpretability of the results found by EIBI researchers. Such limitations include failing to measure treatment fidelity, poor group classification criteria, using dependent measures for classification purposes, and failing to report on possible side effects of EIBI (Matson, 2007). As previously mentioned, few studies of EIBI can be characterized as randomized controlled trials (Matson, 2007; Matson & Smith, 2008; Peters-Scheffer et al., 2011). Thus more methodological rigor is needed in studies examining EIBI effectiveness. Additionally, researchers have rarely examined the effects of EIBI on core symptoms of ASD, comorbidity, or challenging behaviors (Matson, 2007). Instead, measures of intelligence or adaptive skills are typically used. Matson

(2007) stresses that, while the aforementioned meta-analyses provided support for increases in IQ, these results should be reworded as increases in IQ scores, as it is likely that changes in compliance and attention led to changes in IQ scores, rather than true changes in intelligence. Finally, there are a number of child characteristics that may affect the success of EIBI, a possibility often ignored by EIBI researchers (Matson & Smith, 2008). For example, more positive outcome scores are often associated with less severe baseline ASD symptoms, higher IQ, and fewer comorbid problems at pretest (Matson & Smith, 2008). More research is needed examining these individual characteristics and their relationship to EIBI (Matson & Smith, 2008). While researchers have begun extensively examining EIBI, the number of such studies is still very small when compared to the number of studies examining treatment in ASD as a whole (Matson & Smith, 2008). Although most research on EIBI thus far is encouraging, more research is needed.

Purpose

In recent years, the support for early intervention for children with ASD has grown immensely. This support has been augmented by research suggesting the possibility of improved outcomes with earlier intervention implementation (Corsello, 2005; Eikeseth et al., 2007; Eldevik et al., 2006; Eldevik et al., 2009; Fenske et al., 1985; Harris et al., 1991; Howard et al., 2005; Matson, 2007; Matson & Smith, 2008; Matson, Wilkins, & González, 2008; Peters-Scheffer et al., 2011; Remington et al., 2007; Shea, 2005; T. Smith, 1999; Werner et al., 2000). However, in order for early intervention to be implemented, the accurate identification and diagnosis of an ASD must occur. As such, researchers have begun examining ways to improve the assessment of ASD in children below three years of age.

The BISCUIT is an assessment battery that examines diagnostic criteria, symptoms of comorbidity, and challenging behaviors in infants and toddlers aged 17 to 37 months that are considered at-risk for an ASD. Preliminary psychometric studies of the BISCUIT have been promising. On Part 1 of the BISCUIT, sensitivity and specificity for differentiating toddlers with ASD from toddlers with atypical development, using a cutoff score of 17, were found to be 93.4 and 86.6, respectively (Matson, Wilkins, Sevin et al, 2009). Additionally, clinical cutoff scores have been developed for Parts 2 and 3 of the BISCUIT. The current cutoff scores of the BISCUIT do not, however, take into account the age of the child being assessed. While the BISCUIT is only intended for toddlers in a small age range (i.e., 17 to 37 months), a great amount of development and change typically occurs during this time frame (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991). Therefore, examination of even more specific age ranges may be warranted. This is of particular importance when considering that the BISCUIT asks parents to compare their child to other children of the same

age. Many of the core features of ASD may become more apparent as the child ages and falls further behind his or her typically developing peers (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey, Truesdale-Kennedy, & Cassidy, 2009; Mitchell et al., 2006; Werner & Dawson, 2005). It is therefore not surprising that the use of age-based scoring procedures is common practice amongst other measures designed to assess for ASD and other psychological disorders (e.g., Achenbach & Rescorla, 2000; Kim & Lord, 2012; Lord et al., 1994; Matson, Kozlowski, Neal, Worley, & Fodstad, 2011; Reynolds & Kamphaus, 2004; Sparrow et al., 2005). Given this knowledge, the primary aim of the current study was to develop age-based cutoff scores for each part of the BISCUIT and to compare them to full-sample cutoff scores. Three studies were conducted looking at each part of the BISCUIT, respectively.

The development of age-based cutoff scores on the BISCUIT-Part 1 could help to increase the measure's sensitivity and specificity, thereby increasing the likelihood that a child with ASD be identified as such, while at the same time increasing the likelihood that a child who does not have an ASD be identified as such. This would, in turn, increase the likelihood that children receive appropriate services and treatment as early as possible. Additionally, the development of age-based cutoff scores for the BISCUIT-Parts 2 and 3 may help more clearly and accurately identify symptoms of comorbidity and challenging behavior that are of clinical concern, thereby leading to the delivery of appropriate treatment.

Method

Participants

A total of 3118 infants and toddlers aged 17 to 37 months ($M=25.91$, $SD=4.79$) and their caregivers served as the participants for the current studies. The initial sample included 3460 infants and toddlers and their caregivers; however, 42 were removed because age was unknown or fell outside of the target age range (i.e., 17 to 37 months) and 300 were removed because they had not yet been assigned a diagnostic classification (via the process described further below). All participants were enrolled in the State of Louisiana's EarlySteps program. EarlySteps is Louisiana's Early Intervention System under the Individuals with Disabilities Education Act, Part C, which provides services to infants and toddlers and their families from birth to 36 months. Qualification for this program requires the presence of a developmental delay or a medical condition that is likely to result in a developmental delay. The populations served by this program include, but are not limited to, children with ASD, cerebral palsy, epilepsy, Down's syndrome, blindness, asthma, allergies, and global developmental delays.

A visual inspection of the sampling distribution did not reveal clear age groups to use for the current studies. Thus, age groups were developed based on a reading of the relevant literature. Based on the approximate intervals at which developmental milestones are most frequently cited in the literature (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991), children were separated into the following three age cohorts determined by the age at assessment: 17 to 23 months, 24 to 30 months, and 31 to 37 months. Using frequently cited developmental milestones as anchors was beneficial, as informants and clinicians must be able to accurately determine how the child compares to other children of the same age.

Diagnostic classifications of autism, PDD-NOS, or non-ASD related atypical development were

made by a licensed doctoral level psychologist with over 30 years of experience in the area of developmental disabilities (Matson, Wilkins, Sevin et al., 2009). Diagnostic classification was blind to BISCUIT scores and was based on a combination of *DSM-IV-TR* (APA, 2000) criteria, M-CHAT (Robins et al., 2001) scores, and developmental profiles from the Battelle Developmental Inventory – 2nd Edition (BDI-2; Newborg, 2005). Similar methodology has been used elsewhere in the literature for research purposes (Fombonne et al., 2004; Matson, Wilkins, Sharp et al, 2009). These diagnostic groups (i.e., Autism, PDD-NOS, and non-ASD related atypical development) were chosen, as they are the intended population for the BISCUIT (Matson, Wilkins, Sevin et al, 2009). A full breakdown of demographic variables by group can be found in Table 1.

Table 1. Demographic Characteristics per Diagnostic Group and Age Cohort

	17 to 23 months (N=1029)		
	Autism (N=140)	PDD-NOS (N=124)	Non-ASD related atypical development (N=765)
Age (in months), mean (SD)	20.25 (1.72)	20.60 (1.69)	20.45 (1.71)
Gender (%)			
Male	70.00	67.70	67.10
Female	29.30	31.50	32.70
Unspecified	.70	.80	.20
Race/ethnicity (%)			
Caucasian	46.40	43.50	53.20
African-American	43.60	43.50	34.90
Hispanic	1.40	0.00	3.40
Other	8.60	12.90	8.50
	24 to 30 months (N=1460)		
	Autism (N=252)	PDD-NOS (N=192)	Non-ASD related atypical development (N=1016)
Age (in months), mean (SD)	27.19 (2.04)	27.14 (1.98)	26.60 (1.99)
Gender (%)			
Female	25.00	27.10	28.80
Unspecified	.00	1.00	.70

(Table 1 continued)

	24 to 30 months (N=1460)		
	Autism (N=252)	PDD-NOS (N=192)	Non-ASD related atypical development (N=1016)
Race/ethnicity (%)			
Caucasian	50.00	45.80	48.20
African-American	40.50	42.20	39.00
Hispanic	1.60	2.60	3.10
Other or Unspecified	8.00	9.40	9.70
	31 to 37 months (N=629)		
	Autism (N=113)	PDD-NOS (N=71)	Non-ASD related atypical development (N=445)
Age (in months), mean (SD)	32.71 (1.43)	32.79 (1.52)	32.73 (1.44)
Gender (%)			
Male	75.20	66.20	70.80
Female	24.80	33.80	28.50
Unspecified	.00	.00	.70
Race/ethnicity (%)			
Caucasian	47.80	52.10	45.40
African-American	38.10	35.20	39.60
Hispanic	.90	2.80	2.90
Other	13.30	9.80	12.10

Measures

Baby and Infant Screen for Children with aUtism Traits (BISCUIT; Matson, Wilkins, Sevin et al, 2009). The BISCUIT is a three-part battery of parent-report measures designed to assess toddlers aged 17 to 37 months that are considered at risk for an ASD. Part-1 of the BISCUIT is the diagnostic section, which is comprised of 62 questions designed to assess for the diagnostic criteria of ASD. Parents rate items on a 3-point Likert-type scale with respect to how their child compares with typically developing children of the same age. A three factor solution was found via factor analysis: socialization/nonverbal communication, repetitive behaviors/restricted interest and communication (Matson, Boisjoli et al, 2010). Children who score below the cutoff score of 17 are placed in the No ASD/Atypical Development range,

children who score between 17 and 39 are placed in the Possible ASD/PDD-NOS range, and children who score at or above the cutoff score of 39 are placed in the Probable ASD/Autistic Disorder range (Matson, Wilkins, Sharp et al, 2009). Internal reliability of the measure was reported to be .97 (Matson, Wilkins, Sevin et al, 2009). Sensitivity and specificity were found to be .84 and .86, respectively, when differentiating those without a diagnosis of ASD from those with PDD-NOS (Matson, Wilkins, Sevin et al, 2009). Sensitivity and specificity were .84 and .83 when differentiating PDD-NOS from autism (Matson, Wilkins, Sevin et al, 2009).

Part 2 of the BISCUIT assesses for symptoms of comorbidity in infants and toddlers considered at risk for an ASD. The BISCUIT-Part 2 contains 57 items that are similarly scored on a 3-point Likert-type scale. Factor analysis yielded five distinct factors: Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eating/Sleep Problems (Matson, Boisjoli et al, 2011). Cutoffs and norms for each factor have been established for children with and without ASD (Matson, Fodstad, & Mahan, 2009; Matson, Fodstad, Mahan et al, 2009). Internal consistency of the scale was found to be .96 (Matson, Wilkins, Sevin et al, 2009).

The third and final part of the BISCUIT measures and assesses for challenging behaviors that are common amongst infants and toddlers with ASD. The BISCUIT-Part 3 is comprised of 15 items that are again rated on a 3-point Likert-type scale. A three-factor solution was found via factor analysis: Aggressive/Disruptive Behaviors, Stereotypic Behaviors, and SIB (Matson, Boisjoli, Rojahn et al, 2009; Rojahn et al., 2009). Like the BISCUIT-Part 2, cutoffs and norms for each factor have been established for children with and without an ASD (Matson, Fodstad et al, 2010; Rojahn et al., 2009). Internal reliability of the BISCUIT-Part 3 was found to be .91 (Matson, Wilkins, Sevin et al, 2009).

Modified Checklist for Autism in Toddlers (M-CHAT) (Robins et al., 2001). The M-CHAT is a 23-item screen for ASD that is based on parent report. Parents or caregivers answer items on a dichotomous Yes/No scale regarding their child's typical functioning. A screen is considered positive if three or more items are failed. Additionally, failure of two or more of the five "critical" items results in a positive screen. Internal reliability of the entire scale was found to be .85, while it was found to be .83 for the critical items (Robins et al., 2001). Sensitivity and specificity of the M-CHAT were found to be .87 and .99, respectively.

Battelle Developmental Inventory, Second Edition (BDI-2) (Newborg, 2005). The BDI-2 assesses five domains of development in children from birth to 7 years, 11 months: personal/social, adaptive, motor, communication, and cognitive. The measure includes 450 items that are answered by caregivers on a 3-point scale of increasing ability. Scores produce a developmental quotient in each domain, as well as a total developmental quotient, with a mean of 100 and standard deviation of 15. Internal consistency of BDI-2 total developmental quotients ranged from .98 to .99, with all domains and total score demonstrating test-retest reliability above .80 (Newborg, 2005). Validity of the measure has been established with many populations, including children with ASD and DD (Newborg, 2005).

Procedures

The BISCUIT, M-CHAT, and BDI-2 were administered to parents or caregivers in the child's home or daycare setting by professionals whose licensure or certification met requirements for the provision of services through EarlySteps. These professionals came from a variety of disciplines, including psychology, occupational therapy, speech-language pathology, physical therapy, social work, and special education. The professionals received extensive training on the topic of ASD and on each of the included measures to ensure standardized

administration (Matson, Wilkins, Sevin et al, 2009). All informants were the parents or legal guardians of the participating children and provided informed consent. This study received prior approval by the Louisiana State University Institutional Review Board and Louisiana's Office for Citizens with Developmental Disabilities.

Sample Size

An *a priori* analysis was conducted to determine the sample size needed to conduct an ROC analysis, with alpha of .05, power of .80, and an estimated area under the curve (AUC) of .80 (representing good discriminative ability). Using these criteria, the required sample size was 28 per group (Compton, Fuchs, Fuchs, & Bryant, 2006; J.A. Hanley & McNeil, 1982; MedCalc, 2011; Swets, 1988). Elsewhere it has been recommended that a total sample size of 100 be employed for ROC analyses (Metz, 1978). Finally, it has also been recommended that normative studies employ a minimum of approximately 50 participants per group (Bridges & Holler, 2007).

Study 1

Research Design

Study 1 first examined the discriminating ability of the BISCUIT-Part 1 for the overall sample and for each of the target age cohorts (i.e., 17 to 23 months, 24 to 30 months, and 31 to 37 months). This was done to ensure that the BISCUIT-Part 1 was appropriate for each age cohort. This was achieved by conducting a receiver operating characteristics (ROC) analysis for the full sample, as well as for each of the target age cohorts. The discriminating ability of the BISCUIT was determined by the Area Under the Curve (AUC) statistic. AUC values range from 0.5, representing chance performance, to 1.0, representing perfect performance (Compton et al., 2006; Fombonne, 1991; Swets, 1988). An AUC above .90 is considered to represent excellent discriminating ability; .80 to .90 is considered good discriminating ability; .70 to .80 is considered fair discriminating ability; and an AUC below .70 is considered to represent poor discriminating ability (Compton et al., 2006; Swets, 1988). The significance level of each AUC was tested at $\alpha = .05$, with a significant value indicating that the BISCUIT performed significantly better than chance for the target population. For each group with a significant AUC, the cutoff point that maximized sensitivity and specificity was established using the Youden Index, which represents the overall accuracy of the test (Krzanowski & Hand, 2009; Kumar & Indrayan, 2011; Perkins & Schisterman, 2005; Youden, 1950). These analyses were conducted twice for the overall sample and twice for each age cohort: once to establish a cutoff score to differentiate between no diagnosis and PDD-NOS and once to establish a cutoff score to differentiate between PDD-NOS and autism (Matson, Wilkins, Sharp et al, 2009). Thus, a total of eight ROC curves were created and analyzed.

Results

Prior to the ROC analysis, the data was examined for missing values and outliers. Outliers were detected by grouping participants by both diagnostic classification and age cohort and calculating group z -scores for Part 1 of the BISCUIT; z -scores with an absolute value greater than 3.29 were identified as outliers (Field, 2005). Participants missing at least 10% of data points or with scores identified as outliers were excluded from the ROC analysis (Donner, 1982; Field, 2005; Matson, Wilkins, Sharp et al., 2009). From the full sample of 3118 participants, 36 (1.15 %) participants with missing data and 20 (0.64%) participants identified as outliers were removed from the analyses in Study 1. Thus the final sample used in Study 1 included 3062 participants.

Full Sample. First, the ROC analysis was conducted for the full sample to determine the optimal cutoff for discriminating between atypical development and PDD-NOS. The resulting curve had an $AUC=.93$, $p<.01$, representing excellent discriminating ability (Compton et al., 2006; Swets, 1988). Figure 1 shows the curve created by this analysis. To determine the cutoff point that maximized sensitivity and specificity, the Youden Index was used. The Youden Index J is defined as the maximum (sensitivity + specificity – 1) for all possible values (Youden, 1950). J is the point on the curve that falls furthest from the diagonal line that represents chance performance. The optimal cutoff for differentiating between atypical development and PDD-NOS for the full sample was a score greater than 17, $J=.70$. Sensitivity and specificity at this cutoff were .86 and .83, respectively.

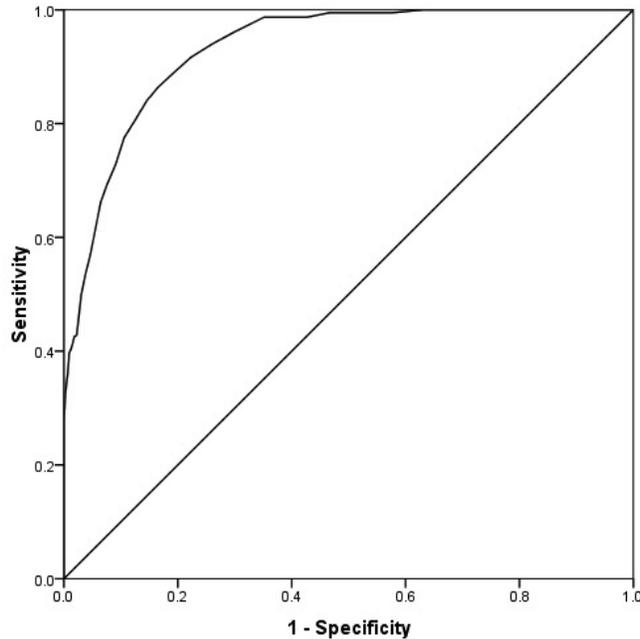


Figure 1. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for the full sample of toddlers with atypical development or PDD-NOS. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Next, these procedures were repeated to determine the optimal cutoff for differentiating PDD-NOS from autism. The AUC was .87, $p < .01$, representing good discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 2. The ROC curve for this analysis can be seen in Figure 2. The optimal cutoff for differentiating between PDD-NOS and autism for the full sample was a score greater than 42, $J = .55$. Sensitivity and specificity at this cutoff were .73 and .82, respectively. Table 2 shows the sensitivity, specificity, and J produced by various possible cutoff points for both curves.

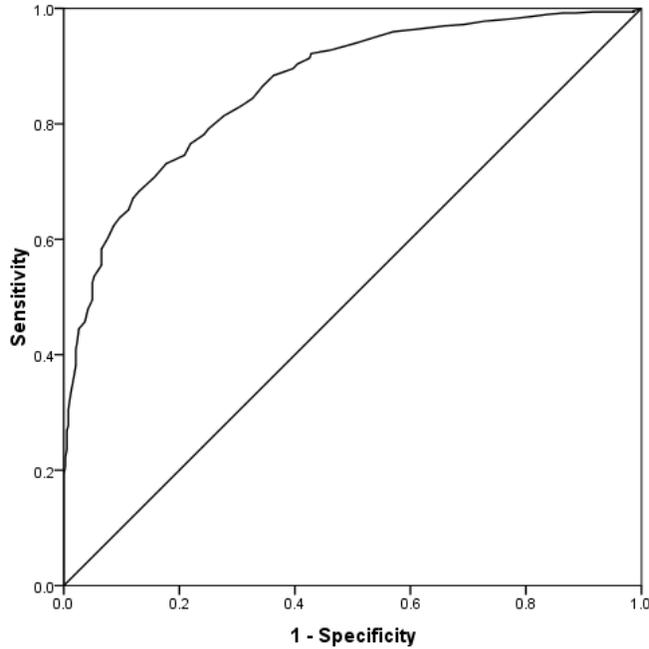


Figure 2. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for the full sample of toddlers with PDD-NOS or autism. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Table 2. Sensitivity, specificity, and Youden Index J for various cutoff points in the full sample

	Cutoff	Sensitivity	Specificity	Youden Index J
Atypical Development vs. PDD-NOS	>14	.9399	.7394	.6793
	>15	.9164	.7771	.6935
	>16	.8851	.8119	.6970
	>17*	.8642	.8344	.6986
	>18	.8407	.8541	.6948
	>19	.8094	.8729	.6823

*Indicates scores chosen as final cutoffs for the full sample

(Table 2 continued)

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
	>20	.7755	.8940	.6695
PDD-NOS vs. Autism	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
	>39	.7816	.7572	.5388
	>40	.7655	.7807	.5462
	>41	.7455	.7911	.5366
	>42*	.7315	.8225	.5539
	>43	.7074	.8433	.5507
	>44	.6834	.8695	.5529
	>45	.6713	.7572	.5388

*Indicates scores chosen as final cutoffs for the full sample

Ages 17 to 23 months. Next, the ROC analyses were repeated, using only infants and toddlers aged 17 to 23 months. This was performed to determine the discriminating ability of the BISCUIT-Part 1 for this age range and, if appropriate, to develop age-specific cutoffs. As with the full sample, the ROC analysis was first conducted to discriminate between atypical development and PDD-NOS. The AUC was .92, $p < .01$, representing excellent discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 3. The optimal cutoff for differentiating between atypical development and PDD-NOS for those aged 17 to 23 months was a score greater than 14, $J = .69$. Sensitivity and specificity at this cutoff were .93 and .76, respectively.

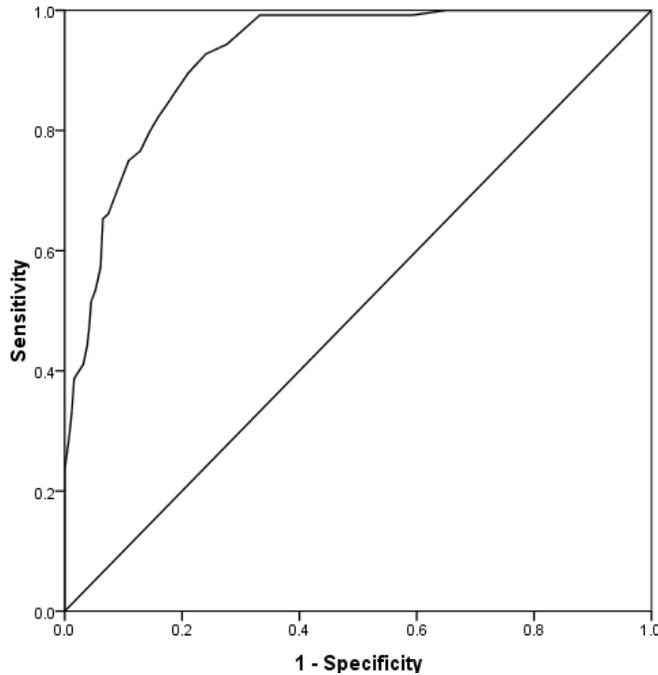


Figure 3. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 17 to 23 months with atypical development or PDD-NOS. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Next, the ROC analyses were repeated to determine the optimal cutoff for discriminating between PDD-NOS and autism in those aged 17 to 23 months. The AUC was .90, $p < .01$, representing good discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 4. The optimal cutoff for differentiating between PDD-NOS and autism for those aged 17 to 23 months was a score greater than 39, $J = .62$. Sensitivity and specificity at this cutoff were .80 and .81, respectively. Table 3 shows the sensitivity, specificity, and J produced by various possible cutoff points for both curves.

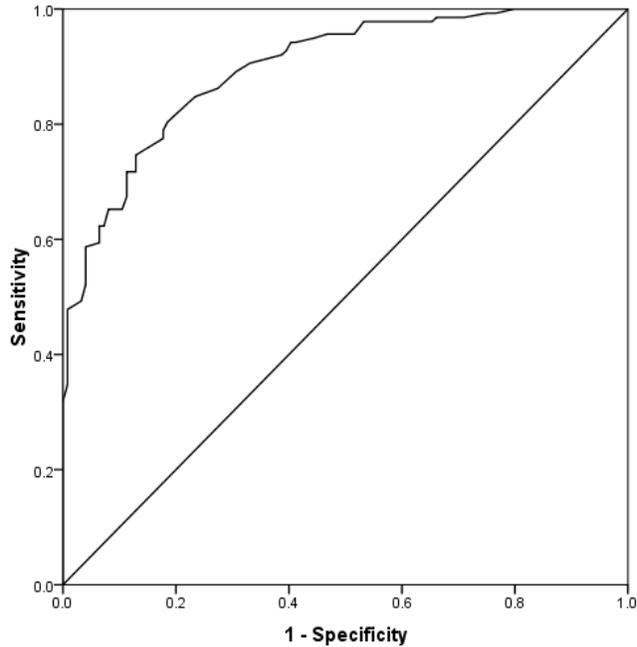


Figure 4. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 17 to 23 months with PDD-NOS or autism. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Table 3. Sensitivity, specificity, and Youden Index J for various cutoff points in infants and toddlers aged 17 to 23 months

	Cutoff	Sensitivity	Specificity	Youden Index J
Atypical Development vs. PDD-NOS	>12	.9919	.6676	.6595
	>13	.9435	.7234	.6669
	>14*	.9274	.7593	.6867
	>15	.8952	.7899	.6851
	>16	.8548	.8178	.6726
	>17^	.8226	.8404	.6630

*Indicates scores chosen as final cutoffs for those aged 17 to 23 months

^Indicates scores previously chosen as final cutoffs for the full sample

(Table 3 continued)

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
	>18	.7984	.8551	.6595
PDD-NOS vs. Autism	>36	.8623	.7258	.5881
	>37	.8478	.7661	.6139
	>38	.8116	.8065	.6181
	>39*	.8043	.8145	.6188
	>40	.7899	.8226	.6125
	>41	.7754	.8226	.5980
	>42^	.7609	.8468	.5881

*Indicates scores chosen as final cutoffs for those aged 17 to 23 months

^Indicates scores previously chosen as final cutoffs for the full sample

Ages 24 to 30 months. Next, the discriminating ability was tested and age-specific cutoffs were developed for infants and toddlers aged 24 to 30 months. The ROC analysis was first conducted to discriminate between atypical development and PDD-NOS. The AUC was .93, $p < .01$, representing excellent discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 5. The optimal cutoff for differentiating between atypical development and PDD-NOS for those aged 24 to 30 months was a score greater than 18, $J = .70$. Sensitivity and specificity at this cutoff were .85 and .85, respectively.

Next, the ROC analyses were repeated to determine the optimal cutoff for discriminating between PDD-NOS and autism in those aged 24 to 30 months. The AUC was .84, $p < .01$, representing good discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 6. The optimal cutoff for differentiating between PDD-NOS and autism for those aged 24 to 30 months was a score greater than 47, $J = .54$. Sensitivity

and specificity at this cutoff were .64 and .90, respectively. Table 4 shows the sensitivity, specificity, and J produced by various possible cutoff points for both curves.

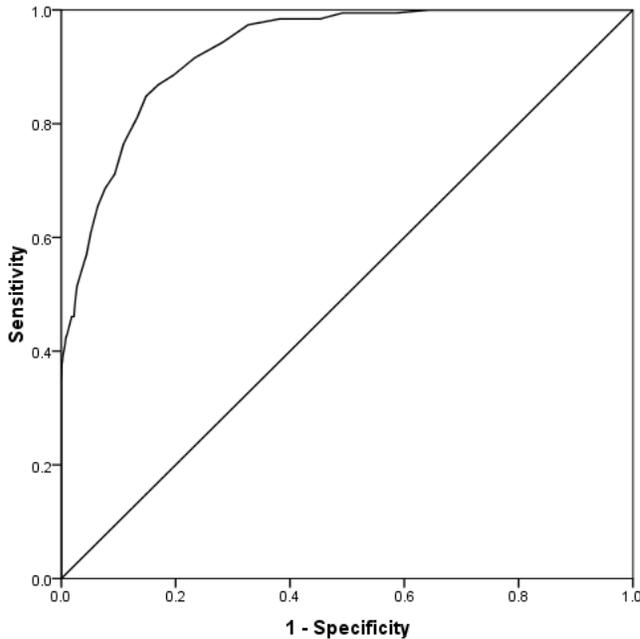


Figure 5. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 24 to 30 months with atypical development or PDD-NOS. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Ages 31 to 37 months. Finally, age specific cutoffs were created for infants and toddlers aged 31 to 37 months. The ROC analysis was first conducted to discriminate between atypical development and PDD-NOS. The AUC was .95, $p < .01$, representing excellent discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 7. The optimal cutoff for differentiating between atypical development and PDD-NOS for

those aged 31 to 37 months was a score greater than 19, $J=.77$. Sensitivity and specificity at this cutoff were .88 and .89, respectively.

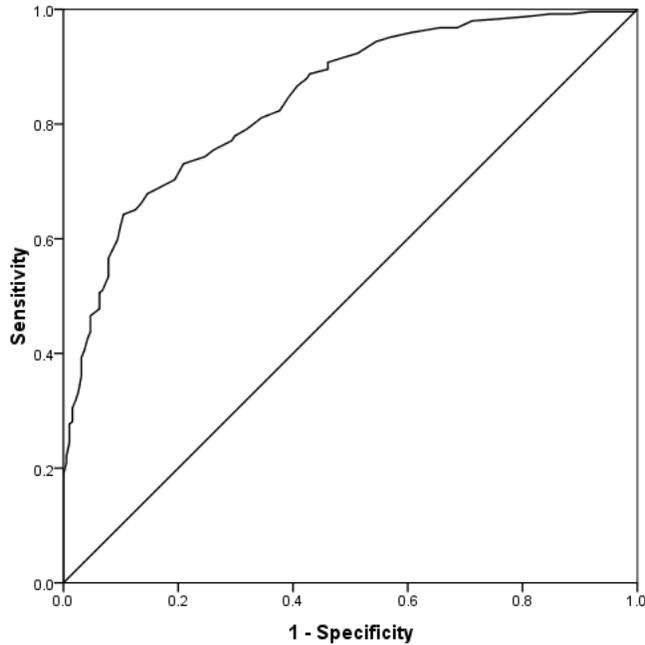


Figure 6. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 24 to 30 months with PDD-NOS or autism. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Table 4. Sensitivity, specificity, and Youden Index J for various cutoff points in infants and toddlers aged 24 to 30 months

	Cutoff	Sensitivity	Specificity	Youden Index J
Atypical Development vs. PDD-NOS	>15	.9162	.7661	.6823
	>16	.8848	.8044	.6892
	>17 [^]	.8691	.8296	.6987

*Indicates scores chosen as final cutoffs for those aged 24 to 30 months

[^]Indicates scores previously chosen as final cutoffs for the full sample

(Table 4 continued)

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
	>18*	.8482	.8518	.7000
	>19	.8115	.8669	.6784
	>20	.7644	.8911	.6555
	>21	.7120	.9062	.6823
PDD-NOS vs. Autism	>42^	.7309	.7906	0.5215
	>45	.6627	.8639	.5266
	>46	.6506	.8743	.5249
	>47*	.6426	.8953	.5379
	>48	.6225	.9005	.523
	>49	.5984	.9058	.5042
	>50	.5663	.9215	.5321

*Indicates scores chosen as final cutoffs for those aged 24 to 30 months

^Indicates scores previously chosen as final cutoffs for the full sample

Next, the ROC analyses were repeated to determine the optimal cutoff for discriminating between PDD-NOS and autism in those aged 31 to 37 months. The AUC was .88, $p < .01$, representing good discriminating ability (Compton et al., 2006; Swets, 1988). The ROC curve for this analysis can be seen in Figure 8. The optimal cutoff for differentiating between PDD-NOS and autism for those aged 31 to 37 months was a score greater than 33, $J = .60$. Sensitivity and specificity at this cutoff were .89 and .71, respectively. Table 5 shows the sensitivity, specificity, and J produced by various possible cutoff points for both curves.

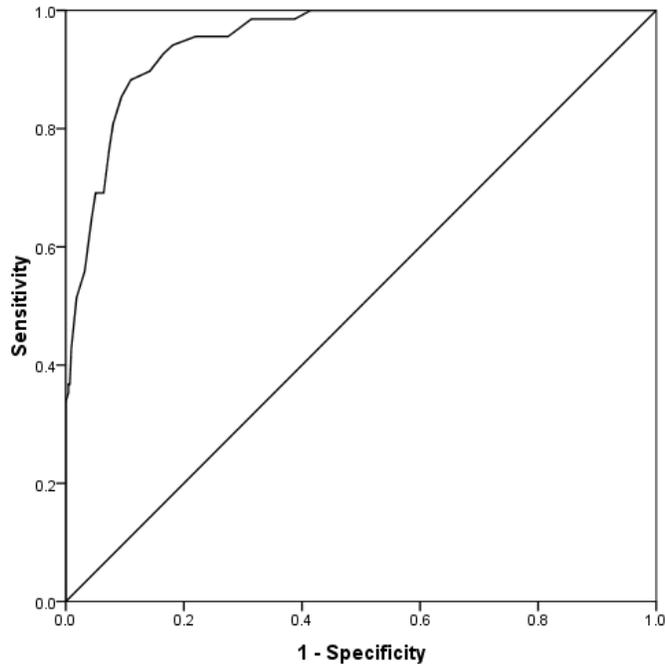


Figure 7. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 31 to 37 months with atypical development or PDD-NOS. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Table 5. Sensitivity, specificity, and Youden Index *J* for various cutoff points in infants and toddlers aged 31 to 37 months

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
Atypical Development vs. PDD-NOS	>16	.9412	.8184	.7596
	>17 [^]	.9265	.8345	.7610
	>18	.8971	.8575	.7546
	>19 [*]	.8824	.8897	.7721
	>20	.8529	.9057	.7586
	>21	.8088	.9195	.7283
	>22	.7647	.9264	.6911

*Indicates scores chosen as final cutoffs for those aged 31 to 37 months

[^]Indicates scores previously chosen as final cutoffs for the full sample

(Table 5 continued)

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
PDD-NOS vs. Autism	>31	.9107	.6471	.5578
	>32	.9018	.6618	.5636
	>33	.8929	.7059	.5988
	>34*	.8750	.7206	.5956
	>35	.8571	.7353	.5924
	>36	.8393	.7353	.5746
	>42^	.6964	.8676	.564

*Indicates scores chosen as final cutoffs for those aged 31 to 37 months

^Indicates scores previously chosen as final cutoffs for the full sample

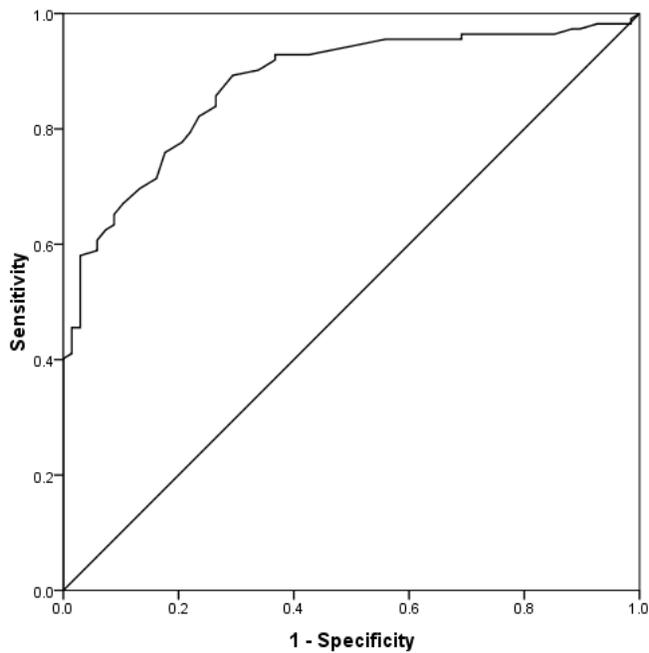


Figure 8. ROC curve representing the trade-offs between sensitivity and 1-specificity for the BISCUIT for toddlers aged 31 to 37 months with PDD-NOS or autism. The diagonal line represents chance performance, while the curved line represents the performance of the BISCUIT.

Discussion

A number of things stand out when looking at the findings of Study 1. Firstly, the BISCUIT-Part 1 demonstrated excellent ability to discriminate between PDD-NOS and non-ASD related atypical development across all age cohorts. Likewise, the BISCUIT-Part 1 demonstrated good ability to discriminate between PDD-NOS and autism across each age cohorts. Additionally, the AUC statistic was significant for each curve, suggesting that the BISCUIT performed significantly better than chance for each age cohort. It should not come as a surprise that the BISCUIT was somewhat better at discriminating between atypical development and PDD-NOS than it was at discriminating between PDD-NOS and autism. Researchers have previously shown that, while there may be some variability in early diagnoses of specific ASDs, early classifications of ASD or non-ASD related atypical development tend to remain more stable (Chawarska et al., 2009; Worley et al., 2011). Overall, these findings lend further support to the wealth of research supporting early assessment of ASD using a psychometrically sound instrument (Adrien et al., 1993; Baron-Cohen et al., 1992; Chawarska et al., 2009; Cox et al., 1999; Lord et al., 2006; Matson, Wilkins, & González, 2008; Matson, Wilkins, Sevin et al, 2009; Mitchell et al., 2006; Osterling & Dawson, 1994; Osterling et al., 2002; Werner et al., 2005; Worley et al., 2011). Particularly, the findings with the youngest age cohort (i.e., those aged 17 to 23 months) provide further evidence that ASD can be reliably assessed before a child's second birthday (Baron-Cohen et al., 1992; Matson et al., in press; Matson, Wilkins, & González, 2008; Matson, Wilkins, Sevin et al, 2009). Additionally, these findings add to the research demonstrating the sound psychometric properties of the BISCUIT-Part 1 (Horovitz & Matson, 2010; LoVullo & Matson, 2012; Matson, Boisjoli et al, 2010; Matson, Fodstad, & Dempsey,

2009b; Matson, Wilkins, & Fodstad, 2011; Matson, Wilkins, Sevin et al, 2009; Matson, Wilkins, Sharp et al, 2009; Worley et al., 2011).

As hypothesized, the optimal cutoff points varied between the age cohorts and the full sample. A summary of the final cutoffs selected for each age group can be found in Table 6. The cutoffs and psychometric properties for the full sample varied somewhat from the original study on the BISCUIT-Part 1 (Matson, Wilkins, Sharp et al., 2009). While the original study found the optimal cutoffs to be scores above 17 and 39, the current study found the optimal cutoffs for the full sample to be scores above 17 and 42. However, given that the current study included over 2000 additional participants, some differences between studies were expected.

Table 6. Selected cutoffs and psychometric properties for each sample

	Cutoff	Sensitivity	Specificity	Youden Index <i>J</i>
Full Sample				
Atypical Development vs. PDD-NOS	>17	.8642	.8344	.6986
PDD-NOS vs. autism	>42	.7315	.8225	.5539
17 to 23 months				
Atypical Development vs. PDD-NOS	>14	.9274	.7593	.6867
PDD-NOS vs. autism	>39	.8043	.8145	.6188
24 to 30				
Atypical Development vs. PDD-NOS	>18	.8482	.8518	.7000
PDD-NOS vs. autism	>47	.6426	.8953	.5379
31 to 37 months				
Atypical Development vs. PDD-NOS	>19	.8824	.8897	.7721
PDD-NOS vs. autism	>34	.8750	.7206	.5956

Given the significant amount of development that occurs between the ages of 17 and 37 months (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991), it is reasonable that one set of cutoff scores was not optimal for the specific age cohorts. In fact, the optimal cutoffs for the full sample were not ideal for any of the individual age cohorts. When looking closer at the cutoffs for differentiating between atypical development and PDD-NOS, it can be seen that, as the age cohort increased, a higher cutoff score was more appropriate. Previous research has suggested that the features of ASD may become more apparent as a child ages, with the child falling further behind his or her peers (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey et al., 2009; Mitchell et al., 2006; Werner & Dawson, 2005). As such, it is fitting that, as a child ages, a higher cutoff score would be more appropriate for discriminating between PDD-NOS and atypical development. Additionally, although the sensitivity and specificity were adequate for all age cohorts, the overall accuracy of the BISCUIT-Part 1, as indicated by the Youden Index J , increased as the age cohort increased. This again supports the notion that the impairments associated with ASD become more pronounced and easier to detect as a child ages (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey et al., 2009; Mitchell et al., 2006; Werner & Dawson, 2005).

The same pattern was not seen when looking at cutoffs for differentiating between PDD-NOS and autism. A U-shaped pattern was seen, with the highest cutoff indicated for the 24 to 30 month age cohort, followed by the 17 to 23 month age cohort. Additionally, the overall accuracy of the BISCUIT-Part 1 when differentiating between PDD-NOS and autism was actually highest for the 17 to 23 month age cohort and lowest for the 24 to 30 month age cohort. These findings may reflect some of the aforementioned variability that exists within early diagnoses of specific

ASD (Chawarska et al., 2009; Worley et al., 2011). One possible explanation for the higher cutoffs found in the younger two age cohorts is that, at younger ages, only children with severe symptoms are given a diagnosis of autism to avoid providing the more stigmatized label of autism to those who have less severe symptoms (Ben-Zeev, Young, & Corrigan, 2010; Calzada, Pistrang, & Mandy, 2012; Neal et al., in press). As the older age cohort approaches the average age of typical diagnosis (DeGiacomo & Fombonne, 1998; Kozlowski et al., 2011; Matson, 2005; Tager-Flusberg & Anderson, 1992), diagnoses of autism may be more likely in those with somewhat less severe symptoms. However, these reasons are merely conjectural and more research is needed to understand why such a pattern was found.

Study 2

Research Design

Study 2 established age-based cutoffs for Part 2 of the BISCUIT, which assesses for symptoms of comorbidity in infants and toddlers considered at risk for an ASD. Cutoffs were determined using the standard deviation from the mean method (Kendall & Grove, 1988; Matson, Fodstad, & Mahan, 2009; Matson, Fodstad et al, 2010; Matson, Fodstad, Mahan et al, 2009; Rojahn et al., 2009). Although the sample is not normally distributed, it is representative of infants and toddlers with ASD and infants and toddlers with atypical development. This methodology followed the same research design used to develop the current cutoffs for Part 2 of the BISCUIT (Kendall & Grove, 1988; Matson, Fodstad, & Mahan, 2009; Matson, Fodstad et al, 2010; Matson, Fodstad, Mahan et al, 2009; Rojahn et al., 2009). Separate cutoffs were established for children with ASD and for children with non-ASD related atypical development, as well as for children in each of the target age cohorts. Therefore, participants were separated into one of six groups for analysis of cutoffs: (1) 17 to 23 months with ASD; (2) 17 to 23 months with non-ASD related atypical development; (3) 24 to 30 months with ASD; (4) 24 to 30 months with non-ASD related atypical development; (5) 31 to 37 months with ASD; and (6) 31 to 37 months with non-ASD related atypical development. Means and standard deviations for the total Part 2 scores, as well as for each subscale, were calculated for each of these groups. Scores within one standard deviation of the group mean were classified as “no/minimal impairments,” scores above one standard deviation and less than or equal to two standard deviations of the group mean were classified as “moderate impairment,” and scores above two standard deviations of the mean were classified as “severe impairment” (Kendall & Grove, 1988; Matson, Fodstad, & Mahan, 2009; Matson, Fodstad et al, 2010; Matson, Fodstad, Mahan et al, 2009; Rojahn et al.,

2009). Calculations used up to two decimal places, while final cutoff scores were rounded down to the nearest whole number (Matson, Fodstad, & Mahan, 2009; Matson, Fodstad et al, 2010; Matson, Fodstad, Mahan et al, 2009; Rojahn et al., 2009).

Results

Prior to the analyses, the data was examined for missing values and outliers. Outliers were detected by calculating group z -scores for Part 2 of the BISCUIT; z -scores with an absolute value greater than 3.29 were identified as outliers (Field, 2005). Participants missing at least 10% of data points or with scores identified as outliers were excluded from the analyses (Donner, 1982; Field, 2005; Matson, Wilkins, Sharp et al., 2009). From the full sample of 3118 participants, 212 (6.80 %) participants with missing data and 39 (1.25%) participants identified as outliers were removed from the analyses in Study 2. Thus the final sample used in Study 2 included 2868 participants.

Ages 17 to 23 months with ASD. First, cutoffs were calculated for toddlers aged 17 to 23 months with an ASD diagnosis. The mean total BISCUIT-Part 2 score for this group was 21.19, with a standard deviation of 16.48. Based on the criteria for creating cutoffs outlined above, total scores between 0 and 37 were classified as “no/minimal impairment,” scores between 38 and 54 were classified as “moderate impairment”, and scores of 55 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCUIT-Part 2 were calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 7.

Table 7. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 17 to 23 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	8.18	8.15	0–16	17–24	≥25
Inattention/impulsivity	7.71	6.09	0–13	14–21	≥22
Avoidance behavior	2.06	3.02	0–5	6–8	≥9
Anxiety/repetitive behavior	1.62	2.59	0–4	5–6	≥7
Eating problems/sleep	1.50	1.93	0–3	4–5	≥6

Ages 17 to 23 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 17 to 23 months with non-ASD related atypical development. The mean total BISCUIT-Part 2 score for this group was 5.63, with a standard deviation of 7.16. As such, total scores between 0 and 12 were classified as “no/minimal impairment,” scores between 13 and 19 were classified as “moderate impairment”, and scores of 20 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCUIT-Part 2 were then calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 8.

Table 8. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 17 to 23 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	2.44	3.76	0–6	7–9	≥10
Inattention/impulsivity	1.89	2.82	0–4	5–7	≥8

(Table 8 continued)

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Avoidance behavior	.39	1.35	0–1	2–3	≥ 4
Anxiety/repetitive behavior	.22	.81	0–1	N/A	≥ 2
Eating problems/sleep	.63	1.32	0–1	1–3	≥ 4

Ages 24 to 30 months with ASD. Cutoffs were next calculated for toddlers aged 24 to 30 months with an ASD diagnosis. The mean total BISCUIT-Part 2 score for this group was 25.26, with a standard deviation of 19.43. Total scores between 0 and 44 were thus classified as “no/minimal impairment,” scores between 45 and 64 were classified as “moderate impairment”, and scores of 65 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCUIT-Part 2 were calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 9.

Table 9. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 24 to 30 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	9.48	8.74	0–18	18–26	≥ 27
Inattention/impulsivity	8.59	6.90	0–15	16–22	≥ 23
Avoidance behavior	2.81	3.64	0–6	7–10	≥ 11
Anxiety/repetitive behavior	2.68	3.52	0–6	7–9	≥ 10
Eating problems/sleep	1.70	2.16	0–3	4–6	≥ 7

Ages 24 to 30 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 24 to 30 months with non-ASD related atypical development. The mean total BISCUIT-Part 2 score for this group was 6.52, with a standard deviation of 8.08. Total scores between 0 and 14 were classified as “no/minimal impairment,” scores between 14 and 22 were classified as “moderate impairment”, and scores of 23 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCUIT-Part 2 were then calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 10.

Table 10. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 24 to 30 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	3.00	4.49	0–7	8–11	≥ 12
Inattention/impulsivity	2.21	3.14	0–5	6–8	≥ 9
Avoidance behavior	.41	1.27	0–1	2–3	≥ 4
Anxiety/repetitive behavior	.25	.79	0–1	N/A	≥ 2
Eating problems/sleep	.56	1.20	0–1	1–2	≥ 3

Ages 31 to 37 months with ASD. Cutoffs were next calculated for toddlers aged 31 to 37 months with an ASD diagnosis. The mean total BISCUIT-Part 2 score for this group was 29.89, with a standard deviation of 20.49. Total scores between 0 and 50 were thus classified as “no/minimal impairment,” scores between 51 and 70 were classified as “moderate impairment”, and scores of 71 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCIUT-Part 2 were calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 11.

Table 11. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 31 to 37 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	11.78	9.31	0–21	22–30	≥ 31
Inattention/impulsivity	10.19	6.90	0–17	18–23	≥ 24
Avoidance behavior	3.11	4.14	0–7	8–11	≥ 12
Anxiety/repetitive behavior	3.13	4.00	0–7	8–11	≥ 12
Eating problems/sleep	1.77	2.15	0–3	4–6	≥ 7

Ages 31 to 37 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 31 to 37 months with non-ASD related atypical development. The mean total BISCUIT-Part 2 score for this group was 5.85, with a standard deviation of 7.26. Total scores between 0 and 13 were classified as “no/minimal impairment,” scores between 14 and 20 were classified as “moderate impairment”, and scores of 21 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCIUT-Part 2 were then calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 12.

Table 12. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 31 to 37 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Tantrum/conduct behavior	2.65	4.04	0–6	6–10	≥ 11
Inattention/impulsivity	1.92	2.70	0–4	5–7	≥ 8
Avoidance behavior	.31	.82	0–1	N/A	≥ 2
Anxiety/repetitive behavior	.33	.94	0–1	1–2	≥ 3
Eating problems/sleep	.55	1.07	0–1	1–2	≥ 3

Discussion

A summary of the total score cutoffs for each group can be found below in Table 13. Like the results of Study 1, these findings show that no single cutoff score is appropriate for children of all ages on the BISCUIT-Part 2. When looking at the total score of those with ASD, one can see that a higher cutoff score was needed as the age cohort increased. A difference in 16 points separated the severe impairment range of the youngest and oldest age cohorts. A similar pattern was shown on nearly every subscale. These results fall in line with the literature showing that, as toddlers with ASD grow older, their symptoms become more pronounced and easier to detect (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey et al., 2009; Mitchell et al., 2006; Werner & Dawson, 2005). These findings suggest that the same is true of symptoms of comorbidity. The typical child with ASD will have more symptoms of comorbidity reported as he or she grows older; therefore, higher cutoffs are needed to indicate significant levels of comorbid symptoms.

The same pattern was not found in children with non-ASD related atypical development. Much less separation was found in the cutoffs indicated for these age cohorts; only three points

separated the highest and lowest severe impairment cutoffs. Additionally, the highest cutoff was indicated for the 24 to 30 month age cohort. Thus, higher cutoffs were not necessarily indicated as the child ages. These findings are not completely unexpected, as children with non-ASD related atypical development would not necessarily be expected to follow the same pattern of increased symptoms as they age. Future researchers may wish to further investigate the emergence of comorbid symptoms in toddlers experiencing non-ASD related delays, perhaps shedding light as to why higher cutoffs are indicated for those in the middle age cohort.

Table 13. Means, standard deviations, and cutoff scores for total scores on the BISCUIT-Part 2 by group

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
17 to 23 months ASD	21.19	16.48	0–37	38–54	≥ 55
17 to 23 months atypical development	5.63	7.16	0–12	13–19	≥ 20
24 to 30 months ASD	25.26	19.43	0–44	45–64	≥ 65
24 to 30 months atypical development	6.52	8.08	0–14	15–22	≥ 23
31 to 37 months ASD	29.89	20.49	0–50	51–70	≥ 71
31 to 37 months atypical development	5.85	7.26	0–13	14–20	≥ 21

Study 3

Research Design

Study 3 employed the exact same research design as Study 2 to establish cutoff scores for Part 3 of the BISCUIT, which assesses for challenging behaviors that are common amongst infants and toddlers with ASD.

Results

Prior to the analyses in Study 3, the data was examined for missing values and outliers. Group z -scores for Part 3 of the BISCUIT were used to detect outliers; z -scores with an absolute value greater than 3.29 were identified as outliers (Field, 2005). Participants missing at least 10% of data points or with scores identified as outliers were excluded from the subsequent analyses (Donner, 1982; Field, 2005; Matson, Wilkins, Sharp et al., 2009). From the full sample of 3118 participants, 48 (1.56 %) participants with missing data and 48 (1.56%) participants identified as outliers were removed from the analyses in Study 3. Thus the final sample used in Study 3 included 3022 participants.

Ages 17 to 23 months with ASD. First, cutoffs for Part 3 of the BISCUIT were calculated for toddlers aged 17 to 23 months with an ASD diagnosis. The mean total BISCUIT-Part 3 score for this group was 5.10, with a standard deviation of 5.53. Based on the criteria for creating cutoffs outlined previously, total scores between 0 and 10 were classified as “no/minimal impairment,” scores between 11 and 16 were classified as “moderate impairment”, and scores of 17 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCUIT-Part 3 were calculated. Using the same methodology as above, cutoffs were then calculated for

each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 14.

Table 14. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 17 to 23 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	3.58	4.52	0–8	8–12	≥ 13
Stereotypies	.99	1.49	0–2	2–3	≥ 4
Self-injurious Behavior	.53	.94	0–1	1–2	≥ 3

Ages 17 to 23 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 17 to 23 months with non-ASD related atypical development. The mean total BISCUIT-Part 3 score for this group was 1.33, with a standard deviation of 2.46. As such, total scores between 0 and 3 were classified as “no/minimal impairment,” scores between 4 and 6 were classified as “moderate impairment”, and scores of 7 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCIUT-Part 2 were then calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 15.

Table 15. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 17 to 23 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	1.07	2.15	0–3	4–5	≥ 6
Stereotypies	.11	.44	0	N/A	≥ 1
Self-injurious Behavior	.15	.47	0	1	≥ 2

Ages 24 to 30 months with ASD. Cutoffs were next calculated for toddlers aged 24 to 30 months with an ASD diagnosis. The mean total BISCUIT-Part 3 score for this group was 6.60, with a standard deviation of 6.77. Total scores between 0 and 13 were thus classified as “no/minimal impairment,” scores between 14 and 20 were classified as “moderate impairment”, and scores of 21 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCUIT-Part 3 were calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 16.

Table 16. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 24 to 30 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	4.74	5.28	0–10	11–15	≥ 16
Stereotypies	1.27	1.72	0–2	2–4	≥ 5
Self-injurious Behavior	.58	1.00	0–1	1–2	≥ 3

Ages 24 to 30 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 24 to 30 months with non-ASD related atypical development. The mean total BISCUIT-Part 3 score for this group was 1.48, with a standard deviation of 2.58. Total scores between 0 and 4 were classified as “no/minimal impairment,” scores between 5 and 6 were classified as “moderate impairment”, and scores of 7 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCUIT-Part 3 were then calculated. Using the same methodology as above, cutoffs were then calculated for

each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 17.

Table 17. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 24 to 30 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	1.25	2.30	0–3	3–5	≥ 6
Stereotypies	.10	.40	0	N/A	≥ 1
Self-injurious Behavior	.12	.40	0	N/A	≥ 1

Ages 31 to 37 months with ASD. Cutoffs were next calculated for toddlers aged 31 to 37 months with an ASD diagnosis. The mean total BISCUIT-Part 3 score for this group was 7.79, with a standard deviation of 6.88. Total scores between 0 and 14 were thus classified as “no/minimal impairment,” scores between 15 and 21 were classified as “moderate impairment”, and scores of 22 and above were classified as “severe impairment.”

Next, the means and standard deviations for each of the five subscales of the BISCUIT-Part 3 were calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 18.

Table 18. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 31 to 37 months with ASD.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	5.81	5.63	0–11	12–17	≥ 18
Stereotypies	1.40	1.71	0–3	3–4	≥ 5
Self-injurious Behavior	.58	.90	0–1	1–2	≥ 3

Ages 31 to 37 months with non-ASD related atypical development. Next, cutoffs were calculated for toddlers aged 31 to 37 months with non-ASD related atypical development. The mean total BISCUIT-Part 3 score for this group was 1.38, with a standard deviation of 2.45. Total scores between 0 and 3 were classified as “no/minimal impairment,” scores between 4 and 6 were classified as “moderate impairment”, and scores of 7 and above were classified as “severe impairment.”

The means and standard deviations for each of the five subscales of the BISCIUT-Part 3 were then calculated. Using the same methodology as above, cutoffs were then calculated for each subscale. A summary of the subscale means, standard deviations, and cutoff scores can be found below in Table 19.

Table 19. Subscale means, standard deviations, and cutoff scores for infants and toddlers aged 31 to 37 months with non-ASD related atypical development.

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Aggressive/Destructive Behavior	1.18	2.16	0–3	4–5	≥6
Stereotypies	.09	.39	0	N/A	≥1
Self-injurious Behavior	.11	.37	0	N/A	≥1

Discussion

A summary of the total cutoff scores for each group can be found below in Table 20. The pattern of results matched those found in Study 2. Among those with ASD, higher total score cutoffs were needed as the age cohort increased. A similar was trend was seen on the subscales, particularly the Aggressive/Destructive Behavior subscale. Less variability was seen on the cutoffs established for the Stereotypies and Self-injurious Behavior subscales, due to low endorsement rates across the age cohorts. The explanation for this trend is likely the same that

has been given for the previous studies: As young children with ASD age, their symptoms, including challenging behaviors, become more marked and distinct from same-aged typically developing peers (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey et al., 2009; Mitchell et al., 2006; Werner & Dawson, 2005).

As in Study 2, the same pattern was not found in children with non-ASD related atypical development. Similar cutoffs were found appropriate across the three age cohorts. This was true of most total score and subscale cutoffs. This is likely due to the overall low endorsement of challenging behaviors in toddlers with non-ASD related atypical development. Therefore, it appears that the use of age-based cutoffs on Part 3 of the BISCUIT is more critical for those with an ASD diagnosis; however, for the sake of consistency, it is recommended that age-based cutoffs be utilized for all administrations of the BISCUIT Part 3.

Table 20. Means, standard deviations, and cutoff scores for total scores on the BISCUIT-Part 3 by group

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
17 to 23 months ASD	5.10	5.53	0–10	11–16	≥ 17
17 to 23 months atypical development	1.33	2.46	0–3	4–6	≥ 7
24 to 30 months ASD	6.60	6.77	0–13	14–20	≥ 21
24 to 30 months atypical development	1.48	2.58	0–4	5–6	≥ 7
31 to 37 months ASD	7.79	6.88	0–14	15–21	≥ 22
31 to 37 months atypical development	1.38	2.45	0–3	4–6	≥ 7

Conclusions

The purpose of the current studies was to examine the utility of age-based scoring procedures on the BISCUIT battery. The BISCUIT is intended for infants and toddlers aged 17 to 37 months, a time in which children are rapidly developing (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Hannemann, 1991). Given the changes that occur due to early development, many psychological measures of young children employ aged-based scoring procedures (e.g., Achenbach & Rescorla, 2000; Kim & Lord, 2012; Lord et al., 1994; Matson, Kozlowski et al., 2011; Reynolds & Kamphaus, 2004; Sparrow et al., 2005). Consideration of age appears especially important in young children with ASD, given that research has shown that, as children with ASD grow older, their deficits and impairments become more pronounced and easier to detect (Charman & Baird, 2002; Desombre et al., 2006; Dixon et al., 2011; Landa & Garrett-Mayer, 2006; McConkey et al., 2009; Mitchell et al., 2006; Werner & Dawson, 2005).

The current studies showed that age-based scoring procedures were appropriate on all parts of the BISCUIT. More specifically, age-based cutoffs increased the overall accuracy of the BISCUIT-Part 1 for each age cohort. The psychometric properties of the measure were improved by using different cutoffs for each age cohort; no single cutoff score was optimal for more than one group. By using the newly developed cutoff scores, researchers and practitioners will be able to more accurately identify and diagnose autism and PDD-NOS in infants and toddlers. This is critical, as accurate identification and diagnosis are needed before appropriate early intervention can take place. While a comprehensive early assessment of ASD should still involve multiple methods from multiple sources, a psychometrically sound diagnostic instrument remains a cornerstone of the assessment process (Chawarska & Bearss, 2008; Matson, Wilkins, Sevin et

al., 2009; Matson, Wilkins, Sharp et al., 2009). Thus any improvements to the psychometric properties of such a measure are critical.

In addition to improving the psychometric properties of the diagnostic portion of the BISCUIT, the current studies have adjusted the cutoffs for the portions examining comorbidity and challenging behaviors. The newly developed cutoffs for Parts 2 and 3 of the BISCUIT will help to more accurately identify those who are experiencing moderate and severe impairments in the areas of comorbidity and challenging behaviors, respectively. In turn, this will help identify specific areas for intervention. Research on EIBI has shown that individuals significantly differ in their response to treatment, and therefore, it is critical that intervention be individually tailored to specific areas of deficit (Anderson & Romanczyk, 1999; Magiati, Charman, & Howlin, 2007; T. Smith, 1999). The newly developed cutoffs for the BISCUIT-Parts 2 and 3 may help in the development of such individualized treatment plans.

The current studies provide a number of areas for future research. While the current studies demonstrated the utility of age-based scoring procedures for all parts of the BISCUIT, future researchers may wish to investigate if other variables, such as race or intellectual functioning, should be considered in the BISCUIT's scoring procedures. For example, the CARS2 has separate scoring procedures for children considered "high-functioning" (Schopler et al., 2010). Inclusion of such variables may further improve the psychometric properties of the measure. Additionally, researchers may wish to adapt the current scoring procedures to be more consistent with the proposed diagnostic revisions of the DSM-V (APA, 2011). The revisions propose to collapse specific ASD diagnoses under one label and to add a level of severity rating. Thus, it may be appropriate to have only one cutoff on the BISCUIT-Part 1 (i.e., one cutoff differentiating atypical development from ASD) and to add a severity rating. Additionally,

researchers may wish to investigate the addition of an observation component to the BISCUIT, as this would provide an additional method and source of diagnostic and clinical information (Chawarska & Bearss, 2008). Such a component would further establish the BISCUIT as a comprehensive battery for assessing ASD in infants and toddlers.

The newly created age-based scoring procedures will help improve the psychometric properties of the BISCUIT. Such improvement is critical, considering the increasing need for early identification of ASD and the potential benefits of early intervention (Corsello, 2005; Eikeseth et al., 2007; Eldevik et al., 2006; Eldevik et al., 2009; Fenske et al., 1985; Harris et al., 1991; Howard et al., 2005; Matson, 2007; Matson & Smith, 2008; Matson, Wilkins, & González, 2008; Peters-Scheffer et al., 2011; Remington et al., 2007; Shea, 2005; T. Smith, 1999; Werner et al., 2000). Further research with the BISCUIT will help continue to ensure that young children with ASD receive the services they need as early as possible.

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