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The Baby and Infant Screen for Children with Autism Traits: A DSM-5 Update

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THE BABY AND INFANT SCREEN FOR CHILDREN WITH AUTISM TRAITS: A DSM-5 UPDATE

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Abstract

Interest surrounding the investigation of autism spectrum disorder (ASD) has increased exponentially since it was initially described over a half-century ago. With this passage of time our conceptualization of the ASD diagnosis has undergone multiple changes. An increasing trend in research has been an emphasis on early identification and intervention. This trend has brought about the creation and adaptation of multiple measures designed to inform early ASD diagnosis. Recently, the ASD diagnostic category underwent significant revisions. In response to revisions, it is necessary to adapt preexisting measures to reflect these significant changes in order to maintain diagnostic accuracy. The *Baby and Infant Screen for Children with aUtIsm Traits* (*BISCUIT*) is a triad of assessments specifically designed to assess infants and toddlers for ASD, comorbid conditions, and challenging behaviors. Initial investigations of each component of the *BISCUIT* have demonstrated that they are reliable and valid in the ASD population. However, the current scoring procedures include individuals diagnosed with both Pervasive Developmental Disorder, Not Otherwise Specified (PDD-NOS) and autistic disorder. The recent restructuring of the ASD category has effectively combined the various ASD diagnoses into a single diagnosis. The current studies identified new cutoff scores that reflect recent changes to the ASD diagnosis and maximized diagnostic sensitivity and specificity for each component of the *BISCUIT* and their individual subscales. Participants were initially separated into two diagnostic groups before being further separated into one of three age cohorts based upon age at time of initial evaluation. This approach allowed for direct comparisons between those with ASD and atypically developing peers while also considering the variable role of development on symptom manifestation. The cutoff scores identified by the current analyses maximized diagnostic sensitivity and specificity. Results of the current analyses are an initial step to demonstrating the utility of the *BISCUIT* for early identification following the revisions implemented by the *Diagnostic and Statistical Manual of Mental Diosrders-5 (DSM-5)*.

Introduction

Autism spectrum disorder (ASD) is often identified as a neurodevelopmental disorder (Barrett et al., 1999; Geschwin & Levitt, 2007; Goldstein, Minshew, Allen, & Seaton, 2002; James et al., 2004; Newschaffer et al., 2007; Samaco, Hogart, & LaSalle, 2005; Shao et al., 2002). Despite consistent agreement about the neurodevelopmental nature of the diagnosis, neuropathological investigations have continuously failed to identify a singular cause or deficit that reliably predicts the presence of ASD (Trevarthen, 2000). Currently, ASD identification and diagnosis is based upon the manifestation of a group of core symptoms which include impairments in communication and socialization, and the presence of restricted interests and/or repetitive behaviors (Bodfish, Symons, Parker, & Lewis, 2000; Fodstad, Matson, Hess, & Neal, 2009; Fombonne et al., 2004; Matson, Dempsey, & Fodstad, 2009; Matson, Mayville, Lott, Bielecki, & Logan, 2003).

There has been a growing interest surrounding the manifestation of ASD in recent years. Unlike some psychological disorders which may be ostracized or garner little attention in the public domain, ASD is a predominant topic amongst researchers, clinical practitioners, and the general public alike (Evans et al., 2001; Fombonne et al., 2004; Gernsbacher, Dawson, & Goldsmith, 2005; Newschaffer & Curran, 2003; Wing & Potter, 2002). Unfortunately, the information being disseminated amongst these groups is not always positive or constructive. For example, Gernsbacher and colleagues (2005) highlighted the negative information displayed to the general public by popular media sources which portrayed ASD as an "epidemic." However, the increased awareness and interest has also given rise to important advancements which include changes in policy, federal funding, and the prevalence and organization of ASD research (Charles, Carpenter, Jenner, & Nicholas, 2008). Collaboration between researchers and practitioners and a growing awareness in the public domain have greatly increased the emphasis on early detection and diagnosis. A dominant factor facilitating this movement has been the observed impact of early interventions (Corsello, 2005; Dawson et al., 2010; Hume, Bellini, & Pratt, 2005; Matson, 2007b; McGee, Morrier, & Daly, 1999; Smith, Groen, & Wynn, 2000). Although the

intensity and modality of early intervention remains a contentious subject of research, the general consensus remains that early intervention in ASD populations is efficacious. However, accurate and reliable assessment and diagnosis is necessary before treatment can be delivered. Professional organizations such as the American Academy of Pediatrics (AAP) and researchers have recommended that children be screened at 18 and 24 months of age for ASD (Johnson & Myers, 2007; Matson & Smith, 2008).

Given the emphasis on early detection and intervention, it is paramount that ASD assessment measures are developed and updated to ensure diagnostic validity and reliability. An appropriate measure should have established psychometric properties utilizing large representative data sets. The *Baby and Infant Screen for Children with aUtism Traits* (*BISCUIT*; Matson, Boisjoli, & Wilkins, 2007; Matson et al., 2009a) was specifically designed to assess infants and toddlers between 17 and 37 months of age. The *BISCUIT* assessment battery consists of a trio of measures which are used for early screening and identification when atypical development is observed in infants (Matson & Tureck, 2012; Matson et al., 2009a). The current study sought to identify new cutoff scores for each of the measures constituting the *BISCUIT* battery based upon the changes to diagnostic criteria that appeared in *Diagnostic and Statistical Muanl of Mential Diosrders-5 (DSM-5)*. The history of ASD, changes in diagnostic criteria, and co-occurring conditions are discussed in addition to the prevalence and etiology of the disorder.

Autism Spectrum Disorder

The origin of the ASD diagnosis as it is thought of today is most often traced back to Kanner (1943). In this seminal text the author provided descriptors of multiple children exhibiting a core group of symptoms which were not readily accounted for by any developmental disorder of the time. Core symptoms included the presence of stereotypic behavior, insistence on sameness, deficits in the formation of appropriate social relationships, and deficits in the acquisition and use of language (Kanner, 1943). Despite variation in symptom severity and presentation, these core symptom features were present in each child.

The word autism has been suggested to be a compound of two Greek words, *autos* which means "self" (Grossman, Carter, & Volkmar, 1997) and *ism* which refers to personal orientation or a state of being (Aitken, Robarts, & Papoudi, 1999). Although this terminology captured the disinterest of individuals with ASD from interacting with others, it did not capture the triad of impairments observed in ASD populations. Kanner (1943) was not the first individual to utilize the term autism in the conceptualization of psychological impairment. In fact, Bleuler (1913) had previously utilized the term to describe secondary symptoms of schizophrenia (detachment from reality). Piaget (1932) also referred to the initial stage of childhood intelligence as autistic thought and the maturation of perceptual relations. Following Kanner's seminal work (1943), research surrounding ASD was controversial due to differences in terminology, diagnostic criteria, and the assessments being developed and used (Rutter, 1972, 1978; Volkmar & Klin, 2005).

Researchers have previously noted that multiple terms and descriptors have been proposed and used intermittently to identify and describe symptoms of ASD such as arrested emotional development (Rank, 1949) and childhood schizophrenia or psychosis (Bender, 1956; Laufer & Gair, 1969; Rutter, 1978). Additional terminology was proposed based upon the hypothesized etiological underpinnings of ASD. For instance, due to the suspected influence of early abnormal cerebral development, Van Krevelen (1971) proposed the term "autismus infantum." Other terminology such as "disintegrative psychosis" was proposed as a means of communicating the observed manifestation of symptoms, specifically the period of regression often reported in ASD populations (Rutter et al., 1969).

Diagnostic confusion was further perpetuated by the diagnostic manuals of the time. Initially, the symptoms of ASD appeared in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; American Psychiatric Association [APA], 1952, 1968) under the diagnosis of childhood schizophrenia. Similarly, the diagnosis of autism was included as a subtype of schizophrenia in the *International Classification of Diseases, 8th Revision* (*ICD-8*; WHO, 1967). It was not until the introduction of the Pervasive Developmental Disorders (PDDs) category in the *Diagnostic and Statistical Manual of Mental*

Disorders - Third edition (*DSM-III*; APA, 1980) that autism and childhood schizophrenia were distinguished in a diagnostic manual (Volkmar, 1998; Volkmar & Klin, 2005). Five diagnoses were included under the PDD category and included atypical PDD, childhood onset PDD, residual childhood onset PDD, infantile autism, and residual infantile autism (Volkmar & Klin, 2005). Criteria for the PDD category incorporated the three core features originally identified by Kanner (1943). However, the proposed diagnostic criteria were also influenced by the work of Rutter (1972, 1978) and Rutter and colleagues (1969) as well as other researchers who identified specific diagnostic criteria for ASD.

In addition to confirming and refining the core symptoms originally identified, Rutter (1978) also proposed that symptom manifestation occurs prior to 30 months of age. However, the specification of an age requirement was one of many problems noted in the debut of the PDDs category. Additional problems included controversy surrounding the classification of autism as a PDD and the diagnostic criteria proposed (Gillberg, 1991a; Volkmar, 1998; Volkmar & Cohen, 1991). For example, to be diagnosed with infantile autism, an individual was required to meet each of the criteria proposed (Volkmar, 1998). Further, researchers argued that the exclusive focus on infant populations excluded older children or higher-functioning individuals (Volkmar, 1998).

The diagnostic category encompassing ASD would undergo multiple changes in subsequent revisions of the *DSM (DSM-III-R, DSM-IV, DSM-IV-TR; APA, 1987, 1994, 2000)* and the *International Classification of Diseases* (*ICD-9*, *ICD-10*; WHO, 1979, 1992) as research expanded and efforts were made to increase diagnostic sensitivity and specificity (Volkmar, Cicchetti, Bregman, & Cohen, 1992). For example, the age requirement appearing in *DSM-III* was subsequently removed in the *DSM-III-R* (Waterhouse, Wing, Spitzer, & Siegel, 1989), only to be reintroduced and increased to 36 months of age in the *DSM-IV* (APA, 1994; Volkmar & Klin, 2005). The release of the *DSM-IV* also included the introduction of specific diagnostic criteria for each ASD diagnosis. Further, the *DSM-IV* also arranged the ASD diagnoses in a hierarchical manner such that a diagnosis for a less severe form of ASD (e.g.,

Asperger's syndrome) was only considered when the individual did not meet criteria for autistic disorder (Mandy, Charman, Gilmour, & Skuse, 2011).

Development of the PDDs category in the *DSM-IV* (APA, 1994) included extensive consideration of the diagnostic criteria for ASD appearing in the *ICD-10* (WHO, 1992) in order to increase agreement and communication amongst researchers and was based upon the data gleaned from field trials (Volkmar, 1998). The *ICD* is a multi-purpose publication with one chapter (Chapter V) dedicated to the classification of mental disorders (Regier, Kuhl, & Kupfer, 2013). Historically the *ICD* and *DSM* have not always been compatible, which impeded international communication and research. An international conference in 1982 led to increased efforts to align these two major classification systems (Jablensky, Sartorius, Hirschfeld, & Pardes, 1983). Multiple revisions are anticipated to appear in the World Health Organization's (WHO) proposed release of the *ICD-11* in 2017 (Regier et al., 2013). The collaboration between the *DSM* and *ICD* working groups is likely to continue as cooperation with members of the *DSM-5* work groups continued during the creation of the *DSM-5* (APA, 2013), and multiple members serve on both *DSM* and *ICD* workgroups (Regier et al., 2013). Criticizing the revisions in the recently released *DSM-5*, Ghaziuddin (2010) suggested that it would be more appropriate to modify the criteria for ASD subtypes instead of completely removing them. This was the initial approach taken by the *ICD* work group in preparation for *ICD-11* (WHO, 2012). However, their beta criteria for ASD are closely aligned with the *DSM-5* criteria at present (WHO, 2014).

Despite active collaboration between the *DSM* and *ICD* work groups, some key discrepancies between the two diagnostic systems have been retained. For instance, the *ICD* includes different criterion requirements and guidelines for the consideration of comorbid diagnoses (Volkmar & Reichow, 2013). One of the most notable differences is the differential approach to formulating diagnostic decisions. Volkmar (1998) noted that the *ICD-10* (WHO, 1992) included both research and clinical guidelines encouraging the use of clinical judgment in case formulation, whereas the *DSM-IV* (APA, 1994) required that the individual meet the minimum symptom requirements specified. Although the *DSM-IV* and *ICD-*

10 remained compatible with previous diagnostic research, they continued to generate controversy amongst researchers. Specifically, the three domain symptom model in the *DSM-IV* (and subsequent *DSM-IV-TR*) and the *ICD-10* provided over 2000 criteria combinations to make an ASD diagnosis (Volkmar & Reichow, 2013). Further, the validity of the *DSM-IV-TR* criteria proposed for Asperger's disorder has previously been questioned (Freeman, Cronin, & Candela, 2002; Mayes & Calhoun, 2003; Tryon, Mayes, Rhodes, & Waldo, 2006). Since its introduction in the *DSM*, researchers have continuously sought to determine how Asperger's disorder could be reliably differentiated from high-functioning autism (HFA; Schopler, 1985; Wing, 1991). Multiple researchers have previously demonstrated that individuals with a previous diagnosis of Asperger's disorder actually met criteria for autistic disorder (Eisenmajer et al., 1996; Howlin, 2003; Szatmari et al., 1995; Tryon et al., 2006). Additional concerns surrounded the Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS) diagnosis. Specifically, concern about the use of vague criteria and no specification of the minimum number of symptoms necessary for a PDD-NOS diagnosis has been expressed (Gibbs, Aldridge, Chandler, Witzlsperger, & Smith, 2012; Witwer & Lecavalier, 2008).

Diagnostic Changes

Although the *ICD-10* (WHO, 1992) remains in use, the *DSM-IV-TR* (APA, 2000) has recently been revised. The release of the *DSM-5* (APA, 2013) included a number of changes to the ASD diagnosis. In direct contention with the *DSM-IV-TR* and *ICD-10*, the *DSM-5* combined the ASD diagnoses (i.e., Asperger's disorder, autistic disorder, PDD-NOS, childhood disintegrative disorder, Rhett's disorder) previously nested within the PDDs category into a single ASD diagnosis (Gibbs et al., 2012; Frazier et al., 2012; Tsai & Ghaziuddin, 2013). The *DSM-5* also made changes to the diagnostic criteria for ASD. As opposed to the trio of core features (i.e., socialization, communication, and restricted and repetitive behaviors and interests [RRBIs]), the *DSM-5* included only two symptom domains (i.e., social communication and interaction, and RRBIs). Factor analytic research was influential in the decision to combine the communication and socialization domains (Boomsma et al., 2008; Gotham, Risi,

Pickles, & Lord, 2007; Lord & Jones, 2012). Despite the controversy surrounding the transition from a triad of core symptoms to a dyad, multiple researchers have demonstrated the construct validity of a two domain ASD model (Boomsma et al., 2008; Georgiades et al., 2007; Lang et al., 2006; Mandy et al., 2012).

In order to meet criteria for an ASD diagnosis in the *DSM-5*, an individual must exhibit deficits for all three symptoms in the social domain, and two of four symptoms within the RRBIs domain. Volkmar and Reichow (2013) noted that the *DSM-5* contains fewer criteria overall, and in the instance of the social/communication domain requires the individual to meet each criteria for a diagnosis. Further, the criteria provided by the *DSM-5* include specific examples of symptoms for each criterion. The inclusion of specific examples among the symptoms appearing in *DSM-5* has historical significance. A significant controversy appearing in the literature during the development of the *DSM-IV* was the removal of the symptom examples that had appeared in the *DSM-III-R*. Volkmar (1998) noted that the inclusion of specific examples decreased the attention given to a symptom construct. The inclusion of specific symptoms in diagnostic criteria was linked to a dimensional approach to diagnosis which also included a rating of the observed symptom severity. This approach is also noted to closely align with the use of structured diagnostic interviews or assessments. Although such measures often demonstrate strong reliability, their relation to a categorical diagnostic approach is indirect (Volkmar, 1998). In line with the previously noted dimensional approach, revisions in the *DSM-5* ASD category also included the specification of the observed degree of impairment (Grzadzinski, Huerta, & Lord, 2013). In addition to a diagnosis, clinicians are required to specify the degree of impairment observed within each symptom domain (i.e., social/communication and RRBIs). Ratings are arranged on a three-point scale (i.e., "*Requiring very substantial support*", "*Requiring substantial support*", and "*Requiring support*") and are also associated with specific examples and individual descriptors (APA, 2013).

Revisions in the *DSM-5* were reportedly made to increase the diagnostic specificity of the ASD diagnosis and its stability across time (APA, 2011, 2013; Gibbs et al., 2012; Grzadzinski et al., 2013).

Multiple researchers have demonstrated an increase in diagnostic specificity based upon *DSM-5* changes to diagnostic criteria (Frazier et al., 2012, McPartland, Reichow, & Volkmar, 2012). However, the same researchers have also reported decreased sensitivity (81% vs. 95%), especially in higher functioning individuals (Frazier et al., 2012; McPartland et al., 2012). Utilizing data from the *DSM-IV* field trials to investigate *DSM-5* criteria, McPartland and colleagues (2012) demonstrated that 40% of children who met *DSM-IV* criteria did not meet *DSM-5* ASD criteria. Investigation of the effects of changes in the *DSM-5* for specific ASD diagnoses (e.g., Asperger's syndrome, PDD-NOS) has suggested that sensitivity and specificity vary dependent upon the group observed (Mattila et al., 2011; Mayes, Black, & Tierney, 2013; McPartland et al., 2012). For example, diagnostic sensitivity for those with autism was observed to be high (98%); however, the same criteria failed to identify 73% of individuals with a previous diagnosis of PDD-NOS (Mayes et al., 2013). Multiple researchers have suggested that a change in symptom requirements would increase sensitivity with minimal effects on specificity (Matson, Hattier, & Williams, 2012; Mayes et al., 2013). The changes proposed by researchers most often consisted of requiring one less symptom from the social communication and social interaction domain (Frazier et al., 2012; Matson et al., 2012). Frazier and colleagues (2012) observed that altering the minimum criteria requirements in the *DSM-5* did not significantly decrease specificity (95% vs. 97%) but increased diagnostic sensitivity (93% vs. 81%).

The changes in the *DSM-5* have caused researchers to express concern about the potential ramifications for individuals and their families (Gibbs et al., 2012; Matson et al., 2012; Mazefsky, McPartland, Gastgeb, & Minshew, 2013; Taheri & Perry, 2012; Wilson et al., 2013; Worley & Matson, 2012). McPartland and colleagues (2012) noted that although the changes did increase diagnostic specificity, they also have negative effects for individuals and their families, researchers, and clinicians. Specifically, these changes may reduce the availability of treatment to individuals who do not meet *DSM-5* criteria. Researchers have reported that as much as 40% of individuals with a previous diagnosis of ASD may not meet *DSM-5* criteria despite presenting with significantly greater numbers of ASD

symptoms than atypically developing peers (Gibbs et al., 2012; Matson et al., 2012; Mattila et al., 2011; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012). Gibbs and colleagues (2012) noted that those children who were diagnosed as ASD under *DSM-IV-TR* criteria most often did not meet *DSM-5* criteria due to a failure to meet the proposed criteria within the RRBIs domain. The authors noted that although the children observed presented with multiple RRBIs, these behaviors were often captured by a single *DSM-5* criterion.

In direct contention with the above research, the American Psychiatric Association (as cited in Gibbs et al., 2012) produced a press release indicating that their own field trials had not resulted in the under-identification of individuals with ASD. The impacts of diagnostic changes are not limited to a single age group and have been observed to affect adults, children, and infants. Further, the changes that have been implemented decreased the applicability of previous research, on-going longitudinal research, and measures developed based upon that research (McPartland et al., 2012).

Current Criteria/Core Features

The recognition of three core symptoms in ASD populations can be traced back to Kanner's early work (1943) describing what would become the core features of the ASD diagnosis. Sigman, Dijamco, Gratier, and Rozga (2004) defined a core deficit as a behavior that distinguishes children with ASD from atypically and typically developing peers. These core symptoms include RRBIs, and deficits in the development and use of social and communication skills (Matson, Dempsey, LoVullo, & Wilkins, 2008). Until the release of the *DSM-5* (APA, 2013), these core symptoms were considered separate domains used in determining diagnosis. The following discussion of core ASD symptoms will be organized to reflect the recent changes to the ASD diagnostic structure appearing in the *DSM-5*.

Social communication and interaction deficits are considered hallmark symptoms of ASD (Bellini, Peters, Benner, & Hopf, 2007; Constantino et al., 2004; Ozonoff & Miller, 1995). Social communication skills include a variety of verbal and non-verbal behaviors utilized in social interactions (Wetherby, Watt, Morgan, & Shumway, 2007). Deficits commonly observed in ASD populations include

a decreased sharing of enjoyment, impaired joint attention skills, use of gestures/pointing, social relationship maintenance, information synthesis, emotion regulation, reciprocity, imitation, preferring to play alone/withdrawal, and perspective taking (Bellini et al., 2007; Cox et al., 1999; Kanner, 1943; Kanner & Eisenberg, 1957; Wing, Gould, & Gillberg, 2011). Deficits in social interactions often also include impairments in social smiling, facial expression, eye contact, social communication, and expressing and receiving affection (Wing et al., 2011). Rutter (1978) noted that the evaluation of eye contact should emphasize the manner in which eye contact is used (e.g., to communicate), not the overall amount.

In ASD populations, deficits in social communication abilities are one of the earliest indicators of ASD (Wetherby et al., 2007). Delay, regression, or the complete absence of language abilities are often reported as a source of initial concern by parents of children with ASD (DeGiacomo & Fombonne, 1998; Howlin & Moore, 1997; Kozlowski, Matson, Horovitz, Worley, & Neal, 2011; Mitchell et al., 2006; Wetherby et al., 2004). Language development is uniquely intertwined with an individual's ability to communicate and socialize. Researchers have consistently demonstrated a strong relationship between an individual's social skills (e.g., joint attention) and future language acquisition (Carpenter, Pennington, & Rogers, 2002; Dawson et al., 2004; Mundy, Sigman, Ungerer, & Sherman, 1987; Stone, Ousley, & Littleford, 1997a).

The expression of communication abilities is noted to be variable in ASD populations (Bartak, Rutter, & Cox, 1975; Kjelgaard & Tager-Flusberg 2001; Luyster, Kadlec, Carter, & Tager-Flusberg, 2008). Individuals may be completely non-verbal and exhibit deficits in non-verbal communication, while others may possess varying degrees of both verbal and non-verbal abilities. Researchers estimate that between 25-50% of individuals with ASD may not develop functional verbal language capabilities (Lord et al., 2004; Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978; Sigman, 1998; Sigman & McGovern, 2005). Even when individuals exhibit verbal communication skills, their use of language may not be functional and include idiosyncratic speech, pronoun reversal, word repetition, and/or echolalia

(Folstein, 1999; Noens & Van Berckelaer-Onnes, 2005; Rutter, 1978; Tager-Flusberg et al., 2009). Echolalia or stereotyped language is identified as the repetitive use of scripts out of context and in a nonfunctional manner (Kanner, 1946; Prizant, 1981). Regardless of communicative ability, individuals with ASD often exhibit deficits in the pragmatic use of communication (Cromer, 1981; Luyster, Kadlec, Carter, & Tager-Flusberg, 2008; Tager-Flusberg, 1985). Pragmatic communication has previously been described as the use of verbal and non-verbal communication that is appropriate for a given social situation (Bates, 1976). Although language use is not always without meaning and may be used to make demands, it is rarely used to socialize (Folstein, 1999).

The effects of deficits in social skills are cumulative and can impair multiple domains (Bellini et al., 2007; Weiss & Harris, 2001). Further, regardless of primary diagnosis, deficits in the use of social skills are significant given their association with prognosis and quality of life (Kapp-Simon, McGuire, Long, & Simon, 2005; Lord & Ventner, 1992; Matson & Swiezy, 1994; Parker & Asher, 1987; Pilling et al., 2002). Poor social skill development is associated with peer rejection and low academic achievement, as well as increased risk for psychopathology (e.g., anxiety, depression, and substance abuse; Bellini, 2006; La Greca & Lopez, 1998; Tantam, 2000; Welsh, Park, Widaman, & O'Neil, 2001). Although social skills deficits do not preclude children with ASD from forming meaningful relationships with others, they do temper their quality (Naber et al., 2007; Travis & Sigman, 1998). Furthermore, these impaired relationships are not self-correcting and may persist across time without appropriate interventions (Matson & Horovitz, 2010; Travis & Sigman, 1998). In ASD populations, Dominick, Davis, Lainhart, Tager-Flusberg and Folstein (2007) found a positive relationship between the presence of deficits in social communication skills and atypical and challenging behaviors (e.g., self-injurious behavior [SIB], temper tantrums, sleep patterns).

Despite the prevalence of these deficits and the negative outcomes associated with their presence, few individuals receive adequate social skills interventions (Hume, Bellini, & Pratt, 2005). Matson and Wilkins (2007) noted that a critical problem in the treatment of social skills deficits is the failure to

consider the role of thorough assessment prior to intervention. Treatment should be informed by the identification of individual strengths and weaknesses. Conceptualization of specific social skills as behavioral cusps may help to inform the development of adequate social skills intervention programs (Bosch & Fuqua, 2001). Identifying specific behavioral targets ahead of time that are feasible and socially relevant and exposing the child to additional social interactions may help to streamline interventions and increase treatment efficacy (Matson & Wilkins, 2007).

A variety of terms are often used interchangeably to describe the broad behavioral domain of RRBIs (e.g., stereotypic behavior, ritualistic behavior, posturing, and self-stimulatory behavior; Bodfish, Symons, Parker, & Lewis, 2000). Stereotypic behavior is broadly defined as repetitive movements that are not associated with an immediate function or purpose (Baumeister & Forehand, 1973; Berkson & Davenport, 1962; Matson, Kiely, & Bamburg, 1997). RRBIs have also been described as a broad class of repetitive behaviors or interests that are inappropriate, rigid, and invariable (Turner, 1999). Rutter (1978) noted that although RRBIs may persist across an individual's lifespan, the features and function of the behavior may differ across time. The manifestation of repetitive behaviors is not unique to ASD populations and may be seen in other disorders such as intellectual disability (ID), obsessive-compulsive disorder, Tourette syndrome, and schizophrenia (Bodfish et al., 2000). RRBIs have also been observed in typically developing infants but often decrease after 12 months of age (Evans et al., 1997; Thelen, 1979). Despite their occurrence in other populations, RRBIs are significantly more prevalent in ASD populations after 24 months of age when compared to typically developing and atypically developing peers (Lord, 1995; Matson et al., 2009).

A variety of RRBIs have been described in ASD populations and include stereotyped or repetitive motor movements (e.g., rocking, full body movements, and hand and/or finger mannerisms; Dawson, Osterling, Meltzoff, & Kuhl, 2000; Klin et al., 2004; Osterling, Dawson, & Munson, 2002; Szatmari et al., 2006) and stereotypical object use, speech, and compulsions (e.g., verbal and nonverbal compulsions or rituals; Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2005). Inappropriate toy play is also prevalent and

may include repetitively lining up or organizing toy items (Bryson et al., 2007; Paterson & Arco, 2007). Individuals with ASD are also noted to utilize idiosyncratic language at significantly greater rates than typically developing peers (Adams, Green, Gilchrist, & Cox, 2002; Volden & Lord, 1991; Volden, Mulcachy, & Holdgrafer, 1997). Idiosyncratic speech is described as the use of verbal speech which is inappropriate or unrelated to the context in which it is used (Hale & Tager-Flusberg, 2005).

In addition to other RRBI symptoms, individuals with ASD may also display narrow or restricted interests (Gabriels et al., 2005) or carry out nonfunctional activities that impair daily functioning. Although some behavioral patterns may have initially served a basic functional purpose, the manner in which they are carried out may become non-functional and idiosyncratic. Insistence on sameness and ritualized patterns of behavior encompass a broad range of behaviors such as marked resistance to environmental change (Rutter, 1978) but may also include rigid and narrow patterns of thought and/or play behavior (DeLong, 1999; Rutter, 1978). Individuals with ASD may exhibit marked resistance to changes in routine and/or the environment which may result in severe behavioral outbursts (Cuccaro et al., 2003; Dettmer, Simpson, Myles, & Ganz, 2000; Schreibman, Whalen, & Stahmer, 2000; Shao et al., 2003; Szatmari et al., 2006). McPartland, Reichow, and Volkmar (2012) noted that fixated interests or activities may be abnormal in intensity and/or area of focus. The intensity of the individual's restricted interests often impede the development of interpersonal relations because they are not shared by others or are not socially relevant (Mercier, Mottron, & Belleville, 2000). Researchers have noted that the manifestation of restricted interests may be particularly difficult to observe in infants and toddlers (Wing et al., 2011).

Unusual sensory responses, interests, and/or sensorimotor abnormalities are commonly observed in ASD populations (Dawson et al., 2000; Gabriels et al., 2005; Klin et al., 2004; Osterling et al., 2002; Williams, 1999). Sensory abnormalities are not limited to interests and activities and may also include abnormal pain responses. Atypical response to pain is a phenomenon commonly noted in ASD populations (Billstedt, Gillberg, & Gillberg, 2007; Cesaroni & Garber, 1991; Leekam, Nieto, Libby,

Wing, & Gould, 2007; Williams, 1999). However, research of this topic has been noted to be methodologically limited due to its current reliance on parent-report and the dearth of standardized assessment measures (Nader, Oberlander, Chambers, & Craig, 2004). Nader and colleagues (2004) reported that children they observed did not differ from typically developing peers with regard to pain experience. Further, researchers have made the distinction between pain sensitivity and pain reactivity (Tordjman et al., 2009). Tordjman and colleagues (2009) reported that although individuals did not exhibit directly observable symptoms of pain they did demonstrate robust physiologic pain responses.

Sensory abnormalities are reportedly present in as much as 90% of the ASD population (Baranek, David, Poe, Stone, & Watson, 2006; Leekam et al., 2007; Ornitz, Guthrie, & Farley, 1978; Volkmar, Cohen, & Paul, 1986). Sensory abnormalities may include impairments in auditory (Bettison, 1994; Dahlgren & Gillberg, 1989; Rosenhall et al., 1999), visual (Baranek, Foster, & Berkson, 1997a; Frankel, Freeman, Ritvo, Chicamin, & Carr, 1976; Hermelin & O'Connor, 1970), and tactile domains (Baranek, Foster, & Berkson, 1997b; Blakemore et al., 2006; Kern et al., 2001; O'Riordan & Passetti, 2006; Wilbarger & Wilbarger, 1991). Auditory deficits may be so severe that the child may be initially considered deaf (Wing, 1966). Deficits may include hyper- or hypo-sensitivity to sounds (Berard, 1993; Bettison, 1994) and abnormal sound processing (Condon, 1975; Courchesne, Akshoomof, & Townsend, 1990). Individuals with ASD may visually fixate on object movement or be hyper- or hypo-sensitive to light (Baranek et al., 1997b; Kern et al., 2001). Leekam and colleagues (2007) observed that sensory abnormalities of smell/taste (e.g., strong preference for certain smells or tastes, craving certain foods, unusual exploration of objects with taste and smell) often distinguished ASD and non-ASD populations. Some researchers have demonstrated that tactile perception does not significantly differ between ASD and typically developing populations (Cascio et al., 2008; O'Riordan & Passetti, 2006).

Although atypically developing children have been observed to exhibit some sensory impairment, it often only occurs in a single sensory domain (Freeman et al., 1981; Leekam et al., 2007; Rogers, Hepburn, & Wehner, 2003; Wing, 1969). In comparison, children with ASD are likely to exhibit multiple

sensory abnormalities across domains (Kientz & Dunn, 1996; Leekam et al., 2007). Regardless of the impacted domain, these sensory abnormalities are noted to be pervasive and impairing across the lifespan (Leekam et al., 2007). Factors such as age and level of intellectual functioning have been shown to mediate the expression of sensory abnormities. Minimal research has been conducted to explore the differences in sensory abnormalities between ASD populations and those with ID only. However, an investigation by Freeman and colleagues (1981) indicated that minimal differences in sensory abnormalities were present between these groups.

RRBIs have immediate and longitunidal negative effects for both the individual and their caretakers. These behaviors are negatively correlated with social development and learning (Koegel, Firestone, Kramme, & Dunlap, 1974; Lovaas, Koegel, Simmons, & Long, 1973). Researchers have also demonstrated that RRBIs impair daily functioning by consuming an individual's time and impeding participation in activities and instruction (Gordon, 2000; Koegel et al., 1974; Lam & Aman, 2007; Varni, Lovaas, Koegel, & Everett, 1979). Sensitivity to sensory input may also negatively impact social interactions (Leekam et al., 2007). Further, if particular behaviors are disrupted or if an individual is prevented from performing a RRBI, he/she may become agitated, noncompliant, and/or anxious (Gordon, 2000).

Comorbid Symptoms

Matson and Nebel-Schwalm (2007a) described comorbidity as the presence of two or more disorders at the same time in the same individual. Researchers investigating psychological conditions in children have repeatedly demonstrated elevated rates of comorbidity when children are diagnosed with attention-deficit/hyperactivity disorder (ADHD; Caron & Rutter, 1991; Kadesjö & Gillberg, 2001), depression (Angold, Costello, & Erkanli, 1999), anxiety (Caron & Rutter, 1991), mania (Lainhart & Folstein, 1994; Matson et al., 1996), and eating disorders (Lewinsohn, Striegel-Moore, & Seeley, 2000). However, similar research in ASD populations has largely been ignored until recently. Aside from those symptoms directly associated with ASD, children with ASD often present with additional impairments

(e.g., activity, attention, and emotion; Lainhart, 1999; Leyfer et al., 2006). Estimates suggest that 65-94% of children with ASD meet criteria for at least one comorbid psychological disorder (Ghaziuddin, Weidmer-Mikhail, & Ghaziuddin, 1998; Mukaddes & Fateh, 2010; Simonoff et al., 2008). In some instances, an individual's symptom expression may be better explained by more than one comorbid disorder. While investigating comorbid conditions, Simonoff and colleagues (2008) reported that nearly half (41%) of their ASD sample met diagnostic criteria for two or more comorbid conditions.

Additional deficits commonly observed in ASD populations include epilepsy (Gillberg, 1991b), anxiety (Davis III et al., 2011), ADHD (Caron & Rutter, 1991), ID (Battaglia & Carey, 2006), visual impairments (Ek, Fernell, Jacobson, & Gillberg, 1998), tics (Canitano & Vivanti, 2007), audiological deficits (Rosenhall et al., 1999), and speech/language disorders (Billstedt, 2000). In addition to psychological impairment, comorbid medical symptoms are also commonly observed in ASD populations (e.g., feeding and sleep difficulties, immune system dysregulation, and seizure activity; Polimeni, Richdale, & Francis, 2005; Tuchman & Rapin, 2002; Warren et al., 1996).

Some researchers have expressed that the consideration of comorbid conditions has negatively impacted our understanding of ASD, yet others have countered that it is necessary and relevant to diagnosis and intervention implementation (Kazdin, 1993; Matson & Nebel-Schwalem, 2007a; Muris et al., 1998; Simonoff et al., 2008). Disagreement about the presence of comorbid conditions is not unique to ASD populations as it has been observed in other disorders (e.g., ID; Matson & Barrett, 1982). The degree of difficulty surrounding differential diagnosis is variable and dependent upon the impairments observed (Matson & Nebel-Schwalm, 2007a; Paclawskyj, Matson, Bamburg, & Baglio, 1997). For instance, symptoms of social anxiety are harder to distinguish from ASD than symptoms of depression.

Regardless of primary diagnosis, the presence of comorbid conditions negatively impacts an individual and their family (Gold, 1993; Leyfer et al., 2006). In addition to core symptoms, the manifestation of challenging behaviors may be related to the manifestation of comorbid conditions (Newschaffer et al., 2007). Comorbid conditions are also associated with significant increases in

impairment, the need for additional interventions, and decreased quality of life (Leyfer et al., 2006; Simonoff et al., 2008). Given the observed benefit of individualized interventions, the identification of comorbid conditions has been suggested to aid case formulation and the identification of additional treatment goals (Leyfer et al., 2006; Simonoff et al., 2008). Measures of comorbid psychopathology may also be used to monitor treatment effects (Matson & Nebel-Schwalm, 2007a).

Intellectual disability. Intellectual functioning has previously been identified as a moderator of ASD symptom expression (Matson et al., 2008). Specifically, the authors observed that a lower IQ was associated with increased severity of ASD symptomology in ID populations. The prevalence of ID in ASD populations is high, with estimates ranging from 40% to 75% (Battaglia & Carey, 2006; Betancur, 2011; Chakrabarti & Fombonne, 2001; Yeargin-Allsopp et al., 2003). The comorbid occurrence of ID in ASD populations is estimated to be 70%, while only 40% of those with ID have a comorbid diagnosis of ASD (La Malfa et al., 2004). However, the co-occurrence of these two conditions is variable and influenced by multiple factors (e.g., diagnostic criteria, population sampled, and ASD diagnoses included; Matson & Shoemaker, 2009). For instance, other researchers reported that only 28% of their sample with an ID diagnosis met criteria for ASD (Bryson, Bradley, Thompson, & Wainwright, 2008). Given the importance of behavior and compliance during the assessment of intellectual functioning, Rapin (2003) cautioned against the strict interpretation of results of intellectual functioning for toddlers. Further, given the limited predictive validity of intellectual functioning assessments in infants and toddlers, a comorbid diagnosis of ID is often deferred until a later age and may be overlooked once an initial ASD diagnosis is given (Charman et al., 2005).

When compared to either condition in isolation, researchers note that the co-occurrence of ID and ASD is associated with significant increases in the severity of symptoms observed (Matson & Shoemaker, 2009). Increases in the severity of ID have also been suggested as a risk factor for ASD (Vig & Jedrysek, 1999). Researchers have suggested that the presence of ID in ASD populations may also negatively impact communication skill aquisition, repetitive behaviors, tantrum behavior, aggression, and SIB

(Allen, 2008; Bartak & Rutter, 1976; Billstedt et al., 2005; Deb & Prasad, 1994; La Malfa et al., 2004; Totsika et al., 2008). These impairments are associated with the provision of care (Van Bourgondien & Schopler, 1990) and negatively impact an individual's quality of life (Allen, 2008; Garcia-Villamisar & Dattilo, 2010). Specifically, the presence of ASD and comorbid ID has been noted to negatively affect integration into traditional residential treatment facilities (Van Bourgondien & Elgar, 1990).

Deficits in intellectual functioning may also negatively affect the consideration of additional comorbid conditions in ASD populations (Long, Wood, & Holmes, 2000). The direct effect of comorbid ASD and ID on the manifestation of additional symptoms of psychopathology is a source of disagreement amongst researchers (LoVullo & Matson, 2009). Researchers have previously failed to find a difference in the manifestation of comorbid symptoms in those with ID and ASD and those with ID only (Tsakanikos, Bouras, Sturmey, & Holt, 2006). Other researchers report elevated rates of comorbid symptoms when ASD co-occurs with ID (Bradley, Summers, Wood, & Bryson, 2004).

Depression. Mood disorders such as depression and bipolar disorder have been observed to frequently co-occur with ASD (Ghaziuddin, Ghaziuddin, & Greden, 2002; Mukaddes & Fateh 2010; Sterling, Dawson, Estes, & Greenson, 2008). However, our current assessment and identification practices may result in an underestimation of their prevalence. Similar to ID populations, the manifestation and expression of depression symptoms may be moderated by the severity of ASD (Ghaziuddin et al., 2002; Matson, Barrett, & Helsel, 1988). For instance, differential rates of depression have been observed in individuals with autism (2%; Ghaziuddin, Tsai, & Ghaziuddin, 1992) when compared to those with Asperger's syndrome (30%; Ghaziuddin et al., 1998; Wing, 1981). Yet this difference may have largely been influenced by participant age as researchers have noted that symptoms of depression increase as children with ASD age (Brereton, Tonge, & Einfeld, 2006).

The early identification and intervention of mood disorder symptoms is an important focus given the negative long-term impact of mood disorders (Cui & Vaillant, 1997). Symptoms of depression may also exacerbate aggressive behaviors, non-compliance, SIB, and withdrawal in children with ASD

(Clarke, Baxter, Perry, & Prasher, 1999; Matson & Nebel-Schwalm, 2007a; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). An advanced understanding of symptom manifestation is currently inhibited by the relative dearth of measures available for the assessment of depression symptoms in ASD populations (Ghaziuddin et al., 2002; Stewart et al., 2006). A majority of the researchers investigating symptoms of depression in ASD populations have relied upon informant report. However, researchers have criticized this approach given the lack of insight into emotions and deficits in communicative abilities (Stewart et al., 2006). Stewart and colleagues (2006) also noted the negative effects of symptom overlap when diagnosing depression in ASD populations. Specifically, the authors suggested that symptoms of depression such as sleep disturbance, decreased appetite, and social withdrawal are commonly observed in those with ASD. Mukaddes and Fateh (2010) also suggested that the underlying causes of depression may vary dependent upon ASD severity, further complicating identification and intervention. Moving forward it will be important to develop assessments appropriate for assessing depression in ASD. Researchers have previously demonstrated the efficacy of behavioral and pharmacological interventions in treating symptoms of depression in those with ASD (Perry, Marston, Hinder, Munden, & Roy, 2001; Sterling et al., 2008).

Attention-deficit/hyperactivity disorder. Research on ADHD in ASD populations has previously been limited due to specifications in diagnostic manuals which prohibited the diagnosis of ADHD in individuals with ASD (Billstedt, 2000). Neuropsychological researchers have reported that individuals with ASD exhibit attentional deficit symptoms similar to those with ADHD (Ehlers et al., 1997; Nydén, Gillberg, Hjelmquist, & Heiman, 1999). Based upon the existing research, the estimated prevalence of ADHD in children with ASD ranges from 31%-70% (Leyfer et al., 2006; Mukaddes & Fateh, 2010; Tani et al., 2005).

The presence of attentional deficits in ASD populations is associated with direct and indirect negative effects. Researchers have previously observed increased rates of challenging behaviors in individuals diagnosed with comorbid ASD and ADHD (Jang et al., 2013; Tureck, Matson, May, &

Turygin, 2013). Konst, Matson, and Turygin (2013) found significantly greater rates of tantrum behaviors in those with comorbid ASD and ADHD when compared to peers with ASD or ADHD only. Researchers have previously observed an increase in the rate of comorbid diagnoses when ASD and ADHD co-occur. Specifically, Simonoff and colleagues (2008) reported that 84% of children with comorbid ASD and ADHD met criteria for at least one additional psychological disorder. Awareness of attentional deficits is crucial given the negative impact these symptoms may have on daily functioning, treatment implementation, and educational attainment (Mukaddes & Fateh, 2010).

Anxiety. Symptoms of anxiety may interfere with a child's development of adaptive skills and increase levels of emotional distress (Muris et al., 1998). Although anxiety symptoms may not be the primary target for itnervention, they are still noted to impact daily functioning and quality of life (Muris et al., 1998). A broad range of anxiety symptoms including separation anxiety, social anxiety, and panic symptoms have been observed in ASD populations (Bellini, 2004; Gillott, Furniss, & Walter, 2001; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000). The effects of anxiety are widespread and have been associated with suicidal ideation, substance abuse, depression, and isolation (Tantam, 2000). Davis III and colleagues (2011) found that anxiety symptoms may vary in severity across the lifespan but are nevertheless present in individuals with ASD. Researchers have also demonstrated that the early identification and treatment of anxiety symptoms may decrease their continuation or severity in adulthood (Davis III, Ollendick, & Nebel-Schwalm, 2008).

Rates of comorbid anxiety disorders and ASD widely vary with estimates ranging from 17 to 84% (de Bruin et al., 2007; Leyfer et al., 2006; Mukaddes & Fateh, 2010; Muris et al., 1998; Sukhodolsky et al., 2008). These rates are significantly elevated compared to the rates observed in typically developing peer populations (8.9%; Bernstein & Borchardt, 1991; Costello, 1989; Muris et al., 1998). The observed variance in prevalence rates may be partially attributed to factors such as participant age, ASD symptom severity, IQ, and the measures administered. Additionally, the wide variety of anxiety disorders (e.g., OCD, social anxiety disorder, and specific phobias) may influence prevalence estimates.

Specific phobias are commonly observed in children with ASD (de Bruin et al., 2007; Leyfer et al., 2006; Mukaddes & Fateh, 2010). Reports from research and evidence from individual case studies indicate that individuals with ASD exhibit significant levels of fear and anxiety (Matson & Love, 1990; Rumsey, Rapoport, & Sceery, 1985). In a systematic review of the research literature on anxiety symptoms in ASD populations, van Steensel, Bögels, and Perrin (2011) reported that specific phobia was one of the most prevalent comorbid conditions. Across the 31 manuscripts reviewed, approximately 30% of participants met criteria for a diagnosis of specific phobia. However, Mukaddes and Fateh (2010) have noted that future research is necessary to determine if specific phobias are distinct and different from the sensory abnormalities observed in ASD populations.

Social anxiety symptoms are also commonly observed in children with ASD. However, differential diagnosis is necessary given the socialization deficits associated with ASD. In this regard, Bellini (2006) noted that social anxiety differs from the indifference towards social situations exhibited by those with ASD. Individuals with comorbid ASD and social anxiety may desire social interaction but experience significant worry or fear due to skills deficits. Prevalence estimates of social anxiety are variable and range from 20-57% (Bellini, 2004; Simonoff et al., 2008; White & Roberson-Nay, 2009). Similar to social anxiety, the comorbid diagnosis of OCD has received criticism due to the observed symptom overlap with the core symptoms of ASD (Mukaddes & Fateh, 2010). However, Ghaziuddin (2005) asserted that a diagnosis of OCD may be warranted when new obsessions or compulsions emerge or if preexisting behaviors change in function or intensity. The reported prevalence of OCD in ASD populations is variable with estimates ranging from 2.8% to 37% (de Bruin et al., 2007; Ghaziuddin et al., 1998; Leyfer et al., 2006).

Eating difficulties. Williams, Dalrymple, and Neal (2000) noted that in addition to core symptoms, individuals with ASD often exhibit highly restricted eating habits and idiosyncratic food preferences. The eating difficulties in ASD populations are varied and not restricted to a particular psychological diagnosis (e.g., anorexia). Commonly reported mealtime problems include food refusal,

food selectivity, pica, choking, gagging, and overeating (Billstedt, 2000; Schwarz, 2003). Food selectivity is commonly observed and has previously been described as food refusal and acceptance of a minimal variety of foods (Bandini et al., 2010). Quantitative research has further demonstrated that when compared to typically developing peers, children with ASD eat fewer different types of food and exhibit more inappropriate behavior during mealtimes (Schreck, Williams, & Smith, 2004). These negative mealtime behaviors are often exacerbated by deficits in communication and/or oral-motor functioning and may be maintained by parental reinforcement (Page & Boucher, 1998; Shaw, Garcia, Thorn, Farley, & Flanagan, 2003).

Factors such as environmental context, food presentation method, and the type of food presented may each be functions contributing to the presence of eating difficulties (Ahearn, Castine, Nault, & Green, 2001; Schreck et al., 2004; Schreck & Williams, 2006). Schreck and colleagues (2004) noted that food selectivity did not appear to be a result of a decreased opportunity to consume a variety of foods. Instead, the authors noted that food selectivity was partially a function of the familial diet; however, this factor accounted for very little variance in a subsequent regression analysis. Contrary to anecdotal evidence, researchers have thus far failed to find evidence supporting hypotheses that food selectivity in ASD populations is related to core ASD symptoms (e.g., sensory deficits; Schreck & Williams, 2006).

Schreck and colleagues (2004) noted that children's eating problems result in additional parental stress. This stress is often heightened by the underlying importance of food consumption and the emphasis on growth monitoring during infancy. Despite the known prevalence of eating problems in ASD population, formal assessment is often deferred unless the individual is identified as failing to thrive (Hutchinson, 1999). Given the potential long-term negative impact that poor nutrition may have on growth and development, researchers have expressed significant concern regarding the deferment of formal assessment until an individual is suspected to be malnourished (Schwarz, 2003).

Sleep habits. Research on sleep problems is problematic due to the lack of a formal definition of what constitutes a sleep problem. Richdale and Schreck (2009) noted that researchers and clinicians often

fail to utilize formal diagnostic sources when evaluating sleep behaviors; they found that researchers often formulate their own operational definitions of sleep behaviors and rely upon parent- and self-report. Although operational definitions of sleep disturbance vary, they most often include three common components, the frequency of the behavior (e.g., waking and/or resistance), duration of the behavior, and persistence (Richadale & Schreck, 2009). In response to criticism surrounding the assessment of sleep disturabance, recent sleep studies have confirmed parent report with procedures such as actigraphy and polysomnography (Allik, Larsson, & Smedje, 2006; Elia et al., 2000; Limoges, Mottron, Bolduc, Berthiaume, & Godbout, 2005; Miano et al., 2007; Wiggs & Stores, 2004).

Prior to the age of 6, sleep difficulties are relatively common in both typically and atypically developing populations. Sheldon, Ferber, and Kryger (2005) reported that approximately 50% of typically developing children exhibit sleep problems. Researchers have reported that approximately 80% of children with ASD exhibit sleep difficulties (Allik et al., 2006; Polimeni et al., 2005; Sheldon et al., 2005). Symptoms consistent with insomnia are often reported in ASD populations (Malow et al., 2006). Additional difficulties may include a decrease in total sleep time (Schreck & Mulick, 2000) and night waking (Malow et al., 2006). Whereas sleep problems in typically developing populations may decrease after age 6, sleep problems in children and young adults with ASD may persist into adulthood in the absence of intervention (Paavonen et al., 2008; Tani et al., 2004). The negative effects of sleep problems are pervasive and include deficits in attention, mood regulation, behavior, health, and cognitive development (Keren, Feldman, & Tyano, 2001; Lavigne et al., 1999; Sadeh, Gruber, & Raviv, 2002).

Assessment of comorbid conditions. The assessment and diagnosis of comorbid conditions in those with ASD is often hindered by deficits in cognition (e.g., executive functioning and information processing; Matson, LoVullo, Rivet, & Boisjoli, 2009; Minshew, Goldstein, & Siegel, 1997; Pennington & Ozonoff, 1996; Tager-Flusberg & Sullivan, 1994) and communication (Leyfer et al., 2006; Lord & Paul, 1997; Simonoff et al., 2008). Additionally, Reiss and Szyszko (1983) noted that the attribution of comorbid symptoms to a primary diagnosis of ASD also contributes to the under identification of

comorbid conditions. Despite the complexity of assessing and identifying comorbid impairments, this information is essential for case formulation and intervention (Goldstein, Naglieri, & Ozonoff, 2008). Aside from intellectual and communication abilities, Ghaziuddin, Ghaziuddin, and Greden (2002) suggested that the presence of comorbid conditions is also a prognostic indicator.

Historically, the identification of comorbid conditions was also interfered by a lack of appropriate measures. Leyfer and colleagues (2006) noted that researchers were often forced to utilize measures that were developed and standardized for the general population. Such measures included the *Conners Rating Scale* (Conners, 1973), the *Child Behavior Checklist* (Achenbach & Edelbrock, 1983), and the *Kiddie Schedule for Affective Disorders and Schizophrenia* (*K-SADS*; Puig-Antich & Chambers, 1978). The use of measures without normative ASD data is less than ideal, given the previously noted negative impact of differences in cognition and communication. Measures such as the *Behavior Problems Inventory* (Rojahn, Matson, Lott, Esbensen, & Smalls, 2001) and the *Developmental Behavior Checklist* (Einfeld & Tonge, 1995) that were designed to assess comorbid conditions in individuals with developmental disabilities are more appropriate; however, their use in ASD populations has not been explicitly validated (Leyfer et al., 2006). The shortage of assessments has led some researchers to revise previously existing measures to create new measures that were appropriate for the ASD population (e.g., *Autism Comorbidity Interview-Present and Lifetime* [*ACI-PL*]; Leyfer et al., 2006) and to create new measures designed specifically for the assessment of comorbid conditions in ASD populations (e.g., *Autism Spectrum Disorder-Comorbid for Children* [*ASD-CC*]; Matson & González, 2007).

Challenging Behaviors

The presence of challenging behaviors is a primary source of referral for psychological evaluation and treatment (Mandell, Novak, & Zubritsky, 2005; Mudford and colleagues, 2008). Challenging behaviors has previously been described as behaviors that significantly limit an individual's access to services, and whose frequency and/or intensity place the individual or those around the individual at risk for bodily harm (Emerson, 2001). In addition to placing the individual at risk for serious mental and

physical impairments, challenging behaviors are associated with significant distress for individuals and their families (Mukaddes & Topcu, 2006; Murphy et al., 2005). Challenging behaviors negatively impact quality of life (Mukaddes & Topcu, 2006) and may limit the services available to an individual (Borthwick-Duffy, Eyman, & White, 1987; Hastings & Brown, 2002; Oliver, Murphy, & Corbett, 1987). Moreover, challenging behaviors may limit an individual's participation in leisure activities and social interactions, further impeding the development of social skills (Chadwick, Walker, Bernard, & Taylor, 2000). Current research suggests that over half (64.3%) of individuals with ASD exhibit challenging behaviors (Murphy, Healy, & Leader, 2009). In the absence of adequate intervention, challenging behaviors may persist across the lifespan in ASD populations Mukaddes & Topcu, 2006; Murphy et al., 2005; Nissen & Haveman, 1997; Oliver et al., 1987).

Research of challenging behaviors in ASD populations most often includes the investigation of behaviors such as stereotypy, SIB, aggression, and noncompliance/property destruction (Baghdadli, Pascal, Grisli, & Aussiloux, 2003; Kiernan & Kiernan, 1994; McClintock, Hall, & Oliver, 2003). Over half of individuals exhibit multiple forms of challenging behaviors (Emerson et al., 2001a) such as property destruction, stereotypy, SIB, and aggression (Borthwick-Duffy, 1994).

Multiple factors are hypothesized to impact the expression of challenging behaviors in ASD populations. For instance, researchers have observed a positive relationship between the severity and intensity of challenging behaviors and the severity of ASD symptomology (Matson, Wilkins, & Macken, 2008; O'Brien & Pearson, 2004; Reese et al., 2005). Furthermore, deficits in communication and social skills are also a risk factor for developing challenging behaviors (Borthwick-Duffy, 1996; Koegel, Koegel, & Surratt, 1992). An interaction between the level of cognitive functioning and the presence of challenging behaviors in those with ASD has also been found. Specifically, the prevalence of challenging behaviors increases with decreases in intellectual functioning (Holden & Gitlesen, 2006; O'Brien & Pearson, 2004; Reese et al., 2005). Researchers have also found that cognitive function may have a differential effect on the exhibition of challenging behaviors. Holden and Gitlesen (2006) observed SIB

to be more common as the severity of ID increased, while aggression was observed to increase as cognitive function increased. These findings have important implications for researchers and clinicians, especially following the diagnostic changes appearing in the *DSM-5* (APA, 2013). Changes to the *DSM-5* require consideration and communication of those specific impairments observed, given that the severity of symptoms is not readily communicated with the use of a single broad ASD diagnosis.

Challenging behavior interventions should be based upon functional assessment (Ellingson, Miltenberger, Stricker, Galensky, & Garlinghouse, 2000). Assessment of behavioral function is conducted to identify those factors that reliably predict and/or maintain the targeted behavior (Horner & Carr, 1997). Behavioral procedures have been found to be effective in the management of challenging behaviors (Downs, Downs, & Rau, 2008; Machalicek, O'Reilly, Beretvas, Sigafoos, & Lancioni, 2007; Ringdahl, Call, Mews, Boelter, & Christensen, 2008). Specific behavioral intervention strategies include the use of differential reinforcement procedures (Braithwaite & Richdale; Durand, 1999; Taylor, Hoch, & Weissman, 2005) and alteration of the environment (i.e., altering behavioral antecedents; Kuoch & Mirenda, 2003; Prupas & Reid, 2001; Schmit, Alper, Raschke, & Ryndak, 2000). Additional treatment approaches in ASD populations have also included the use of atypical antipsychotics such as risperidone (McCracken et al., 2002). However, there is limited research currently available to demonstrate the efficacy and safety of pharmacological interventions in children with ASD. Moreover, pharmacological interventions often result in short-term side effects such as drowsiness, drooling, fatigue, dizziness, and increased appetite and long-term side effects such as tardive dyskinesia (McCracken et al., 2002).

Aggression. Use of the term aggression encompasses a wide variety of behaviors including kicking, slapping, pushing/pulling, pinching/scratching, and punching (Harris, 1993). Aggressive behaviors are common in ASD populations (Billstedt, 2000) and occur most often in adolescent and adult populations (Horrigan & Barnhill, 1998). Procedures derived from positive behavior support and applied behavior analyses are often used to treat aggressive behaviors (Carr & Horner, 2007; Carr et al., 2002).

Intervention targets are often derived from functional assessments (Matson et al., 2009; Matson & Nebel-Schwalm, 2007a).

Self-injurious behavior. Matson and Nebel-Schwalm (2007a) noted that SIB is not a symptom considered for the diagnosis of ASD although it is a common comorbid condition. The term "SIB" encompasses a wide variety of behaviors including head hitting, biting, scratching, and punching/slapping (Emerson et al., 2001b). Additional forms of SIB such as head banging, hair pulling, and biting are often prevalent in individuals with comorbid ASD and ID (Gillberg & Coleman, 1992). Baghdadli and colleagues (2003) demonstrated that deficits in expressive and receptive communication skills may moderate SIBs. Estimates of the prevalence of SIBs in ASD populations are variable and dependent upon the populations sampled but range from 20%-71% (Ando & Yoshimura, 1979; Janicki & Jacobson, 1983).

Noncompliance/property destruction. Noncompliance is a prevalent challenging problem observed in individuals with developmental disabilities (Schoen, 1983). Luiselli (2009) defined non-compliant behavior as a failure to perform specific behaviors following a request. Noncompliance may be further identified as either an active (e.g., aggression) or passive behavior (e.g., ignoring; Luiselli, 2009). Regardless of form and function, noncompliance in ASD populations has particularly negative effects. Compliance is directly related to the exhibition of additional challenging behaviors such as SIB, tantrums, and aggression (Cataldo, Ward, Russo, Riordan, & Bennett, 1986; Russo, Cataldo, & Cushing, 1981). Noncompliance further complicates the introduction and shaping of appropriate adaptive behaviors and skills (Luiselli, 2009). Interventions used to increase compliance include procedures such as behavioral momentum (Mace et al., 1988) and time-out (Handen et al., 1992).

Assessment of challenging behaviors. A growing amount of research has focused on the prevalence of challenging behaviors in ASD populations (Carcani-Rathewell, Rabe-Hasketh, & Santosh, 2006). When compared to typically developing peers, adults with ASD are four times as likely to exhibit challenging behaviors (McCarthy et al., 2010). Despite the increased prevalence, the influence of

multiple factors (e.g., age, gender, and ASD severity) on the manifestation of challenging behaviors is not clear (McCarthy et al., 2010). The assessment of challenging behaviors is an important initial step in intervention (Matson & Nebel-Schwalm, 2007b). Initially a broad assessment helps to identify target behaviors for which specific follow-up assessments may be used to identify information relevant to intervention (e.g., maintaining variables and rate). Preliminary research has suggested that in the absence of intervention challenging behaviors are stable and persistent for children and adolescents (Horner, Carr, Strain, Todd, & Reed, 2002; Matson, Mahan, Hess, Fodstad, & Neal, 2010). The presence of challenging behaviors is negatively associated with long-term outcome and is positively associated with an increase in the use of pharmacologic interventions (Advokat, Mayville, & Matson, 2000), an elevated risk for injury (Lee, Harrington, Chang, & Connors, 2008), and poor public perception (Morton & Campbell, 2008).

Matson and Nebel-Schwalm (2007b) suggested that the negative effect of challenging behaviors on development is poorly understood and cautioned against a simplistic approach to challenging behavior assessment given the variety of factors (e.g., settings and reinforcement) that directly influence their maintenance. The assessment of challenging behaviors is necessary when developing effective interventions and provides an objective measure of treatment effects (Ellingson et al., 2000; Matson, & Minshawi, 2007; Matson & Nebel-Schwalm, 2007b). Despite the prevalence of challenging behaviors there are few measures currently available that have adequate normative samples and demonstrate good psychometric properties to assess children with ASD (Matson & Nebel-Schwalm, 2007b).

Measures currently used to assess challenging behaviors in ASD populations include the *PDD Behavior Inventory* (*PPDBI*; Cohen, 2003; Cohen, Schmidt-Lackner, Romanczyk, & Sudhalter, 2003), the *Overt Aggression Scale* (*OAS*; Hellings et al., 2005; Yudofsky et al., 1986), the *ASD-PBC* (Matson & González, 2007), and the *Behavior Problems Inventory* (*BPI*; Rojahn et al., 2001; Sturmey, Fink, & Sevin, 1993; Sturmey, Sevin, & Williams, 1995). The *Disability Assessment Schedule* (*DAS-B*; Holmes, Shah, & Wing, 1983) has also been used to assess challenging behaviors in ASD populations; however, this scale is intended for use in ID populations (McCarthy et al., 2010).

Prevalence

Prevalence has been defined by as the estimated total number of cases of a disease or disorder at a single point in time (Dorland, 1994). The prevalence rate for ASD has continuously increased across time. Previous inconsistencies in the application of ASD diagnoses according to the *DSM-IV-TR* may have been responsible for the variability observed in recent estimates of ASD prevalence (Mahjouri & Lord, 2012). Factors external to ASD and diagnostic variability are also hypothesized to significantly influence estimated prevalence rates: resource allocation, awareness, a growing body of research, changes in research methodology, and changes in diagnostic criteria (Mahjouri & Lord, 2012; Saracino, Noseworthy, Steiman, Reisinger, & Fombonne, 2010). Mahjouri and Lord (2012) suggested that the allocation of state and federal resources for programs that serve individuals with ASD has increased research in this area and efforts to identify individuals with ASD across multiple sites (Palmer, Blanchard, Jean, & Mandell, 2005).

During the 1980's the estimated prevalence rate of ASD was reported to be 5 per 10,000 individuals (Gillberg, Steffenburg, & Schaumann, 1991). More recent estimates of ASD prevalence have ranged from 181/10,000 (Kawamura, Takahashi, & Ishii, 2008) to 70/10,000 (Saracino et al., 2010). Based upon a multi-site study carried out by the Autism and Developmental Disabilities Monitoring Network (AADM), the Centers for Disease Control (CDC) estimated that 11.3 in 1000 children met *DSM-IV-TR* criteria for ASD (Baio, 2012). In the investigation carried out by the AADM, large variability in prevalence estimates was noted across sites despite the utilization of a standardized assessment approach (Baio, 2012). Specifically, estimates ranged from 4.8 per 1000 children to 21.2 per 1000 children.

As noted previously, multiple researchers have estimated that diagnostic changes in the *DSM-5* may significantly alter the prevalence of ASD (Gibbs et al., 2012; Mahjouri & Lord, 2012; Matson et al., 2012a, b; Mattila et al., 2011; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012).

With the recent release of the *DSM-5*, no research is currently available to verify estimates made based upon the criteria proposed prior to the final release of the *DSM-5*.

Etiology

The heterogeneity of ASD remains a point of controversy as evidenced by the recent changes in the *DSM-5* (APA, 2013). This variability may be clearly observed in the different symptom profiles observed in ASD populations. Although some children may exhibit symptoms of ASD from early infancy, estimates suggest that nearly half may only begin exhibiting symptoms following a period of regression in areas such as verbal and nonverbal communication and social skills (Geschwind & Levitt, 2007; Rogers, 2004). Multiple researchers have documented periods of regression in ASD populations. On average this period of regression occurs during the second year of life and may affect both verbal and non-verbal communication skills (e.g., use of words, and pointing or imitation skills; Volkmar & Cohen, 1989; Werner & Dawson, 2005). Further, there is significant variability in the severity of ASD symptoms and in the manifestation of comorbid disorders (e.g., ID, obsessive-compulsive disorder, ADHD; Newschaffer et al., 2007).

As research and awareness of ASD has increased, so have attempts to identify causal factors associated with the disorder. Initial hypotheses regarding the underlying etiology were controversial (Matson & Minshawi, 2006). For example, Kanner (1943) suggested a link between ASD and the personalities of parents who exhibited limited social abilities. Along the same psychodynamic approach, the idea of "refrigerator mothers" being a causal factor of ASD prevailed for a period of time (Bettelheim, 1967). Additional psychoanalytic theories concerning mother-child dyads as the cause for ASD were promoted by both Mahler (1968) and Tustin (1981). Following advancements in both research practices and theoretical orientation, these early theories have been replaced by learning, neurobiologic, and genetic theories (Matson & Minshawi, 2006). However, not all of the emerging theories have been supported by scientific evidence. Although it has since been retracted, research by Wakefield et al. (1998) suggesting that measles-mumps-rubella (MMR) vaccination was associated with ASD generated significant

controversy among researchers, professionals, and parents (DeStefano & Thompson, 2004; Takahashi et al., 2003). Despite the retraction and failures to replicate the original study (Honda, Shimizu, & Rutter, 2005; Takahashi et al., 2003), the negative effects have persisted (D'Souza, Fombonne, & Ward, 2006; Wilson, Mills, Ross, McGowan, & Jadad, 2003). In a survey of parents with children with ASD, nearly 30% of respondents endorsed the belief that an immunization was responsible for their child's disorder (Harrington, Patrick, Edwards, & Brand, 2006). In a survey of parents with infants, Smith, Yarwood and Salisbury (2007) found that 10% of parents surveyed still consider the MMR to be a high risk factor. Brown and colleagues (2010) noted that multiple factors influence parental decisions regarding vaccinations. The authors noted that researchers and practitioners must work to address concerns and be prepared to discuss the appropriate research supporting vaccinations when necessary.

Genetic theories. Epidemiology researchers have previously suggested a strong genetic role in ASD populations (Betancur, 2011; Chudley, 2004; Cook, 2001; Muhle, Trentacoste, & Rapin, 2004). In ASD populations, the estimated variance in phenotype accounted for by the genotype (i.e., heritability) is 90% (Bailey et al., 1995; Schaefer & Mendelsohn, 2008). Further, parents and siblings of individuals with ASD show significantly greater rates of ASD symptoms than control groups (Bishop et al., 2004; Bolton et al., 1994). Additional support for a genetic component is derived from the elevated concordance rates observed in monozygotic twins (70%) when compared to dizygotic twins (3%; Betancur, 2011; Cook, 2001; Lotspeich & Ciaranello, 1993; Muhle et al., 2004; Spence, 2004). Since concordance among monozygotic twins is less than 100%, it is hypothesized that a multi factor inheritance is likely to be the cause (Herbert, Sharp, & Gaudiano, 2002).

As previously indicated there is a wide degree of variability in ASD symptom manifestation. Geschwind and Levitt (2007) suggested that ASD should not be considered a unidimensional syndrome given its heterogeneity. Instead, they hypothesized that a variety of etiologic factors are associated with the manifestation of ASD symptoms (Abrahams & Geschwind, 2008). Chromosomal loci commonly associated with ASD symptom expression are present at: 2q, 7q 22-31, 13q, 15q11-13, 16p, and 17q 11

(Betancur, 2011; Cook et al., 1998; Falk & Casas, 2007; Goizet et al., 2000; Liu et al., 2001; Nakamine et al., 2008; Yonan et al., 2003). The investigation of specific genetic factors has identified chromosomal abnormalities that may increase the risk of developing ASD. For example, a variation of the HOXA1 gene on chromosome 7 has been suggested to double the risk of developing ASD (Rodier, 2000). However, with the exclusion of chromosomes 14 and 20, each of the autosomes and gonosomes has been associated with the expression of ASD symptoms (Gillberg, 1998). Based upon this observation the author concluded that ASD is likely caused by the mutation of several different genes across multiple chromosomes. Although the increased prevalence of ASD among males also suggests a genetic factor, extensive research utilizing whole genome screens of the X chromosome in ASD populations has identified only minor links to date (Schaefer et al., 2008).

Individuals with specific genetic disorders (e.g., fragile X syndrome, neurofibromatosis, tuberous sclerosis, angelman syndrome, and phenylketonuria) are at an increased risk for developing ASD (Blomquist et al., 1985; Cook et al., 1997; Matsuura et al., 1997; Peters, Beaudet, Madduri, & Bacino, 2004; Santangelo & Folstein, 1999; Trottier, Srivastava, & Walker, 1999). However, despite the elevated levels of risk associated with these syndromes, they do not individually account for more than 2% of identified ASD cases (Abrahams & Geschwind, 2008). In a similar manner, the increased prevalence of comorbid ID (40-70% of those with ASD) also hints at underlying genetic factors (Battaglia & Carey, 2006; Betancur, 2011; Chakrabarti & Fombonne, 2001; Chudley et al., 1998; Gillberg, 1998; Mauk, 1993; Sponheim & Skjeldal, 1998; Yeargin-Allsopp et al., 2003). Abrahams and Geschwind (2008) noted that the lack of a single overarching genetic cause is reminiscent of ID which is also associated with multiple contributing factors. Researchers have previously noted that 4-34.1% of those with ID demonstrate chromosomal abnormalities (Xu & Chen, 2003). While discussing genetic factors, Betancur (2011) highlighted the overlap of genes and loci identified in ASD populations that have also been identified in those with ID and/or epilepsy.

The use of clinical geneticists has increased recently as genetic consultation is sought to provide insight into specific factors that may have led to the presence of ASD symptoms (Schaefer et al., 2008). When considering genetic consultation, individuals should consider the heterogeneity of genetic factors, cost, and the variability of existing tests (Schaefer et al., 2008). On average, chromosomal analyses are noted to produce higher diagnostic yields than other genetic analyses in ASD populations (Reddy, 2005; Shevell et al., 2001; Weidmer-Mikhail, Sheldon, & Ghaziuddin, 1998). However, the use of genetic evaluation to identify copy number variations, genetic syndromes, and mutations is only successful in approximately 6-20% of ASD cases (Abdul-Rahman & Hudgins, 2006; Abrahams & Geschwind, 2008; Battaglia & Carey, 2006; Betancur, 2011; Schaefer & Lutz, 2006; Shevell, Majnemer, Rosenbaum, & Abrahamowicz, 2001).

Non-genetic risk factors. In utero experiences have also been linked to an increased risk for ASD (Herbert et al., 2002). Specifically, researchers have previously suggested that exposure to ethanol, thalidomide, rubella infection, and valproic acid during pregnancy may each elevate the risk for developing ASD (Rodier, 2000). Although the casual link has been reported, investigations of each of these factors have only accounted for a minute portion of the overall ASD population (Herbert et al., 2002).

Given the sex differences in ASD populations, Baron-Cohen (2002) hypothesized that ASD represents an extreme variation of the male-typical cognitions and behaviors due to exposure to elevated levels of testosterone during prenatal development (Knickmeyer & Baron-Cohen, 2006). Consistent with this hypothesis, Auyeung and colleagues (2009) found a strong positive association with levels of fetal testosterone and ASD symptom manifestation. However, this line of research is in its infancy, and further studies are needed (Knickmeyer & Baron-Cohen, 2006).

Neurobiological theories. Current neurobiological research may be separated into two broad domain areas, cellular accounts and hypotheses surrounding regional or systematic abnormalities (Abrahams & Geschwind, 2008). Cellular explanations include factors such as atypical brain

connectivity and abnormalities in synaptic functioning (Bear, Huber, & Warren, 2004; Chugani, 2004; Dölen et al., 2007; Geschwind & Levitt, 2007; Krey & Dolmetsch, 2007; Tabuchi et al., 2007; Zoghbi, 2003). A wide variety of regional hypotheses including impaired cerebellum functioning (Hebert et al., 2005), abnormal mirror neuron activity (Dapretto et al., 2006; Ramachandran & Oberman, 2006), epigentic factors and theories (Hogart, Nagarajan, Patzel, Yasui, & LaSalle, 2007; Jiang et al., 2004), and hyperactivation of the immune system have been proposed (Vargas, Nascimbene, Krishnan, Zimmerman, & Pardo, 2005). Research investigating each of the above theories is in its infancy and continues to be conducted (Abrahams & Geschwind, 2008).

Macrocephaly is a condition associated with a head circumference that is 1.88 standard deviations greater than the normative data for an individual's gender and age (Fidler, Bailey, & Smalley, 2000). According to its definition and diagnostic criteria, macrocephaly occurs in only 3% of the world's population (Herbert, 2005). However, nearly 20% of those diagnosed with macrocephaly are also diagnosed with ASD (Aylward, Minshew, Field, Sparks, & Singh, 2002; Deutsch & Joseph 2003; Fombonne, Roge, Claverie, Courty, & Fremolle, 1999; Lainhart et al., 1997; Stevenson, Schroer, Skinner, Fender, & Simensen, 1997). Researchers have also noted atypical patterns of brain growth in individuals with ASD (Herbert, 2005). Specifically, they have noted accelerated growth in brain volume early in development but not at later points when it is typically observed (Aylward et al., 2002; Courchesne et al., 2001). The abnormal growth is most often attributed to atypical increases in white matter (Courchesne et al., 2001; Herbert et al., 2003a). However, replication of these initial observations has not been consistent (Lotspeich et al., 2004). Despite the overlap with macrocephaly, this condition does not solely account for the deficits observed in ASD (Lainhart et al., 1997; Miles, Hadden, Takahashi, & Hillman, 2000) and is not specific to ASD populations (Gaziuddin, Zaccagnini, Tsai, & Elardo, 1999; Herbert et al., 2003b).

Mirror neurons are activated when we perform actions and see others perform the same action (Ramachandran & Oberman, 2006; Rizzolatti & Craighero, 2004). These neurons are hypothesized to help us infer other's intentions and are thus integral components in social interaction (e.g., imitation,

empathy, self-awareness). Multiple researchers have demonstrated deficits in mirror neuron functioning in ASD populations (Iacoboni & Dapretto, 2006; Nishitani, Avikainen, & Hari, 2004; Oberman et al., 2005; Theoret et al., 2005). Hirstein, Iversen, and Ramachandran (2001) proposed that atypical connectivity surrounding the amygdala, limbic structures, frontal lobes, and sensory input results in abnormal emotional responding. However, this hypothesis has been criticized because it fails to explain the presence of RRBIs symptoms required for a diagnosis of ASD (Ramachandran & Oberman, 2006).

Infants and Toddlers

Typical developmental progression. Prior to a discussion of atypical development, it is important to present a brief overview of typical developmental progression. The increased focus on the early identification of ASD and other developmental disorders has forced researchers to create guidelines which aid in systematically differentiating between typical, delayed, and atypical development in infants and toddlers. Given the heterogeneity observed in developmental progression (Dodson & Alexander, 1986; Green & Palfrey, 2002; Shelov & Altmann, 2009; Shelov & Hannemann, 1991), these guidelines often encompass a wide age range and are not clear-cut.

In 2004 the CDC created a program to educate parents and health care professionals to help monitor a child's cognitive, communication, motor, emotional, and social development. The CDC's information was largely derived from the work of the American Academy of Pediatrics (2008) and Shelov and Altmann (2009). The following information was derived from the works of Fenson and colleages (1994), Green and Palfrey (2002), and Shelov and Altmann (2009) unless otherwise noted. It is important to note that these guidelines do not represent firm rules that clearly differentiate typical and atypical development (Fenson et al., 1994; Green & Palfrey, 2002; Shelov & Altmann, 2009).

During the first year of life, initial developments in communication include cooing, babbling, and imitating simple sounds. The infant may build upon these initial skills by stringing babbling into a series of consonants and using sounds to communicate emotion. At one year children should begin responding to simple requests and utilizing/imitating simple gestures (e.g., shaking head "no"). At this point, their

babbling should begin to incorporate alterations in tone, and they may begin to imitate/approximate simple words presented by a parent or caretaker. During this period, children may also begin communicating using simple words or phrases (e.g., "mama"). As children approach their second birthday, their vocabulary should consist of approximately 15 to 20 words.

Socially, children should begin social smiling when engaging with parents and caretakers during the first year. Closer to their first birthday, infants should begin to respond to their name, exhibit an interest in others, and enjoy and seek out simple interaction games (e.g., peek-a-boo) with preferred persons. Children's emotional state may change depending on their familiarity with individuals or the situation at hand. During this period, children should also begin to display a preference for certain objects or persons. Children may also begin to use sounds and physical actions to gain caretaker's attention and exhibit an understanding of social cues.

The presence of RRBIs within the first year of life is not a symptom clearly differentiating typical from atypical development. Researchers have previously reported that both typically and atypically developing children exhibit various forms of RRBIs (Evans et al., 1997; Morgan, Wetherby, & Barber, 2008). Further, the severity and frequency of these behaviors do not significantly differ across populations (Baranek, 1999; Osterling et al., 2002). Although RRBIs are present during the first year, typically developing children begin to exhibit them less frequently after their first birthday (Evans et al., 1997; Thelen, 1979).

By 2 years of age children may begin to point to identify objects such as toys or body parts when they are named. In addition to imitating simple words heard in conversation and following basic instructions, children should also begin to verbally identify objects as they are presented or identified. At this point children should begin to identify the names of familiar persons and may begin to communicate using simple sentences. Following their second birthday their vocabulary is expanding rapidly and includes approximately 50 words.

Around 2 years of age children may become visibly excited when interacting with peers and display affection for others. In addition to parallel play, children may also begin to seek out and incorporate their peers in activities and display turn taking behavior. Their imitation skills may increase to include novel or more difficult behaviors/activities or expand upon previously learned behaviors.

At this age, the manifestation of RRBIs in typically developing children has decreased and differs significantly in frequency from those with ASD (Lord, 1995; Matson et al., 2009). However, typically developing children may continue to exhibit some RRBIs (Richler, Bishop, Kleinke, & Lord, 2007). Further, Leekam and colleagues (2007) also suggested that gender may influence the exhibition of RRBIs. Specifically, within this age range the authors observed that typically developing males exhibited more RRBIs than typically developing females.

At 3 years of age children should be able to communicate their age, gender, and name when prompted. In addition to naming familiar objects, they should also be able to identify preferred peers. Their speech should be intelligible and readily understood by unfamiliar persons. Further, they should be able to converse with others using complex sentences and be able to tell simple stories. Their vocabulary has increased to include as many as 1,000 words, and they are beginning to display a basic understanding of grammar. At this point they should understand prepositions (e.g., under) and begin to incorporate the use of personal pronouns into their speech.

Socially, children should be able to take turns in games and begin to understand possessive pronouns (e.g., mine, hers). At this age children should display a wide range of facial expressions that align with the environmental context and their emotional state. Although they may exhibit discomfort following major changes in routines, they should readily separate from parents/caretakers. Children should also be able to readily imitate both peer and adult behavior and engage in make-believe or pretend play.

Some researchers have argued that typically developing children continue to exhibit RRBIs up until 4 years of age and decline thereafter (Evans et al., 1997; MacDonald et al., 2007). Despite the

disagreement about when RRBIs decline in typically developing children, researchers assert that there is a clear and quantifiable difference in the expression of RRBIs in ASD populations compared to their typically developing peers (Bodfish et al., 2000). MacDonald and colleagues (2007) noted strong differences in the exhibition of repetitive behavior in typically developing toddlers by 2 years of age. However, the authors noted that these differences became significantly greater by age 3.

Atypical developmental progression. Little is known about the developmental course of infants with ASD during their first year of life (Werner et al., 2000). Given the noted variability in typical development, it is difficult to identify atypical development during the first year of life as a reliable predictor of later ASD diagnosis (Werner et al., 2000). However, as children age, initial developmental delays become pronounced deficits; that are associated with significant impairments in daily functioning and impede further development (Desombre et al., 2006; Landa & Garrett-Mayer, 2006; Werner et al., 2000). The study of early developmental progression is hindered by a conservative diagnostic approach, as ASD is often not diagnosed until a child is 3 or 4 years of age (DeGiacomo & Fombonne, 1998; Kozlowski et al., 2011; Matson, 2005).

Existing information on early ASD development has been derived from three primary sources of information: retrospective parent-report, high-risk infant studies, and video review (Wetherby et al., 2007). Given the unreliability of retrospective parent-report (Zwaigenbaum et al, 2005; Zwaigenbaum et al., 2007), researchers have previously employed two alternative methodologies to study atypical development during the first year of life. One approach invovles analyzing home-video footage of an infant or toddler later diagnosed with ASD (Osterling & Dawson, 1994; Werner et al., 2000). Although this approach has been criticized due to methodological flaws (i.e., contextual limitations, selection bias), it has still yielded valuable insight into atypical development during the first year of life (Zwaigenbaum et al., 2007). A second approach involves the analysis of the developmental course of newborns identified as high-risk for developing ASD (i.e., having an older sibling with ASD; Bryson et al., 2007;

Zwaigenbaum et al., 2007). Despite noted limitations, these retrospective and prospective approaches have provided preliminary insight into the developmental course of ASD.

A majority of parents first express concern about their child's development around 18 months of age (Howlin & Moore, 1997; Siegel, Pliner, Eschler, & Elliot, 1988). During their first year of life, children with ASD may display limited facial expressions and imitation capabilities (Adrien et al., 1993; De Giacomo & Fombonne, 1998; Sigman & Ungerer, 1984). Zwaigenbaum and colleagues (2005) noted that infants with ASD often exhibit multiple visual impairments (e.g., eye-contact, impaired visual attention, visual tracking). When compared to typically developing peers, infants with ASD engage in less social smiling, imitation, and exploration of their environment (Baranek, 1999; Losche, 1990; Osterling & Dawson, 1994; Zwaigenbaum et al., 2005). Bryson and colleagues (2007) reported that infants with ASD display less interest in interacting with others. During this period researchers have also noted that children with ASD exhibit significant deficits in receptive (e.g., response to name) and expressive (e.g., pointing) joint attention skills (Osterling & Dawson, 1994).

During the second year of life developmental delays become more pronounced (Desombre et al., 2006; Landa & Garrett-Mayer, 2006; Werner et al., 2000). Impairments in social skills include decreased sharing of enjoyment or interests (Lord, 1995; Wetherby et al., 2004) and deficits or the complete absence of social referencing abilities (e.g., coordination of gaze and emotional expression to share affective states; Wetherby et al., 2007). Deficits in joint attention skills include deficits in following other's attentional focus, directing other's attention, and a failure to integrate vision and attention (Stone, Ousley, Yoder, Hogan, & Hepburn, 1997b; Wetherby, Prizant, & Hutchinson, 1998). The development of verbal communication skills varies significantly among toddlers with ASD (Tager-Flusberg, Joseph, & Folstein, 2001; Wetherby et al., 2004). In comparison to typically and atypically developing peers, toddlers with ASD exhibit deficits in their use of babbling and single word (Werner & Dawson, 2005).

Toddlers with ASD may also regress in skill development between their first and third birthdays (Goldberg et al., 2003). As much as 40% of toddlers with ASD will exhibit a period of regression with

regard to the use of expressive language (Hoshino et al., 1987; Kurita, 1985, 1996; Rutter & Lord, 1987). Deficits in verbal communication are often compounded by impairments in non-verbal communication. Toddlers with ASD often fail to employ gestures such as declarative pointing, head nodding, waving, and showing to communicate (Loveland & Landry, 1986; Stone et al., 1997b; Werner & Dawson, 2005; Wetherby et al., 1998). During this developmental period deficits in age-appropriate play (i.e., using a toy in the intended manner) become increasingly evident. In addition toddlers with ASD may also exhibit impairments in make-believe or pretend play (Dawson & Adams, 1984; Stone et al., 1990; Wetherby et al., 1998).

Infantile Onset of Psychopathology

Within the *DSM-5* (APA, 2013), the ASD diagnosis is classified as a neurodevelopmental disorder. The *DSM-5* includes a total of 17 neurodevelopmental disorders including ASD, ADHD, ID, communication disorders, specific learning disorders, and motor disorders. The classification of ASD as a neurodevelopmental disorder is commensurate with researchers who have repeatedly demonstrated the presence of symptoms within the first year of life (DeGiacomo & Fombonne, 1998; Dixon, Granpeesheh, Tarbox, & Smith, 2011; Osterling, Dawson, & Munson, 2002; Werner, Dawson, Osterling, & Dinno, 2000). Researchers have suggested that abnormal brain development initially occurs during the prenatal period in ASD populations (Bauman & Kemper, 2003; Rodier, 2002). Yet these abnormalities may continue after birth as well, as indicated by the atypical patterns of brain maturation previously discussed (e.g., Courchesne et al., 2001; Courchesne & Pierce, 2005; Lainhart et al., 1997).

Rydz and colleagues (2006) estimated that 12-16% of children are identified as having a developmental disability. An individual is identified as developmentally delayed when they are two or more standard deviations below average in one or more developmental domains (e.g., speech/language, cognition, gross/fine motor, social/personal) on a standardized, norm-referenced assessment of adaptive functioning (Shevell et al., 2003). The typical approach to assessing developmental progression entails a review of developmental history and discussion of parental concerns (Illingworth, 2013; Sand,

Silverstein, Glascoe, Gupta, Tonniges, & O'Connor, 2005). However, multiple researchers have questioned the sensitivity of this approach (Glascoe, 2005; Hamilton, 2006). Aylward (2009) demonstrated that exclusive reliance upon this method failed to identify as much as 45% of those with developmental disabilities. This method prevails despite the policy put in place by the American Academy of Pediatrics (2001) which recommends regular screening of infants and toddlers. Researchers have demonstrated that systematic screening and testing significantly improves the identification of atypical development (Guevara et al., 2013).

Early Identification

A majority of parents report that they expressed concern about their child's development before 18 months of age (Siegel et al., 1988). Despite this information, the average diagnosis is not made until nearly two years after parents initially expressed concern (Bryson, Rogers, & Fombonne, 2003). Screening administered by a pediatrician is typically the only evaluation children receive prior to school enrollment (Kleinman et al., 2008). Parents of children identified as atypically developing report being evaluated by an average of three professionals during the time preceding initial diagnosis of their child. At this point a large amount of research is available to support the efficacy and stability of an ASD diagnosis around 24 months of age (Charman & Baird, 2002; Eaves & Ho, 2004; Howlin & Moore, 1997; Wetherby et al., 2007). Additional research has noted that children as young as 18 months may be reliably identified in severe cases (Baird et al., 2001; Baron-Cohen, Cox, Baird, Swettenham, & Nighingale, 1996). Further, these early diagnoses have also been observed to be stable and reliable across time (Lord, 1995; Stone et al., 1999). Diagnostic differentiation has been an important factor in early diagnosis. Multiple behaviors such as joint attention (Lewy & Dawson, 1992; McEvoy, Rogers, & Pennington, 1993; Mundy, Sigman, Ungerer, & Sherman, 1986), imitation (Sigman & Ungerer, 1984; Stone, Lemanek, Fishel, Fernandez, & Altemeier, 1990), and functional play (Mundy et al., 1986; Sigman & Ungerer, 1984; Stone et al., 1990) are noted to help differentiate children with developmental delays from those with ASD.

Despite the evidence of diagnostic reliability at younger ages (Charman & Baird, 2002; Cox et al., 1999; Filipek et al., 2000; Lord, 1995; Lord & Risi, 2000; Moore & Goodson, 2003; Stone et al., 1999), the average age of ASD diagnosis remains closer to 4 years of age (Howlin & Moore, 1997; Mandell, Novak, & Zubritsky, 2005; Siegel, Pliner, Eschler, & Elliott, 1988). Mandell and colleagues (2005) identified multiple factors that impede early diagnosis. Specifically, they found that living in rural areas; low socio-economic status, abnormal pain response, and hearing impairment were each associated with delaying the initial ASD diagnosis. Researchers have also noted that ethnicity may indirectly affect early identification. Specifically, Mandell, Listerud, Levy, and Pinto-Martin (2002) reported that African-American children are diagnosed three years later on average when compared to peers from other ethnic backgrounds. Additional factors that interfere with early referral and evaluation include delays in service provision and a relative lack of qualified providers (Dosreis, Weiner, Johnson, & Newschaffer, 2006; Wiggins, Baio, & Rice, 2006; Zwaigenbaum & Stone, 2006). Factors such as the presence of stereotypic behaviors or severe language deficits led to diagnosis a year earlier on average. An additional factor that is associated with earlier diagnosis is service provider selection. Individuals referred to a specialist were diagnosed nearly a half a year earlier than children who sought services from multiple primary care physicians (Mandell et al., 2005). In a recent national survey, professionals working with infants and toddlers with ASD reported that 88% of them diagnosed toddlers with ASD between 18 and 35 months of age (Shaw & Hatton, 2009).

Researchers' attempts to accurately identify ASD symptoms at earlier points in development have also increased (Charman & Howlin, 2003; Filipek et al., 2000; Lord et al., 2005). Initially, retrospective studies based on parental recall of a child's first year of life were analyzed (Dahlgren & Gillberg, 1989; Ornitz et al., 1978). Researchers suggested that nearly half of the parents interviewed recalled abnormalities such as lack of eye contact and limited reaction to parental attempts to play and interact (De Giacomo & Fombonne, 1998; Gillberg et al., 1990; Hoshino et al., 1987; Rogers & DiLalla, 1990; Volkmar, Stier, & Cohen, 1985). Although exploratory in nature, multiple confounding factors (e.g.,

inaccuracy, bias) were present in the research of retrospective parent-report (Osterling & Dawson, 1994; Reznick, 2006). Since then multiple researchers have utilized alternative techniques (e.g., home videos) to analyze the manifestation of ASD symptoms in infants (Adrien et al., 1993; Osterling & Dawson, 1994). Osterling and Dawson (1994) were able to reliably identify children that would be later diagnosed with ASD. In comparison to control groups, children later diagnosed with ASD were noted to differ both qualitatively and quantitatively (Lösche, 1990). The use of home videos to analyze infantile populations had methodological flaws as well. These videos are often not standardized, do not present the same information across children, and provide only a limited sample of behavior (Osterling & Dawson, 1994; Reznick, 2006). Early attempts focused on sensorimotor and cognitive deficits, failing to specifically assess ASD symptomology to allow for differential diagnosis (Osterling & Dawson, 1994). More recently, researchers have demonstrated that behaviors observed around 12 months of age in home videos may distinguish those who will later be diagnosed with ASD from typically developing peers, yet the reliability of this approach was reported to increase by 18 months of age (Baranek, 1999; Baranek et al., 2005). Reznick (2006) cautioned against efforts to diagnose children younger than 18 months due to the limited validity and sensitivity of those measures currently available. Further, the natural heterogeneity surrounding skill development around 12 months in the typically developing population further complicates identifying atypical development and differential diagnosis (Reznick, 2006).

The increased interest surrounding ASD has led to significant policy changes which emphasize screening for developmental delays during well-baby visits (Johnson & Myers, 2007). The American Academies of Neurology, Pediatrics, and Child and Adolescent Psychiatry advocate the use of a two-stage screening procedure for ASD (Bryson et al., 2003). According to the proposed guidelines, initial screening should assess for the presence of global developmental delays, while follow-up assessment should include assessment of symptoms specific to ASD (e.g., socialization and language development; Bryson et al., 2003). Wetherby and colleagues (2004) demonstrated the utility of screening assessments for identifying atypically developing children for follow-up assessment. Multiple measures (e.g., the

Infant-Toddler Checklist (*ITC*; Wetherby, Goldstein, Cleary, Allen, & Kublin, 2003; Wetherby & Prizant, 2002), the *Screening Tool for Autism in Two-Year-Olds* (*STAT*; Stone, Coonrod, & Ousley, 2000), the *Modified Checklist for Autism in Toddlers* (*M-CHAT*; Robins, Fein, Barton, & Green, 2001); and the *Baby and Infant Screen for Children with aUtIsm Traits* (*BISCUIT*; Matson, Boisjoli, & Wilkins, 2007) have since been developed and implemented as screens for ASD symptomology.

Early identification measures. Despite the availability of screening measures, researchers have continuously noted the dearth of diagnostic measures with sound psychometric properties that are appropriate for infants and toddlers (Baird et al., 2001; Baron-Cohen et al., 2000; Filipek et al., 2000; Zwaigenbaum et al., 2005). Glascoe (2005) suggested that sensitivity for a single administration of a diagnostic screener should be between 70 and 80%. The following is a review of measures that are commonly employed to screen for ASD in infant and toddlers and their psychometric properties.

The *Infant-Toddler Checklist* (*ITC*; Wetherby et al., 2003; Wetherby & Prizant, 2002) is an individual component of the broader *Communication and Symbolic Behavior Scales - Developmental Profile* assessment (*CSBS-DP*; Wetherby & Prizant, 2002). Comprised of 24 items, the *ITC* is a broad developmental screener focusing on deficits in language and communication. Normative data for the *ITC* was derived from 2000 infants and toddlers ranging from 6 to 24 months of age (Wetherby & Prizant, 2002). However, positive predictive value for identifying developmental delay is generally low for younger age groups and was 79% for participants between 21-24 months of age. Wetherby and colleagues (2003) reported that the sensitivity and specificity of the *ITC* was 87% and 75%, respectively. However, the *ITC* does not provide for differential diagnosis. Specifically, a positive result from an *ITC* screening does not distinguish between the presence of a communication delay and ASD (Wetherby et al., 2008).

The *Screening Tool for Autism in Two-Year-Olds* (*STAT*; Stone & Ousley, 1997) is an interactive measure that was designed specifically to differentiate children with developmental delays from those with ASD (Stone et al., 2000). The *STAT* is especially useful in assessing lower functioning populations,

as the items (n=12) do not require language comprehension skills (Stone et al., 2000). The recommended age range of the *STAT* is 24 to 35 months. During validation research, Stone and colleagues (2000) observed that the *STAT* has a specificity of 86% and sensitivity of 83%. Stone, Coonrod, Turner, and Pozdol (2004) indicated that the *STAT* has good test-retest reliability and inter-obersver agreement. Additional research has suggested that the *STAT* may be used to identify children younger than 24 months of age with modification of the cutoff scores (Stone, McMahon, & Henderson, 2008). Specifically, the authors noted that with cutoff score modification, the sensitivity was 93% and specificity was 83% in infants ages 14 months and older.

The *Checklist for Autism in Toddlers* (*CHAT*) has previously been used as a screen for ASD in children as young as 18 months (Baron-Cohen et al., 1992). It combines both informant-report (nine items) and direct observation (five items). Although it is noted to exhibit good specificity, it has been noted to demonstrate poor sensitivity (Reznick, 2006). In 2001, the *CHAT* was revised and reintroduced as the *Modified Checklist for Autism in Toddlers (M-CHAT*; Robins et al., 2001). Revisions included the removal of the direct observation component and shifted the target population to include toddlers 16 to 30 months of age (Robins et al., 2001). The *M-CHAT* contains a total of 23 items which are rated in a "yes/no" format by informants (Robins et al., 2001). Kleinman and colleagues (2008) reported that the *M-CHAT* has a positive predictive value of 74%, which was consistent with earlier work by Robins and colleagues (2001). Matson and colleagues (2009b) reported that the *M-CHAT* had a specificity of 87% and sensitivity of 74%. These values are consistent with previous investigations of the *M-CHAT* (Eaves et al., 2006; Wong et al., 2004).

Despite the above revisions, researchers continue to question the psychometric properties of the *M-CHAT* due to its restricted normative sample, limited item content, and response format (Reznick, 2006). Eaves, Wingert, and Ho (2006) questioned the specificity of the *M-CHAT* in high-risk samples (i.e., individuals previously identified as "at-risk" for ASD) and suggested that the measure should not be used to rule out an ASD diagnosis. Ventola and colleagues (2007) noted that the *M-CHAT* failed to

differentiate between those with ASD and those with other developmental disorders. Further, Matson and colleagues (2009b) noted that the *M-CHAT* failed to reliably differentiate between ASD subtypes.

The *Quantitative Checklist for Autism in Toddlers* (*Q-CHAT*; Allison et al., 2008) is the latest revision of the *CHAT*. The *Q-CHAT* represents a shift in diagnostic approach from categorical to dimensional (Allison, Auyeung, & Baron-Cohen, 2012). This revision contains 25 items that are scored on a 5-point Likert-type scale. Item scoring was altered from the "yes-no" format in an attempt to improve reliability. Informant response is based upon the extent to which the target behavior is observed (Allison et al., 2012). Allison and colleagues (2008) observed that the *Q-CHAT* is a valid measure of ASD symptoms and demonstrates adequate test-retest reliability. However, these results should be interpreted with caution given the low response rate observed (33%) and the length of time separating administrations (one month; Allison et al., 2008).

The *BISCUIT* (Matson et al., 2007) is an informant-based assessment battery for infants and toddlers 17 to 37 months of age. Composed of three individual assessments, the *BISCUIT* may be used to assess ASD symptomology (*Baby and Infant Screen for Children with aUtIsm Traits - Part 1, BISCUIT-Part 1*), comorbid psychopathology (*Baby and Infant Screen for Children with aUtIsm Traits - Part 2, BISCUIT-Part-2*), and challenging behaviors (*Baby and Infant Screen for Children with aUtIsm Traits - Part 3, BISCUIT-Part 3*). The entire *BISCUIT* battery may be administered to a parent, caregiver, or guardian in approximately 30 minutes. Scale development and item selection were based on the methodology recommended by Devellis (1991) and Crocker and Algina (1986). Each portion of the *BISCUIT* is read to a parent/caregiver who is then asked to compare their child to a same-aged peer. Items are rated as "0" = not different; no impairment, "1" = somewhat different; mild different, and "2" = very different; severe impairment.

The *BISCUIT-Part 1* contains 62 items and was designed to gather information relevant to the diagnosis of autistic disorder and PDD-NOS but may also be employed to monitor symptom manifestation during intervention (Matson et al., 2007). Researchers have previously demonstrated the

diagnostic sensitivity (93%) and specificity (86%) of the *BISCUIT-Part 1*, which has a positive predictive value of 88% (Matson, Wilkins, Sharp, Knight, Sevin, & Boisjoli, 2009b). Matson, Wilkins, and Fodstad (2011) reported that the *BISCUIT-Part 1* also demonstrated good convergent validity with the *M-CHAT*. The *BISCUIT-Part 1* also demonstrates excellent internal consistency (97%; Matson et al., 2009a).

Early Intervention

Given the complex etiology and current lack of prenatal procedures available to identify the presence of ASD, significant amounts of research have instead focused on early identification and intervention (Galli, Carminati, Gerber, Baud, & Baud, 2007). Ramey and Ramey (1998) broadly identified early intervention as a method used to enrich a toddler's development through the implementation of a variety of activities. Early identification is beneficial for multiple reasons inclduing the early diagnosis and identification of comorbid conditions, increased understanding of the services and support available, and earlier enrollment in the appropriate interventions (Dover & Le Couteur, 2007).

The emphasis placed upon early identification is largely driven by researchers who have demonstrated the necessity and efficacy of early intervention in ASD populations (Dawson & Osterling, 1997; Myers & Johnson, 2007; Reichow & Wolery, 2009). In contrast with childhood interventions, the emphasis of early intervention often entails intervention during the second and third year of life. Researchers have demonstrated that intervention provided at age 3 has a significantly greater impact than the same intervention occurring after 5 years of age (Woods & Wetherby, 2003). However, some caution is necessary. Despite the increased stress upon early identification, Matson, Wilkins, and Gonzalez (2008) emphasized that early identification is advantageous only if it aids in the provision of care, has predictive validity, and is reliable. Further, the goals and efficacy of treatment should be understood and clearly communicated to parents and caretakers. Despite various claims, Volkmar (1998) argued that autism is not a transient entity; symptom severity may decrease following treatment, but this does not mean an individual has been "cured." Instead, the early intervention process is multidimensional and ongoing. Prior to the provision of services, a thorough assessment is necessary to identify targets for

intervention (Ramey & Ramey, 1998). Further, early diagnosis and intervention should be complemented by continuous assessment in order to monitor change and treatment efficacy (Dover & Le Couteur, 2007).

Early intervention programs most often target social and communication skills and may also seek to reduce comorbid symptoms and challeninging behaviors (Howlin, Magiati, & Charman, 2009). Although the field surrounding ASD includes alternative therapies such as chelation therapy, acupuncture, and play therapy, these therapies often have limited empirical support (Green, Pituch, Itchon, Choi, O'Reilly, & Sigafoos, 2006; Howlin et al., 2009). At present few of the proposed ASD interventions are empirically supported with early intensive behavioral interventions (EIBI) being an exception. Researchers have previously identified EIBI as the only "well-established" ASD intervention currently available (Rogers & Vismara, 2008).

Early intensive behavioral interventions. A major component of early behavioral intervention has been the use of operant conditioning techniques (Cannella, O'Reilly, & Lancioni, 2006; Green et al., 2006; Matson & Minshawi, 2006). EIBI programs also incorporate multiple components from the field of applied behavior analysis (ABA) such as discrete trial training and pivotal response training (Maurice, Green, & Luce, 1996; Schreibman & Koegel, 2005). However, a variety of intervention techniques have been used. Interventions such as differential reinforcement (Harris, Handleman, & Fong, 1987), time-out (Durand & Carr, 1987), or a combination of the two (Rolider & Van Houten, 1985) are more acceptable than other techniques such as electric shock (Linscheid, Iwata, Ricketts, Williams, & Griffin, 1990).

Researchers have used ABA techniques for treating symptoms of ASD for nearly 50 years (Matson & Smith, 2008). Initially, this approach most often consisted of targeting a single symptom or challenging behavior at a time. Although traditional behavior analytic research focused upon specific behaviors (Matson, 2007b), their methodologies have been increasingly adapted to create broader ASD intervention programs. In 1973, Lovaas and colleagues published research which simultaneously targeted a broad group of behaviors and symptoms with a combination of individually validated treatment

methods. This research was significant as it emphasized an organized approach to concurrently treating multiple symptoms with an array of interventions.

Multiple factors such as symptom severity, participant age, and comorbid psychopathology influence the interpretation of results from EIBI interventions (Matson & Smith, 2008; Symes, Remington, Brown, & Hastings, 2006). Agreement concerning the intensity and duration of intervention necessary to produce positive treatment effects is largely absent. However, given the heterogeneity of symptoms observed and targets for intervention, a uniform approach may never be fully reached. At present general consensus suggests that intervention should occur prior to 36 months of age and include between 20 and 40 hours of intervention per week (Howlin et al., 2009; Matson et al., 2008; Matson & Konst, in press).

Matson (2007) noted that to date a majority of research has failed to include measures of core symptoms of ASD as an outcome measure. This oversight has generated a significant amount of controversy in EIBI research. Despite criticism for focusing on cognitive functioning as an outcome of early intervention, researchers continue to consistently report changes in IQ as an outcome variable (Howlin et al., 2009). Analysis of changes in core ASD symptoms (e.g., social communication) are not as prevalent but have increased in recent years (e.g., Howlin et al., 2007; Kasari, Freeman, & Paparella, 2006; Yoder & Stone, 2006).

Early intensive behavioral intervention evidence. Given the large amount of interventions currently available and new ones that continue to be introduced and implemented without empirical support, Matson and Smith (2008) noted the importance of acceptance of EIBI by parent groups. In a 2007 survey, parents of children with ASD indicated that they were currently using between four and six therapies to treat their child, and had previously tried as many as nine different interventions (Goin-Kochel, Myers, & MacKintosh, 2007).

Following two years of EIBI, individuals receiving treatment exhibited significantly greater gains in multiple domains (i.e., daily living skills, intelligence, social skills, and language) when compared to a

control group (Remington et al., 2007). The authors also reported that parental stress was not negatively impacted by enrollment in EIBI. Early interventions have also been suggested to help decrease the development of secondary deficits and challenging behaviors (Bryson et al., 2003). Multiple meta-analyses have been carried out surrounding the use of EIBI in ASD populations. Researchers have reported that EIBI is an effective intervention for ASD (Eldevik et al., 2009; Howlin et al., 2009; Makrygianni & Reed, 2010; Reichow & Wolery, 2009; Virues-Ortega, 2010). Following a systematic review of EIBI literature, Eldevik and colleagues (2009) observed that on average studies reported that EIBI had a large effect on IQ (*Hedge's g* = 1.10) and a moderate effect on adaptive behaviors (*Hedge's g* = .66).

Research of early brain development has also been presented as evidence supporting the efficacy of early intervention in ASD populations (Shore, 1997). Researchers have suggested that the timing of intervention is crucial in consideration of brain plasticity. Huttenlocher (1994) hypothesized that the success of early intervention may be due in part to increased brain plasticity. Additional researchers have noted that early experiences may help shape and develop neural connections (Fischer & Rose, 1994). Positive predictors of treatment outcome have also been noted to include communication skills and intellectual functioning (Howlin, 1997). In addition to evidence directly supporting early intervention, Shore (1997) reported that the absence of stimulation and/or the accumulation of negative experiences during early development may have significant negative longitudinal ramifications.

Aside from immediate advantages of EIBI, Chasson and colleagues (2007) estimated that early intervention may also help to negate or decrease the demand for long-term special education resources. Upon reaching school-age, children with ASD often receive services through special education programs. Special education programs are variable in both the services rendered and populations they treat (e.g., ID, Down's syndrome). Despite their prevalence, researchers have largely failed to observe significant gains in cognitive, social, language functioning, and adaptive behaviors for children with ASD who have been enrolled in special education programs (Freeman et al., 1985; Howard et al., 2005; Smith et al., 2000).

Purpose

The knowledge base related to ASD has seen a substantial increase since Kanner's (1943) initial description of the disorder. This growth in knowledge has resulted in an increased emphasis on the importance of early identification and intervention. Researchers have demonstrated the increased efficacy of ASD interventions in toddlers (2-3 years of age) compared to the same interventions delayed by two years (Fenske, Zalenski, Krantz, & McClannahan, 1985; Lovaas & Smith, 1988). However, the benefit of early intervention is only possible following accurate identification. Accurate identification is reliant upon the continuation of research surrounding ASD assessments as new information and changes to the ASD diagnostic category are introduced.

The *BISCUIT-Part-1* is a measure often used to assess toddlers between 17 and 37 months of age for symptoms of ASD (Matson, Wilkins, & Fodstad, 2011). Two additional components of the *BISCUIT* assessment battery are also advantageous for early intervention as they assess symptoms of comorbid psychopathology (*BISCUIT-Part 2*; Matson, Boisjoli, Hess, & Wilkins, 2011) and challenging behaviors (*BISCUIT-Part 3*; Rojahn et al., 2009). A growing body of research has demonstrated the validity and reliability of each individual component of the *BISCUIT* assessment battery in ASD populations (Matson et al., 2009b).

A cutoff score of 17 was identified as ideal during the development of the *BISCUIT-Part 1* (Matson et al., 2009b). Researchers reported that this cutoff score provided good sensitivity (84%) and specificity (86%) when differentiating toddlers with atypical development from those with PDD-NOS. A separate cutoff score (i.e., 39) was identified to differentiate between toddlers with PDD-NOS and those with autistic disorder (sensitivity, 84% and specificity, 83%; Matson et al., 2009b). The standard deviation from the central tendency method approach was employed to identify cutoff score ranges for the remaining components of the *BISCUIT* battery (Matson, Fodstad, Mahan, & Rojahn, 2010; Matson, Fodstad, Mahan, & Sevin, 2009). More recently, researchers revised the cutoff scores of each *BISCUIT* component to better reflect the effects of age and development upon symptom manifestation (Horovitz &

Matson, 2013a, 2013b, 2014). This approach is commonly employed in measures of psychological impairment and cognitive functioning, especially for infants and toddlers (Achenbach & Rescorla, 2000; Matson, Kozlowski, Neal, Worley, & Fodstad, 2011).

Horovitz and Matson (2013a, 2013b, 2014) separated toddlers into three age-bands: 17-23 months, 24-30 months, and 31-37 months. The authors then calculated two cutoff scores for each respective age group. One cutoff score was identified to differentiate atypical development from PDD-NOS, and a separate score was identified to distinguish between PDD-NOS and autistic disorder (Horovitz & Matson, 2014). Based upon their analyses, the following cutoff scores were identified for the *BISCUIT-Part 1*. For the 17-23 month age range scores less than 14 were identified as optimal to differentiate atypical development from PDD-NOS (sensitivity, 93% and specificity, 76%) and scores greater than 39 differentiated those with PDD-NOS from those with autistic disorder (sensitivity, 80% and specificity, 81%). For the 24-30 month age range scores less than 18 were identified as optimal to differentiate atypical development from PDD-NOS (sensitivity, 85% and specificity, 85%) and scores greater than 47 were observed to differentiate those with PDD-NOS from those with autistic disorder (sensitivity, 64% and specificity, 90%). For the 31-37 month age range scores less than 19 were identified as optimal to differentiate atypical development from PDD-NOS (sensitivity, 88% and specificity, 89%; Horovitz & Matson, 2014) and scores greater than 34 differentiated those with PDD-NOS from those with autistic disorder (sensitivity, 88% and specificity, 72%).

Recent changes to the ASD diagnostic category and the symptoms required for diagnosis have prompted a revaluation of the cutoff scores for the *BISCUIT*. Changes to the ASD diagnostic category in the *DSM-5* (APA, 2013) have modified the diagnostic criteria and removed the PDD-NOS diagnosis. Researchers investigating the implications of the changes to the ASD symptom structure and diagnostic categories have suggested that as many as 40% of children may no longer meet criteria for an ASD diagnosis (Gibbs et al., 2012; Mattila et al., 2011; Matson et al., 2012a, 2012b; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012). Given the observed differences in previous *BISCUIT*-

Part 1 cutoff scores used to differentiate autistic disorder and PDD-NOS, it is clear that a difference in symptom manifestation was apparent between these two groups (Matson et al., 2009b). The combination of these two diagnostic groups and changes in diagnostic criteria required additional evaluation of the cutoff scores for the *BISCUIT* assessment battery.

Given the positive effects of early intervention, a valid and reliable approach to early identification is paramount. Differential diagnosis is important to identify those individuals who would most benefit from participation in intervention services as early as possible. In addition to differential diagnosis, the assessment of challenging behaviors (*BISCUIT-Part 3*) and comorbid conditions (*BISCUIT-Part 2*) were also updated to reflect diagnostic changes as this information may be integrated into case conceptualization, treatment plans, and future research. Although the *BISCUIT-Part 2* and *Part 3* have not previously differentiated between PDD-NOS and autistic disorder, the recent diagnostic changes may result in some individuals previously diagnosed with ASD as no longer meeting diagnostic criteria. Given this information, it was necessary to evaluate the cutoff scores for all three components of the *BISCUIT* assessment battery.

Study One

A total of three studies are included in the current analysis. The first study focused on the identification of cutoff scores for the *BISCUIT-Part 1* that best discriminated between toddlers with ASD and atypical development. Initially, a Receiver Operating Characteristic analysis for the total sample was conducted to identify a cutoff score that differentiated between ASD and atypical development regardless of participant age. To better account for differences in developmental progression, age-based cutoff scores (i.e., 17-23 months, 24-30 months, and 31-37 months) were also calculated. This analysis was used to determine the effect of individual age cohorts on the discriminating ability of the *BISCUIT-Part 1* and to determine if age-based scoring procedures were appropriate. The discriminative abilities of the *BISCUIT-Part 1* were further analyzed by calculating the Area Under Curve (AUC). The AUC was selected to evaluate the sensitivity and specificity of the *BISCUIT-Part 1*.

Study Two

The second study focused on identifying cutoff score ranges for the *BISCUIT-Part 2* that best discriminated the severity of comorbid psychopathology symptoms within diagnostic groups (i.e., ASD and atypical development). Although the *BISCUIT-Part 2* scoring procedures have not previously differentiated between different ASD diagnoses, the previous cutoff scores were calculated using individuals with a PDD-NOS and/or autistic disorder diagnosis. Utilizing the standard deviation from central tendency method, new cutoff scores were calculated for the total score of the *BISCUIT-Part 2* and each factor (n=5). The cutoff scores identified differentiated between symptom severity for the three age groups within each diagnostic category. Age-based cutoff scores were calculated to examine the impact of age on the manifestation of comorbid symptoms and advance previous *BISCUIT-Part 2* research (Horovitz & Matson, 2013a).

Study Three

The third study sought to identify cutoff scores for the *BISCUIT-Part 3* that best discriminated the severity of challenging behaviors within each diagnostic group. The third component of the *BISCUIT* assessment battery is used to identify challenging behaviors frequently identified in individuals with ASD (i.e., aggressive/disruptive behavior, stereotypic behavior, and SIB). Similar to the *BISCUIT-Part 2*, the *BISCUIT-Part 3* did not previously differentiate between ASD diagnoses. New cutoff scores were created for the *BISCUIT-Part 3* by employing the standard deviation from the central tendency method. A range of cutoff scores was calculated for the *BISCUIT-Part 3* total score and each subscale. Cutoff scores differentiating symptom severity were identified for each age group within each diagnostic category. Age-based scoring procedures were included to account for the impact of age upon the exhibition of challenging behaviors and to advance previous *BISCUIT-Part 3* research (Horovitz & Matson, 2013b).

Method

Participants

Participants in each study were infants and toddlers that received services from Louisiana's EarlySteps program between 2006 and 2013. EarlySteps is a component of Louisiana's Early Intervention System which was implemented in 2004 as part of the Individuals with Disabilities Education Act, Part C. EarlySteps was designed to provide services to families of infants less than 36 months of age with medical conditions that may cause, or are associated with, developmental delays. In the absence of a diagnosed medical condition, the child may also qualify for services if they exhibit significant delays (i.e., 1.5 standard deviations) in two or more developmental domains (e.g., vision, cognition, hearing, communication). Participants admitted to the program are eligible to receive multiple services (e.g., audiology, psychological, nutrition, speech therapy, and occupational therapy) based upon the impairments observed during initial and follow-up evaluations.

A total of 6,860 participants were initially identified for inclusion in the current analyses. However, 12 participants were removed because their age was unidentified, or they fell outside of the normative age range of the *BISCUIT* (i.e., 17-37 months). The remaining sample included 6,848 participants. Individuals were separated into two categories based upon primary diagnosis (ASD or atypical development). Participants placed in the atypical development group included those exhibiting conditions such as global developmental delay, epilepsy, Klinefelter's syndrome, cerebral palsy, and Down's syndrome. The identification of these conditions was based upon informant report or the results of their EarlySteps evaluation. Data included in the current analyses were collected during periods when both the *DSM-IV-TR* (APA, 2004) and *DSM-5* (APA, 2013) were used to make diagnostic decisions. Due to the significant changes to the ASD category, a discussion of the diagnostic process used for the current studies is warranted. The *DSM-5* explicitly states that individuals with any pre-existing ASD diagnosis (e.g., PDD-NOS, autistic disorder) according to the *DSM-IV-TR* may retain their ASD diagnosis (APA, 2013). However, in order to make an ASD diagnosis for the current analyses, a licensed clinical

psychologist reviewed each individual's scores on the *M-CHAT* (Robins et al., 2001) and the *Battelle Developmental Inventory-Second Edition* (*BDI-2*; Newborg, 2005) and evaluated their performance based upon the *DSM-5* diagnostic criteria for ASD (APA, 2013). Using this procedure, an ASD diagnosis was based upon the observed manifestation of symptoms and was not made based upon a previous *DSM-IV-TR* ASD diagnosis.

While making diagnostic decisions, the clinical psychologist was blind to the individual's *DSM-IV-TR* diagnoses and their performance on the *BISCUIT* assessment battery. Of those participants identified, 943 participants met *DSM-5* criteria for an ASD diagnosis and 5,905 were identified as atypically developing. Individual diagnostic categories were then further separated into groups based upon participant age at the time of initial assessment. Participant age was used to separate participants into one of three groups within each diagnostic group: 17 to 23 months, 24 to 30 months, and 31 to 37 months. The use of age cohorts was selected to advance research recently conducted on the *BISCUIT* (Horovitz & Matson, 2013a, 2013b, 2014). The age groups were also utilized to reflect the achievement of developmental milestones to allow for refined peer comparisons (Green & Palfrey, 2002; Horovitz & Matson, 2014; Shelov & Hannemann, 1991). The selected age-bands represent an equal distribution of participant age and capture the full range of ages assessed by the *BISCUIT*. In addition to information regarding diagnostic assessment, demographic information was also collected for each participant and is presented in Table 1.

Participants must have met select criteria prior to inclusion in the current analyses. The participant must have been administered at least one portion of the *BISCUIT* assessment battery. However, participants were evaluated separately for each study so the absence of one component of the *BISCUIT* battery did not exclude them from consideration in the remaining studies. At the time of evaluation the participant must have been within the normative age range of the *BISCUIT* (i.e., 17 to 37 months; Matson et al., 2011). Finally, to be included in the current analyses, the child must have met *DSM-5* criteria for an ASD diagnosis or have been identified as atypically developing.

Table 1
Participant Demographics

17- 23 months (n = 2,414)		
	Atypical (n = 2,151)	**ASD** (n = 263)
Age	20.35 (1.81)	20.44 (1.81)
Race/ethnicity (%)		
African-American	34.50	45.30
Caucasian	54.90	45.30
Hispanic	5.10	3.10
Other	5.50	6.30
Gender (%)		
Female	34.80	26.80
Male	65.20	73.20
24-30 months (n = 3,273)		
	Atypical (n = 2,786)	**ASD** (n = 487)
Age	26.67 (1.97)	26.79 (1.95)
Race/ethnicity (%)		
African-American	38.60	40.50
Caucasian	50.20	49.30
Hispanic	4.50	4.50
Other	6.70	5.70
Gender (%)		
Female	30.90	23.20
Male	69.10	76.80
31-37 months (n = 1,161)		
	Atypical (n = 968)	**ASD** (n = 193)
Age	32.69 (1.42)	32.49 (1.30)
Race/ethnicity (%)		
African-American	38.10	37.80
Caucasian	49.30	45.10
Hispanic	2.90	5.70
Other	9.70	11.40
Gender (%)		
Female	26.50	21.40
Male	73.50	78.60

Note: Participant age is reported in months. Standard deviations are provided in parentheses directly beside the mean age.

Measures

The *BISCUIT* assessment battery is comprised of three measures designed to assess for symptoms of ASD (*BISCUIT-Part 1*), comorbid psychopathology (*BISCUIT-Part 2*), and challenging behaviors (*BISCUIT-Part 3*) in infants between the ages of 17 and 37 months (Matson et al., 2007). The entire battery was initially validated in a large sample of atypically developing infants and toddlers. This

sample included toddlers with an ASD diagnosis. The *BISCUIT-Part 1* contains 62 items that assess three broad symptom domains: communication, socialization/nonverbal communication, and restricted interests/repetitive behaviors (Matson et al., 2009a). During evaluation, parents or caretakers are asked to compare their child to a typically developing peer and rate each item based upon the degree of impairment or difference in behavior present (Matson et al., 2007). An appendix of developmentally appropriate behaviors typical for toddlers within this age range accompanies the assessment battery as a reference for informants. A Likert-type scale is used to code each item, a rating of "2" is ascribed to behavior that is *very different* from their peers, and a rating of "0" reflects *no difference* in behavior relative to their peers (Matson et al., 2007). Administration of the entire battery requires approximately 20-30 minutes (Matson et al., 2011). Researchers have previously demonstrated the diagnostic sensitivity (93%) and specificity (86%) of the *BISCUIT-Part 1*, which has a positive predictive value of 89% (Matson et al., 2009b). Analysis of the reliability of the *BISCUIT* battery indicates that each subscale demonstrates excellent internal consistency with values ranging from 91% (*BISCUIT-Part 3*) to 97% (*BISCUIT-Part 1*; Matson et al., 2009a).

The early identification of comorbid symptoms is also beneficial due to the increased prevalence of comorbid psychopathological conditions observed in ASD populations (Gadow, DeVincent, Pomeroy, & Azizian, 2004; Ghaziuddin, Tsai, & Ghaziuddin, 1992; Konst & Matson, 2014; Konst, Matson, & Turygin, 2013b). The *BISCUIT-Part 2* contains a total of 65 items which are administered to a parent or caregiver by a trained professional. Factor analytic research of the *BISCUIT-Part 2* identified a total of five factors (i.e., tantrum/conduct behavior, inattention/impulsivity, eating/sleeping problems, avoidance behavior, and anxiety/repetitive behavior) related to common psychological impairments observed in ASD populations (Matson et al., 2011). Given the observed differences in the manifestation of comorbid symptoms, separate cutoff scores were created for those with ASD and those identified as atypically developing (Matson et al., 2009c). More recently, Horovitz and Matson (2013a) created age-based cutoff

scores for each diagnostic category. Revisions based upon participant age included the creation of cutoff scores for each subscale and the *BISCUIT-Part 2* total score.

The *BISCUIT-Part 3* is the third scale included in the *BISCUIT* battery of assessments (Matson et al., 2007). It contains 15 items that are used to assess the presence and severity of challenging behaviors. Items were arranged into three categories of challenging behavior based upon factor analytic research: aggressive/disruptive behavior, stereotypic behavior, and SIB (Matson, Boisjoli, Rojahn, & Hess, 2009). Recently, Horovitz and Matson (2013b) created cutoff scores for the *BISCUIT-Part 3* to account for the influence of participant age and developmental course. The authors identified age-based cutoff scores for each factor of the *BISCUIT-Part 3* and the total score.

The *Battelle Developmental Inventory, Second Edition* (*BDI-2*; Newborg, 2005) is a standardized measure of five developmental domains (i.e., social/personal, motor, communication, cognition, and adaptive; Newborg, 2005). The scale includes normative data for children from birth to 7 years and 11 months of age. The *BDI-2* employs three different methods to gather relevant information for each of the 450 items. Assessment administration includes a structured test format and, depending upon the setting and child's capabilities, the evaluator may supplement this information with informant report and direct observation (Bliss, 2007). Item administration is initially informed based upon participant age, but may vary dependent upon their individual level of functioning for each domain. The internal consistency coefficients and inter-rater reliability were observed to be excellent, ranging from 90 to 99% across the individual domains. Test-retest reliability of the *BDI-2* was observed to be above 80% in general, but was reported to vary across domains and age groups (Bliss, 2007).

The *Modified Checklist for Autism in Toddlers* (*M-CHAT*; Robins et al., 2001) is an informant based screener designed to assess toddlers for ASD symptoms. The *M-CHAT* consists of 23 items, with six items identified as critical indicators. Item ratings are based upon "yes" or "no" responses. A toddler is identified for follow-up assessment if they "fail" three of the total items, or two or more of the critical items. The *M-CHAT* demonstrates good sensitivity (87%) and excellent specificity (99%), with a positive

predictive value of 80% (Robins et al., 2001). The measure is also reported to demonstrate good internal consistency (85%; Kleinman et al., 2008; Robins et al., 2001).

Procedure

Participants in the current studies were drawn from a pre-existing database. These individuals were placed into one of two diagnostic categories (i.e., atypical development, ASD) in the database following their participation in the EarlySteps program. As part of their participation in EarlySteps, parents, caregivers, or legal guardians of an infant or toddler were initially administered the *BDI-2*, *BISCUIT*, and *M-CHAT*. Evaluations were conducted by professionals employed by the EarlySteps program. Evaluators had a minimum of a bachelor's degree, but up to a doctoral degree in professional disciplines such as education, speech/language pathology, psychology, early childhood development, and social work. In addition to their primary education, evaluators also held licenses and/or certifications related to their respective fields. Evaluators also received formal training in the administration of standardized assessments and training relevant to each specific measure included in the assessment battery. Informed parental consent to participate in research was given prior to assessment administration. Prior to the collection of data, the current study was approved by the Louisiana State University Institutional Review Board and Louisiana's Office for Citizens with Developmental Disabilities. Following evaluation, the data collected was coded and entered into a secure electronic database for further analysis.

Preliminary Statistics

Study One. Prior to analysis, the dataset was reviewed for missing items and invalid responses. Participants missing more than 10% of the responses for a given component of the *BISCUIT* were removed from further analysis in each respective study (Donner, 1982; Field, 2009). In order to preserve sample size and score distribution, outliers were identified by grouping participants according to diagnostic group and age group. For Study One, group z-scores for the *BISCUIT-Part 1* total score were calculated for each group (n=6). Those z-scores with an absolute value greater than 3.29 were identified

as outliers and removed from the dataset (Field, 2005). The initial sample for Study One included 6,848 participants. However, 32 (0.46%) participants were removed due to missing values and an additional 86 (1.26%) participants were subsequently removed after being identified as outliers. The final sample for Study One included a total of 6,730 participants, with 940 having a *DSM-5* ASD diagnosis. A diagram of participant flow for Study One is presented in Figure 1.

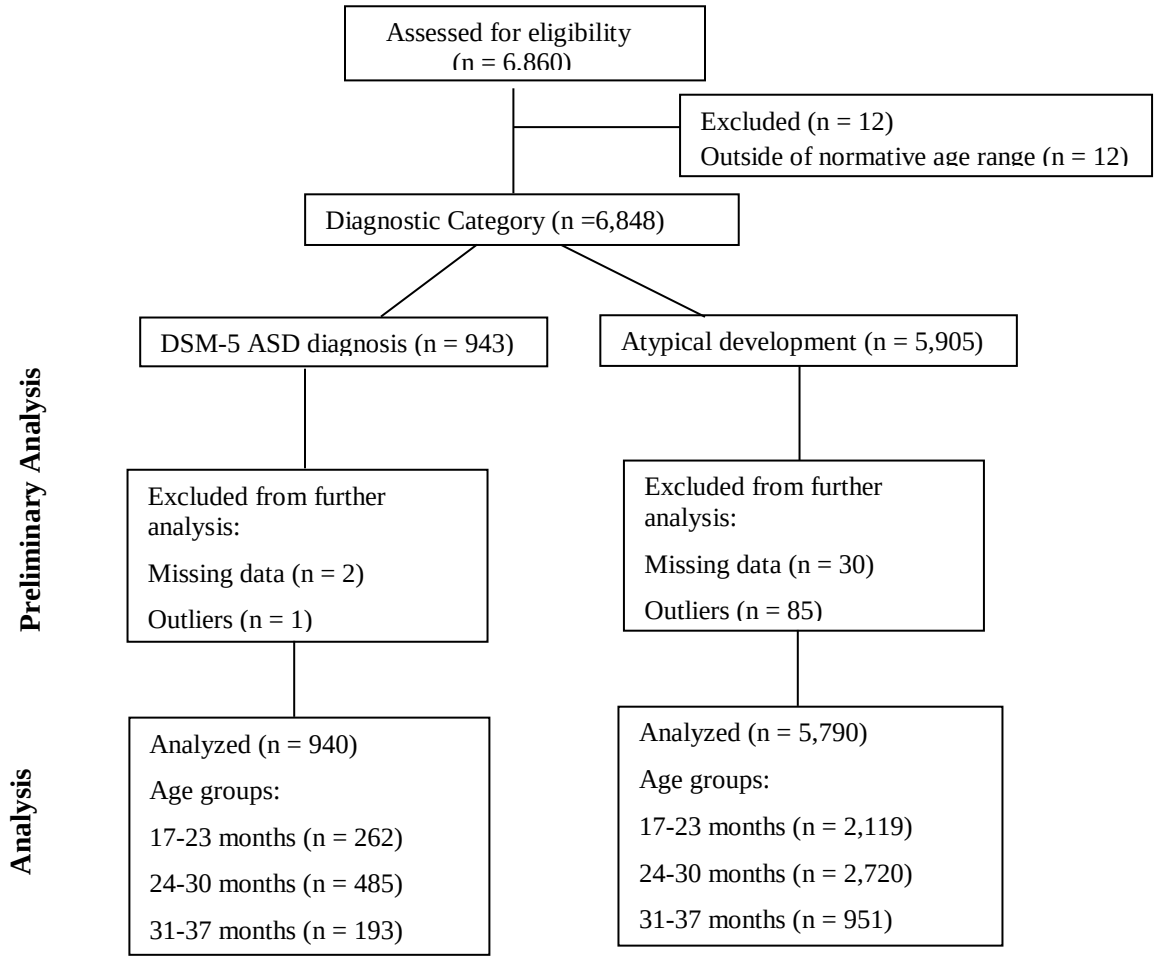

Figure 1. Study One Consort diagram

Study Two. The total score for each *BISCUIT-Part 2* item was analyzed for missing data and extreme values (Cohen, 2008; Field, 2013). To preserve sample size and score distribution, outliers were identified by grouping participants by diagnostic group and age group. As in Study One, group z-scores for the *BISCUIT-Part 2* total score were calculated for each diagnostic and age group (n=6). Those z-scores with an absolute value greater than 3.29 were identified as outliers and removed from the dataset (Field, 2005). The initial sample for Study Two included 3,527 participants. However, 229 (6.49%) participants were removed due to missing values, and an additional 53 (1.61%) participants were subsequently removed after being identified as outliers. The final sample for Study Two included a total of 3,245 participants, with 503 participants meeting criteria for ASD based upon *DSM-5* criteria. A diagram of participant flow for Study Two is presented in Figure 2.

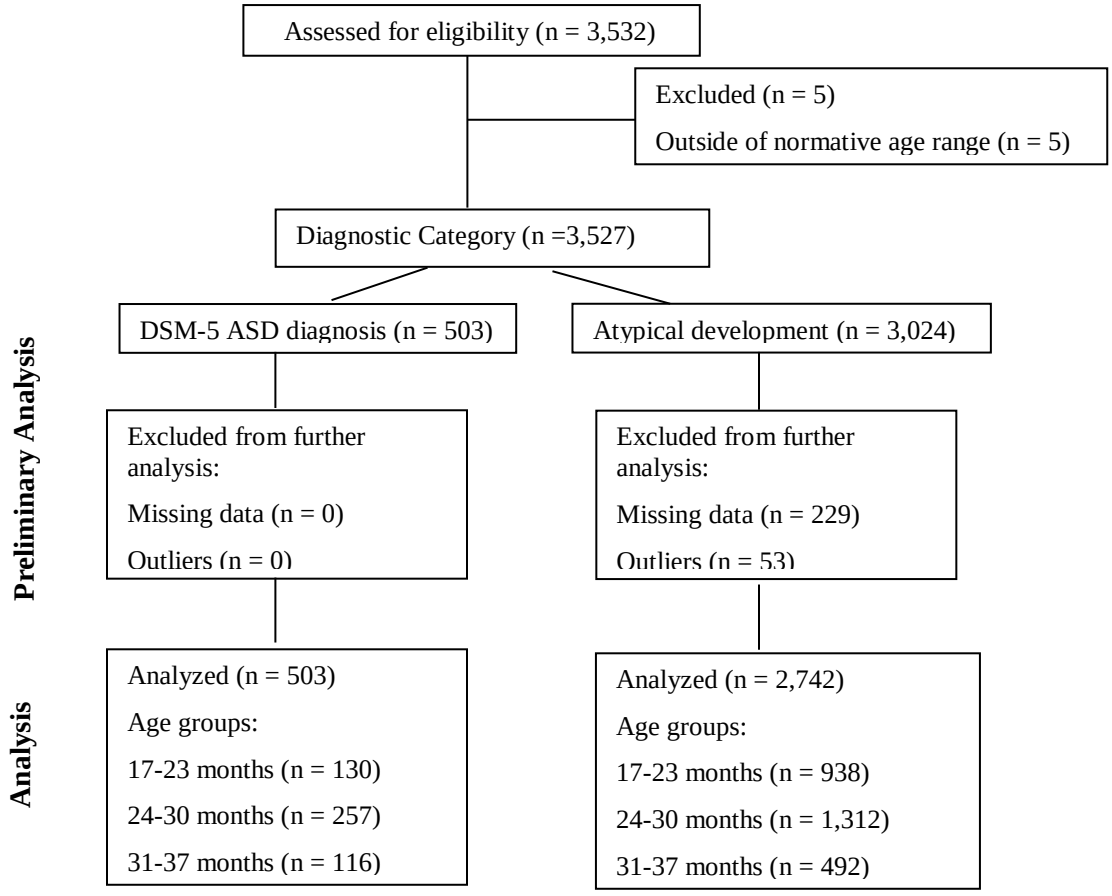

Figure 2. Study Two Consort diagram

Study Three. The analysis used to identify outliers and participants with missing data for the *BISCUIT-Part 3* was identical to the analysis carried out in Studies One and Two. The initial sample for Study Three included 3,455 participants. However, one participant was removed due to missing values, and an additional 57 (1.65%) participants were subsequently removed after being identified as outliers. The final sample for Study Three included a total of 3,397 participants, with 567 individuals meeting *DSM-5* criteria for ASD. A diagram of participant flow for Study Three is presented in Figure 3.

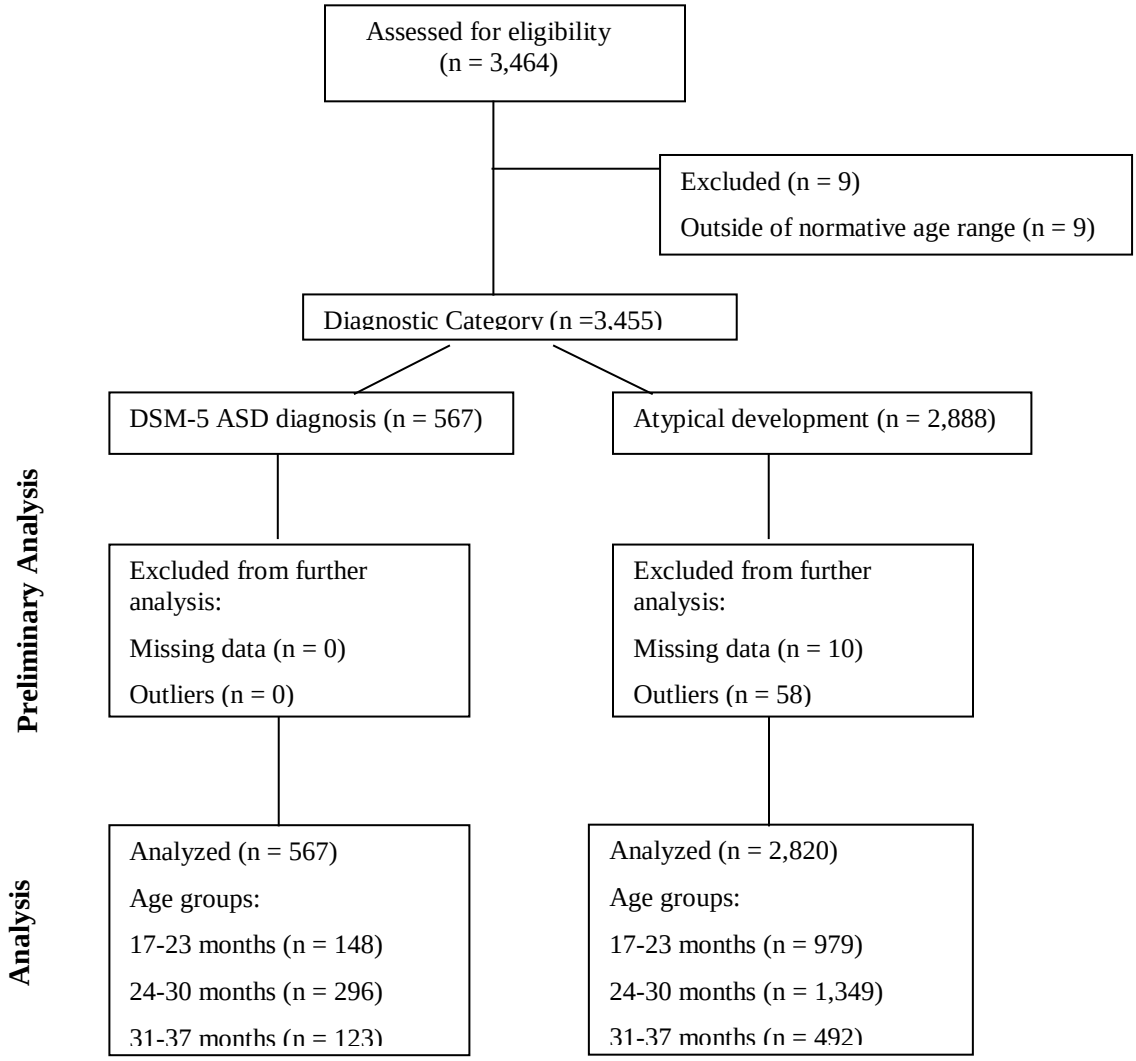

Figure 3. Study Three Consort diagram

Imputation. Prior to further analysis, a Little's Missing Completely At Random test (MCAR) was carried out (Little, 1988). The MCAR was not significant and interpreted as indicating that the missing data was missing at random, and not related to purposeful omission or the dependent variables (Tabachnick & Fidell, 2007; Van Ness, Murphy, Araujo, Pisani, & Allore, 2007).

A multiple imputation procedure was carried out for individuals identified as missing less than 10% of responses. The multiple imputation procedure involves multiple steps for estimating missing item values and is reported to maintain sampling variability (Tabachnick & Fidell, 2007). First, a logistic regression was carried out utilizing diagnostic category, developmental quotient, and participant age as predictor variables. The logistic regression then utilized these variables to create an equation for estimating missing item values for each diagnostic category (Tabachnick & Fidell, 2007). A random sample was then drawn (with replacement) from the cases without missing variables. The identified variable distribution for each missing item was then used to provide an estimate for the missing items in five random samples (with replacement). The average imputed variable across all five samples for each missing item value was then included in a sixth dataset (Mehrotra, Li, Liu, & Lu, 2012; Rubin, 1987). The sixth dataset was then used to carry out Studies One, Two, and Three.

Study One

Statistical Analyses

The first study focused on identifying cutoff scores for the *BISCUIT-Part 1* that best discriminated between toddlers with ASD and those identified as developing atypically. Initially, a ROC analysis for the total sample was carried out. This analysis was used to identify a cutoff score that reliably differentiated between ASD and atypical development regardless of participant age. Following the initial evaluation of the total sample, a ROC analysis was carried out for each proposed age cohort (i.e., 17-23 months, 24-30 months, and 31-37 months). These analyses were used to determine the effect of an individual's age and development on the discriminating ability of the *BISCUIT-Part 1* and to explore the utility of age-based scoring procedures. ROC curve analyses are an empirical method used to inform decisions regarding the threshold of a diagnostic test (Metz, 1978). A ROC curve is a graphical display used to evaluate the discriminatory accuracy of a given measure over all possible cutoff values for a given sample (Fluss, Faraggi, & Reiser, 2005; Metz, 1978). The ROC curve evaluation is carried out independent of condition prevalence and decision threshold effects because it is constructed based upon the comparison of the true positive fraction and the false positive fraction (Metz, 1978). ROC curve analyses have previously been used in the development of multiple screening and assessment measures (Glascoe & Byrne, 1993; Meisels, Henderson, Liaw, Browning, & Have, 1993).

The performance of the *BISCUIT-Part 1* was further analyzed by calculating the AUC. The AUC was used as a measure of discrimination by evaluating the overall sensitivity and specificity of all cutoff scores identified by the ROC analyses. An AUC value of 1 indicates perfect accuracy, while a value of .50 indicates that the test operates at a level equivalent to random chance. Values between .80 and .90 are suggested to represent good discriminative ability, while values between .90 and 1 are considered to represent excellent discriminative ability (Hanley & McNeil, 1982, 1983; Zou et al., 2007). An alpha level of .05 was used to determine the significance of each AUC value. Finally, cutoff scores for the total sample and each age cohort were then identified utilizing the Youden Index *J* (Youden, 1950). The

Youden Index *J* was used to calculate the cutoff value that maximizes the tradeoff between sensitivity and specificity when weighing both equally (Faraggi, 2000; Greiner, Pfeiffer, & Smith, 2000; Perkins & Schisterman, 2005; Reiser, 2000). Saah and Hoover (1997) identified diagnostic sensitivity as the percentage of participants correctly identified as having a given condition. Conversely, diagnostic specificity is the percentage of participants correctly identified by a measure as not meeting criteria for a specific condition. Greiner and colleagues (2000) noted that the emphasis on both diagnostic sensitivity and specificity allows for the consideration of all information gathered by a diagnostic assessment.

In order to further evaluate the applicability of age-based scoring procedures for the *BISCUIT-Part 1*, the positive predictive value (PPV) and negative predictive value (NPV) were calculated. These scores were calculated for the total sample and each age group. Altman and Bland (1994) defined PPV as the proportion of patients who are correctly diagnosed and have positive test results. The NPV is the proportion of individuals who are correctly diagnosed and do not have positive results.

Results

Total sample. A ROC analysis was initially carried out for the full sample regardless of participant age to analyze the discriminative ability of the *BISCUIT-Part 1*. This initial analysis produced the curve depicted in Figure 4. The calculated ROC curve had an *AUC* =.96, $p<.01$, which is considered to represent excellent diagnostic discrimination (Hanley & McNeil, 1982, 1983; Zou et al., 2007). The Youden Index *J* was also calculated to identify the cutoff point that optimized the tradeoff between diagnostic sensitivity and specificity while weighting both equally. In Figure 4, Youden Index *J* is considered the point of the ROC curve that falls furthest from the line indicating chance performance. This calculation was carried out for all possible cutoff scores utilizing the following formula; sensitivity + specificity – 1 (Youden, 1950). Prior to cutoff score selection, the sensitivity, specificity, and Youden Index *J* were each examined. Table 2 depicts cutoff scores for the total sample and their respective sensitivity, specificity, and Youden Index *J*. For the full sample the optimal cutoff value for differentiating between atypical development and ASD was a score greater than or equal to 25, Youden

Index *J* = .81. This cutoff value was associated with excellent diagnostic sensitivity (94%) and good diagnostic specificity (87%). The PPV for the total sample cutoff score of 25 was 56%, while the NPV was 99%.

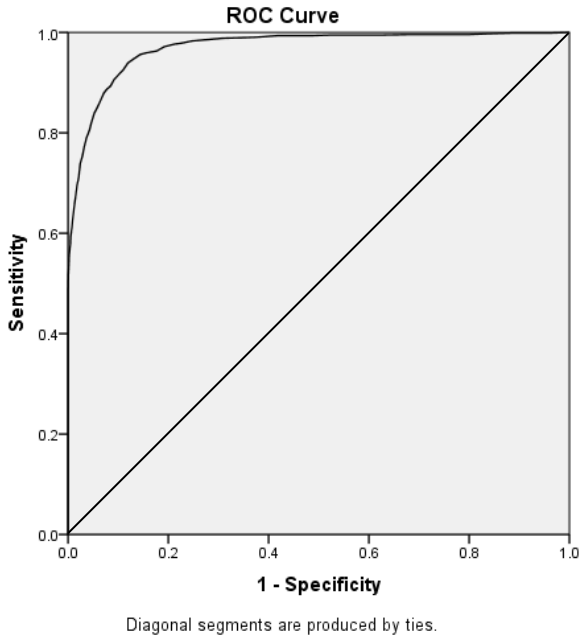

Figure 4. ROC curve depicting the trade-off between diagnostic sensitivity and 1-specificity for range of *BISCUIT-Part 1* cutoff scores for the complete sample.

Table 2
Potential Cutoff Scores for the Total Sample

	Cutoff score	Sensitivity	Specificity	Youden Index *J*
Atypical development Vs. ASD	≥22	.9597	.8304	.7901
	≥23	.9565	.8430	.7995
	≥24	.9480	.8558	.8038
	≥25*	.9395	.8676	.8071
	≥26	.9257	.8767	.8024
	≥27	.9161	.8859	.8020
	≥28	.9066	.8946	.8012

* selected cutoff score

Participants 17 to 23 months of age. The analyses carried out for the total sample were also conducted for each age group. These analyses were necessary to observe any potential difference in symptom manifestation across age groups and determine the appropriateness of age-based scoring procedures for the *BISCUIT-Part 1*. The ROC analysis for the 17 to 23 month age group produced the

curve appearing in Figure 5. The *BISCUIT-Part 1* demonstrated excellent discriminating ability within this age group (*AUC* = .97, $p < .01$; Hanley & McNeil, 1982, 1983; Zou et al., 2007). The optimal cutoff point identified for this age group was a score greater than or equal to 27 (Youden Index *J* =.84). This cutoff score was associated with excellent diagnostic sensitivity (95%) and good diagnostic specificity (89%). The PPV for this age group was 74% and the NPV was 97%. Table 3 depicts potential cutoff scores considered for this age group and their respective sensitivity, specificity, and Youden Index *J*.

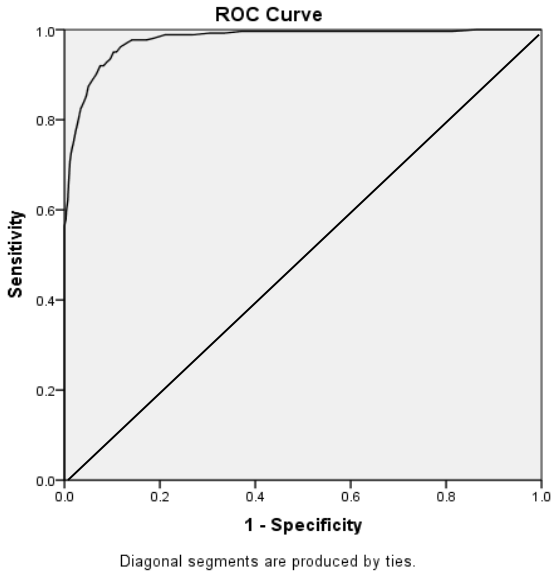

Figure 5. ROC curve depicting the trade-off between diagnostic sensitivity and 1-specificity for range of *BISCUIT-Part 1* cutoff scores for participants 17-23 months of age.

Table 3
Potential Cutoff Scores for Toddlers 17-23 Months of Age

	Cutoff score	Sensitivity	Specificity	Youden Index *J*
Atypical development Vs. ASD	≥24	.9696	.8610	.8306
	≥25	.9620	.8727	.8347
	≥26	.9506	.8811	.8317
	≥27*	.9506	.8871	.8377
	≥28	.9354	.8937	.8291
	≥29	.9278	.9016	.8294
	≥30	.9202	.9076	.8278

* selected cutoff score

Participants 24 to 30 months of age. Next a ROC analysis was carried out for the 24 to 30 month age group to identify the cutoff score that differentiates atypical development from ASD. The

ROC analysis for this age group produced the curve presented in Figure 6. The *BISCUIT-Part 1* was observed to demonstrate excellent discriminating ability within this age group (*AUC* = .96, $p < .01$; Hanley & McNeil, 1982, 1983; Zou et al., 2007). Table 4 depicts potential cutoff scores considered for this age group and their respective sensitivity, specificity, and Youden Index *J*. The optimal cutoff point identified for this age group was a score greater than or equal to 25 (Youden Index *J* =.81). This cutoff score was associated with excellent diagnostic sensitivity (94%) and good diagnostic specificity (87%). The PPV for this age group based upon the selected cutoff score was 56%, while the NPV was 99%.

Table 4
Potential Cutoff Scores for Toddlers 24-30 Months of Age

	Cutoff score	Sensitivity	Specificity	Youden Index *J*
Atypical development Vs. ASD	≥22	.9630	.8254	.7884
	≥23	.9609	.8388	.7997
	≥24	.9547	.8532	.8079
	≥25*	.9444	.8662	.8107
	≥26	.9321	.8745	.8066
	≥27	.9177	.8850	.8027
	≥28	.9115	.8941	.8056

* selected cutoff score

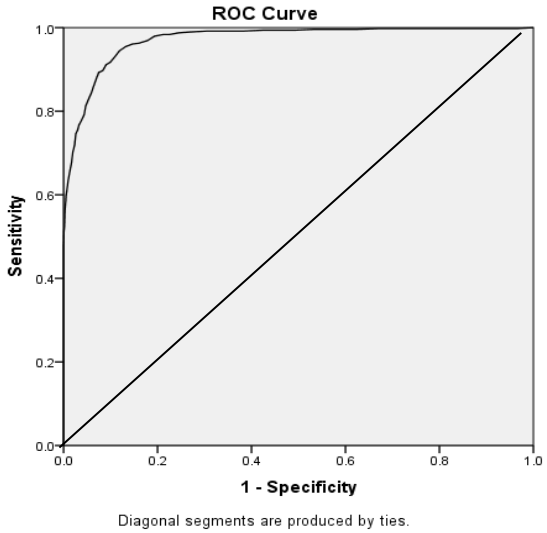

Figure 6. ROC curve depicting the trade-off between diagnostic sensitivity and 1-specificity for range of *BISCUIT-Part 1* cutoff scores for participants 24-30 months of age.

Participants 31 to 37 months of age. Finally, a ROC analysis was also carried out for those participants between 31 and 37 months of age. The ROC analysis for this age group produced the curve

presented in Figure 7. The *BISCUIT-Part 1* was observed to demonstrate excellent discriminating ability within this age group (*AUC* = .95, $p < .01$; Hanley & McNeil, 1982, 1983; Zou et al., 2007). Table 5 depicts potential cutoff scores considered for this age group and their respective sensitivity, specificity, and Youden Index *J*. The optimal cutoff point identified for this age group was a score greater-then or equal to 23 (Youden Index *J* = .75). This cutoff score was associated with excellent diagnostic sensitivity (92%) and good specificity (84%). The PPV for this age group based upon the selected cutoff score was 84%, while the NPV was 97%.

Table 5
Potential Cutoff Scores for Toddlers 31-37 Months of Age

	Cutoff score	Sensitivity	Specificity	Youden Index *J*
Atypical development Vs. ASD	≥20	.9378	.7915	.7293
	≥21	.9275	.8091	.7366
	≥22	.9275	.8299	.7574
	≥23*	.9171	.8413	.7584
	≥24	.9016	.8517	.7533
	≥25	.8964	.8600	.7564
	≥26	.8756	.8734	.7490

* selected cutoff score

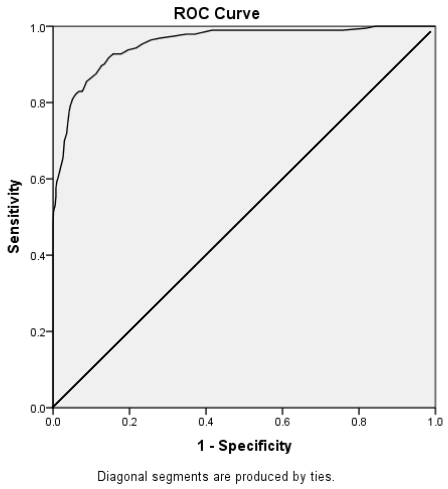

Figure 7. ROC curve depicting the trade-off between diagnostic sensitivity and 1-specificity for range of *BISCUIT-Part 1* cutoff scores for participants 24-30 months of age.

Discussion

Researchers have previously questioned the validity of the PDD-NOS diagnosis based upon the descriptor provided in the *DSM-IV-TR* (Luteijn et al., 2000b; Walker et al., 2004). Specifically, Luteijn and colleagues (2000b) criticized the diagnostic criteria for PDD-NOS, suggesting that it was unclear and ambiguous, which led to differences in interpretation and application. The current research is based upon the *DSM-5* ASD diagnosis which removed the need for differentiation between the various ASD diagnoses. The identification of new cutoff scores for the *BISCUIT-Part 1* utilizing a large normative population with *DSM-5* diagnoses provided a scoring approach that is in line with the current diagnostic criteria (APA, 2013).

Previous investigations have demonstrated the discriminative abilities of the *BISCUIT-Part 1*with and without the use of age-based scoring procedures (Horovitz & Matson, 2014; Matson et al., 2009b). Given that the proposed study did not alter the content of the *BISCUIT-Part 1*, it was hypothesized that the measure would continue to discriminate between diagnostic groups at levels significantly greater than chance. This hypothesis was confirmed by the ROC analyses carried out in Study One. The AUC statistic was significant for the total sample and each individual age group suggesting that the *BISCUIT-Part 1* discriminates between atypical development and ASD at levels greater than chance regardless of participant age. With AUC values ranging from .95 (31-37 months of age) to .97 (17-23 months of age), the cutoff scores selected for the *BISCUIT-Part 1* are considered to demonstrate excellent discriminative ability for the total sample and each respective age group (Hanley & McNeil, 1982, 1983; Zou et al., 2007). Furthermore, the AUC values observed were greater than those reported for cutoff scores previously used for the *BISCUIT-Part 1* (*AUC* =.84 to .95; Horovitz & Matson, 2014). In order to utilize all of the information gathered during an assessment, the Youden Index *J* was calculated to identify cutoff scores that placed a balanced emphasis on diagnostic sensitivity and specificity. As depicted in Table 6, the selected cutoff scores for the *BISCUIT-Part 1* demonstrate excellent diagnostic sensitivity and good diagnostic specificity.

Table 6

BISCUIT-Part 1 Selected Cutoff Scores

Atypical development Vs. ASD	Cutoff score	Sensitivity	Specificity	Youden Index *J*
Total sample	≥25	.9395	.8676	.8071
17-23 months	≥27	.9506	.8871	.8377
24-30 months	≥25	.9444	.8662	.8107
31-37 months	≥23	.9171	.8413	.7584

It was hypothesized that the computed analyses would identify new cutoff scores and increase the diagnostic sensitivity and specificity of the *BISCUIT-Part 1*. Given the removal of a diagnostic category, a direct comparison between previously identified cutoff scores for the *BISCUIT-Part 1* and the results of the current analysis was not possible within the confines of the current dataset. However, a general discussion is warranted. Previously selected cutoff scores for the *BISCUIT-Part 1* included diagnostic sensitivity values ranging from 64 to 93% and diagnostic specificity values ranging from 72 to 89%. Those cutoff scores identified by the current analysis demonstrate less variability and are higher on average with regard to diagnostic sensitivity (92 to 95%) and diagnostic specificity (84 to 89%). The Youden Index *J* values (*J* = .76 - .84) identified by the current analysis was also greater than those previously identified for distinguishing between PDD-NOS and atypical development (*J* = .70) and between PDD-NOS and autistic disorder (*J* = .55; Horovitz & Matson, 2014).

A multitude of factors likely contributed to the observed increases in diagnostic sensitivity and specificity. The *DSM-5* committee members indicated the goal of restructuring the ASD category was to increase diagnostic sensitivity and specificity (APA, 2011, 2013; Frazier et al., 2012; Gibbs et al., 2012; Grzadzinski et al., 2013; McPartland et al., 2012). To do so, the committee altered the criteria necessary for an ASD diagnosis (Boomsma et al., 2008; Gotham et al., 2007; Lord & Jones, 2012) and consolodated previous ASD diagnostic groups (Gibbs et al., 2012; Frazier et al., 2012; Tsai & Ghaziuddin, 2013). Further, the sample used in the current analysis (N = 6,730 participants, 940 of whom met criteria for a *DSM-5* ASD diagnosis) was roughly twice the size of the sample (N = 3,062) utilized by Horovitz and Matson (2014). The standardization sample previously used included a total of 892 participants with

ASD. This included 505 participants who met criteria for autistic disorder and 387 participants who met criteria for PDD-NOS.

Aside from analyzing potential cutoff scores for the total sample, Study One also analyzed and selected cutoff scores for three separate age ranges (17-23, 24-30, and 31-37 months of age). The selection of age-based cutoff scores was included to advance previous research (Horovitz & Matson, 2014) and to allow for refined peer comparisons (Green & Palfrey, 2002; Horovitz & Matson, 2014; Shelov & Hannemann, 1991). Relative to the cutoff scores generated by the age-based analysis previously conducted (Horovitz & Matson, 2014), the current analysis identified cutoff scores that increased the diagnostic sensitivity and specificity of the *BISCUIT-Part 1*. Tables 2, 3, 4, and 5, allow for a visual analysis of the changes in diagnostic sensitivity and specificity across a range of potential cutoff scores for the total sample and each age group. Given the influence of developmental progression, it was hypothesized that a range of cutoff scores based upon participant age would better differentiate between atypical development and ASD than a single cutoff score for the total sample.

The hypothesis that cutoff scores for individual age groups would differ from the cutoff scores identified for the total sample was partially upheld. Specifically, the cutoff scores identified for the 17-23 and 31-37 month age groups did differ from the cutoff score identified for the total sample. For example, the use of the cutoff score identified for the total sample (i.e., 25) for the 17-23 month old group would result in an increase in diagnostic sensitivity and a decrease in specificity. The reverse scenario was observed for the 31-37 month age group, as a cutoff score of 25 decreased diagnostic sensitivity and increased diagnostic specificity. However, the cutoff score identified for the total sample, regardless of participant age, is largely consistent with the cutoff score and psychometric properties identified for the 24-30 month age group. This is likely due to sample distribution, as the 24-30 month age group was the largest single group for both diagnostic categories. The identified cutoff scores for each age group were selected utilizing the *Youden Index J* in order to give equal weight to diagnostic sensitivity and specificity (Faraggi, 2000; Greiner, Pfeiffer, & Smith, 2000; Perkins & Schisterman, 2005; Reiser, 2000). Given the

observed variation in cutoff scores, the use of age-based cutoff scores should be continued to maximize the psychometric properties of the *BISCUIT-Part 1*. Further, the use of age-based scoring procedures resulted in PPVs and NPVs that were greater than or equal to those calculated for the total sample cutoff score.

Previous researchers have noted that in the absence of intervention, the severity of ASD symptoms increases as children age (Charman et al., 2005; Moore & Goodson, 2003; Starr et al., 2003). Based upon this finding, it was hypothesized that the cutoff scores necessary to distinguish between diagnostic groups would increase with participant age. This hypothesis was not confirmed by the results of the current research. In fact, the *BISCUIT-Part 1* score necessary to distinguish between participants with atypical development and ASD gradually decreased across age groups. However, this does not refute previous research suggesting that ASD symptom severity increases in the absence of intervention. Rather, the results of the current study suggest that the symptom severity necessary for differential diagnosis decreases as individual's age. This finding is consistent with previous research. For example, researchers have found that atypically developing children exhibit some symptoms consistent with ASD (i.e., RRBIs) early in the lifespan, but the frequency of this behavior decreases after the second birthday. Both atypically and typically developing toddlers are reported to exhibit RRBIs during the first year of life (Evans et al., 1997; Morgan, Wetherby, & Barber, 2008). Researchers have also demonstrated that the manifestation of RRBIs does not significantly differ across populations during the first year of life (Baranek, 1999; Osterling et al., 2002). The frequency and severity of RRBIs is reported to be significantly different by two years of age (Lord, 1995; Matson et al., 2009). However, it should be noted that typically developing children do continue to exhibit some RRBIs even after their second birthday (Richler et al., 2007).

With regard to the current results, this pattern of manifestation could result in decreasing score elevations for atypically developing children as their age increases, decreasing the score necessary to differentiate between ASD and atypical development. Further, the general variability of development

may also lead to the need for a higher cutoff score for younger participants relative to older participant groups. For example, researchers have noted significant variation in the development of language, communication, and social skills for toddlers during their first year of life which may complicate differential diagnosis (Landa, Holman, & Garrett-Mayer, 2007; Maestro et al., 2005). However participants closer to 24 months of age may be more readily distinguished from their peers as language and social skills impairments become more discrepant (Baghdadliet al., 2003; Charman et al., 1997; De Giacomo & Fombonne, 1998; Landa & Garrett-Mayer, 2006; Sullivan et al. Landa, 2007; Wetherby et al., 2004).

From a broader standpoint, this research continues to demonstrate the ability of clinicians and researchers to accurately identify ASD in infants and toddlers (Baird et al., 2001; Baron-Cohen et al., 1996; Charman & Baird, 2002; Eaves & Ho, 2004; Howlin & Moore, 1997; Wetherby et al., 2007). Previously, researchers have suggested that developers of diagnostic screeners should strive for diagnostic sensitivity between 70 and 80% (Glascoe, 2005). The diagnostic sensitivity and specificity for each of the selected cutoff scores from the current analyses demonstrate the utility of the *BISCUIT-Part 1* as a screening measure for ASD despite recent diagnostic changes. The identification of new cutoff scores for the *BISCUIT-Part 1* utilizing a large normative population with *DSM-5* diagnoses provided a scoring approach that is in line with the current diagnostic criteria (APA, 2013).

Study Two

Statistical Analyses

The second study focused on the identification of cutoff scores for the *BISCUIT-Part 2* that best discriminate the severity of comorbid psychopathology symptoms for each diagnostic group (i.e., ASD and atypical development). Although previous calculations of the cutoff scores for the *BISCUIT-Part 2* did not differentiate between ASD diagnostic categories, they were calculated based upon the symptom manifestation observed within individuals with PDD-NOS and autistic disorder. Researchers have previously reported that recent changes to the ASD diagnostic category may cause some individuals previously diagnosed with ASD to no longer meet diagnostic criteria (Gibbs et al., 2012; Matson et al., 2012a, 2012b; Mattila et al., 2011; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012). New cutoff scores were calculated based upon the symptom manifestation observed for individuals meeting *DSM-5* criteria for a diagnosis of ASD. Cutoff scores were calculated for the *BISCUIT-Part 2* total score and each factor (n=5) for the total sample and each age group (n=3) across each diagnostic category. Age-based scoring procedures were included to capture differences in symptom manifestation across developmental periods and to remain consistent with those procedures previously used for the development of the *BISCUIT*.

The standard deviation from the central tendency method was used to calculate cutoff scores for the *BISCUIT-Part 2*. This method has previously been employed to develop scores for the adolescent version of the *BISCUIT*, the *ASD-CC* (Thorson & Matson, 2012), as well as previous versions of the *BISCUIT-Part 2* (Horovitz & Matson, 2013a; Matson et al., 2009c). The standard deviation from the central tendency method has also been used in the development of cutoff scores for other clinical measures such as the *Psychological Assessment Tool 2.0* (Pai et al., 2008).

Following the correction for outliers, the mean and standard deviation for the total score and each of the five factors of the *BISCUIT-Part 2* were calculated for each diagnostic category and their respective age groups. Cutoff scores were then calculated for each age group within each diagnostic

category. Given that scoring of the *BISCUIT-Part 2* utilizes whole numbers, the calculated means, standard deviations, and cutoff scores were rounded to the nearest whole number when necessary. This process created a total of six cutoff scores for each of the six groups (e.g., ASD, 17-23 months; atypical development, 17-23 months). Previously researchers have indicated that scores greater than two standard deviations from the mean are of clinical significance (Jacobson & Traux, 1991). Based upon this criterion, scores identified as being more than two standard deviations above the mean were identified as falling in the *severe impairment* range. Scores between one and two standard deviations above the respective group mean were classified as indicating *moderate impairment*. Finally, scores within one standard deviation of the mean were identified and described as indicating *no/minimal impairment*.

Results

Total sample. Initially, cutoff scores were calculated for each diagnostic category regardless of participant age. For the ASD diagnostic category, the mean level of symptom endorsement was 32.49, with a standard deviation of 20.85. Based upon the procedures outline above, a total score on the *BISCUIT-Part 2* less than or equal to 53 for participants with ASD was considered to be indicative of *no/minimal impairment*. A total score greater than or equal to 54 and less than or equal to 74 was classified as indicating *moderate impairment*. Finally, a total score greater than or equal to 75 was classified as indicating *severe impairment*. The cutoff score ranges each of the *BISCUIT-Part 2* factors are presented in Table 7.

Table 7
BISCUIT-Part 2 Cutoff Scores for the Total ASD Sample

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total	32.49	20.85	0-53	54-74	≥75
Tantrum/conduct behavior	10.96	7.83	0-19	20-27	≥28
Inattention/impulsivity	8.88	5.59	0-14	15-20	≥21
Avoidance behavior	2.72	3.04	0-6	7-9	≥10
Anxiety/repetitive behavior	4.21	3.36	0-8	9-11	≥12
Eating/sleep problems	2.86	1.79	0-5	6-7	8

These procedures were also carried out for the atypically developing diagnostic category, regardless of participant age. The average value of symptom endorsement for this group was 8.83, with a standard deviation of 11.92. A score less than or equal to 21 was classified as indicating *no/minimal impairment*. A score greater than or equal to 22 and less than or equal to 33 was classified as indicating *moderate impairment*, while a total score greater than or equal to 34 was classified as indicating *severe impairment*. The cutoff scores ranges for each *BISCUIT-Part 2* factor appear in Table 8.

Table 8
BISCUIT-Part 2 Total Sample Cutoff Scores for Atypically Developing Participants

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	8.83	11.92	0-21	22-33	≥34
Tantrum/conduct behavior	3.23	4.76	0-8	9-13	≥14
Inattention/impulsivity	2.63	3.42	0-6	7-10	≥11
Avoidance behavior	.59	1.39	0-2	3-4	≥5
Anxiety/repetitive behavior	.97	1.72	0-3	4-5	≥6
Eating/sleep problems	.69	1.21	0-2	3-4	≥7

Participants 17 to 23 months of age. Previously, researchers have observed that rates of comorbid psychopathology increase across time (Konst & Matson, 2013). In order to account for the impact of age and development on the manifestation of symptoms of comorbid psychopathology, cutoff scores were calculated for each diagnostic category across each age group. This age-based differentiation was also carried out to be consistent with previous versions of the *BISCUIT-Part 2*. Initially, cutoff scores were calculated for the *BISCUIT-Part 2* total score. For the ASD diagnostic group, the mean level of total symptom endorsement was 29.58, while the standard deviation was 18.80. A total score less than or equal to 48 was considered to indicate *no/minimal impairment*. A score greater than 48 but less than or equal to 67 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 68 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 2* factors were calculated utilizing the same methodology and are presented in Table 9.

Table 9

BISCUIT-Part 2 Cutoff Scores for ASD ParticipantsASD 17-23 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	29.58	18.80	0-48	49-67	≥68
Tantrum/conduct behavior	9.72	7.39	0-17	18-25	≥26
Inattention/impulsivity	8.42	5.39	0-14	15-19	≥20
Avoidance behavior	2.54	2.75	0-5	6-8	≥9
Anxiety/repetitive behavior	3.85	2.96	0-7	8-10	≥11
Eating/sleep problems	2.91	1.85	0-5	6-7	8

These procedures were then repeated based upon the symptom endorsement observed for participants 17-23 months of age in the atypically developing group. The average total score for this group was 7.95, while the standard deviation was 10.61. Based upon the standard deviation from the central tendency method, a score less than or equal to 19 was classified as indicating *no/minimal impairment*. A total score greater than 19, but less than or equal to 29, was classified as indicating *moderate impairment*. Finally, a total score greater than or equal to 30 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 2* factors are presented in Table 10.

Table 10
BISCUIT-Part 2 Cutoff Scores for Atypically Developing Participants17-23 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	7.95	10.61	0-19	20-29	≥30
Tantrum/conduct behavior	2.92	4.38	0-7	8-9	≥10
Inattention/impulsivity	2.33	3.07	0-5	6-8	≥9
Avoidance behavior	0.49	1.20	0-2	2-3	≥4
Anxiety/repetitive behavior	0.91	1.54	0-2	3-4	≥5
Eating/sleep problems	0.64	1.10	0-2	3-4	≥5

Participants 24 to 30 months of age. Initially cutoff scores were calculated for the *BISCUIT-Part 2* total score. For the ASD diagnostic category, the average level of total symptom endorsement was 32.31 with a standard deviation of 21.49. Based upon the procedures and criteria outlined above a total score less than or equal to 54 was classified as indicating *no/minimal impairment*. A score greater than 54 and less than or equal to 75 was classified as representing *moderate impairment*. Lastly, a total score greater than or equal to 76 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 2* factors are presented in Table 11.

Table 11
BISCUIT-Part 2 Cutoff Scores for ASD Participants 24-30 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	32.31	21.49	0-54	55-75	≥76
Tantrum/conduct behavior	10.86	7.72	0-19	20-26	≥27
Inattention/impulsivity	8.83	5.57	0-14	15-20	≥21
Avoidance behavior	2.85	2.97	0-6	7-9	≥10
Anxiety/repetitive behavior	4.30	3.52	0-8	9-11	≥12
Eating/sleep problems	2.83	1.79	0-5	6-7	8

In a similar manner, cutoff scores were calculated for the *BISCUIT-Part 2* total score for those participants identified as atypically developing without an ASD diagnosis. Calculations were based upon the mean level of total symptom endorsement for this age group (M = 9.22) and the observed standard deviation (i.e., 12.34). A total score less than or equal to 22 was considered to represent *no/minimal impairment*. A score greater than 22 and less than or equal to 34 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 35 was classified as indicating *severe impairment*. The cutoff score ranges for each *BISCUIT-Part 2* factor are presented in Table 12.

Table 12
BISCUIT-Part 2 Cutoff Scores for Atypically Developing Participants 24-30 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	9.22	12.34	0-22	23-34	≥35
Tantrum/conduct behavior	3.34	4.85	0-8	9-13	≥14
Inattention/impulsivity	2.76	3.53	0-6	7-10	≥11
Avoidance behavior	0.62	1.44	0-2	3-4	≥5
Anxiety/repetitive behavior	0.99	1.79	0-3	4-5	≥6
Eating/sleep problems	0.71	1.26	0-2	2-3	≥4

Participants 31 to 37 months of age. Cutoff scores were calculated for the *BISCUIT-Part 2* total score for those participants who were between 31 and 37 months of age and diagnosed with ASD. Cutoff score calculations were based upon the mean level of total symptom endorsement for this age group (M = 36.14) and the standard deviation (i.e., 21.18). Based upon the procedures and criteria outlined above, a total score less than or equal to 57 was identified as representing *no/minimal impairment*. A score greater than 57 and less than or equal to 79 was classified as indicating *moderate*

impairment. Lastly, a total score greater than or equal to 80 was identified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 2* factors are presented in Table 13.

Table 13
BISCUIT-Part 2 Cutoff Scores for ASD Participants 31-37 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	36.14	21.18	0-57	58-79	≥80
Tantrum/conduct behavior	12.69	8.37	0-21	22-29	≥30
Inattention/impulsivity	9.54	5.87	0-15	16-21	≥22
Avoidance behavior	2.63	3.54	0-6	7-10	≥11
Anxiety/repetitive behavior	4.46	3.40	0-8	9-11	≥12
Eating/sleep problems	2.86	1.75	0-5	6-7	8

Lastly, these analyses were then carried out for those participants identified as atypically developing without an ASD diagnosis. Calculations were based upon the mean level of total symptom endorsement (M = 9.46) and standard deviation (i.e., 13.05). A total score less than or equal to 23 was considered to represent *no/minimal impairment*. Scores greater than 23 and less than or equal to 36 are suggested to indicate *moderate impairment*. Lastly, a total score greater than or equal to 37 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 2* factors are presented in Table 14.

Table 14
BISCUIT-Part 2 Cutoff Scores for Atypically Developing Participants 31-37 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	9.46	13.05	0-23	24-36	≥37
Tantrum/conduct behavior	3.58	5.18	0-9	10-14	≥15
Inattention/impulsivity	2.87	3.75	0-7	8-10	≥11
Avoidance behavior	0.68	1.57	0-2	3-4	≥5
Anxiety/repetitive behavior	1.03	1.84	0-3	4-5	≥6
Eating/sleep problems	0.76	1.25	0-2	3-4	≥5

Discussion

Researchers have previously observed elevated rates of comorbid psychological symptoms in ASD populations when compared to their atypically developing peers (Konst & Matson, 2014). Increased rates of comorbid psychopathology are often observed in ASD populations regardless of age and level of functioning (Billstedt, 2000; Hofvander et al., 2009; LoVullo & Matson, 2009; Matson & Nebel-Schwalm, 2007a; Simonoff et al., 2008). However, direct comparisons of the manifestation of comorbid symptoms within ASD populations have identified significantly greater rates of comorbid symptoms in those with autistic disorder in comparison to those with PDD-NOS (Matson et al., 2009c). Given the difference in symptom manifestation within the ASD category and between those with ASD and their atypically developing peers, it was hypothesized that the cutoff scores for the *BISCUIT-Part 2* would increase for each diagnostic group following the *DSM-5* diagnostic revisions.

The range of cutoff scores for the *BISCUIT-Part 2* total score, relative to each age group, is presented in Table 15. On average, the cutoff scores identified for each age group within the current analysis were 11 points higher than those identified previously for individuals with ASD. With regard to the individual atypically developing age groups, those total scores identified by the current analysis were an average of 13 points higher than those previously identified for the *BISCUIT-Part 2* (Horovitz & Matson, 2013a). These results directly support the hypothesized increase in cutoff scores necessary for the *BISCUIT-Part 2*. With regard to specific subscales, the largest average change was a three-point increase for the atypically developing age groups observed on the anxiety subscale. For the ASD population, the largest average change in cutoff score was an increase of three points on the tantrum/conduct behavior subscale. The observed increase in cutoff score ranges is consistent with previous research suggesting that the *DSM-5* diagnostic changes identify individuals with greater levels of impairment (Matson, Belva et al., 2012; Worley & Matson, 2012).

Table 15
Selected Cutoff scores for the BISCUIT-Part 2 Total Score

Diagnostic group	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
ASD					
Total Sample	32.49	20.85	0-53	54-74	≥75
17-23 months	29.58	18.80	0-48	49-67	≥68
24-30 months	32.31	21.49	0-54	55-75	≥76
31-37 months	36.14	21.18	0-57	58-79	≥80
Atypical development					
Total sample	8.83	11.92	0-21	22-33	≥34
17-23 months	7.95	10.61	0-19	20-29	≥30
24-30 months	9.22	12.34	0-22	23-34	≥35
31-37 months	9.46	13.05	0-23	24-36	≥37

It was hypothesized that the further separation of diagnostic groups into age groups would be necessary in order to account for developmental differences in the manifestation of comorbid symptoms for individuals with ASD. Konst and Matson (2013) demonstrated that the exhibition of comorbid symptoms increased significantly for individuals with ASD across two administrations of the *BISCUIT-Part 2*. The authors did not observe the same pattern of change in an atypically developing control group. Based upon previous research, it was also hypothesized that the cutoff scores for those with ASD would increase with age. Both hypotheses were confirmed by the results of Study Two. With regard to total score, the cutoff score suggesting severe impairment was 12 points higher for the 31-37 month age group when compared to the cutoff score selected for the 17-23 month age group. A similar pattern was observed for the identified cutoff scores for four of the five subscales of the *BISCUIT-Part 2*. The cutoff scores identified for the eating/sleep problems subscale did not differ for any age group. However, this may be largely due to a ceiling effect. The eating/sleep problems subscale contains only four items, with a maximum score of eight points. A score of eight was identified as the maximum cutoff score for each ASD age group. Given the observed trend of symptom manifestation and the prevalence of eating and sleep problems observed in ASD populations (Ledford & Gast, 2006; Schreck, Williams, & Smith, 2004), future researchers should evaluate the addition of extra items to further assess these impairments. The

addition of more items may help capture a wider range of symptom manifestation and differentiate symptom severity.

Within the atypically developing group, the total score cutoffs increased across each age cohort. Specifically, the total score cutoff indicating severe impairment was seven points less for the 17-23 month age group than the cutoff score identified for the 31-37 month age group. However, the observed increase in cutoff scores was not evenly dispersed across the *BISCUIT-Part 2* subscales. A total of five points separated the youngest and oldest age group cutoff scores representing severe impairment for the tantrum/conduct behavior subscale. The identified cutoff scores for three of five subscales continued to increase across age groups, though to a lesser degree. This trend of increasing severity of comorbid symptoms is similar to the trend observed in those cutoff scores identified for previous atypically developing populations on the *BISCUIT-Part 2* (Horovitz & Matson, 2013a). However, the observed increase in the manifestation of comorbid symptoms across age groups is not consistent with previously observed trends in atypically developing populations (Konst & Matson, 2013). The authors reported that the manifestation of comorbid symptoms did not significantly change across two administrations of the *BISCUIT-Part 2*. Although the observed increases in cutoff scores from the current analysis are unlikely to be significantly different statistically, they do suggest an overall trend of increasing symptom severity.

It is important to also note that there are significant differences between the two studies which impede a direct comparison. First, the current sample is larger than that used by Konst and Matson (N = 205; 2013). Secondly, their research involved two assessments of the same person across time, not different individuals being assessed at various points in development. Finally, the previous researchers combined the ASD diagnostic groups (i.e., PDD-NOS and autistic disorder), whereas the current analysis included the direct implementation of the *DSM-5* diagnostic criteria. Although the identification of contributing factors is beyond the scope of the current analysis, it is possible that changes in diagnostic criteria and the ASD categories are associated with the observed shift in symptom manifestation. Mayes and colleagues (2013) suggested that as much as 73% of individuals with a previous PDD-NOS diagnosis

would no longer meet *DSM-5* criteria. The implementation of *DSM-5* changes may have led to some individuals who previously met criteria for PDD-NOS to be placed in the atypical development group. This factor may have contributed to the observed differences in symptom manifestation relative to previous research. Currently, there is limited longitudinal research investigating comorbid symptoms in atypically developing toddlers. Future researchers may wish to examine comorbid symptoms in infants and toddlers to identify additional symptoms that may have a negative impact on an individual's quality of life.

Study Three

Statistical Analyses

Previously, the cutoff scores for the *BISCUIT-Part 3* did not differentiate between ASD diagnostic groups. However, the cutoff scores for the ASD group were calculated based upon the symptom manifestation observed in individuals with PDD-NOS and autistic disorder. Individuals who previously met criteria for an ASD diagnosis may no longer meet *DSM-5* criteria due to the changes to the ASD diagnostic category and diagnostic criteria (Gibbs et al., 2012; Mattila et al., 2011; Matson et al., 2012a, 2012b; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012). Study Three focused on identifying cutoff scores for the *BISCUIT-Part 3* that best discriminated the severity of challenging behaviors within each diagnostic group (i.e., ASD and atypical development). New cutoff scores were created for the total score of the *BISCUIT-Part 3* and its three factors for each age group within each diagnostic category. Age-based scoring procedures were included to remain consistent with previous scoring procedures and to capture the effects of development on the expression of challenging behaviors. Cutoff scores for the *BISCUIT-Part 3* were identified based upon the standard deviation from the central tendency method previously described in Study Two. This method was also employed in earlier research which created the previous cutoff scores for the *BISCUIT-Part 3* (Horovitz & Matson, 2013b; Matson et al., 2010).

The third component of the *BISCUIT* assessment battery is used to evaluate challenging behaviors frequently identified in atypically developing populations. For the purposes of the current analysis, the means and standard deviations for the total score and for each of the *BISCUIT-Part 3* factors (i.e., aggressive/disruptive behavior, stereotypic behavior, and SIB) were calculated for each individual diagnostic age group. This created a total of six groups for which cutoff scores were calculated for the total score and each individual factor (n=3). Calculated means, standard deviations, and cutoff scores were rounded to the nearest whole number when necessary. Scores greater than two standard deviations from the mean were considered to be of clinical significance (Jacobson & Traux, 1991). Scores identified

as being within one standard deviation of the calculated group mean were identified as falling in the *no/minimal impairment* range. A classification of *moderate impairment* was ascribed to scores between one and two standard deviations above the respective group mean. Finally, those scores more than two standard deviations above the calculated mean were classified as indicating *severe impairment*.

Results

Total sample. First, cutoff scores were calculated for each diagnostic category regardless of participant age. For the ASD diagnostic category, the mean level of challenging behavior symptom endorsement was 8.39, while the standard deviation was 7.16. In accordance with the standard deviation from central tendency method, a total score less than or equal to 16 was considered to be indicative of *minimal impairment*. A total score greater than or equal to 17 and less than or equal to 23 was classified as indicating *moderate impairment*. Finally, a total score greater than or equal to 24 was classified as indicating *severe impairment* for individuals diagnosed with ASD. The cutoff score ranges for the total ASD sample for each of the *BISCUIT-Part 3* factors are presented in Table 15.

Table 15
Total ASD Sample BISCUIT-Part 3 Cutoff Scores

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	8.39	7.16	0-16	17-23	≥24
Aggressive/destructive behaviors	5.10	4.67	0-10	11-14	≥15
Stereotypies	1.39	1.75	0-3	4-5	6
SIB	0.71	1.03	0-2	3	4

These analyses were also carried out for the atypical development group. The average total score on the *BISCUIT-Part 3* for this group, regardless of participant age, was 2.32, while the standard deviation was 4.22. Based upon the criteria outlined above, a total score less than or equal to seven was identified as representing *no/minimal impairment*. A total score greater than or equal to eight, but less than or equal to 11 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 12 was classified as indicating *severe impairment*. The cutoff score ranges for the total atypical development sample for each of the *BISCUIT-Part 3* factors are presented in Table 16.

Table 16
Total Sample BISCUIT-Part 3 Cutoff Scores for Atypical Development

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	2.32	4.22	0-7	8-11	≥12
Aggressive/destructive behaviors	1.54	2.81	0-4	5-7	≥8
Stereotypies	0.42	1.01	0-1	2-3	≥4
SIB	0.20	0.58	0-1	2-3	4

Participants 17 to 23 months of age. Initially cutoff scores were calculated for the *BISCUIT-Part 3* total score. For the ASD diagnostic category, the mean level of total symptom endorsement was 7.09, while the standard deviation was 6.57. Based upon the procedures and criteria outlined above, a total score less than or equal to 14 was considered to fall within the *no/minimal impairment* range. A score greater than 14 and less than or equal to 20 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 21 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 3* factors are displayed in Table 17.

Table 17
BISCUIT-Part 3 Cutoff Scores for ASD Participants 17-23 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	7.09	6.57	0-14	15-20	≥21
Aggressive/destructive behaviors	4.22	4.44	0-9	10-13	≥14
Stereotypies	0.93	1.46	0-2	3-4	≥5
SIB	0.72	1.04	0-2	3	4

These procedures were then carried out for the total score from the *BISCUIT-Part 3* for those participants identified as atypically developing in the 17 to 23 month age range. Calculations were based upon the mean level of total symptom endorsement for this age group (M = 2.13) and the observed standard deviation (i.e., 3.97). A total score less than or equal to six was identified as representing *no/minimal impairment*. A score greater than or equal to seven and less than or equal to 10 was classified as indicating *moderate impairment*. Finally, a total score greater than or equal to 11 was suggested to

indicate *severe impairment*. The cutoff score ranges for each of the three *BISCUIT-Part 3* factors were calculated utilizing identical methodology and are presented in Table 18.

Table 18
BISCUIT-Part 3 Cutoff Scores for Atypically Developing Participants 17-23 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	2.13	3.97	0-6	7-10	≥11
Aggressive/destructive behaviors	1.46	2.72	0-4	5-7	≥8
Stereotypies	0.34	0.91	0-1	2-3	≥4
SIB	0.21	0.58	0-1	2-3	4

Participants 24 to 30 months of age. Identical procedures were repeated for those individuals 24 to 30 months of age with an ASD diagnosis. For the ASD diagnostic category, the mean level of total symptom endorsement was 8.60, with a standard deviation of 7.27. A total score less than or equal to 16 was suggested to indicate *no/minimal impairment*. A score greater than or equal to 17 and less than or equal to 23 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 24 was identified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 3* factors are presented in Table 19.

Table 19
BISCUIT-Part 3 Cutoff Scores for ASD Participants 24-30 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	8.60	7.27	0-16	17-23	≥24
Aggressive/destructive behaviors	5.29	4.71	0-10	11-15	≥16
Stereotypies	1.40	1.79	0-3	4-5	6
SIB	0.70	1.04	0-2	3	4

These procedures were then carried out for those participants identified as atypically developing in the 24 to 30 month age range. The average total score of the *BISCUIT-Part 3* for this age group (M = 2.39) and the standard deviation (i.e., 4.31) were used to calculate cutoff score ranges. A total score less than or equal to seven was identified as representing *no/minimal impairment*. A score greater than or equal to eight and less than or equal to 11 was classified as indicating *moderate impairment*. Lastly, a

total score greater than or equal to 12 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 3* factors are presented in Table 20.

Table 20
BISCUIT-Part 3 Cutoff Scores for Atypically Developing Participants 24-30 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	2.39	4.31	0-7	8-11	≥12
Aggressive/destructive behaviors	1.56	2.82	0-4	5-7	≥8
Stereotypies	0.45	1.04	0-1	2-3	≥4
SIB	0.21	0.61	0-1	2-3	4

Participants 31 to 37 months of age. Finally, these procedures were repeated for those individuals 31 to 37 months of age. For the ASD diagnostic category, the mean level of total symptom endorsement was 9.46, with a standard deviation of 7.42. Based upon the standard deviation from central tendency method, a total score less than or equal to 17 was identified as representing *no/minimal impairment*. A score greater than or equal to 18 and less than or equal to 24 was classified as indicating *moderate impairment*. Lastly, a total score greater than or equal to 25 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 3* factors are displayed in Table 21.

Table 21
BISCUIT-Part 3 Cutoff Scores for ASD Participants 31-37 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	9.46	7.42	0-17	18-24	≥25
Aggressive/destructive behaviors	5.72	4.76	0-11	12-15	≥16
Stereotypies	1.90	1.84	0-4	5	6
SIB	0.73	1.01	0-2	3	4

Lastly, these procedures were carried out for those participants who were 31 to 37 months of age and identified as atypically developing. The average total score on the *BISCUIT-Part 3* for this age group (M = 2.51) and the standard deviation (i.e., 4.41) were used to calculate cutoff scores. A total score less than or equal to seven was identified as representing *no/minimal impairment*. A score greater than or equal to eight and less than or equal to 11 was classified as indicating *moderate impairment*. Finally, a

total score greater than or equal to 12 was classified as indicating *severe impairment*. The cutoff score ranges for each of the *BISCUIT-Part 3* factors are presented in Table 22.

Table 22
BISCUIT-Part 3 Cutoff Scores for Atypically Developing Participants 24-30 Months of Age

	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
Total score	2.51	4.41	0-7	8-11	≥12
Aggressive/destructive behaviors	1.67	2.96	0-5	6-8	≥9
Stereotypies	0.52	1.10	0-2	3-4	≥5
SIB	0.16	0.46	0-1	2-3	4

Discussion

Challenging behaviors have been previously identified as one of the most prevalent reasons for psychological referrals (Robb, 2010). Lecavalier (2006) noted that the presence of ASD was associated with elevated rates of challenging behaviors. In comparison to typically and atypically developing peers, individuals with ASD exhibit significantly greater rates of challenging behaviors (Konst et al., 2013a; Matson, 2009; Tureck et al., 2013). However, differences within previous diagnostic groups have suggested that the severity of ASD symptom manifestation may moderate the exhibition of challenging behaviors. Sipes and colleagues (2011) reported that children with autistic disorder exhibited significantly greater amounts of tantrum behaviors compared to peers with PDD-NOS and those identified as atypically developing. Additional researchers have reported that regardless of primary diagnosis, elevated levels of ASD symptoms were positively associated with the presence of challenging behaviors (Konst et al., 2013).

Researchers have previously reported an increase in the expression of challenging behaviors as children with ASD age (Charman et al., 2005). Lainhart (1999) hypothesized that an increase in the exhibition of challenging behaviors may be associated with increased frustration due to communication deficits. Other researchers have suggested that social skills deficits, increased demands, and the establishment of punishment and reinforcement procedures (e.g., negative reinforcement) may also alter the expression of challenging behaviors (Cheng et al., 2009; Gutierrez et al., 2009; Lacroix et al., 2009;

Matson, Neal et al., 2010; Matson & Sipes, 2010). Based upon this research, it was hypothesized that cutoff scores would increase with participant age for those participants diagnosed with ASD.

Table 23 depicts the identified cutoff score ranges for the *BISCUIT-Part 3* total score for each diagnostic group, as well as each age group. The results of Study Three are consistent with the hypothesis that cutoff scores would increase for individuals with ASD relative to participant age. The cutoff score for the 31-37 month age group total score was four points higher than that observed for the 17-23 month age group. This pattern was also consistent across two of the three *BISCUIT-Part 3* subscales. As can be seen in Tables 17, 19, and 21, the identified cutoff scores increased as participant age increased for the aggressive/destructive behavior and stereotypic behavior subscales. These results are consistent with previous research demonstrating an increase in the manifestation of challenging behavior across the lifespan (Charman et al., 2005; Horovitz & Matson, 2013b; Matson et al., 2010). However, it should be noted that the cutoff scores identified for the SIB subscale remained consistent for participants with ASD regardless of age. This observation may most likely be attributed to a restriction of range. The SIB subscale contains only two items with a maximum score of four points. Given this restriction of range, it is possible that the observed range of symptom severity is not completely representative of the SIB severity observed in the ASD population. However, the mean level of symptom endorsement across each age group (.70 - .73) does not suggest that this was a significant factor in the current analyses.

Table 23
BISCUIT-Part 3 Total Score Ranges

Diagnostic group	Mean	Standard deviation	No/minimal impairment	Moderate impairment	Severe impairment
ASD					
Total sample	8.39	7.16	0-16	17-23	≥24
17-23 months	7.09	6.57	0-14	15-20	≥21
24-30 months	8.60	7.27	0-16	17-23	≥24
31-37 months	9.46	7.42	0-17	18-24	≥25
Atypical development					
Total sample	2.32	4.22	0-7	8-11	≥12
17-23 months	2.13	3.97	0-6	7-10	≥11
24-30 months	2.39	4.31	0-7	8-11	≥12
31-37 months	2.51	4.41	0-7	8-11	≥12

Recent investigations of the influence of age upon the manifestation of challenging behaviors in atypically developing populations have suggested that these symptoms remain relatively stable across time. Some researchers have reported that challenging behaviors such as stereotypic behaviors actually decrease with age in typically and atypically developing toddlers (Evans et al., 1997; Thelen, 1979). Given the observed trend in previous research, it was hypothesized that the cutoff scores for atypically developing toddlers identified by Study Three would not increase across age groups. A visual analysis of the score ranges depicted in Table 23 confirmed this hypothesis. Across the three age groups the total score cutoff changed by a total of one point, with the 24-30 and 31-37 month age groups demonstrating no change. This pattern was upheld across each age group for each subscale of the *BISCUIT-Part 3*. Despite the relative lack of change in cutoff scores across age groups, it is suggested that the use of age-based cutoff scores be maintained to be consistent with the remaining portions of the *BISCUIT* assessment battery.

The cutoff scores identified for participants with ASD remained relatively consistent with the most recent revision of the *BISCUIT-Part 3* cutoff scores (Horovitz & Matson, 2013b). This was not the case for the atypical development group. Specifically, the total score cutoff identified by the current analysis for the *BISCUIT-Part 3* increased by a total of five points. Further, the average cutoff score identified for each subscale increased by three points relative to those identified by Horovitz and Matson (2013b). These results are consistent with the concern raised by previous researchers (Matson, Belva, et al., 2012; Worley & Matson, 2012). Specifically, these researchers reported that those individuals with a *DSM-IV-TR* diagnosis of ASD that did not meet *DSM-5* diagnostic criteria exhibited significantly greater symptoms than their atypically developing peers without an ASD diagnosis. With regard to the current analysis, the addition of individuals who no longer met criteria for ASD may have contributed to the observed increase in the expression of challenging behaviors for the atypically developing group.

Conclusion

Stone and colleagues (1999) emphasized that the delivery of efficacious interventions is contingent upon the reliable identification of ASD. With regard to both identification and intervention, researchers have continuously placed a growing emphasis on the term *early*. This trend has stemmed from an accumulation of research demonstrating the decreased efficacy of the same intervention when treatment delivery is delayed by as little as two years (Fenske et al., 1985; Lovaas & Smith, 1988). With respect to assessment, an emphasis on diagnostic sensitivity and specificity has had a significant impact on the development of measures used to identify ASD. Most recently, the *DSM-5* was published and included significant revisions to the ASD diagnostic category (APA, 2013; Gibbs et al., 2012; Frazier et al., 2012; Tsai & Ghaziuddin, 2013). Specifically, the diagnostic categories and criteria for diagnosis were modified in an attempt to increase diagnostic sensitivity and specificity (APA, 2011, 2013; Gibbs et al., 2012; Grzadzinski et al., 2013).

One measure designed to assess infants and toddlers for ASD is the *BISCUIT* assessment battery. This battery includes three separate measures used to assess for ASD symptomology, comorbid psychopathology, and challenging behaviors. Researchers have previously demonstrated the psychometric properties of each *BISCUIT* component using a large normative sample that included individuals with ASD (Horovitz & Matson, 2013a, 2013b, 2014; Matson et al., 2009b; Matson, Fodstad et al., 2010). Given the significance of the changes to the ASD category appearing in the *DSM-5*, it is imperative to update ASD assessments. The current analyses sought to update the scoring procedures for each component of the *BISCUIT* assessment battery using a large sample of infants and toddlers.

The results of Study One advanced previous research of the *BISCUIT-Part 1* in multiple ways. The updated scoring procedures not only increased the diagnostic sensitivity and specificity of the *BISCUIT-Part 1*, but they also demonstrated the continued importance of age-based scoring procedures. Relative to recent research by Horovitz and Matson (2014), the cutoff scores identified in Study One resulted in an increase in diagnostic sensitivity and specificity for each cutoff score across each age

group. The use of age based cutoff scores also resulted in greater PPV and NPV for each age group relative to the PPV and NPV calculated for the total sample. Further, the current results also demonstrated the utility of the *BISCUIT-Part 1* as an assessment measure of ASD in light of recent diagnostic changes. On average, the cutoff scores identified in Study One provided greater diagnostic sensitivity (94%) and specificity (87%) when compared to other early identification measures. For example, Wetherby and colleagues (2003) reported that the *ITC* has a sensitivity of 87% and specificity of 75%. Additional measures commonly used for the early identification of ASD include the *STAT* (sensitivity, 93% and specificity, 83%; Stone et al., 2000) and *M-CHAT* (sensitivity, 74% and specificity, 87%; Matson et al., 2009b). Given the importance of early identification and intervention, the strong psychometric properties of the *BISCUIT-Part 1* make it a valuable component of a diagnostic evaluation. With these scoring updates, the *BISCUIT-Part 1* is one of the few early identification measures with research demonstrating its consistency with the *DSM-5*.

Additional analyses also updated the scoring procedures for the *BISCUIT-Parts 2* and *3*. Although these measures did not previously distinguish between PDD-NOS and autistic disorder, the normative sample used to create cutoff scores for the ASD category included participants with a PDD-NOS or an autistic disorder diagnosis. Given the observed impact of *DSM-5* changes on the PDD-NOS population (Gibbs et al., 2012; Mattila et al., 2011; Matson et al., 2012a, b; Mayes et al., 2013; McPartland et al., 2012; Worley & Matson, 2012), it was necessary to analyze both measures to update the cutoff scores used. The total score cutoff scores identified for the *BISCUIT-Part 2* increased by more than 10 points for each diagnostic category. With regard to specific subscales, the largest average change observed for the ASD population was the tantrum/conduct behavior subscale. With regard to the atypical development group, the largest average increase observed was related to the anxiety subscale. The *BISCUIT-Part 3* cutoff scores identified for the ASD category increased but were relatively consistent with those observed by Horovitz and Matson (2013b). However, the cutoff scores for the atypical development group increased relative to previously identified cutoff scores. The accurate identification of

symptoms of comorbid psychopathology and challenging behaviors is a pivotal component of an evaluation and is necessary for the delivery of efficacious treatment (Gold, 1993; Kazdin, 1993; Leyfer et al., 2006; Matson & Nebel-Schwalem, 2007a; Muris et al., 1998; Simonoff et al., 2008). Aside from the symptom manifestation observed for the atypically developing group on the *BISCUIT-Part 3*, the use of age-based scoring procedures increased diagnostic accuracy for each component of the *BISCUIT* battery. Across each study, those cutoff scores identified without regard for participant age were not appropriate for more than one age group. Based upon these results, the continued use of age-based cutoff scores is appropriate and may also serve to enhance scale interpretation and the development of individualized interventions.

Future research should continue to monitor the impact of changes appearing in the *DSM-5* and how these changes influence individuals no longer meeting criteria for ASD. This should include monitoring symptoms of comorbid psychopathology and challenging behaviors. Future researchers should continue to assess comorbid symptoms and challenging behaviors in this population to increase our knowledge of the early expression of these symptoms. Increased understanding of the emergence of these symptoms would aid in the development of early identification measures and inform treatment approaches. The results of Study Two suggested that further research may be necessary for the eating/sleep problems subscale of the *BISCUIT-Part 2*. Specifically, each ASD age group elevated the subscale to the maximum level possible given the current amount of items for this subscale. This restriction of range may have limited differentiation across age groups and may fail to capture the true variation in severity of eating and sleeping problems during assessment. The importance of this research is underscored by the prevalence of sleeping and eating problems in ASD populations (Ledford & Gast, 2006; Schreck, Williams, & Smith, 2004). A similar situation was identified by Study Three. The range of symptom endorsement for the SIB subscale reached the maximum possible score for each age group within the ASD category. Given the negative impact of SIB and its prevalence in ASD populations

(Ando & Yoshimura, 1979; Janicki & Jacobson, 1983), future researchers should consider the identification of additional items to be included in the *BISCUIT-Part 3*.

Additional research on early identification in general and the *BISCUIT* battery specifically may include the exploration of additional factors that may influence diagnosis. Aside from age and developmental progression, factors such as ethnicity and gender have also been suggested to be potential factors that influence the early identification of ASD (Mandell et al., 2002). Researchers have reported that relative to caucasian Americans, ethnic minorities are less likely to be diagnosed with ASD (Kogan et al., 2008; Palmer et al., 2010; Rosenberg et al., 2009). A review of the demographic information presented in Table 1 does not immediately suggest that this was the case for the current analyses. Specifically, the demographic information for individuals meeting criteria for ASD was consistent with recent information from the US Census Bureau (2013). As a state, 32% of Louisiana residents identified themselves as African-American, 64% as caucasian, 4.7% as hispanic, and 1.5% as Asian. However, it is important to note that the nature of the services provided by EarlySteps is unique and different from the populations analyzed by previous researchers. Konst and Matson (in press) noted that a family's referral to a traditional clinic setting had multiple factors such as transportation, cost, and waitlists that may act as barriers to seeking services. Therefore, future research analyzing factors that hinder the availability of psychological services to minority groups remains paramount. Researchers have also previously identified intellectual functioning as a factor that may moderate ASD symptom severity (Matson et al., 2008). Given the prevalence of comorbid ID in ASD populations (Battaglia & Carey, 2006; Betancur, 2011; Chakrabarti & Fombonne, 2001; Yeargin-Allsopp et al., 2003), the influence of intellectual functioning on assessment and early identification should also be examined.

As our conceptualization of ASD continues to develop and progress, it will be necessary to also update those measures used to assess ASD symptomology across the lifespan. The continued development of ASD assessment measures is important for accurate identification, the delivery of interventions, financial reimbursement, and the advancement of future research. The current analyses are

an initial step toward demonstrating the utility of the *BISCUIT* assessment battery following the recent revisions to the ASD category in the *DSM-5*. However, the factors contributing to this increase in sensitivity and specificity should not be overlooked. Specifically, the complete removal of a contentious diagnostic category (i.e., PDD-NOS) has far reaching implications for families, researchers, and clinicians. The specific ramifications of these changes will only become more salient with further research.

References

Abdul-Rahman, O. A., & Hudgins, L. (2006). The diagnostic utility of a genetics evaluation in children with pervasive developmental disorders. *Genetics in Medicine, 8*(1), 50-54.

Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews Genetics*, *9*(5), 341-355.

Achenbach, T. M., & Edelbrock, C. S. (1983). *Manual for the child behavior checklist and revised child behavior profile*. Burlington, VT: University of Vermont Department of Psychiatry.

Achenbach, T. M., & Rescorla, L. (2000). *Child behavior checklist*. Burlington, VT: ASEBA.

Adams, C., Green, J., Gilchrist, A., & Cox, A. (2002). Conversational behavior of children with Asperger syndrome and conduct disorder. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 43*, 679-690.

Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., & Sauvage, D. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry*, *32*(3), 617-626.

Advokat, C. D., Mayville, E. A., & Matson, J. L. (2000). Side effect profiles of atypical antipsychotics, typical antipsychotics, or no psychotropic medications in persons with mental retardation. *Research in Developmental Disabilities*, *21*(1), 75-84.

Ahearn, W. H., Castine, T., Nault, K., & Green, G. (2001). An assessment of food acceptance in children with autism or pervasive developmental disorder-not otherwise specified. *Journal of Autism and Developmental Disorders*, *31*(5), 505-511.

Allen, D. (2008). The relationship between challenging behaviour and mental ill-health in people with intellectual disabilities: A review of current theories and evidence. *Journal of Intellectual Disabilities, 12*, 267–294.

Allik, H., Larsson, J. O., & Smedje, H. (2006). Sleep patterns of school-age children with Asperger syndrome or high-functioning autism. *Journal of Autism and Developmental Disorders*, *36*(5), 585-595.

Allison, C., Auyeung, B., & Baron-Cohen, S. (2012). Toward brief "red flags" for autism screening: The short autism spectrum quotient and the short quantitative checklist in 1,000 cases and 3,000 controls. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(2), 202-212.

Allison, C., Baron-Cohen, S., Wheelwright, S., Charman, T., Richler, J., Pasco, G., & Brayne, C. (2008). The Q-CHAT (Quantitative CHecklist for Autism in Toddlers): A normally distributed quantitative measure of autistic traits at 18–24 months of age: Preliminary report. *Journal of Autism and Developmental Disorders*, *38*(8), 1414-1425.

Altman, D. G., & Bland, J. M. (1994). Statistics Notes: Diagnostic tests 2: Predictive values. *British Medical Journal, 309*(6947), 102.

American Academy of Pediatrics. (2001). Technical report: The pediatrician's role in the diagnosis and management of autism spectrum disorder. *Pediatrics, 107,* 602–607.

American Academy of Pediatrics. (2008). *Bright futures: Guidelines for health supervision of infants, children, and adolescents.* Elk Grove Village, IL: American Academy of Pediatrics.

American Psychiatric Association (1952). *Diagnostic and statistical manual of mental disorders* (DSM-I). Washington, DC: Author.

American Psychiatric Association (1968). *Diagnostic and statistical manual of mental disorders* (2nd edition, DSM-II). Washington, DC: Author.

American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders* (3rd edition, DSM-III). Washington, DC: Author.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd edition–revised, DSM-III-R). Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th edition, DSM-IV). Washington, DC: Author.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th edition-text revision, DSM-IV-TR). Washington, DC: Author.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th edition, DSM-5). Washington, DC: Author.

Ando, H., & Yoshimura, I. (1979). Effects of age on communication skill levels and prevalence of maladaptive behaviors in autistic and mentally retarded children. *Journal of Autism and Developmental Disorders*, *9*(1), 83-93.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*(1), 57-87.

Arnold, L. E., Aman, M. G., Martin, A., Collier-Crespin, A., Vitiello, B., Tierney, E., . . . Vokmar, F. (2000). Assessment in multisite randomized clinical trials of patients with autistic disorder: The autism RUPP Network. *Journal of Autism and Developmental Disorders*, *30*(2), 99-111.

Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K., & Hackett, G. (2009). Fetal testosterone and autistic traits. *British Journal of Psychology*, *100*(1), 1-22.

Aylward, E. H., Minshew, N. J., Field, K., Sparks, B. F., & Singh, N. (2002). Effects of age on brain volume and head circumference in autism. *Neurology*, *59*(2), 175-183.

Aylward, G. P. (2009). Developmental screening and assessment: What are we thinking?. *Journal of Developmental and Behavioral Pediatrics*, *30*(2), 169-173.

Baghdadli, A., Pascal, C., Grisi, S., & Aussilloux, C. (2003). Risk factors for self-injurious behaviours among 222 young children with autistic disorders. *Journal of Intellectual Disability Research*, *47*(8), 622-627.

Baghdadli, A., Picot, M. C., Pascal, C., Pry, R., & Aussilloux, C. (2003). Relationship between age of recognition of first disturbances and severity in young children with autism. *European Child and Adolescent Psychiatry*, *12*(3), 122-127.

Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine, 25*, 63-77.

Baio, J. (2012). Prevalence of autism spectrum disorders: Autism and developmental disabilities monitoring network, 14 sites, United States, 2008. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, *61*(3).

Baird, G., Cass, H., & Slonims, V. (2003). Diagnosis of autism. *British Medical Journal, 327*, 488-493.

Baird, G., Charman, T., Cox, A., Baron-Cohen, S., Swettenham, J., Wheelwright, S., & Drew, A. (2001). Screening and surveillance for autism and pervasive developmental disorders. *Archives of Disease in Childhood, 84*(6), 468-475.

Bandini, L. G., Anderson, S. E., Curtin, C., Cermak, S., Evans, E. W., Scampini, R., . . . Must, A. (2010). Food selectivity in children with autism spectrum disorders and typically developing children. *The Journal of Pediatrics*, *157*(2), 259-264.

Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders, 29*, 213-224.

Baranek, G. T., Barnett, C. R., Adams, E. M., Wolcott, N. A., Watson, L. R., & Crais, E. R. (2005). Object play in infants with autism: Methodological issues in retrospective video analysis. *The American Journal of Occupational Therapy*, *59*(1), 20-30.

Baranek, G. T., David, F. J., Poe, M. D., Stone, W. D., & Watson L. R. (2006). Sensory experiences questionnaire: Discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry, 47*, 591-601.

Baranek, G. T., Foster, L. G., & Berkson, G. (1997a). Sensory defensiveness in persons with developmental disabilities. *Occupational Health and Industrial Medicine*, *37*(3).

Baranek, G. T., Foster, L. G., & Berkson, G. (1997b). Tactile defensiveness and stereotyped behaviors. *The American Journal of Occupational Therapy*, *51*(2), 91-95.

Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *The British Journal of Psychiatry*, *161*(6), 839-843.

Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., . . . Charman, T. (1996). Psychological markers in the detection of autism in infancy in a large population. *The British Journal of Psychiatry*, *168*(2), 158-163.

Barrett, S., Beck, J. C., Bernier, R., Bisson, E., Braun, T. A., Casavant, T. L., . . . Slager, S. (1999). An autosomal genomic screen for autism. Collaborative linkage study of autism. *American Journal of Medical Genetics*, *88*(6), 609-615.

Bartak, L., & Rutter, M. (1976). Differences between mentally retarded and normally intelligent autistic children. *Journal of Autism and Childhood Schizophrenia*, *6*(2), 109-120.

Bartak, L., Rutter, M., & Cox, A. (1975). A comparative study of infantile autism and specific developmental receptive language disorder. *British Journal of Psychiatry, 126*, 127–145.

Bates, E. (1976). *Language in context.* New York, NY: Academic Press.

Battaglia, A., & Carey, J. C. (2006). Etiologic yield of autistic spectrum disorders: a prospective study. *American Journal of Medical Genetics Part C, 142*(1), 3-7.

Bauman, M. L., & Kemper, T. L. (2003). The neuropathology of the autism spectrum disorders: What have we learned?. *Novartis Eound*, 112-22.

Baumeister, A. A., & Forehand, R. (1973). Stereotyped acts. In N. R. Ellis (Ed.), *International Review of Research in Mental Retardation* (Vol. 6, pp. 55–96). New York: Academic Press.

Bear, M. F., Huber, K. M., & Warren, S. T. (2004). The mGluR theory of fragile X mental retardation. *Trends in Neurosciences*, *27*(7), 370-377.

Bellini, S. (2004). Social skill deficits and anxiety in high functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities, 19,* 78–86.

Bellini, S. (2006). The development of social anxiety in adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, *21*(3), 138-145.

Bellini, S., Peters, J. K., Benner, L., & Hopf, A. (2007). A meta-analysis of school-based social skills interventions for children with autism spectrum disorders. *Remedial and Special Education*, *28*(3), 153-162.

Bender, L. (1956). Childhood schizophrenia: Symposium, 1955: 2. Schizophrenia in childhood—Its recognition, description and treatment. *American Journal of Orthopsychiatry*, *26*(3), 499.

Berard, G. (1993). *Hearing equals behavior.* New Canaan, CT: Keats.

Berkson, G., & Davenport, R. K. (1962). Stereotyped movements of mental defectives: Initial survey. *American Journal of Mental Deficiency, 66*, 849–852.

Bernstein, G. A., & Borchardt, C. M. (1991). Anxiety disorders of childhood and adolescence: A critical review. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*(4), 519-532.

Betancur, C. (2011). Etiological heterogeneity in autism spectrum disorders: more than 100 genetic and genomic disorders and still counting. *Brain Research, 1380*, 42-77.

Bettison, S. (1994). "Auditory Training" as a treatment for sound sensitivity in autism: Preliminary results. *Special Education Perspectives, 3*(1).

Bettelheim, B. (1967). *Empty fortress*. Simon and Schuster.

Billstedt, E. (2000). Autism and Asperger syndrome: Coexistence with other clinical disorders. *Acta Psychiatrica Scandinavica*, *102*(5), 321-330.

Billstedt, E., Gillberg, I., & Gillberg, C. (2007). Autism in adults: symptom patterns and early childhood predictors. Use of the DISCO in a community sample followed from childhood. *Journal of Child Psychology and Psychiatry*, *48*(11), 1102-1110.

Bishop, D. V., Maybery, M., Maley, A., Wong, D., Hill, W., & Hallmayer, J. (2004). Using self-report to identify the broad phenotype in parents of children with autistic spectrum disorders: A study using the Autism-Spectrum Quotient. *Journal of Child Psychology and Psychiatry*, *45*(8), 1431-1436.

Blakemore, S. J., Tavassoli, T., Calo, S., Thomas, R. M., Catmur, C., Frith, U., et al. (2006). Tactile sensitivity in Asperger syndrome. *Brain and Cognition, 61*, 5–13.

Bleuler, E. (1913). Autistic thinking. *The American Journal of Insanity, 69*(5), 873-886.

Bliss, S. L. (2007). Test Review: Newborg, J.(2005). Battelle Developmental Inventory-Itasca, IL: Riverside. [Review of the *Battelle Developmental Inventory*, by J. Newborg]. *Journal of Psychoeducational Assessment*.

Blomquist, H. K., Bohman, M., Edvinsson, S. O., Gillberg, C., Gustavson, K. H., Holmgren, G., & Wahlström, J. (1985). Frequency of the fragile X syndrome in infantile autism. *Clinical Genetics*, *27*(2), 113-117.

Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, *30*(3), 237-243.

Bolton, P., Macdonald, H., Pickles, A., Rios, P., Goode, S., Crowson, M., . . . Rutter, M. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry*, *35*(5), 877-900.

Boomsma, A., Van Lang, N. D. J., De Jonge, M. V., De Bildt, A. A., Van Engeland, H., & Minderaa, R. B. (2008). A new symptom model for autism cross-validated in an independent sample. *Journal of Child Psychology and Psychiatry*, *49*(8), 809-816.

Borthwick-Duffy, S. A. (1994). Prevalence of destructive behaviors: A study of aggression, self-injury, and property destruction. *SAGE Focus Editions*, *170*, 3-3.

Borthwick-Duffy, S. A. (1996). Evaluation and quality of life: Special considerations for persons with mental retardation. In R. L. Schalock & G. N. Siperstein (Eds.), *Quality of life. Vol. I: Conceptualization and measurement* (pp. 105–120). Washington, DC: American Association on Mental Retardation.

Borthwick-Duffy, S. A., Eyman, R. K., & White, J. F. (1987). Client characteristics and residential placement patterns. *American Journal of Mental Deficiency*.

Bosch, S., & Fuqua, R. W. (2001). Behavioral cusps: A model for selecting target behaviors. *Journal of Applied Behavior Analysis*, *34*(1), 123.

Bradley, E. A., Summers, J. A., Wood, H. L., & Bryson, S. E. (2004). Comparing rates of psychiatric and behavior disorders in adolescents and young adults with severe intellectual disability with and without autism. *Journal of Autism and Developmental Disorders*, *34*(2), 151-161.

Braithwaite, K. L., & Richdale, A. L. (2000). Functional communication training to replace challenging behaviors across two behavioral outcomes. *Behavioral Interventions*, *15*(1), 21-36.

Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, *36*(7), 863-870.

Brown, K. F., Kroll, J. S., Hudson, M. J., Ramsay, M., Green, J., Long, S. J., . . . Sevdalis, N. (2010). Factors underlying parental decisions about combination childhood vaccinations including MMR: A systematic review. *Vaccine*, *28*(26), 4235-4248.

Bryson, S. E., Bradley, E. A., Thompson, A., & Wainwright, A. (2008). Prevalence of autism among adolescents with intellectual disabilities. *Canadian Journal of Psychiatry*, *53*(7), 449-459.

Bryson, S. E., Rogers, S. J., & Fombonne, E. (2003). In Review. *Canadian Journal of Psychiatry*, *48*, 506-516.

Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., & McDermott, C. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders*, *37*(1), 12-24.

Canitano, R., & Vivanti, G. (2007). Tics and Tourette syndrome in autism spectrum disorders. *Autism*, *11*(1), 19-28.

Cannella, H. I., O'Reilly, M. F., & Lancioni, G. E. (2006). Treatment of hand mouthing in individuals with severe to profound developmental disabilities: A review of the literature. *Research in Developmental Disabilities*, *27*(5), 529-544.

Carcani-Rathwell, I., Rabe-Hasketh, S., & Santosh, P. J. (2006). Repetitive and stereotyped behaviours in pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, *47*(6), 573-581.

Caron, C., & Rutter, M. (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry*, *32*(7), 1063-1080.

Carpenter, M., Pennington, B., & Rogers, S. (2002). Interrelations among social-cognitive skills in young children with autism. *Journal of Autism and Developmental Disorders*, *32*(2), 91– 106.

Carr, E. G., Dunlap, G., Horner, R. H., Koegel, R. L., Turnbull, A. P., Sailor, W., . . . Fox, L. (2002). Positive behavior support evolution of an applied science. *Journal of Positive Behavior Interventions*, *4*(1), 4-16.

Carr, E. G., & Horner, R. H. (2007). The expanding vision of positive behavior support research perspectives on happiness, helpfulness, hopefulness. *Journal of Positive Behavior Interventions*, *9*(1), 3-14.

Cascio, C., McGlone, F., Folger, S., Tannan, V., Baranek, G., Pelphrey, K. A., & Essick, G. (2008). Tactile perception in adults with autism: a multidimensional psychophysical study. *Journal of Autism and Developmental Disorders*, *38*(1), 127-137.

Case-Smith, J., & Arbesman, M. (2008). Evidence-based review of interventions for autism used in or of relevance to occupational therapy. *American Journal of Occupational Therapy*, *62*(4), 416-429.

Cataldo, M. F., Ward, E. M., Russo, D. C., Riordan, M., & Bennett, D. (1986). Compliance and correlated problem behavior in children: Effects of contingent and noncontingent reinforcement. *Analysis and Intervention in Developmental Disabilities, 6*, 265-282.

Cesaroni, L., & Garber, M. (1991). Exploring the experience of autism through first hand accounts. *Journal of Autism and Developmental Disorders, 21*(3), 303–313.

Chadwick, O., Walker, N., Bernard, S., & Taylor, E. (2000). Factors affecting the risk of behaviour problems in children with severe intellectual disability. *Journal of Intellectual Disability Research, 44*(2), 108-123.

Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association*, *285*(24), 3093-3099.

Charles, J. M., Carpenter, L. A., Jenner, W., & Nicholas, J. S. (2008). Recent advances in autism spectrum disorders. *The International Journal of Psychiatry in Medicine*, *38*(2), 133-140.

Charman, T., & Baird, G. (2002). Practitioner review: Diagnosis of autism spectrum disorder in 2-and 3-year-old children. *Journal of Child Psychology and Psychiatry*, *43*(3), 289-305.

Charman, T., & Howlin, P. (2003). Research into early intervention for children with autism and related disorders: Methodological and design issues. *Autism, 7*, 217–225.

Charman, T., Swettenham, J., Baron-Cohen, S., Cox, A., Baird, G., & Drew, A. (1997). Infants with autism: an investigation of empathy, pretend play, joint attention, and imitation. *Developmental Psychology*, *33*(5), 781.

Charman, T., Taylor, E., Drew, A., Cockerill, H., Brown, J. A., & Baird, G. (2005). Outcome at 7 years of children diagnosed with autism at age 2: Predictive validity of assessments conducted at 2 and 3 years of age and pattern of symptom change over time. *Journal of Child Psychology and Psychiatry, 46*(5), 500-513.

Chasson, G. S., Harris, G. E., & Neely, W. J. (2007). Cost comparison of early intensive behavioral intervention and special education for children with autism. *Journal of Child and Family Studies, 16*(3), 401-413.

Chawarska, K., & Volkmar, F. R. (2004). Autism in infancy and early childhood. *Handbook of Autism and Pervasive Developmental Disorders, 1*(3), 223-246.

Cheng, H. C., Chen, H. Y., Tsai, C. L., Chen, Y. J., & Cherng, R. J. (2009). Comorbidity of motor and language impairments in preschool children of Taiwan. *Research in Developmental Disabilities*, *30*(5), 1054-1061.

Chudley, A. E. (2004). Genetic landmarks through philately–autism spectrum disorders: A genetic update. *Clinical Genetics*, *65*(5), 352-357.

Chudley, A. E., Gutierrez, E., Jocelyn, L. J., & Chodirker, B. N. (1998). Outcomes of genetic evaluation in children with pervasive developmental disorder. *Journal of Developmental and Behavioral Pediatrics*, *19*(5), 321-325.

Chugani, D. C. (2004). Serotonin in autism and pediatric epilepsies. *Mental Retardation and Developmental Disabilities Research Reviews*, *10*(2), 112-116.

Clarke, D., Baxter, M., Perry, D., & Prasher, V. (1999). The diagnosis of affective and psychotic disorders in adults with autism: Seven case reports. *Autism*, *3*(2), 149-164.

Cohen, B. H. (2008). *Explaining psychological statistics*. Hoboken, NJ: John Wiley & Sons.

Cohen, I. L. (2003). Criterion-related validity of the PDD Behavior Inventory. *Journal of Autism and Developmental Disorders*, *33*(1), 47-53.

Cohen, I. L., Schmidt-Lackner, S., Romanczyk, R., & Sudhalter, V. (2003). The PDD Behavior Inventory: a rating scale for assessing response to intervention in children with pervasive developmental disorder. *Journal of Autism and Developmental Disorders*, *33*(1), 31-45.

Cohen, J. (1992). A Power Primer. *Psychological Bulletin*, *112*(1), 155-159.

Condon, W. S. (1975). Multiple response to sound in dysfunctional children. *Journal of Autism and Childhood Schizophrenia*, *5*, 37-56.

Conners, C. K. (1973). Rating scales for use in drug studies. *Psychopharmacology Bulletin*, *9*, 24–29.

Constantino, J. N., Gruber, C. P., Davis, S., Hayes, S., Passanante, N., & Przybeck, T. (2004). The factor structure of autistic traits. *Journal of Child Psychology and Psychiatry*, *45*(4), 719-726.

Cook Jr, E. H. (2001). Genetics of autism. *Child and Adolescent Psychiatric Clinics of North America*, *10*(2), 333-350.

Cook Jr, E. H., Courchesne, R. Y., Cox, N. J., Lord, C., Gonen, D., Guter, S. J., . . . Courchesne, E. (1998). Linkage-disequilibrium mapping of autistic disorder, with 15q11-13 markers. *The American Journal of Human Genetics*, *62*(5), 1077-1083.

Cook Jr, E. H., Lindgren, V., Leventhal, B. L., Courchesne, R., Lincoln, A., Shulman, C., . . . Courchesne, E. (1997). Autism or atypical autism in maternally but not paternally derived proximal 15q duplication. *American Journal of Human Genetics*, *60*(4), 928.

Corsello, C. M. (2005). Early intervention in autism. *Infants and Young Children*, *18*(2), 74-85.

Costello, E. J. (1989). Child psychiatric disorders and their correlates: A primary care pediatric sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *28*(6), 851-855.

Courchesne, E., Akshoomof, N. A., & Townsend, J. (1990). Recent advances in autism. *Current*

Opinion in Paediatrics, 2, 685-693.

Courchesne, E., Karns, C. M., Davis, H. R., Ziccardi, R., Carper, R. A., Tigue, Z. D., . . . Courchesne, R. Y. (2001). Unusual brain growth patterns in early life in patients with autistic disorder an MRI study. *Neurology*, *57*(2), 245-254.

Courchesne, E., & Pierce, K. (2005). Brain overgrowth in autism during a critical time in development: Implications for frontal pyramidal neuron and interneuron development and connectivity. *International Journal of Developmental Neuroscience*, *23*(2), 153-170.

Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., . . . Wheelwright, S. (1999). Autism spectrum disorders at 20 and 42 months of age: Stability of clinical and ADI-R diagnosis. *Journal of Child Psychology and Psychiatry*, *40*(5), 719-732.

Crocker, L., & Algina, J. (1986). *Introduction to classical and modern test theory*. Orlando, FL: Holt, Rinehart and Winston.

Cromer, R. F. (1981). Developmental language disorders: Cognitive processes, semantics, pragmatics, phonology, and syntax. *Journal of Autism and Developmental Disorders*, *11*(1), 57-74.

Cuccaro, M. L., Shao, Y., Grubber, J., Slifer, M., Wolpert, C. M., Donnelly, S. L., … & Pericak-Vance, M. A. (2003). Factor analysis of restricted and repetitive behaviours in autism using the Autism Diagnostic Interview-R. *Child Psychiatry and Human Development, 34*, 3–17.

Cui, X. J., & Vaillant, G. E. (1997). Does depression generate negative life events?. *The Journal of Nervous and Mental Disease*, *185*(3), 145-150.

D'Souza, Y., Fombonne, E., & Ward, B. J. (2006). No evidence of persisting measles virus in peripheral blood mononuclear cells from children with autism spectrum disorder. *Pediatrics*, *118*(4), 1664-1675.

Dahlgren, S. V, & Gillberg, C. (1989). Symptoms in the first two years of life: A preliminary population study of infantile autism. *European Archives of Psychiatry and Neurological Sciences, 238*, 169–174.

Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., & Iacoboni, M. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, *9*(1), 28-30.

Davis III, T. E., Hess, J. A., Moree, B. N., Fodstad, J. C., Dempsey, T., Jenkins, W. S., & Matson, J. L. (2011). Anxiety symptoms across the lifespan in people diagnosed with autistic disorder. *Research in Autism Spectrum Disorders*, *5*(1), 112-118.

Davis III, T. E., Ollendick, T. H., & Nebel-Schwalm, M. (2008). Intellectual ability and achievement in anxiety-disordered children: A clarification and extension of the literature. *Journal of Psychopathology and Behavioral Assessment*, *30*(1), 43-51.

Dawson, G., & Adams, A. (1984). Imitation and social responsiveness in autistic children. *Journal of Abnormal Child Psychology, 12*, 209–226.

Dawson, G., & Osterling, J. (1997). Early intervention in autism. In M. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 307–326). Baltimore, MD: Brookes Publishing Co.

Dawson, G., Osterling, J., Meltzoff, A. N., & Kuhl, P. (2000). Case study of the development of an infant with autism from birth to two years of age. *Journal of Applied Developmental Psychology, 21*, 299–313.

Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., . . . Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: the Early Start Denver Model. *Pediatrics, 125*(1), 17-23.

Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology, 40*(2), 271–283.

De Bildt, A., Sytema, S., Ketelaars, C., Kraijer, D., Mulder, E., Volkmar, F., & Minderaa, R. (2004). Interrelationship between autism diagnostic observation schedule-generic (ADOS-G), autism diagnostic interview-revised (ADI-R), and the diagnostic and statistical manual of mental disorders (DSM-IV-TR) classification in children and adolescents with mental retardation. *Journal of Autism and Developmental Disorders*, *34*(2), 129-137.

de Bruin, E. I., Ferdinand, R. F., Meester, S., de Nijs, P. F., & Verheij, F. (2007). High rates of psychiatric co-morbidity in PDD-NOS. *Journal of Autism and Developmental Disorders*, *37*(5), 877-886.

Deb, S., & Prasad, K. B. (1994). The prevalence of autistic disorder among children with a learning disability. *The British Journal of Psychiatry*, *165*(3), 395-399.

DeGiacomo, A., & Fombonne, E. (1998). Parental recognition of developmental abnormalities in autism. *European Journal of Child and Adolescent Psychiatry, 7*(3), 131–136.

DeGiacomo, A., & Fombonne, E. (1998). Parental recognition of developmental abnormalities in autism. *European Child and Adolescent Psychiatry*, *7*(3), 131-136.

DeLong, G. R. (1999). Autism new data suggest a new hypothesis. *Neurology*, *52*(5), 911-911.

Desombre, H., Malvy, J., Roux, S., de Villard, R., Sauvage, D., Dalery, J., & Lenoir, P. (2006). Autism and developmental delay. *European Child and Adolescent Psychiatry*, *15*(6), 343-351.

DeStefano, F., & Thompson, W. W. (2004). MMR vaccine and autism: an update of the scientific evidence. *Expert Review of Vaccines, 3*(1), 19-22.

Dettmer, S., Simpson, R. L., Myles, B. S., & Ganz, J. B. (2000). The use of visual supports to facilitate transitions of students with autism. *Focus on Autism and Other Developmental Disabilities*, *15*(3), 163-169.

Deutsch, C. K., & Joseph, R. M. (2003). Brief report: cognitive correlates of enlarged head circumference in children with autism. *Journal of Autism and Developmental Disorders*, *33*(2), 209-215.

DeVellis, R. F. (1991). *Scale development: Theory and applications*. Newbury Park, CA: Sage.

DiLalla, D. L., & Rogers, S. J. (1994). Domains of the Childhood Autism Rating Scale: Relevance for diagnosis and treatment. *Journal of Autism and Developmental Disorders, 24*, 115-128.

Dixon, D. R., Granpeesheh, D., Tarbox, J., & Smith, M. N. (2011). Early detection of autism spectrum disorders. In *International Handbook of Autism and Pervasive Developmental Disorders* (pp. 197-214). Springer New York.

Dodson, F., & Alexander, A. (1986). *Your child: Birth to age 6*. Simon & Schuster.

Dölen, G., Osterweil, E., Rao, B. S., Smith, G. B., Auerbach, B. D., Chattarji, S., & Bear, M. F. (2007). Correction of fragile X syndrome in mice. *Neuron*, *56*(6), 955-962.

Dominick, K. C., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities*, *28*(2), 145-162.

Donner, A. (1982). The relative effectiveness of procedures commonly used in multiple regression analysis for dealing with missing values. *The American Statistician, 36*, 378–381.

Dorland, W. A. (1994). *Dorland's Illustrated Medical Dictionary, 28th edition*. Philadelphia: WB Saunders Company.

Dosreis, S., Weiner, C. L., Johnson, L., & Newschaffer, C. J. (2006). Autism spectrum disorder screening and management practices among general pediatric providers. *Journal of Developmental and Behavioral Pediatrics*, *27*(2), S88-S94.

Dover, C. J., & Le Couteur, A. (2007). How to diagnose autism. *Archives of Disease in Childhood*, *92*(6), 540-545.

Downs, A., Downs, R. C., & Rau, K. (2008). Effects of training and feedback on discrete trial teaching skills and student performance. *Research in Developmental Disabilities*, *29*(3), 235-246.

Durand, V. M. (1999). Functional communication training using assistive devices: Recruiting natural communities of reinforcement. *Journal of Applied Behavior Analysis*, *32*(3), 247-267.

Durand, V. M., & Carr, E. G. (1987). Social influences on "self-stimulatory" behavior: Analysis and treatment application. *Journal of Applied Behavior Analysis, 20*, 119-132.

Eaves, L. C., & Ho, H. H. (2004). The very early identification of autism: Outcome to age 4 1/2–5. *Journal of Autism and Developmental Disorders*, *34*(4), 367-378.

Eaves, L. C., Wingert, H., & Ho, H. H. (2006). Screening for autism Agreement with diagnosis. *Autism, 10*(3), 229-242.

Ehlers, S., Nydén, A., Gillberg, C., Sandberg, A. D., Dahlgren, S. O., Hjelmquist, E., & Odén, A. (1997). Asperger syndrome, autism and attention disorders: A comparative study of the cognitive profiles of 120 children. *Journal of Child Psychology and Psychiatry*, *38*(2), 207-217.

Eikeseth, S., Smith, T., Jahr, E., & Eldevik, S. (2002). Intensive behavioral treatment at school for 4- to 7-year-old children with autism: A 1-year comparison controlled study. *Behavior Modification, 26,* 49 - 68.

Einfeld, S. L., & Tonge, B. J. (1995). The Developmental Behavior Checklist: The development and validation of an instrument to assess behavioral and emotional disturbance in children and adolescents with mental retardation. *Journal of Autism and Developmental Disorders, 25*(2), 81-104.

Eisenmajer, R., Prior, M., Leekam, S., Wing, L., Gould, J., Welham, M., & Ong, B. (1996). Comparison of clinical symptoms in autism and Asperger's disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*(11), 1523-1531.

Ek, U., Fernell, E., Jacobson, L., & Gillberg, C. (1998). Relation between blindness due to retinopathy of prematurity and autistic spectrum disorders: A population-based study. *Developmental Medicine and Child Neurology, 40*(5), 297.

Eldevik, S., Hastings, R. P., Hughes, J. C., Jahr, E., Eikeseth, S., & Cross, S. (2009). Meta-analysis of early intensive behavioral intervention for children with autism. *Journal of Clinical Child and Adolescent Psychology, 38*(3), 439-450.

Elia, M., Ferri, R., Musumeci, S. A., Del Gracco, S., Bottitta, M., Scuderi, C., . . . Grubar, J. C. (2000). Sleep in subjects with autistic disorder: A neurophysiological and psychological study. *Brain and Development, 22*(2), 88-92.

Ellingson, S. A., Miltenberger, R. G., Stricker, J., Galensky, T. L., & Garlinghouse, M. (2000). Functional assessment and intervention for challenging behaviors in the classroom by general classroom teachers. *Journal of Positive Behavior Interventions, 2*(2), 85-97.

Emerson, E. (2001). *Challenging behaviour: Analysis and intervention in people with severe intellectual disabilities*. Cambridge, England: Cambridge University Press.

Emerson, E., Kiernan, C., Alborz, A., Reeves, D., Mason, H., Swarbrick, R., . . . Hatton, C. (2001a). The prevalence of challenging behaviors: a total population study. *Research in Developmental Disabilities, 22*(1), 77-93.

Emerson, E., Kiernan, C., Alborz, A., Reeves, D., Mason, H., Swarbrick, R., . . . Hatton, C. (2001b). Predicting the persistence of severe self-injurious behavior. *Research in Developmental Disabilities, 22*(1), 67-75.

Evans, D. W., Leckman, J. F., Carter, A., Reznick, J. S., Henshaw, D., King, R. A., & Pauls, D. (1997). Ritual, habit and perfectionism: The prevalence and development of compulsive-like behavior in normal young children. *Child Development 68,* 58–68.

Evans, M., Stoddart, H., Condon, L., Freeman, E., Grizzell, M., & Mullen, R. (2001). Parents' perspectives on the MMR immunization: A focus group study. *The British Journal of General Practice, 51*(472), 904.

Falk, R. E., & Casas, K. A. (2007). Chromosome 2q37 deletion: clinical and molecular aspects. *American Journal of Medical Genetics, 145*(4), 357-371.

Faraggi, D. (2000). The effect of random measurement error on receiver operating characteristic (ROC) curves. *Statistics in Medicine 19*, 61–70.

Fenske, E. C., Zalenski, S., Krantz, P. J., & McClannahan, L. E. (1985). Age at intervention and treatment outcome for autistic children in a comprehensive intervention program. *Analysis and Intervention in Developmental Disabilities, 5*, 7-31.

Fenson, L., Dale, P. S., Reznick, J. S., Bates, E., Thal, D. J., Pethick, S. J., . . . Stiles, J. (1994). Variability in early communicative development. *Monographs of the Society for Research in Child Development*, *59*(5), 1-185.

Fidler, D. J., Bailey, J. N., & Smalley, S. L. (2000). Macrocephaly in autism and other pervasive developmental disorders. *Developmental Medicine and Child Neurology*, *42*(11), 737-740.

Field, A. (2009). *Discovering statistics using SPSS* (3rd edition). London, England: Sage Publications.

Field, A. (2013). *Discovering statistics using IBM SPSS statistics*. London, England: Sage Publications.

Filipek, P. A., Accardo, P. J., Ashwal, S., Baranek, G. T., Cook, E. H., Dawson, G., . . . Volkmar, F. R. (2000). Practice parameter: Screening and diagnosis of autism report of the quality standards subcommittee of the American academy of neurology and the child neurology society. *Neurology*, *55*(4), 468-479.

Fischer, K., & Rose, S. (1994). Dynamic development of coordination of components in brain and behavior: A framework for theory. In G. Dawson & K. Fischer (Eds.), *Human behavior and brain development* (pp. 3–66). New York, NY: Guilford.

Fluss, R., Faraggi, D., & Reiser, B. (2005). Estimation of the Youden Index and its associated cutoff point. *Biometrical Journal*, *47*(4), 458-472.

Fodstad, J. C., Matson, J. L., Hess, J., & Neal, D. (2009). Social and communication behaviours in infants and toddlers with autism and pervasive developmental disorder-not otherwise specified. *Developmental Neurorehabilitation*, *12*(3), 152-157.

Folstein, S. (1999). Autism. *International Review of Psychiatry*, *11*, 269-277.

Fombonne, E., Heavey, L., Smeeth, L., Rodrigues, L. C., Cook, C., Smith, P. G., . . . Hall, A. J. (2004). Validation of the diagnosis of autism in general practitioner records. *BMC Public Health*, *4*(1), 5.

Fombonne, E., Roge, B., Claverie, J., Courty, S., & Fremolle, J. (1999). Microcephaly and macrocephaly in autism. *Journal of Autism and Developmental Disorders*, *29*(2), 113-119.

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

Freeman, B. J., Cronin, P., & Candela, P. (2002). Asperger syndrome or autistic disorder? The diagnostic dilemma. *Focus on Autism and Other Developmental Disabilities*, *17*(3), 145-151.

Freeman, B. J., Rahbar, B., Ritvo, E. R., Bice, T. L., Yokota, A., & Ritvo, R. (1991). The stability of cognitive and behavioral parameters in autism: A twelve-year prospective study. *Journal of the American Academy of Child Psychiatry*, *30*, 479–482.

Freeman, B. J., Ritvo, E. R., Needleman, R., & Yokota, A. (1985). The stability of cognitive and linguistic parameters in autism: A five-year prospective study. *Journal of the American Academy of Child Psychiatry*, *24*, 459–464.

Freeman, B. J., Ritvo, E. R., Schroth, P. C., Tonick, I., Guthrie, D., & Wake, L. (1981). Behavioral characteristics of high and low-IQ autistic children. *American Journal of Psychiatry, 138*(1), 25–29.

Frith, U. (1992) *Autism: Explaining the Enigma*. Oxford: Blackwell.

Gabriels, R. L., Cuccaro, M. L., Hill, D. E., Ivers, B. J., & Goldson, E. (2005). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, *26*(2), 169-181.

Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic comparison samples. *Journal of Autism and Developmental Disorders, 34*, 379–393.

Galli Carminati, G., Gerber, F., Baud, M. A., & Baud, O. (2007). Evaluating the effects of a structured program for adults with autism spectrum disorders and intellectual disabilities. *Research in Autism Spectrum Disorders, 1*(3), 256-265.

Garcia-Villamisar, D. A., & Dattilo, J. (2010). Effects of a leisure programme on quality of life and stress of individuals with ASD. *Journal of Intellectual Disability Research, 54*, 611–619.

Garfin, D. G., McCallon, D., & Cox, R. (1988). Validity and reliability of the Childhood Autism Rating Scale with autistic adolescents. *Journal of Autism and Developmental Disorders, 18*, 367-378.

Georgiades, S., Szatmari, P., Zwaigenbaum, L., Duku, E., Bryson, S., Roberts, W., . . . Mahoney, W. (2007). Structure of the autism symptom phenotype: A proposed multidimensional model. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*(2), 188-196.

Gernsbacher, M. A., Dawson, M., & Goldsmith, H. H. (2005). Three reasons not to believe in an autism epidemic. *Current Directions in Psychological Science*, *14*(2), 55-58.

Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: developmental disconnection syndromes. *Current Opinion in Neurobiology*, *17*(1), 103-111.

Ghaziuddin, M. (2005). *Mental health aspects of autism and Asperger syndrome*. London, England: Jessica Kingsley Publishers.

Ghaziuddin, M. (2010). Brief report: Should the DSM V drop Asperger syndrome?. *Journal of Autism and Developmental Disorders*, *40*(9), 1146-1148.

Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, *32*(4), 299-306.

Ghaziuddin, M., Tsai, L., & Ghaziuddin, N. (1992). Comorbidity of autistic disorder. *European Journal of Child and Adolescent Psychiatry, 1*, 209–213.

Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Comorbidity of Asperger syndrome: A preliminary report. *Journal of Intellectual Disability Research*, *42*(4), 279-283.

Ghaziuddin, M., Zaccagnini, J., Tsai, L., & Elardo, S. (1999). Is megalencephaly specific to autism?. *Journal of Intellectual Disability Research*, *43*(4), 279-282.

Gibbs, V., Aldridge, F., Chandler, F., Witzlsperger, E., & Smith, K. (2012). Brief report: An exploratory study comparing diagnostic outcomes for autism spectrum disorders under DSM-IV-TR with the proposed DSM-5 revision. *Journal of Autism and Developmental Disorders*, *42*(8), 1750-1756.

Gillberg, C. (1991a). Debate and argument: Is autism a pervasive developmental disorder?. *Journal of Child Psychology and Psychiatry, 32*(7), 1169-1170.

Gillberg, C. (1991b). Clinical and neurobiological aspects of Asperger syndrome in six family studies. *Autism and Asperger syndrome*, 122-146.

Gillberg, C. (1998). Chromosomal disorders and autism. *Journal of Autism and Developmental Disorders*, *28*(5), 415-425.

Gillberg, C., & Coleman, M. (1992). *The biology of the autistic syndromes*. London, England: Mac Keith Press.

Gillberg, C., Steffenburg, S., & Schaumann, H. (1991). Is autism more common now than ten years ago?. *The British Journal of Psychiatry*, *158*(3), 403-409.

Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism, 5*, 277–286.

Glascoe, F. P. (2005). Screening for developmental and behavioral problems. *Mental Retardation and Developmental Disabilities Research Reviews*, *11*(3), 173-179.

Glascoe, F. P., & Byrne, K. E. (1993). The accuracy of three developmental screening tests. *Journal of Early Intervention*, *17*(4), 368-379.

Goin-Kochel, R. P., Myers, B. J., & Mackintosh, V. H. (2007). Parental reports on the use of treatments and therapies for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *1*(3), 195-209.

Goizet, C., Excoffier, E., Taine, L., Taupiac, E., El Moneim, A. A., Arveiler, B., . . . Lacombe, D. (2000). Case with autistic syndrome and chromosome 22q13. 3 deletion detected by FISH. *American Journal of Medical Genetics*, *96*(6), 839-844.

Gold, N. (1993). Depression and social adjustment in siblings of boys with autism. *Journal of Autism and Developmental Disorders*, *23*(1), 147-163.

Goldberg, W. A., Osann, K., Filipek, P. A., Laulhere, T., Jarvis, K., Modahl, C., . . . Spence, M. A. (2003). Language and other regression: Assessment and timing. *Journal of Autism and Developmental Disorders*, *33*(6), 607-616.

Goldstein, G., Minshew, N. J., Allen, D. N., & Seaton, B. E. (2002). High-functioning autism and schizophrenia: a comparison of an early and late onset neurodevelopmental disorder. *Archives of Clinical Neuropsychology*, *17*(5), 461-475.

Goldstein, S., Naglieri, J. A., & Ozonoff, S. (Eds.). (2008). *Assessment of autism spectrum disorders*. New York, NY: Guilford Press.

Gordon, C. T. (2000). Commentary: Considerations on the pharmacological treatment of compulsions and stereotypies with serotonin reuptake inhibitors in pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 30*, 437–438.

Gorsuch, R. L. (1983). *Factor analysis* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates

Gotham, K., Risi, S., Pickles, A., & Lord, C. (2007). The Autism Diagnostic Observation Schedule: Revised algorithms for improved diagnostic validity. *Journal of Autism and Developmental Disorders*, *37*(4), 613-627.

Green, M., & Palfrey, J. S. (Eds.). (2002). *Bright futures: Guidelines for health supervision of infants, children, and adolescents* (2nd ed., revised). Arlington, VA: National Center for Education in Maternal and Child Health.

Green, V. A., Pituch, K. A., Itchon, J., Choi, A., O'Reilly, M., & Sigafoos, J. (2006). Internet survey of treatments used by parents of children with autism. *Research in Developmental Disabilities*, *27*(1), 70-84.

Greiner, M., Pfeiffer, D., & Smith, R. D. (2000). Principals and practical application of the receiver operating characteristic analysis for diagnostic tests. *Preventive Veterinary Medicine 45*, 23–41.

Grossman, J. B., Carter, A., & Volkmar, F. R. (1997). Social behavior in autism. *Annals of the New York Academy of Sciences*, *807*(1), 440-454.

Grzadzinski, R., Huerta, M., & Lord, C. (2013). DSM-5 and autism spectrum disorders (ASDs): An opportunity for identifying ASD subtypes. *Molecular Autism*, *4*(1), 1-6.

Guevara, J. P., Gerdes, M., Localio, R., Huang, Y. V., Pinto-Martin, J., Minkovitz, C. S., . . . Pati, S. (2013). Effectiveness of developmental screening in an urban setting. *Pediatrics*, *131*(1), 30-37.

Gutierrez Jr, A., Hale, M. N., O'Brien, H. A., Fischer, A. J., Durocher, J. S., & Alessandri, M. (2009). Evaluating the effectiveness of two commonly used discrete trial procedures for teaching receptive discrimination to young children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *3*(3), 630-638.

Hale, C. M., & Tager-Flusberg, H. (2005). Brief report: The relationship between discourse deficits and autism symptomatology. *Journal of Autism and Developmental Disorders*, *35*(4), 519-524.

Hamilton, S. (2006). Screening for developmental delay: Reliable, easy-to-use tools. *Journal of Family Practice, 55*(5), 415.

Handen, B. L., Parrish, J. M., McClung, T. J., Kerwin, M. E., & Evans, L. D. (1992). Using guided compliance versus time out to promote child compliance: A preliminary comparative analysis in an analogue context. *Research in Developmental Disabilities, 13*(2), 157-170.

Handleman, J., & Harris, S. L. (Eds.). (2001). *Preschool education programs for children with autism*. Austin, TX: Pro-Ed.

Hanley, J. A., & McNeil, B. J. (1982). The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology*, *143*(1), 29-36.

Hanley, J. A., & McNeil, B. J. (1983). A method of comparing the areas under receiver operating characteristic curves derived from the same cases. *Radiology*, *148*(3), 839-843.

Harrington, J. W., Patrick, P. A., Edwards, K. S., & Brand, D. A. (2006). Parental beliefs about autism Implications for the treating physician. *Autism, 10*(5), 452-462.

Harris, P. (1993). The nature and extent of aggressive behaviour amongst people with learning difficulties (mental handicap) in a single health district. *Journal of Intellectual Disability Research, 37*(3), 221-242.

Harris, S. L., Handleman, J. S., & Fong, P. L. (1987). Imitation of self-stimulation: Impact on the autistic child's behavior and affect. *Child and Family Behavior Therapy*, *9*(1-2), 1-21.

Hastings, R. P., & Brown, T. (2002). Coping strategies and the impact of challenging behaviors on special educators' burnout. *Journal Information*, *40*(2).

Hellings, J. A., Nickel, E. J., Weckbaugh, M., McCarter, K., Mosier, M., & Schroeder, S. R. (2005). The overt aggression scale for rating aggression in outpatient youth with autistic disorder: Preliminary findings. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *17*(1), 29-35.

Herbert, J. D., Sharp, I. R., & Gaudiano, B. A. (2002). Separating fact from fiction in the etiology and treatment of autism: A scientific review of the evidence. *Scientific Review of Mental Health Practice, 1*(1), 23-43.

Herbert, M. R. (2005). Large brains in autism: the challenge of pervasive abnormality. *The Neuroscientist, 11*(5), 417-440.

Herbert, M. R., Ziegler, D. A., Deutsch, C. K., O'brien, L. M., Lange, N., Bakardjiev, A., . . . Caviness, V. S. (2003a). Dissociations of cerebral cortex, subcortical and cerebral white matter volumes in autistic boys. *Brain*, *126*(5), 1182-1192.

Herbert, M. R., Ziegler, D. A., Makris, N., Bakardjiev, A., Hodgson, J., Adrien, K. T., . . . Caviness, V. S. (2003b). Larger brain and white matter volumes in children with developmental language disorder. *Developmental Science, 6*(4), 11-22.

Hirstein, W., Iversen, P., & Ramachandran, V. S. (2001). Autonomic responses of autistic children to people and objects. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *268*(1479), 1883-1888.

Hofvander, B., Delorme, R., Chaste, P., Nydén, A., Wentz, E., Ståhlberg, O., . . . Leboyer, M. (2009). Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. *BMC Psychiatry*, *9*(1), 35.

Hogart, A., Nagarajan, R. P., Patzel, K. A., Yasui, D. H., & LaSalle, J. M. (2007). 15q11-13 GABAA receptor genes are normally biallelically expressed in brain yet are subject to epigenetic dysregulation in autism-spectrum disorders. *Human Molecular Genetics*, *16*(6), 691-703.

Holden, B., & Gitlesen, J. P. (2006). A total population study of challenging behaviour in the county of Hedmark, Norway: Prevalence, and risk markers. *Research in Developmental Disabilities*, *27*(4), 456-465.

Holmes, N., Shah, A., & Wing, L. (1982). The Disability Assessment Schedule: A brief screening device for use with the mentally retarded. *Psychological Medicine*, *12*(04), 879-890.

Honda, H., Shimizu, Y., & Rutter, M. (2005). No effect of MMR withdrawal on the incidence of autism: a total population study. *Journal of Child Psychology and Psychiatry*, *46*(6), 572-579.

Horner, R. H., & Carr, E. G. (1997). Behavioral support for students with severe disabilities functional assessment and comprehensive intervention. *The Journal of Special Education*, *31*(1), 84-104.

Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism: A research synthesis. *Journal of Autism and Developmental Disorders*, *32*(5), 423-446.

Horovitz, M., & Matson, J. L. (2013a). The baby and infant screen for children with autism traits-part 2: The development of age-based cutoffs. *Developmental Neurorehabilitation*, (0), 1-8.

Horovitz, M., & Matson, J. L. (2013b). The Baby and Infant Screen for Children with aUtIsm Traits-Part 3: The development of age-based scoring procedures. *Research in Autism Spectrum Disorders*, *7*(11), 1291-1299.

Horovitz, M. & Matson, J. L. (2014). The Baby and Infant Screen for Children with aUtIsm Traits- Part 1: Age-based scoring procedures. *Journal of Developmental and Physical Disabilities, 26*, 1-22.

Horrigan, J. P., & Barnhill, L. J. (1998). Does guanfacine trigger mania in children?. *Journal of Child and Adolescent Psychopharmacology*, *8*(2), 149-150.

Hoshino, Y., Kaneko, M., Yashima, Y., Kumashiro, H., Volkmar, F. R., & Cohen, D. J. (1987). Clinical features of autistic children with setback course in their infancy. *Psychiatry and Clinical Neurosciences*, *41*(2), 237-245.

Howard, J. S., Sparkman, C. R., Cohen, H. G., Green, G., & Stanislaw, H. (2005). A comparison of intensive behavior analytic and eclectic treatments for young children with autism. *Research in Developmental Disabilities*, *26*, 359–383.

Howlin, P. (1997). Prognosis in autism: Do specialist treatments affect long-term outcome? *European Child and Adolescent Psychiatry, 6*: 55–72.

Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: implications for the differentiation between autism and Asperger syndrome. *Journal of Autism and Developmental Disorders*, *33*(1), 3-13.

Howlin, P., Gordon, K., Pasco, G., Wade, A., & Charman, T. (2007). A group randomised, controlled trial of the Picture Exchange Communication System for children with autism. *Journal of Child Psychology and Psychiatry, 48,* 473–481.

Howlin, P., Magiati, I., & Charman, T. (2009). Systematic review of early intensive behavioral interventions for children with autism. *Journal Information*, *114*(1).

Howlin, P., & Moore, A. (1997). Diagnosis in autism: A survey of over 1200 patients in the UK. *Autism, 1*, 135-162.

Hume, K., Bellini, S., & Pratt, C. (2005). The usage and perceived outcomes of early intervention and early childhood programs for young children with autism spectrum disorder. *Topics in Early Childhood Special Education*, *25*(4), 195-207.

Hutchinson, H. (1999). Feeding problems in young children: Report of three cases and review of the literature. *Journal of Human Nutrition and Dietetics, 12,* 337–343.

Huttenlocher, P. R. (1994). Synaptogenesis in human cerebral cortex. In G. Dawson & K. W. Fischer (Eds.), *Human behavior and the developing brain* (pp. 137–152). New York, NY: Guilford.

Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience, 7*(12), 942-951.

Illingworth, R. S. (2013). *The development of the infant and the young child: Normal and abnormal*. Cambridge, MA: Elsevier Health Sciences.

Jablensky, A., Sartorius, N. E., Hirschfeld, R., & Pardes, H. (1983). Diagnosis and classification of mental disorders and alcohol-and drug-related problems: A research agenda for the 1980s. *Psychological Medicine*.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, *59*(1), 12.

James, S. J., Cutler, P., Melnyk, S., Jernigan, S., Janak, L., Gaylor, D. W., & Neubrander, J. A. (2004). Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. *The American Journal of Clinical Nutrition*, *80*(6), 1611-1617.

Jang, J., Matson, J. L., Williams, L. W., Tureck, K., Goldin, R. L., & Cervantes, P. E. (2013). Rates of comorbid symptoms in children with ASD, ADHD, and comorbid ASD and ADHD. *Research in Developmental Disabilities*, *34*(8), 2369-2378.

Janicki, M. P., & Jacobson, J. W. (1983). Selected clinical features and service characteristics of autistic adults. *Psychological Reports*, *52*(2), 387-390.

Jiang, Y. H., Sahoo, T., Michaelis, R. C., Bercovich, D., Bressler, J., Kashork, C. D., . . . Beaudet, A. L. (2004). A mixed epigenetic/genetic model for oligogenic inheritance of autism with a limited role for UBE3A. *American Journal of Medical Genetics*, *131*(1), 1-10.

Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. *Pediatrics*, *120*(5), 1183-1215.

Kadesjö, B., & Gillberg, C. (2001). The comorbidity of ADHD in the general population of Swedish school-age children. *Journal of Child Psychology and Psychiatry*, *42*(4), 487-492.

Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child, 2*, 217-250.

Kapp-Simon, K. A., McGuire, D. E., Long, B. C., & Simon, D. J. (2005). Addressing quality of life issues in adolescents: Social skills interventions. *The Cleft Palate-Craniofacial Journal*, *42*(1), 45-50.

Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry, 47*, 611–620.

Kawamura, Y., Takahashi, O., & Ishii, T. (2008). Reevaluating the incidence of pervasive developmental disorders: impact of elevated rates of detection through implementation of an integrated system of screening in Toyota, Japan. *Psychiatry and Clinical Neurosciences*, *62*(2), 152-159.

Kazdin, A. E. (1993). Replication and extension of behavioral treatment of autistic disorder. *American Journal of Mental Retardation, 97*, 377–378.

Keren, M., Feldman, R., & Tyano, S. (2001). Diagnoses and interactive patterns of infants referred to a community-based infant mental health clinic. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*(1), 27-35.

Kern, J. K., Miller, V. S., Cauller, L. J., Kendall, R., Mehta, J., & Dodd, M. (2001). The effectiveness of N,N-dimethylglycine in autism/PDD. *Journal of Child Neurology, 16*, 169–173.

Kientz, M. A., & Dunn, W. (1996). A comparison of the performance of children with and without autism on the sensory profile. *The American Journal of Occupational Therapy, 51*(7), 530–537.

Kiernan, C., & Kiernan, D. (1994). Challenging behaviour in schools for pupils with severe learning difficulties. *Mental Handicap Research*, *7*(3), 177-201.

Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism, 4*, 117–132.

King, M. D., & Bearman, P. S. (2011). Socioeconomic status and the increased prevalence of autism in California. *American Sociological Review*, *76*(2), 320-346.

Kjelgaard, M. M., & Tager-Flusberg, H. (2001). An investigation of language impairment in autism: Implications for genetic subgroups. *Language and Cognitive Processes, 16*(2/3), 287–308.

Kleinman, J. M., Robins, D. L., Ventola, P. E., Pandey, J., Boorstein, H. C., Esser, E. L., . . . Fein, D. (2008). The modified checklist for autism in toddlers: A follow-up study investigating the early

detection of autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *38*(5), 827-839.

Klin, A., Chawarska, K., Paul, R., Rubin, E., Morgan, T., Wiesner, L., & Volkmar, F. (2004). Autism in a 15-month-old child. *American Journal of Psychiatry*, *161*(11), 1981-1988.

Knickmeyer, R. C., & Baron-Cohen, S. (2006). Topical review: Fetal testosterone and sex differences in typical social development and in autism. *Journal of Child Neurology*, *21*(10), 825-845.

Koegel, L. K., Koegel, R. L., & Surratt, A. (1992). Language intervention and disruptive behavior in preschool children with autism. *Journal of Autism and Developmental Disorders, 22,* 141–153.

Koegel, R. L., Firestone, P. B., Kramme, K. W., & Dunlap, G. (1974). Increasing spontaneous play by suppressing self-stimulation in autistic children. *Journal of Applied Behavior Analysis, 7*, 521–528.

Kogan, M. D., Strickland, B. B., Blumberg, S. J., Singh, G. K., Perrin, J. M., & van Dyck, P. C. (2008). A national profile of the health care experiences and family impact of autism spectrum disorder among children in the United States, 2005–2006. *Pediatrics*, *122*(6), 1149-1158.

Konst, M. J., & Matson, J. L. (in press). Early Identification of children at risk for autism spectrum disorder. *Research in Autism Spectrum Disorders*.

Konst, M. J., & Matson, J. L. (2014). Comorbid psychopathology symptom rates in infants and toddlers with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders*, *8*(2), 147-155.

Konst, M. J., Matson, J. L., & Turygin, N. (2013a). Comparing the rates of tantrum behavior in children with ASD and ADHD as well as children with comorbid ASD and ADHD diagnoses. *Research in Autism Spectrum Disorders*, *7*(11), 1339-1345.

Konst, M. J., Matson, J. L., & Turygin, N. (2013b). Exploration of the correlation between autism spectrum disorder symptomology and tantrum behaviors. *Research in Autism Spectrum Disorders*, *7*(9), 1068-1074.

Kozlowski, A. M., Matson, J. L., Horovitz, M., Worley, J. A., & Neal, D. (2011). Parents' first concerns of their child's development in toddlers with autism spectrum disorders. *Developmental Neurorehabilitation, 14,* 72-78.

Krey, J. F., & Dolmetsch, R. E. (2007). Molecular mechanisms of autism: a possible role for Ca 2+ signaling. *Current Opinion in Neurobiology*, *17*(1), 112-119.

Krug, D. A., Arick, J., & Almond, P. (1980). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. *Journal of Child Psychology and Psychiatry*, *21*(3), 221-229.

Kuoch, H., & Mirenda, P. (2003). Social story interventions for young children with autism spectrum disorders. *Focus on Autism and other Developmental Disabilities*, *18*(4), 219-227.

Kurita, H. (1985). Infantile autism with speech loss before the age of 30 months. *Journal of the American Academy of Child Psychiatry, 24,* 191–196.

Kurita, H. (1996). Specificity and developmental consequences of speech loss in children with pervasive developmental disorders. *Psychiatry and Clinical Neurosciences, 50,* 181–184.

La Greca, A. M., & Lopez, N. (1998). Social anxiety among adolescents: Linkages with peer relations and friendships. *Journal of Abnormal Child Psychology, 26*(2), 83-94.

La Malfa, G., Lassi, S., Bertelli, M., Salvini, R., & Placidi, G. F. (2004). Autism and intellectual disability: a study of prevalence on a sample of the Italian population. *Journal of Intellectual Disability Research, 48*, 262–267.

Lacroix, A., Guidetti, M., Rogé, B., & Reilly, J. (2009). Recognition of emotional and nonemotional facial expressions: A comparison between Williams syndrome and autism. *Research in Developmental Disabilities, 30*(5), 976-985.

Lainhart, J. E. (1999). Psychiatric problems in individuals with autism, their parents and siblings. *International Review of Psychiatry, 11*, 278–298.

Lainhart, J. E., & Folstein, S. E. (1994). Affective disorders in people with autism: A review of published cases. *Journal of Autism and Developmental Disorders, 24*(5), 587-601.

Lainhart, J. E., Piven, J., Wzorek, M., Landa, R., Santangelo, S. L., Coon, H., & Folstein, S. E. (1997). Macrocephaly in children and adults with autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(2), 282-290.

Lam, K. S., & Aman, M. G. (2007). The Repetitive Behavior Scale-Revised: Independent validation in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37*(5), 855-866.

Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry, 47*(6), 629-638.

Landa, R. J., Holman, K. C., & Garrett-Mayer, E. (2007). Social and communication development in toddlers with early and later diagnosis of autism spectrum disorders. *Archives of General Psychiatry, 64*(7), 853-864.

Lang, N. D., Boomsma, A., Sytema, S., Bildt, A. A., Kraijer, D. W., Ketelaars, C., & Minderaa, R. B. (2006). Structural equation analysis of a hypothesised symptom model in the autism spectrum. *Journal of Child Psychology and Psychiatry, 47*(1), 37-44.

Laufer, M. W., & Gair, D. S. (1969). Childhood schizophrenia. In L. Bellak & L. Loeb (Eds.). *The Schizophrenic Syndrome*. New York, NY: Grune and Stratton.

Lavigne, J. V., Arend, R., Rosenbaum, D., Smith, A., Weissbluth, M., Binns, H. J., & Christoffel, K. K. (1999). Sleep and behavior problems among preschoolers. *Journal of Developmental and Behavioral Pediatrics, 20*(3), 164-169.

Le Couteur, A., Rutter, M., Lord, C., Rios, P., Robertson, S., Holdgrafer, M., & McLennan, J. (1989). Autism diagnostic interview: a standardized investigator-based instrument. *Journal of Autism and Developmental Disorders, 19*(3), 363-387.

Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders*, *36*(8), 1101-1114.

Ledford, J. R., & Gast, D. L. (2006). Feeding problems in children with autism spectrum disorders a review. *Focus on Autism and Other Developmental Disabilities*, *21*(3), 153-166.

Lee, L. C., Harrington, R. A., Chang, J. J., & Connors, S. L. (2008). Increased risk of injury in children with developmental disabilities. *Research in Developmental Disabilities*, *29*(3), 247-255.

Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders*, *37*(5), 894-910.

Lewinsohn, P. M., Striegel-Moore, R. H., & Seeley, J. R. (2000). Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(10), 1284-1292.

Lewy, A. L., & Dawson, G. (1992). Social stimulation and joint attention in young autistic children. *Journal of Abnormal Child Psychology, 20,* 555–566.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., . . . Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders*, *36*(7), 849-861.

Limoges, É., Mottron, L., Bolduc, C., Berthiaume, C., & Godbout, R. (2005). Atypical sleep architecture and the autism phenotype. *Brain*, *128*(5), 1049-1061.

Linscheid, T. R., Iwata, B. A., Ricketts, R. W., Williams, D. E., & Griffin, J. C. (1990). Clinical evaluation of the self-injurious behavior inhibiting system (sibis). *Journal of Applied Behavior Analysis, 23*(1), 53-78.

Little, R. J. A. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of American Statistical Association, 83*(404), 1198–1202.

Liu, J., Nyholt, D. R., Magnussen, P., Parano, E., Pavone, P., Geschwind, D., . . . Autism Genetic Resource Exchange Consortium (2001). A genomewide screen for autism susceptibility loci. *The American Journal of Human Genetics*, *69*(2), 327-340.

Long, K.,Wood, H., & Holmes, N. (2000). Presentation, assessment and treatment of depression in a young woman with learning disability and autism. *British Journal of Learning Disabilities, 28,* 102–108.

Lord, C. (1995). Follow-up of two-year olds referred for possible autism. *Journal of Child Psychology and Psychiatry, 36,* 1365–1685.

Lord, C., & Jones, R. M. (2012). Annual Research Review: Re-thinking the classification of autism spectrum disorders. *Journal of Child Psychology and Psychiatry, 53*(5), 490-509.

Lord, C., & Paul, R. (1997). Language and communication in autism. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of Autism and Pervasive Developmental Disorders, 2nd edition.* (pp. 195–225). New York, NY: Wiley.

Lord, C., & Risi, S. (2000). Diagnosis of autism spectrum disorders in young children. In A. M. Wetherby & B. M. Prizant (Eds.), *Autism spectrum disorders: A transactional developmental perspective* (pp. 11-30). Baltimore, MD: Brooks Publishing Co.

Lord, C., & Ventner, A. (1992). Outcome and follow-up studies of high-functioning autistic individuals. In E. Schopler & G. Mesibov (Eds.), *High-functioning individuals with autism* (pp. 187–199). New York, NY: Plenum Press.

Lord, C., Risi, S., & Pickles, A. (2004). Trajectory of language development in autistic spectrum disorders. In M. Rice & S. Warren (Eds.), *Developmental language disorders: From phenotypes to etiologies* (pp. 7–29). Mahwah, NJ: Lawrence Erlbaum Associates.

Lord, C., Risi, S., Lambrecht, L., Cook Jr, E. H., Leventhal, B. L., DiLavore, P. C., . . . Rutter, M. (2000). The Autism Diagnostic Observation Schedule—Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30*(3), 205-223.

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism diagnostic observation schedule: ADOS*. Los Angeles, CA: Western Psychological Services.

Lord, C., Rutter, M., & LeCouteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*, 659–685.

Lösche, G. (1990). Sensorimotor and action development in autistic children from infancy to early childhood. *Journal of Child Psychology and Psychiatry*, *31*(5), 749-761.

Lotspeich, L. J., & Ciaranello, R. D. (1993). The neurobiology and genetics of infantile autism. *International Review of Neurobiology*, 87-89.

Lotspeich, L. J., Kwon, H., Schumann, C. M., Fryer, S. L., Goodlin-Jones, B. L., Buonocore, M. H., . . . Reiss, A. L. (2004). Investigation of neuroanatomical differences between autism and asperger syndrome. *Archives of General Psychiatry*, *61*(3), 291-298.

Lovaas, O. I., & Smith, T. (1988). Intensive behavioral treatment for young autistic children. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 2). New York, NY: Plenum Press.

Lovaas, O. I., Koegel, R., Simmons, J. Q., & Long, J. S. (1973). Some generalization and follow-up measures on autistic children in behavior therapy. *Journal of Applied Behavior Analysis*, *6*(1), 131-165.

Loveland, K., & Landry, S. (1986). Joint attention and language in autism and developmental language delay. *Journal of Autism and Developmental Disorders, 16*, 335–349.

LoVullo, S. V., & Matson, J. L. (2009). Comorbid psychopathology in adults with autism spectrum disorders and intellectual disabilities. *Research in Developmental Disabilities*, *30*(6), 1288-1296.

Luiselli, J. K. (2009). Aggression and noncompliance. In J. L. Matson (Ed.), *Applied behavior analysis for children with autism spectrum disorders* (pp. 175-187). New York, NY: Springer.

Luteijn, E., Luteijn, F., Jackson, S., Volkmar, F., & Minderaa, R. (2000a). The Children's Social Behavior Questionnaire for milder variants of PDD problems: Evaluation of the psychometric characteristics. *Journal of Autism and Developmental Disorders*, *30*(4), 317-330.

Luteijn, E. F., Serra, M., Jackson, S., Steenhuis, M. P., Althaus, M., Volkmar, F., & Minderaa, R. (2000b). How unspecified are disorders of children with a pervasive developmental disorder not otherwise specified? A study of social problems in children with PDD-NOS and ADHD. *European Child and Adolescent Psychiatry*, *9*(3), 168-179.

Luyster, R. J., Kadlec, M. B., Carter, A., & Tager-Flusberg, H. (2008). Language assessment and development in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *38*(8), 1426-1438.

MacDonald, R., Green, G., Mansfield, R., Geckeler, A., Gardenier, N., Anderson, J., . . . Sanchez, J. (2007). Stereotypy in young children with autism and typically developing children. *Research in Developmental Disabilities*, *28*(3), 266-277.

Mace, F. C., Hock, M. L., Lalli, J. S., West, B. J., Belfiore, P., Pinter, E., & Brown, D. K. (1988). Behavioral momentum in the treatment of noncompliance. *Journal of Applied Behavior Analysis*, *21*(2), 123-141.

Machalicek, W., O'Reilly, M. F., Beretvas, N., Sigafoos, J., & Lancioni, G. E. (2007). A review of interventions to reduce challenging behavior in school settings for students with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *1*(3), 229-246.

Maenner, M. J., Arneson, C. L., & Durkin, M. S. (2009). Socioeconomic disparity in the prevalence of autism spectrum disorder in Wisconsin. *Wisconsin Medical Journal*, *108*(5), 37-39.

Maestro, S., Muratori, F., Cesari, A., Cavallaro, M. C., Paziente, A., Pecini, C., ... & Sommario, C. (2005). Course of autism signs in the first year of life. *Psychopathology*, *38*(1), 26-31.

Mahjouri, S., & Lord, C. E. (2012). What the DSM-5 portends for research, diagnosis, and treatment of autism spectrum disorders. *Current Psychiatry Reports*, *14*(6), 739-747.

Mahler, M. (1968). *On human symbiosis and the vicissitudes of individuation*. New York, NY: International Universities Press.

Makrygianni, M. K., & Reed, P. (2010). A meta-analytic review of the effectiveness of behavioural early intervention programs for children with autism spectrum disorders. *Research in Autism Spectrum Disorders*, *4*, 577–593.

Malow, B. A., Marzec, M. L., McGrew, S. G., Wang, L., Henderson, L. M., & Stone, W. L. (2006). Characterizing sleep in children with autism spectrum disorders: A multidimensional approach. *Sleep*, *29*(12), 15-63.

Mandell, D. S., & Palmer, R. (2005). Differences among states in the identification of autistic spectrum disorders. *Archives of Pediatrics and Adolescent Medicine*, *159*(3), 266-269.

Mandell, D. S., Listerud, J., Levy, S. E., & Pinto-Martin, J. A. (2002). Race differences in the age at diagnosis among Medicaid-eligible children with autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*(12), 1447-1453.

Mandell, D. S., Novak, M. M., & Zubritsky, C. D. (2005). Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics*, *116*(6), 1480-1486.

Mandy, W. P., Charman, T., & Skuse, D. H. (2012). Testing the Construct Validity of Proposed Criteria for DSM-5 Autism Spectrum Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(1), 41-50.

Matson, J. L. (2009). Aggression and tantrums in children with autism: A review of behavioral treatments and maintaining variables. *Journal of Mental Health Research in Intellectual Disabilities*, *2*(3), 169-187.

Matson, J. L. (2007a). Current status of differential diagnosis for children with autism spectrum disorders. *Research in Developmental Disabilities*, *28*(2), 109-118.

Matson, J. L. (2007b). Determining treatment outcome in early intervention programs for autism spectrum disorders: A critical analysis of measurement issues in learning based interventions. *Research in Developmental Disabilities*, *28*(2), 207-218.

Matson, J. L., Baglio, C. S., Smiroldo, B. B., Hamilton, M., Packlowskyj, T., Williams, D., & Kirkpatrick-Sanchez, S. (1996). Characteristics of autism as assessed by the diagnostic assessment for the severely handicapped—II (DASH-II). *Research in Developmental Disabilities*, *17*(2), 135-143.

Matson, J. L., & Barrett, R. P. (1982). *Psychopathology in the mentally retarded*. New York, NY: Grane and Stratton.

Matson, J. L., Barrett, R. P., & Helsel, W. J. (1988). Depression in mentally retarded children. *Research in Developmental Disabilities*, *9*, 39–46.

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

Matson, J. L., Boisjoli, J. A., Hess, J. A., & Wilkins, J. (2011). Comorbid psychopathology factor structure on the Baby and Infant Screen for Children with aUtIsm Traits-Part 2 (BISCUIT-Part 2). *Research in Autism Spectrum Disorders*, *5*(1), 426-432.

Matson, J. L., Boisjoli, J., Rojahn, J., & Hess, J. (2009). A factor analysis of challenging behaviors assessed with the Baby and Infant Screen for Children with aUtism Traits BISCUIT-Part 3. *Research in Autism Spectrum Disorders*, *3*(3), 714-722.

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

Matson, J. L., Dempsey, T., & Fodstad, J. C. (2009). Stereotypies and repetitive/restrictive behaviours in infants with autism and pervasive developmental disorder. *Developmental Neurorehabilitation*, *12*(3), 122-127.

Matson, J. L., Dempsey, T., LoVullo, S. V., & Wilkins, J. (2008). The effects of intellectual functioning on the range of core symptoms of autism spectrum disorders. *Research in Developmental Disabilities*, *29*(4), 341-350.

Matson, J. L., Fodstad, J. C., Mahan, S., & Rojahn, J. (2010). Cut-offs, norms and patterns of problem behaviours in children with developmental disabilities on the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT-Part 3).*Developmental Neurorehabilitation*, *13*(1), 3-9.

Matson, J. L., Fodstad, J. C., Mahan, S., & Sevin, J. A. (2009c). Cutoffs, norms, and patterns of comorbid difficulties in children with an ASD on the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT-Part 2). *Research in Autism Spectrum Disorders*, *3*(4), 977-988.

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

Matson, J. L., González, M. L., & Rivet, T. T. (2008). Reliability of the autism spectrum disorder-behavior problems for children (ASD-BPC). *Research in Autism Spectrum Disorders*, *2*(4), 696-706.

Matson, J. L., González, M. L., & Wilkins, J. (2009). Validity study of the autism spectrum disorders-diagnostic for children (ASD-DC). *Research in Autism Spectrum Disorders*, *3*(1), 196-206.

Matson, J. L., González, M. L., Wilkins, J., & Rivet, T. T. (2008). Reliability of the autism spectrum disorder-diagnostic for children (ASD-DC). *Research in Autism Spectrum Disorders*, *2*(3), 533-545.

Matson, J. L., Hattier, M. A., & Williams, L. W. (2012). How does relaxing the algorithm for autism affect DSM-V prevalence rates?. *Journal of Autism and Developmental Disorders*, *42*(8), 1549-1556.

Matson, J. L., & Horovitz, M. (2010). Stability of autism spectrum disorders symptoms over time. *Journal of Developmental and Physical Disabilities*, *22*, 331-342.

Matson, J. L., Kiely, S. L., & Bamburg, J. W. (1997). The effect of stereotypies on adaptive skills as assessed with the DASH-II and Vineland Adaptive Behavior Scales. *Research in Developmental Disabilities*, *18*(6), 471-476.

Matson, J. L., & Konst, M. J. (in press). Early intervention for autism: Who provides treatment and in what settings. *Research in Autism Spectrum Disorders*.

Matson, J. L., Kozlowski, A. M., Hattier, M. A., Horovitz, M., & Sipes, M. (2012b). DSM-IV vs DSM-5 diagnostic criteria for toddlers with autism. *Developmental Neurorehabilitation*, *15*(3), 185-190.

Matson, J. L., Kozlowski, A. M., Neal, D., Worley, J. A., & Fodstad, J. C. (2011). Cutoffs for the Matson Evaluation of Social Skills with Youngsters-II (MESSY-II) for typically developing children and for children diagnosed with autism spectrum disorders. *Research in Autism Spectrum Disorders, 5*, 798-802.

Matson, J. L., & Love, S. R. (1990). A comparison of parent-reported fear for autistic and nonhandicapped age-matched children and youth. *Journal of Intellectual and Developmental Disability, 16*(4), 349-357.

Matson, J. L., LoVullo, S. V., Rivet, T. T., & Boisjoli, J. A. (2009). Validity of the autism spectrum disorder-comorbid for children (ASD-CC). *Research in Autism Spectrum Disorders, 3*(2), 345-357.

Matson, J. L., Mahan, S., Hess, J. A., Fodstad, J. C., & Neal, D. (2010). Progression of challenging behaviors in children and adolescents with autism spectrum disorders as measured by the Autism Spectrum Disorders-Problem Behaviors for Children (ASD-PBC). *Research in Autism Spectrum Disorders, 4*(3), 400-404.

Matson, J. L., Mayville, E. A., Lott, J. D., Bielecki, J., & Logan, R. (2003). A comparison of social and adaptive functioning in persons with psychosis, autism, and severe or profound mental retardation. *Journal of Developmental and Physical Disabilities, 15*(1), 57-65.

Matson, J. L., & Minshawi, N. F. (2006). *Early intervention for autism spectrum disorders: A critical analysis* (Vol. 1). Cambridge, MA: Elsevier Science.

Matson, J. L., & Minshawi, N. F. (2007). Functional assessment of challenging behavior: Toward a strategy for applied settings. *Research in Developmental Disabilities, 28*(4), 353-361.

Matson, J. L., Neal, D., Fodstad, J. C., & Hess, J. A. (2010). The relation of social behaviours and challenging behaviours in infants and toddlers with autism spectrum disorders. *Developmental Neurorehabilitation, 13*(3), 164-169.

Matson, J. L., & Nebel-Schwalm, M. S. (2007a). Comorbid psychopathology with autism spectrum disorder in children: An overview. *Research in Developmental Disabilities, 28*(4), 341-352.

Matson, J. L., & Nebel-Schwalm, M. S. (2007b). Assessing challenging behaviors in children with autism spectrum disorders: A review. *Research in Developmental Disabilities, 28*(6), 567-579.

Matson, J. L., & Shoemaker, M. (2009). Intellectual disability and its relationship to autism spectrum disorders. *Research in Developmental Disabilities, 30*(6), 1107-1114.

Matson, J. L., & Sipes, M. (2010). Methods of early diagnosis and tracking for autism and pervasive developmental disorder not otherwise specified (PDDNOS). *Journal of Developmental and Physical Disabilities, 22*(4), 343-358.

Matson, J. L., & Smith, K. R. (2008). Current status of intensive behavioral interventions for young children with autism and PDD-NOS. *Research in Autism Spectrum Disorders, 2*(1), 60-74.

Matson, J. L., & Swiezy, N. (1994). Social skills training with autistic children. *Autism in Children and Adults: Etiology, Assessment and Intervention*, 241-260.

Matson, J. L., & Tureck, K. (2012). Early diagnosis of autism: Current status of the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT-Parts 1, 2, and 3). *Research in Autism Spectrum Disorders*, *6*(3), 1135-1141.

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

Matson, J. L., Wilkins, J., & Fodstad, J. C. (2011). The validity of the baby and infant screen for children with autism traits: Part 1 (BISCUIT: Part 1). *Journal of Autism and Developmental Disorders*, *41*(9), 1139-1146.

Matson, J. L., Wilkins, J., & Gonzalez, M. (2008). Early identification and diagnosis in autism spectrum disorders in young children and infants: How early is too early?. *Research in Autism Spectrum Disorders*, *2*(1), 75-84.

Matson, J. L., Wilkins, J., & Macken, J. (2008). The relationship of challenging behaviors to severity and symptoms of autism spectrum disorders. *Journal of Mental Health Research in Intellectual Disabilities*, *2*(1), 29-44.

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

Matson, J. L., Wilkins, J., Sharp, B., Knight, C., Sevin, J. A., & Boisjoli, J. A. (2009b). Sensitivity and specificity of the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT): Validity and cutoff scores for autism and PDD-NOS in toddlers. *Research in Autism Spectrum Disorders*, *3*(4), 924-930.

Matsuura, T., Sutcliffe, J. S., Fang, P., Galjaard, R. J., Jiang, Y. H., Benton, C. S., . . . Beaudet, A. L. (1997). De novo truncating mutations in E6-AP ubiquitin-protein ligase gene (UBE3A) in Angelman syndrome. *Nature Genetics*, *15*(1), 74-77.

Mattila, M. L., Kielinen, M., Linna, S. L., Jussila, K., Ebeling, H., Bloigu, R., . . . Moilanen, I. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison With DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(6), 583-592.

Mauk, J. E. (1993). Autism and pervasive developmental disorders. *Pediatric Clinics of North America*, *40*(3), 567-578.

Maurice, C., Green, G., & Foxx, R. M. (Eds.). (2001). *Making a difference: Behavioral intervention for autism*. Austin, Tx: Pro-Ed.

Mayes, S. D., & Calhoun, S. L. (2003). Relationship between Asperger syndrome and high-functioning autism. *Learning and Behavior Problems in Asperger Syndrome*, 15-34.

Mayes, S. D., Black, A., & Tierney, C. D. (2013). DSM-5 under-identifies PDDNOS: Diagnostic agreement between the DSM-5, DSM-IV, and Checklist for Autism Spectrum Disorder. *Research in Autism Spectrum Disorders*, *7*(2), 298-306.

Mazefsky, C. A., & Oswald, D. P. (2006). The discriminative ability and diagnostic utility of the ADOS-G, ADI-R, and GARS for children in a clinical setting. *Autism*, *10*(6), 533-549.

Mazefsky, C. A., McPartland, J. C., Gastgeb, H. Z., & Minshew, N. J. (2013). Brief report: Comparability of DSM-IV and DSM-5 ASD research samples. *Journal of Autism and Developmental Disorders*, *43*(5), 1236-1242.

McCarthy, J., Hemmings, C., Kravariti, E., Dworzynski, K., Holt, G., Bouras, N., & Tsakanikos, E. (2010). Challenging behavior and co-morbid psychopathology in adults with intellectual disability and autism spectrum disorders. *Research in Developmental Disabilities*, *31*(2), 362-366.

McClintock, K., Hall, S., & Oliver, C. (2003). Risk markers associated with challenging behaviours in people with intellectual disabilities: A meta-analytic study. *Journal of Intellectual Disability Research*, *47*(6), 405-416.

McCracken, J. T., McGough, J., Shah, B., Cronin, P., Hong, D., Aman, M. G., . . . McMahon, D. (2002). Risperidone in children with autism and serious behavioral problems. *New England Journal of Medicine*, *347*(5), 314-321.

McEvoy, R. E., Rogers, S. J., & Pennington, B. F. (1993). Executive function and social communication deficits in young autistic children. *Journal of Child Psychology and Psychiatry*, *34*, 563–578.

McGee, G. G., Morrier, M. J., & Daly, T. (1999). An incidental teaching approach to early intervention for toddlers with autism. *Research and Practice for Persons with Severe Disabilities*, *24*(3), 133-146.

McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(4), 368-383.

MedCalc (Version 13.3.3) [Computer software]. Mariakerke, Belgium: MedCalc Software bvba.

Meisels, S. J., Henderson, L. W., Liaw, F. R., Browning, K., & Have, T. T. (1993). New evidence for the effectiveness of the early screening inventory. *Early Childhood Research Quarterly*, *8*(3), 327-346.

Mercier, C., Mottron, L., & Belleville, S. (2000). A psychosocial study on restricted interests in high functioning persons with pervasive developmental disorders. *Autism*, *4*(4), 406-425.

Mesibov, G. B., Schopler, E., Schaffer, B., & Michal, N. (1989). Use of the Childhood Autism Rating Scale with autistic adolescents and adults. *Journal of the American Academy of Child and Adolescent Psychiatry, 28*, 538-541.

Metz, C. E. (1978). Basic principles of ROC analysis. *Seminars in Nuclear Medicine, 8*(4), 283-298.

Miano, S., Bruni, O., Elia, M., Trovato, A., Smerieri, A., Verrillo, E., . . . Ferri, R. (2007). Sleep in children with autistic spectrum disorder: A questionnaire and polysomnographic study. *Sleep Medicine, 9*(1), 64-70.

Mildenberger, K., Sitter, S., Noterdaeme, M., & Amorosa, H. (2001). The use of the ADI-R as a diagnostic tool in the differential diagnosis of children with infantile autism and children with a receptive language disorder. *European Child and Adolescent Psychiatry*, *10*(4), 248-255.

Miles, J. H., Hadden, L. L., Takahashi, T. N., & Hillman, R. E. (2000). Head circumference is an independent clinical finding associated with autism. *American Journal of Medical Genetics*, *95*(4), 339-350.

Minshew, N. J., Goldstein, G., & Siegel, D. J. (1997). Neuropsychologic functioning in autism: Profile of a complex information processing disorder. *Journal of the International Neuropsychological Society, 3*, 303–316.

Mitchell, S., Brian, J., Zwaigenbaum, L., Roberts, W., Szatmari, P., Smith, I., & Bryson, S. (2006). Early language and communication development of infants later diagnosed with autism spectrum disorder. *Journal of Developmental and Behavioral Pediatrics*, *27*(2), 69-78.

Moore, V., & Goodson, S. (2003). How well does early diagnosis of autism stand the test of time? Follow-up study of children assessed for autism at age 2 and development of an early diagnostic service. *Autism*, *7*(1), 47-63.

Morgan, L., Wetherby, A. M., & Barber, A. (2008). Repetitive and stereotyped movements in children with autism spectrum disorders late in the second year of life. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 49*, 826– 837.

Morton, J. F., & Campbell, J. M. (2008). Information source affects peers' initial attitudes toward autism. *Research in Developmental Disabilities*, *29*(3), 189-201.

Mudford, O. C., Arnold-Saritepe, A. M., Phillips, K. J., Locke, J. M., Ho, I. C. S., & Taylor, S. A. (2008). Challenging behaviors. *Clinical Assessment and Intervention for Autism Spectrum Disorders*, 267-297.

Muhle, R., Trentacoste, S. V., & Rapin, I. (2004). The genetics of autism. *Pediatrics*, *113*(5), 472-486.

Mukaddes, N. M., & Fateh, R. (2010). High rates of psychiatric co-morbidity in individuals with Asperger's disorder. *World Journal of Biological Psychiatry*, *11*(22), 486-492.

Mukaddes, N. M., & Topcu, Z. (2006). Case report: Homicide by a 10-year-old girl with autistic disorder. *Journal of Autism and Developmental Disorders*, *36*(4), 471-474.

Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1986). Defining the social deficits of autism: The contribution of non-verbal communication measures. *Journal of Child Psychology and Psychiatry, 27,* 657–669.

Mundy, P., Sigman, M., Ungerer, J., & Sherman, T. (1987). Nonverbal communication and play correlates of language development in autistic children. *Journal of Autism and Developmental Disorders*, *17*(3), 349–364.

Muris, P., Steerneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, *12*(4), 387-393.

Murphy, G. H., Beadle-Brown, J., Wing, L., Gould, J., Shah, A., & Holmes, N. (2005b). Chronicity of challenging behaviours in people with severe intellectual disabilities and/or autism: A total population sample. *Journal of Autism and Developmental Disorders*, *35*(4), 405-418.

Murphy, O., Healy, O., & Leader, G. (2009). Risk factors for challenging behaviors among 157 children with autism spectrum disorder in Ireland. *Research in Autism Spectrum Disorders*, *3*(2), 474-482.

Myers, S. M., & Johnson, C. P. (2007). American academy of pediatrics council on children with disabilities. Management of children with autism spectrum disorders. *Pediatrics, 120*, 1162–1182.

Naber, F., Swinkels, S., Buitelaar, J., Bakermans-Kranenburg, M., Van IJzendoorn, M., Dietz, C., . . . Van Engeland, H. (2007). Attachment in toddlers with autism and other developmental disorders. *Journal of Autism and Developmental Disorders, 37*, 1123-1138.

Nader, R., Oberlander, T. F., Chambers, C. T., & Craig, K. D. (2004). Expression of pain in children with autism. *The Clinical Journal of Pain*, *20*(2), 88-97.

Nakamine, A., Ouchanov, L., Jiménez, P., Manghi, E. R., Esquivel, M., Monge, S., . . . McInnes, L. A. (2008). Duplication of 17 (p11. 2p11. 2) in a male child with autism and severe language delay. *American Journal of Medical Genetics Part A*, *146*(5), 636-643.

Newborg, J. (2005). *Battele developmental inventory-second edition*. Itasca, IL: Riverside.

Newschaffer, C. J., & Curran, L. K. (2003). Autism: An emerging public health problem. *Public Health Reports*, *118*(5), 393.

Newschaffer, C. J., Croen, L. A., Daniels, J., Giarelli, E., Grether, J. K., Levy, S. E., . . . Windham, G. C. (2007). The epidemiology of autism spectrum disorders. *Annual Review of Public Health*, *28*, 235-258.

Nishitani, N., Avikainen, S., & Hari, R. (2004). Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Annals of Neurology*, *55*(4), 558-562.

Nissen, J. M. J. F., & Haveman, M. J. (1997). Mortality and avoidable death in people with severe self-injurious behaviour: Results of a Dutch study. *Journal of Intellectual Disability Research*, *41*(3), 252-257.

Noens, I., & Van Berckelaer-Onnes, I. A. (2005). Captured by details: Sense-making, language and communication in autism. *Journal of Communication Disorders, 38*, 123-141.

Nydén, A., Gillberg, C., Hjelmquist, E., & Heiman, M. (1999). Executive function/attention deficits in boys with Asperger syndrome, attention disorder and reading/writing disorder. *Autism*, *3*(3), 213-228.

O'Brien, G., & Pearson, J. (2004). Autism and learning disability. *Autism*, *8*(2), 125-140.

O'Riordan, M., & Passetti, F. (2006). Discrimination in autism within different sensory modalities. *Journal of Autism and Developmental Disorders, 36*, 665–675.

Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, *24*(2), 190-198.

Obuchowski, N. A. (2000). Sample size tables for receiver operating characteristic studies. *American Journal of Roentgenology*, *175*(3), 603-608.

Obuchowski, N. A., Lieber, M. L., & Wians, F. H. (2004). ROC curves in clinical chemistry: Uses, misuses, and possible solutions. *Clinical Chemistry*, *50*(7), 1118-1125.

Obuchowski, N. A., & McClish, D. K. (1997). Sample size determination for diagnostic accuracy studies involving binormal ROC curve indices. *Statistics in Medicine*, *16*(13), 1529-1542.

Oliver, C., Murphy, G. H., & Corbett, J. A. (1987). Self-injurious behaviour in people with mental handicap: A total population study. *Journal of Intellectual Disability Research*, *31*(2), 147-162.

Ornitz, E. M., Guthrie, D., & Farley, A. J. (1978). The early symptoms of childhood autism. In G. Serban (Ed.), *Cognitive Defects in the Development of Mental Illness* (pp. 24–42). New York: Brunner/Mazel.

Orr, J. M., Sackett, P. R., & Dubois, C. L. (1991). Outlier detection and treatment in I/O psychology: A survey of researcher beliefs and an empirical illustration. *Personnel Psychology*, *44*(3), 473-486.

Osterling, J., & Dawson, G. (1994). Early recognition of children with autism: A study of first birthday home videotapes. *Journal of Autism and Developmental Disorders*, *24*(3), 247-257.

Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology*, *14*(2), 239-251.

Ozonoff, S., Goodlin-Jones, B. L., & Solomon, M. (2005). Evidence-based assessment of autism spectrum disorders in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, *34*(3), 523-540.

Ozonoff, S., & Miller, J. N. (1995). Teaching theory of mind: A new approach to social skills training for individuals with autism. *Journal of Autism and Developmental Disorders*, *25*(4), 415-433.

Paavonen, E. J., Vehkalahti, K., Vanhala, R., von Wendt, L., Nieminen-von Wendt, T., & Aronen, E. T. (2008). Sleep in children with Asperger syndrome. *Journal of Autism and Developmental Disorders*, *38*(1), 41-51.

Paclawskyj, T. R., Matson, J. L., Bamburg, J. W., & Baglio, C. S. (1997). A comparison of the Diagnostic Assessment for the Severely Handicapped-II (DASH-II) and the Aberrant Behavior Checklist (ABC). *Research in Developmental Disabilities*, *18*(4), 289-298.

Page, J., & Boucher, J. (1998). Motor impairments in children with autistic disorder. *Child Language Teaching and Therapy, 14*, 233–259.

Pai, A. L., Patiño-Fernández, A. M., McSherry, M., Beele, D., Alderfer, M. A., Reilly, A. T., . . . Kazak, A. E. (2008). The Psychosocial Assessment Tool (PAT2. 0): Psychometric properties of a

screener for psychosocial distress in families of children newly diagnosed with cancer. *Journal of Pediatric Psychology*, *33*(1), 50-62.

Palmer, R. F., Blanchard, S., Jean, C. R., & Mandell, D. S. (2005). School district resources and identification of children with autistic disorder. *American Journal of Public Health*, *95*(1), 125.

Palmer, R. F., Walker, T., Mandell, D., Bayles, B., & Miller, C. S. (2010). Explaining low rates of autism among Hispanic schoolchildren in Texas. *American Journal of Public Health*, *100*(2), 270.

Parker, J. G., & Asher, S. R. (1987). Peer relations and later personal adjustment: Are low-accepted children at risk?. *Psychological Bulletin*, *102*(3), 357.

Paterson, C. R., & Arco, L. (2007). Using video modeling for generalizing toy play in children with autism. *Behavior Modification*, *31*(5), 660-681.

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 37*, 51–87.

Pereira, A., Riesgo, R. S., & Wagner, M. B. (2008). Childhood autism: translation and validation of the Childhood Autism Rating Scale for use in Brazil. *Jornal De Pediatria*, *84*(6), 487-494.

Perkins, N. J., & Schisterman, E. F. (2005). The Youden Index and the Optimal Cut-Point Corrected for Measurement Error. *Biometrical Journal*, *47*(4), 428-441.

Perry, A., Condillac, R. A., Freeman, N. L., Dunn-Geier, J., & Belair, J. (2005). Multi-site study of the Childhood Autism Rating Scale (CARS) in five clinical groups of young children. *Journal of Autism and Developmental Disorders*, *35*(5), 625-634.

Perry, A., & Freeman, N. L. (1996, March). *Psychometric properties of the Childhood Autism Rating Scale* (*CARS*). Poster presented at the Ontario Association on Developmental Disabilities Research Special Interest Group Research Day, Toronto, ON.

Perry, D. W., Marston, G. M., Hinder, S. A., Munden, A. C., & Roy, A. (2001). The phenomenology of depressive illness in people with a learning disability and autism. *Autism, 5*, 265–275.

Peters, S. U., Beaudet, A. L., Madduri, N., & Bacino, C. A. (2004). Autism in Angelman syndrome: Implications for autism research. *Clinical Genetics*, *66*(6), 530-536.

Pilling, S., Bebbington, P., Kuipers, E., Garety, P., Geddes, J., Martindale, B., . . . Morgan, C. (2002). Psychological treatments in schizophrenia: II. Meta-analyses of randomized controlled trials of social skills training and cognitive remediation. *Psychological Medicine*, *32*(05), 783-791.

Polimeni, M. A., Richdale, A. L., & Francis, A. J. P. (2005). A survey of sleep problems in autism, Asperger's disorder and typically developing children. *Journal of Intellectual Disability Research, 49*(4), 260-268.

Prizant, B., & Duchan, J. (1981). The functions of immediate echolalia in autistic children. *Journal of Speech and Hearing Disorders, 46*, 241–249.

Prupas, A., & Reid, G. (2001). Effects of Exercise Frequency on Stereotypic Behaviors of Children with Developmental Disabilities. *Education and Training in Mental Retardation and Developmental Disabilities, 36*(2), 196-206.

Puig-Antich, J., & Chambers, W. (1978). *The schedule for affective disorders and schizophrenia for school-age children* (*Kiddie-SADS*). New York, NY: New York State Psychiatric Institute.

Ramachandran, V. S., & Oberman, L. M. (2006). Broken mirrors: A theory of autism. *Scientific American, 295*(5), 62-69.

Ramey, C. T., & Ramey, S. L. (1998). Early intervention and early experience. *American Psychologist, 53*(2), 109.

Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *American Journal of Orthopsychiatry, 19*(1), 130.

Rapin, I. (2003). Value and limitations of preschool cognitive tests, with an emphasis on longitudinal study of children on the autistic spectrum. *Brain and Development, 25*(8), 546-548.

Reddy, K. S. (2005). Cytogenetic abnormalities and fragile-X syndrome in Autism Spectrum Disorder. *BMC Medical Genetics, 6*(1), 3.

Reese, R. M., Richman, D. M., Belmont, J. M., & Morse, P. (2005). Functional characteristics of disruptive behavior in developmentally disabled children with and without autism. *Journal of Autism and Developmental Disorders, 35*(4), 419-428.

Regier, D. A., Kuhl, E. A., & Kupfer, D. J. (2013). The DSM-5: Classification and criteria changes. *World Psychiatry, 12*(2), 92-98.

Reichow, B., & Wolery, M. (2009). Comprehensive synthesis of early intensive behavioral interventions for young children with autism based on the UCLA young autism project model. *Journal of Autism and Developmental Disorders, 39*(1), 23-41.

Reiser, B. (2000). Measuring the effectiveness of diagnostic markers in the presence of measurement error through the use of ROC curves. *Statistics in Medicine 19*(16), 2115–29.

Reiss, S., & Szyszko, J. (1983). Diagnostic overshadowing and professional experience with mentally retarded persons. *American Journal of Mental Deficiency*.

Remington, B., Hastings, R. P., Kovshoff, H., degli Espinosa, F., Jahr, E., Brown, T., . . . Ward, N. (2007). Early intensive behavioral intervention: Outcomes for children with autism and their parents after two years. *American Journal on Mental Retardation, 112*(6), 418-438.

Reznick, J. S. (2006). Early identification of autism: how early can we go?. *Seminars in Speech and Language, 27*(3), 143-160.

Richdale, A. L., & Prior, M. R. (1995). The sleep/wake rhythm in children with autism. *European Child and Adolescent Psychiatry, 4*(3), 175-186.

Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: Prevalence, nature, and possible biopsychosocial etiologies. *Sleep Medicine Reviews, 13*(6), 403-411.

Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and repetitive behaviors in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37,* 73-85.

Ringdahl, J. E., Call, N. A., Mews, J. B., Boelter, E. W., & Christensen, T. J. (2008). Assessment and treatment of aggressive behavior without a clear social function. *Research in Developmental Disabilities, 29*(4), 351-362.

Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27,* 169-192.

Robb, A. S. (2010). Managing irritability and aggression in autism spectrum disorders in children and adolescents. *Developmental Disabilities Research Reviews, 16*(3), 258-264.

Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The Modified Checklist for Autism in Toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 31*(2), 131-144.

Rodier, P. M. (2000). The early origins of autism. *Scientific American, 282*(2), 56.

Rodier, P. M. (2002). Converging evidence for brain stem injury in autism. *Development and Psychopathology, 14*(03), 537-557.

Rogers, S. J. (2004). Developmental regression in autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews, 10*(2), 139-143.

Rogers, S. J., & DiLalla, D. L. (1990). Age of symptom onset in young children with pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*(6), 863-872.

Rogers, S., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders, 33*(6), 631–642.

Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child and Adolescent Psychology, 37*(1), 8-38.

Rojahn, J. (1984). Self-injurious behavior in institutionalized, severely/profoundly retarded adults: Prevalence and staff agreement. *Journal of Behavioral Assessment, 6,* 13–27.

Rojahn, J., Matson, J. L., Lott, D., Esbensen, A. J., & Smalls, Y. (2001). The Behavior Problems Inventory: An instrument for the assessment of self-injury, stereotyped behavior, and aggression/destruction in individuals with developmental disabilities. *Journal of Autism and Developmental Disorders, 31*(6), 577-588.

Rojahn, J., Matson, J. L., Mahan, S., Fodstad, J. C., Knight, C., Sevin, J. A., & Sharp, B. (2009). Cutoffs, norms, and patterns of problem behaviors in children with an ASD on the Baby and Infant Screen

for Children with aUtIsm Traits (BISCUIT-Part 3). *Research in Autism Spectrum Disorders*, *3*(4), 989-998.

Rolider, A., & Houten, R. (1985). Movement suppression time-out for undesirable behavior in psychotic and severely develop mentally delayed children. *Journal of Applied Behavior Analysis*, *18*(4), 275-288.

Rosenberg, R. E., Daniels, A. M., Law, J. K., Law, P. A., & Kaufmann, W. E. (2009). Trends in autism spectrum disorder diagnoses: 1994–2007. *Journal of Autism and Developmental Disorders*, *39*(8), 1099-1111.

Rosenhall, U., Nordin, V., Sandström, M., Ahlsen, G., & Gillberg, C. (1999). Autism and hearing loss. *Journal of Autism and Developmental Disorders*, *29*(5), 349-357.

Rumsey, J. M., Rapoport, J. L., & Sceery, W. R. (1985). Autistic children as adults: Psychiatric, social, and behavioral outcomes. *Journal of the American Academy of Child Psychiatry*, *24*(4), 465-473.

Russo, D. C., Cataldo, M. F., & Cushing, P. J. (1981). Compliance training and behavioral covariation in the treatment of multiple behavior problems. *Journal of Applied Behavior Analysis*, *14*, 209-222.

Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia*, 2, 315-337.

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

Rutter, M., Le Couteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles, CA: Western Psychological Services.

Rutter, M., Lebovici, S., Eisenberg, L., Sneznevskij, A. V., Sadoun, R., Brooke, E., & Lin, T. Y. *(*1969*).* A triaxial classification of mental disorders in children. *Journal of Child Psychology and Psychiatry, 10*, 41-61.

Rutter, M., & Lord, C. (1987). Language disorders associated with psychiatric disturbances. In W. Yule & M. Rutter (Eds.), *Language development and disorders* (pp. 206–233). Philadelphia, Pa: Lippincott.

Rydz, D., Srour, M., Oskoui, M., Marget, N., Shiller, M., Birnbaum, R., . . . Shevell, M. I. (2006). Screening for developmental delay in the setting of a community pediatric clinic: A prospective assessment of parent-report questionnaires. *Pediatrics*, *118*(4), 1178-1186.

Saah, A. J., & Hoover, D. R. (1997). "Sensitivity" and "specificity" reconsidered: the meaning of these terms in analytical and diagnostic settings. *Annals of Internal Medicine*, *126*(1), 91-94.

Sadeh, A., Gruber, R., & Raviv, A. (2002). Sleep, neurobehavioral functioning, and behavior problems in school-age children. *Child Development*, *73*(2), 405-417.

Samaco, R. C., Hogart, A., & LaSalle, J. M. (2005). Epigenetic overlap in autism-spectrum neurodevelopmental disorders: MECP2 deficiency causes reduced expression of UBE3A and GABRB3. *Human Molecular Genetics*, *14*(4), 483-492.

Sand, N., Silverstein, M., Glascoe, F. P., Gupta, V. B., Tonniges, T. P., & O'Connor, K. G. (2005). Pediatricians' reported practices regarding developmental screening: Do guidelines work? Do they help?. *Pediatrics, 116*(1), 174-179.

Santangelo, S. L., & Folstein, S. E. (1999). Autism: A genetic perspective. *Neurodevelopmental Disorders*, 431-447.

Saracino, J., Noseworthy, J., Steiman, M., Reisinger, L., & Fombonne, E. (2010). Diagnostic and assessment issues in autism surveillance and prevalence. *Journal of Developmental and Physical Disabilities, 22*(4), 317-330.

Schaefer, G. B., & Lutz, R. E. (2006). Diagnostic yield in the clinical genetic evaluation of autism spectrum disorders. *Genetics in Medicine, 8*(9), 549-556.

Schaefer, G. B., Mendelsohn, N. J., & Professional Practice and Guidelines Committee (2008). Clinical genetics evaluation in identifying the etiology of autism spectrum disorders. *Genetics in Medicine, 10*(4), 301-305.

Schmit, J., Alper, S., Raschke, D., & Ryndak, D. (2000). Effects of using a photographic cueing package during routine school transitions with a child who has autism. *Mental Retardation, 38*(2), 131-137.

Schoen, S. F. (1983). The status of compliance technology: Implications for programming. *The Journal of Special Education, 17*, 483-496.

Schoonjans, F., Zalata, A., Depuydt, C. E., & Comhaire, F. H. (1995). MedCalc: A new computer program for medical statistics. *Computer Methods and Programs in Biomedicine, 48*(3), 257-262.

Schopler, E. (1985). Convergence of learning disability, higher-level autism, and Asperger's syndrome. *Journal of Autism and Developmental Disorders, 15*(4), 359-360.

Schopler, E. C., Reichler, R., & Renner, B. (1988). *The childhood autism rating scale* (*CARS*). Los Angeles, CA: Western Psychological Services.

Schopler, E. C., Van Bourgondien, M., Wellman, J., & Love, S. (2010). *Childhood autism rating scale—second edition* (*CARS2*) *manual*. Los Angeles, CA: Western Psychological Services.

Schreck, K. A., & Mulick, J. A. (2000). Parental report of sleep problems in children with autism. *Journal of Autism and Developmental Disorders, 30*(2), 127-135.

Schreck, K. A., & Williams, K. (2006). Food preferences and factors influencing food selectivity for children with autism spectrum disorders. *Research in Developmental Disabilities, 27*(4), 353-363.

Schreck, K. A., Williams, K., & Smith, A. F. (2004). A comparison of eating behaviors between children with and without autism. *Journal of Autism and Developmental Disorders, 34*(4), 433-438.

Schreibman, L., & Koegel, R. L. (2005). Training for parents of children with autism: Pivotal responses, generalization, and individualization of interventions. In E. D. Hibbs & P. S. Jensen (Eds.), *Psychosocial treatment for child and adolescent disorders: Empirically based strategies for*

clinical practice (2nd edition, pp. 605–631). Washington, DC: American Psychological Association.

Schreibman, L., Whalen, C., & Stahmer, A. C. (2000). The use of video priming to reduce disruptive transition behavior in children with autism. *Journal of Positive Behavior Interventions, 2*(1), 3-11.

Schwarz, S. M. (2003). Feeding disorders in children with developmental disabilities. *Infants and Young Children, 16,* 317–330.

Sevin, J. A., Matson, J. L., Coe, D. A., Fee, V. E., & Sevin, B. M. (1991). A comparison and evaluation of three commonly used autism scales. *Journal of Autism and Developmental Disorders*, *21*(4), 417-432.

Shao, Y., Cuccaro, M. L., Hauser, E. R., Raiford, K. L., Menold, M. M., Wolpert, C. M., … & Pericak-Vance, M. A. (2003). Fine mapping of autistic disorder to chromosome 15q11-q13 by use of phenotypic subtypes. *American Journal of Human Genetics, 72*, 539– 548.

Shao, Y., Raiford, K. L., Wolpert, C. M., Cope, H. A., Ravan, S. A., Ashley-Koch, A. A., . . . Pericak-Vance, M. A. (2002). Phenotypic homogeneity provides increased support for linkage on chromosome 2 in autistic disorder. *American Journal of Human Genetics*, *70*(4), 1058-1061.

Shaw, E., & Hatton, D. (2009). Screening and early identification of autism spectrum disorders updated queries: An occasional paper compiling states' approaches to current topics. *National Early Childhood Technical Assistance Center (NECTAC)*.

Shaw, R. J., Garcia, M., Thorn, M., Farley, C. A., & Flanagan, G. (2003). Treatment of feeding disorders in children with Down syndrome. *Clinical Child Psychology and Psychiatry, 8,* 105–117.

Sheldon, S. H., Ferber, R., & Kryger, M. H. (Eds.). (2005). *Principles and Practice of Pediatric Sleep Medicine*. Elsevier Health Sciences.

Shelov, S. P., & Altmann, T. (Eds.). (2009). *Caring for your baby and young child: Birth to age 5* (5th ed.). New York, NY: Bantam Books.

Shelov, S. P., & Hannemann, R. E. (Eds.). (1991). *Caring for your baby and young child: Birth to age 5*. New York, NY: Bantam Books.

Shevell, M. I., Ashwal, S., Donley, D., Flint, J., Gingold, M., Hirtz, D., . . . Sheth, R. D. (2003). Practice parameter: Evaluation of the child with global developmental delay report of the quality standards subcommittee of the American Academy of Neurology and the practice committee of the Child Neurology Society. *Neurology, 60*(3), 367-380.

Shevell, M. I., Majnemer, A., Rosenbaum, P., & Abrahamowicz, M. (2001). Etiologic yield of autistic spectrum disorders: a prospective study. *Journal of Child Neurology*, *16*(7), 509-512.

Shore, R. (1997). *Rethinking the brain: New insights into early development*. New York, NY: Families and Work Institute.

Siegel, B., Pliner, C., Eschler, J., & Elliot, G. (1988). How children with autism are diagnosed: Difficulties in identification of children with multiple developmental delays. *Developmental and Behavioral Pediatrics, 9*, 199–204.

Sigman, M. (1998). The Emanuel Miller memorial lecture 1997: Change and continuity in the development of children with autism. *Journal of Child Psychology and Psychiatry, 19*(6), 817–827.

Sigman, M., & McGovern, C. (2005). Improvement in cognitive and language skills from preschool to adolescence in autism. *Journal of Autism and Developmental Disorders, 35*(1), 15–23.

Sigman, M., & Ungerer, J. A. (1984). Cognitive and language skills in autistic, mentally retarded, and normal children. *Developmental Psychology, 20,* 293–302.

Sigman, M., Dijamco, A., Gratier, M., & Rozga, A. (2004). Early detection of core deficits in autism. *Mental Retardation and Developmental Disabilities Research Reviews, 10*, 221–233.

Sikora, D. M., Hall, T. A., Hartley, S. L., Gerrard-Morris, A. E., & Cagle, S. (2008). Does parent report of behavior differ across ADOS-G classifications: Analysis of scores from the CBCL and GARS. *Journal of Autism and Developmental Disorders*, *38*(3), 440-448.

Simonoff, E., Pickles, A., Charman, T., Chandler, S., Loucas, T., & Baird, G. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(8), 921-929.

Sipes, M., Rojahn, J., Turygin, N., Matson, J. L., & Tureck, K. (2011). Comparison of problem behaviors in atypically developing infants and toddlers as assessed with the Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT). *Developmental Neurorehabilitation, 14*, 261-266.

Sleep, P. (2006). Behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep*, *29*(10), 1263.

Smith, A., Yarwood, J., & Salisbury, D. M. (2007). Tracking mothers' attitudes to MMR immunization 1996–2006. *Vaccine*, *25*(20), 3996-4002.

Smith, T., Groen, A. D., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *American Journal on Mental Retardation*, *105*(4), 269-285.

Spence, S. J. (2004). The genetics of autism. *Seminars in Pediatric* Neurology, 11(3), 196-204.

Sponheim, E., & Skjeldal, O. (1998). Autism and related disorders: Epidemiological findings in a Norwegian study using ICD-10 diagnostic criteria. *Journal of Autism and Developmental Disorders*, *28*(3), 217-227.

Starr, E., Szatmari, P., Bryson, S., & Zwaigenbaum, L. (2003). Stability and change among high-functioning children with pervasive developmental disorders: A 2-year outcome study. *Journal of Autism and Developmental Disorders, 33*, 15–22.

Stella, J., Mundy, P., & Tuchman, R. (1999). Social and nonsocial factors in the Childhood Autism Rating Scale. *Journal of Autism and Developmental Disorders, 29*(4), 307-317.

Sterling, L., Dawson, G., Estes, A., & Greenson, J. (2008). Characteristics associated with presence of depressive symptoms in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 38*(6), 1011-1018.

Stevenson, R. E., Schroer, R. J., Skinner, C., Fender, D., & Simensen, R. J. (1997). Autism and macrocephaly. *The Lancet, 349*(9067), 1744-1745.

Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism, 10*(1), 103-116.

Stone, W. L., Coonrod, E. E., & Ousley, O. Y. (2000). Brief report: screening tool for autism in two-year-olds (STAT): Development and preliminary data. *Journal of Autism and Developmental Disorders, 30*(6), 607-612.

Stone, W. L., Coonrod, E. E., Turner, L. M., & Pozdol, S. L. (2004). Psychometric properties of the STAT for early autism screening. *Journal of Autism and Developmental Disorders, 34*(6), 691-701.

Stone, W. L., Lee, E. B., Ashford, L., Brissie, J., Hepburn, S. L., Coonrod, E. E., & Weiss, B. H. (1999). Can autism be diagnosed accurately in children under 3 years?. *Journal of Child Psychology and Psychiatry, 40*(2), 219-226.

Stone, W. L., Lemanek, K. L., Fishel, P. T., Fernandez, M. C., & Altemeier, W. A. (1990). Play and imitation skills in the diagnosis of autism in young children. *Pediatrics, 86,* 267–272.

Stone, W. L., McMahon, C. R., & Henderson, L. M. (2008). Use of the Screening Tool for Autism in Two-Year-Olds (STAT) for children under 24 months: An exploratory study. *Autism, 12*(5), 557-573.

Stone, W. L. & Ousley, O. Y. (1997). *STAT Manual: Screening Tool for Autism in Two-Year-Olds.* Unpublished manuscript, Vanderbilt University.

Stone, W., Ousley, O. Y., & Littleford, C. (1997a). Motor imitation in young children with autism: What's the object? *Journal of Abnormal Child Psychology, 25*(6), 475–485.

Stone, W., Ousley, O., Yoder, P., Hogan, K., & Hepburn, S. (1997b). Nonverbal communication in 2- and 3-year old children with autism. *Journal of Autism and Developmental Disorders, 27*, 677–696.

Sturmey, P., Fink, C., & Sevin, J. A. (1993). The behavior problem inventory: A replication and extension of its psychometric properties. *Journal of Developmental and Physical Disabilities, 5*(4), 327-336.

Sturmey, P., Matson., J. L., & Sevin, J. A. (1992). Brief report: Analysis of the internal consistency of three autism scales. *Journal of Autism and Developmental Disorders, 22*, 321-328.

Sturmey, P., Sevin, J. A., & Williams, D. E. (1995). The Behavior Problem Inventory: A further replication of its factor structure. *Journal of Intellectual Disability Research, 39*(4), 353-356.

Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L. E., Aman, M. G., McDougle, C. J., . . . Vitiello, B. (2008). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology, 36*(1), 117-128.

Sullivan, M., Finelli, J., Marvin, A., Garrett-Mayer, E., Bauman, M., & Landa, R. (2007). Response to joint attention in toddlers at risk for autism spectrum disorder: a prospective study. *Journal of Autism and Developmental Disorders*, *37*(1), 37-48.

Symes, M. D., Remington, B., Brown, T., & Hastings, R. P. (2006). Early intensive behavioral intervention for children with autism: Therapists' perspectives on achieving procedural fidelity. *Research in Developmental Disabilities*, *27*(1), 30-42.

Szatmari, P. (1992). The validity of autistic spectrum disorders: a literature review. *Journal of Autism and Developmental Disorders, 22*(4), 583-600.

Szatmari, P., Georgiades, S., Bryson, S., Zwaigenbaum, L., Roberts, W., Mahoney, W., . . . Tuff, L. (2006). Investigating the structure of the restricted, repetitive behaviours and interests domain of autism. *Journal of Child Psychology and Psychiatry*, *47*(6), 582-590.

Tabachnick, B., Fidell, L. (2007). *Using multivariate statistics: Fifth edition*. Boston, MA: Pearson Education, Inc.

Tabuchi, K., Blundell, J., Etherton, M. R., Hammer, R. E., Liu, X., Powell, C. M., & Südhof, T. C. (2007). A neuroligin-3 mutation implicated in autism increases inhibitory synaptic transmission in mice. *Science*, *318*(5847), 71-76.

Tachimori, H., Osada, H., & Kurita, H. (2003). Childhood Autism Rating Scale− Tokyo Version for screening pervasive developmental disorders. *Psychiatry and Clinical Neurosciences*, *57*(1), 113-118.

Tager-Flusberg, H. (1985). Psycholinguistic approaches to language and communication in autism. In E. Schopler & G. B. Mesibov (Eds.), *Communication problems in autism* (pp. 69-87). New York, NY: Springer Publishing.

Tager-Flusberg, H., Joseph, R., & Folstein, S. (2001). Current directions in research on autism. *Mental Retardation and Developmental Disabilities Research Reviews, 7*, 21–29.

Tager-Flusberg, H., Rogers, S., Cooper, J., Landa, R., Lord, C., Paul, R., . . . Yoder, P. (2009). Defining spoken language benchmarks and selecting measures of expressive language development for young children with autism spectrum disorders. *Journal of Speech, Language, and Hearing Research*, *52*(3), 643-652.

Tager-Flusberg, H., & Sullivan, K. (1994). Predicting and explaining behavior: A comparison of autistic, mentally retarded and normal children. *Journal of Child Psychology and Psychiatry, 35*, 1059–1075.

Taheri, A., & Perry, A. (2012). Exploring the proposed DSM-5 criteria in a clinical sample. *Journal of Autism and Developmental Disorders*, *42*(9), 1810-1817.

Takahashi, H., Suzumura, S., Shirakizawa, F., Wada, N., Tanaka-Taya, K., Arai, S., . . . Sato, T. (2003). An epidemiological study on Japanese autism concerning routine childhood immunization history. *Japanese Journal of Infectious Diseases*, *56*(3), 114-117.

Tani, P., Lindberg, N., Appelberg, B., Nieminen-von Wendt, T., von Wendt, L., & Porkka-Heiskanen, T. (2005). Childhood inattention and hyperactivity symptoms self-reported by adults with Asperger syndrome. *Psychopathology*, *39*(1), 49-54.

Tani, P., Lindberg, N., Nieminen-von Wendt, T., von Wendt, L., Virkkala, J., Appelberg, B. O. R., & Porkka-Heiskanen, T. (2004). Sleep in young adults with Asperger syndrome. *Neuropsychobiology*, *50*(2), 147-152.

Tantam, D. (2000). Psychological disorder in adolescents and adults with Asperger syndrome. *Autism, 4*, 47–62.

Taylor, B. A., Hoch, H., & Weissman, M. (2005). The analysis and treatment of vocal stereotypy in a child with autism. *Behavioral Interventions*, *20*(4), 239-253.

Thelen, E. (1979). Rhythmical stereotypies in normal human infants. *Animal Behavior, 27*, 699–715.

Theoret, H., Halligan, E., Kobayashi, M., Fregni, F., Tager-Flusberg, H., & Pascual-Leone, A. (2005). Impaired motor facilitation during action observation in individuals with autism spectrum disorder. *Current Biology*, *15*(3), 84-85.

Thorner, R. M., & Remein, Q. R. (1961). Principles and Procedures in the Evaluation of Screening for Disease. *Public Health Service Publications*, (846).

Thorson, R. T., & Matson, J. L. (2012). Cutoff scores for the Autism Spectrum Disorder–Comorbid for Children (ASD-CC). *Research in Autism Spectrum Disorders*, *6*(1), 556-559.

Tordjman, S., Anderson, G. M., Botbol, M., Brailly-Tabard, S., Perez-Diaz, F., Graignic, R., . . . Bronsard, G. (2009). Pain reactivity and plasma β-endorphin in children and adolescents with autistic disorder. *PLoS One*, *4*(8), 52-89.

Totsika, V., Toogood, S., Hastings, R. P., & Lewis, S. (2008). Persistence of challenging behaviours in adults with intellectual disability over a period of 11 years. *Journal of Intellectual Disability Research, 52*, 446–57.

Travis, L., & Sigman, M. (1998). Social deficits and interpersonal relationships in autism. *Mental Retardation and Developmental Disabilities Research Reviews, 4*, 65-72.

Trevarthen, C. (2000). Autism as a neurodevelopmental disorder affecting communication and learning in early childhood: prenatal origins, post-natal course and effective educational support. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, *63*(1), 41-46.

Trevarthen, C., Aitken, K., Robarts, J., & Papoudi, D. (Eds.). (1999). *Children with autism*. London, England: Kingsley Publications.

Trottier, G., Srivastava, L., & Walker, C. D. (1999). Etiology of infantile autism: a review of recent advances in genetic and neurobiological research. *Journal of Psychiatry and Neuroscience*, *24*(2), 103.

Tryon, P. A., Mayes, S. D., Rhodes, R. L., & Waldo, M. (2006). Can Asperger's disorder be differentiated from autism using DSM-IV criteria?. *Focus on Autism and Other Developmental Disabilities*, *21*(1), 2-6.

Tsai, L. Y., & Ghaziuddin, M. (2013). Dsm-5 ASD moves forward into the past. *Journal of Autism and Developmental Disorders, 44*(2) , 1-10.

Tsakanikos, E., Bouras, N., Sturmey, P., & Holt, G. (2006). Psychiatric co-morbidity and gender differences in intellectual disability. *Journal of Intellectual Disability Research*, *50*(8), 582-587.

Tuchman, R., & Rapin, I. (2002). Epilepsy in autism. *The Lancet Neurology*, *1*(6), 352-358.

Tureck, K., Matson, J. L., May, A., Davis III, T. E., & Whiting, S. E. (2013). Investigation of the rates of comorbid symptoms in children with ADHD compared to children with ASD. *Journal of Developmental and Physical Disabilities*, *25*(4), 405-417.

Tureck, K., Matson, J. L., May, A., & Turygin, N. (2013). Externalizing and tantrum behaviours in children with ASD and ADHD compared to children with ADHD. *Developmental Neurorehabilitation*, *16*(1), 52-57.

Turner, M. (1999). Annotation: Repetitive behaviour in autism: A review of psychological research. *Journal of Child Psychology and Psychiatry*, *40*(6), 839-849.

Tustin, F. (1981). *Autistic states in children*. Boston, MA: Routledge.

U.S. Census Bureau, Population Division (2013). Annual Estimates of the Resident Population by Sex, Race, and Hispanic Origin for the United States, States, and Counties: April 1, 2010 to July 1, 2012. Retrieved from http://louisiana.gov/Explore/Demographics_and_Geography/

Van Bourgondien, M. E., & Elgar, S. (1990). The relationship between existing residential services and the needs of autistic adults. *Journal of Autism and Developmental Disorders*, *20*(3), 299-308.

Van Bourgondien, M. E., & Schopler, E. (1990). Critical issues in the residential care of people with autism. *Journal of Autism and Developmental Disorders*, *20*, 391–99.

Van Daalen, E., Kemner, C., Dietz, C., Swinkels, S. H., Buitelaar, J. K., & Van Engeland, H. (2009). Inter-rater reliability and stability of diagnoses of autism spectrum disorder in children identified through screening at a very young age. *European Child and Adolescent Psychiatry*, *18*(11), 663-674.

Van Krevelen, D. A. (1971). Early infantile autism and autistic psychopathy. *Journal of Autism and Childhood Schizophrenia*, *1*(1), 82-86.

Van Ness, P. H., Murphy, T. E., Araujo, K. B., Pisani, M. A., & Allore, H. G. (2007). The use of missingness screens in clinical epidemiologic research has implications for regression modeling. *Journal of Clinical Epidemiology*, *60*(12), 1239-1245.

van Steensel, F. J., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review, 14*(3), 302-317.

Vargas, D. L., Nascimbene, C., Krishnan, C., Zimmerman, A. W., & Pardo, C. A. (2005). Neuroglial activation and neuroinflammation in the brain of patients with autism. *Annals of Neurology, 57*(1), 67-81.

Varni, J. W., Lovaas, O. I., Koegel, R. L., & Everett, N. L. (1979). An analysis of observational learning in autistic and normal children. *Journal of Abnormal Child Psychology, 7*, 31–43.

Ventola, P., Kleinman, J., Pandey, J., Wilson, L., Esser, E., Boorstein, H., . . . Fein, D. (2007). Differentiating between autism spectrum disorders and other developmental disabilities in children who failed a screening instrument for ASD. *Journal of Autism and Developmental Disorders, 37*(3), 425-436.

Vig, S., & Jedrysek, E. (1999). Autistic features in young children with significant cognitive impairment: Autism or mental retardation?. *Journal of Autism and Developmental Disorders, 29*(3), 235-248.

Virues-Ortega, J. (2010). Applied behavior analytic intervention for autism in early childhood: Meta-analysis, meta-regression and dose-response meta-analysis of multiple outcomes. *Clinical Psychology Review, 30*, 387–399.

Volden, J., & Lord, C. (1991). Neologisms and idiosyncratic language in autistic speakers. *Journal of Autism and Developmental Disorders, 21*, 109–131.

Volden, J., Mulcahy, R. F., & Holdgrafer, G. (1997). Pragmatic language disorder and perspective taking in autistic speakers. *Applied Psycholinguistics, 18*, 181–198.

Volkmar, F. R. (1998). Categorical approaches to the diagnosis of autism an overview of DSM-IV and ICD-10. *Autism, 2*(1), 45-59.

Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders, 22*(4), 483-492.

Volkmar, F. R., Cicchetti, D. V., Dykens, E., Sparrow, S. S., Leckman, J. F., & Cohen, D. J. (1988). An evaluation of the autism behavior checklist. *Journal of Autism and Developmental Disorders, 18*(1), 81-97.

Volkmar, F. R., & Cohen, D. J. (1989). Disintegrative disorder or "late onset" autism. *Journal of Child Psychology and Psychiatry, 30*(5), 717-724.

Volkmar, F. R., & Cohen, D. J. (1991). Debate and argument: The utility of the term pervasive developmental disorder. *Journal of Child Psychology and Psychiatry, 32*(7), 1171-1172.

Volkmar, F. R., Cohen, D., & Paul, R. (1986). An evaluation of DSM-III criteria for infantile autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 25*, 190–197.

Volkmar, F. R., & Klin, A. (2005). Issues in the classification of autism and related conditions. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.). *Handbook of Autism and Pervasive Developmental Disorders 3rd edition* (pp. 5-41). Hoboken, NJ: John Wiley & Sons, Inc.

Volkmar, F. R., & Reichow, B. (2013). Autism in DSM-5: Progress and challenges. *Molecular Autism*, *4*(1), 13.

Volkmar, F. R., Stier, D. M., & Cohen, D. J. (1985). Age of recognition of pervasive developmental disorder. *American Journal of Psychiatry*, *142*(12), 1450-1452.

Wadden, N. P., Bryson, S. E., & Rodger, R. S. (1991). A closer look at the Autism Behavior Checklist: Discriminant validity and factor structure. *Journal of Autism and Developmental Disorders*, *21*(4), 529-541.

Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., . . . Walker-Smith, J. A. (1998). RETRACTED: Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, *351*(9103), 637-641.

Walker, D. R., Thompson, A., Zwaigenbaum, L., Goldberg, J., Bryson, S. E., Mahoney, W. J., . . . Szatmari, P. (2004). Specifying PDD-NOS: a comparison of PDD-NOS, Asperger syndrome, and autism. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(2), 172-180.

Warren, R. P., Singh, V. K., Averett, R. E., Odell, J. D., Maciulis, A., Burger, R. A., . . . Warren, W. L. (1996). Immunogenetic studies in autism and related disorders. *Molecular and Chemical Neuropathology*, *28*(1-3), 77-81.

Waterhouse, L., Wing, L., Spitzer, R., & Siegel, B. (1989). Pervasive developmental disorders: From DSM-III to DSM-III-R. *Journal of Autism and Developmental Disorders*, *22*, 525-549.

Weidmer-Mikhail, E., Sheldon, S., & Ghaziuddin, M. (1998). Chromosomes in autism and related pervasive developmental disorders: A cytogenetic study. *Journal of Intellectual Disability Research*, *42*(1), 8-12.

Weiss, M. J. (1999). Differential rates of skill acquisition and outcomes of early intensive behavioral intervention for autism. *Behavioral Interventions*, *14*(1), 3-22.

Weiss, M. J., & Harris, S. L. (2001). Teaching social skills to people with autism. *Behavior Modification*, *25*(5), 785-802.

Welsh, M., Parke, R. D., Widaman, K., & O'Neil, R. (2001). Linkages between children's social and academic competence: A longitudinal analysis. *Journal of School Psychology*, *39*(6), 463-482.

Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry*, *62*, 889–895.

Werner, E., Dawson, G., Osterling, J., & Dinno, N. (2000). Brief report: Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. *Journal of Autism and Developmental Disorders*, *30*(2), 157-162.

Wetherby, A. M., Brosnan-Maddox, S., Peace, V., & Newton, L. (2008). Validation of the Infant—Toddler Checklist as a broadband screener for autism spectrum disorders from 9 to 24 months of age. *Autism*, *12*(5), 487-511.

Wetherby, A. M., Goldstein, H., Cleary, J., Allen, L., & Kublin, K. (2003). Early identification of children with communication disorders: Concurrent and predictive validity of the CSBS developmental profile. *Infants and Young Children*, *16*(2), 161-174.

Wetherby, A. M., & Prizant, B. (2002). *Communication and symbolic behavior scales developmental profile–First normed edition*. Baltimore, MD: Brookes.

Wetherby, A. M., Prizant, B. M., & Hutchinson, T. (1998). Communicative, social-affective, and symbolic profiles of young children with autism and pervasive developmental disorder. *American Journal of Speech-Language Pathology*, *7*, 79–91.

Wetherby, A. M., Watt, N., Morgan, L., & Shumway, S. (2007). Social communication profiles of children with autism spectrum disorders late in the second year of life. *Journal of Autism and Developmental Disorders*, *37*(5), 960-975.

Wetherby, A. M., Woods, J., Allen, L., Cleary, J., Dickinson, H., & Lord, C. (2004). Early indicators of autism spectrum disorders in the second year of life. *Journal of Autism and Developmental Disorders*, *34*(5), 473-493.

White, S. W., & Roberson-Nay, R. (2009). Anxiety, social deficits, and loneliness in youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *39*(7), 1006–1013.

Wiggins, L. D., Baio, J., & Rice, C. (2006). Examination of the time between first evaluation and first autism spectrum diagnosis in a population-based sample. *Journal of Developmental and Behavioral Pediatrics*, *27*(2), 79-87.

Wiggs, L., & Stores, G. (2004). Sleep patterns and sleep disorders in children with autistic spectrum disorders: insights using parent report and actigraphy. *Developmental Medicine and Child Neurology*, *46*(6), 372-380.

Wilbarger, P., & Wilbarger, J. L. (1991). *Sensory defensiveness in children aged 2-12: An intervention guide for parents and other caretakers*. Minneapolis, MN: Avanti Educational Programs.

Williams, D. (1999). *Autism and sensing: The unlost instinct*. London, England: Jessica Kingsley Publishers.

Williams, P. G., Dalrymple, N., & Neal, J. (2000). Eating habits of children with autism. *Pediatric Nursing*, *26*(3), 259.

Wilson, C. E., Gillan, N., Spain, D., Robertson, D., Roberts, G., Murphy, C. M., . . . Murphy, D. G. (2013). Comparison of ICD-10R, DSM-IV-TR and DSM-5 in an adult autism spectrum disorder diagnostic clinic. *Journal of Autism and Developmental Disorders*, *43*(11), 2515-2525.

Wilson, K., Mills, E., Ross, C., McGowan, J., & Jadad, A. (2003). Association of autistic spectrum disorder and the measles, mumps, and rubella vaccine: A systematic review of current epidemiological evidence. *Archives of Pediatrics and Adolescent Medicine*, *157*(7), 628-634.

Wing, J. K. (1966). Diagnosis, epidemiology, aetiology. In J. K. Wing (Ed.), *Early childhood autism. Clinical educational and social aspects.* London, England: Pergamon.

Wing, L. (1969). The handicaps of autistic children: A comparative study. *Journal of Child Psychology and Psychiatry, 10*, 1–40.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine, 11*, 115–119.

Wing, L. (1991). The relationship between Asperger's syndrome and Kanner's autism. *Autism and Asperger syndrome*, 93-121.

Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: is the prevalence rising?. *Mental Retardation and Developmental Disabilities Research Reviews*, *8*(3), 151-161.

Wing, L., Gould, J., & Gillberg, C. (2011). Autism spectrum disorders in the DSM-V: Better or worse than the DSM-IV?. *Research in Developmental Disabilities*, *32*(2), 768-773.

Wing, L., Leekam, S. R., Libby, S. J., Gould, J., & Larcombe, M. (2002). The diagnostic interview for social and communication disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry*, *43*(3), 307-325.

Witwer, A. N., & Lecavalier, L. (2008). Examining the validity of autism spectrum disorder subtypes. *Journal of Autism and Developmental Disorders*, *38*(9), 1611-1624.

Wong, V., Hui, L., Lee, W., Leung, L., Ho, P., Lau, W., Fung, C., & Chung, B. (2004). A modified screening tool for autism (Checklist for Autism in Toddlers [CHAT-23]) for Chinese children. *Pediatrics, 114*, 166–176.

Woods, J. J., & Wetherby, A. M. (2003). Early identification of and intervention for infants and toddlers who are at risk for autism spectrum disorder. *Language, Speech and Hearing Services in Schools*, *34*(3).

World Health Organisation (1967). *Manual of the Inter-national Statistical Classification of Diseases, Injuries and Causes of Death - Eighth Revision*. Geneva, Switzerland: World Health Organisation.

World Health Organization (1979. *The ICD-9 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines*. Geneva, Switzerland: World Health Organization.

World Health Organization (1992). *The ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines*. Geneva, Switzerland: World Health Organization.

World Health Organization (2012). ICD 11 Beta Draft: Autism spectrum disorders and related disorders. Retrieved from http://id.who.int/icd/entity/523143960.

World Health Organization (2014). ICD 11 Beta Draft: Autism spectrum disorders and related disorders. Retrieved from http://id.who.int/icd/entity/437815624.

Worley, J. A., & Matson, J. L. (2012). Comparing symptoms of autism spectrum disorders using the current DSM-IV-TR diagnostic criteria and the proposed DSM-V diagnostic criteria. *Research in Autism Spectrum Disorders*, *6*(2), 965-970.

Xu, J., & Chen, Z. (2003). Advances in molecular cytogenetics for the evaluation of mental retardation. *American Journal of Medical Genetics Part C*, 117(1), 15-24.

Yeargin-Allsopp, M., Rice, C., Karapurkar, T., Doernberg, N., Boyle, C., & Murphy, C. (2003). Prevalence of autism in a US metropolitan area. *Journal of the American Medical Association*, *289*(1), 49-55.

Yoder, P. J., & Stone, W. L. (2006). Randomized comparison of two communication interventions for preschoolers with autism spectrum disorders. *Journal of Consulting and Clinical Psychology, 74,* 426–235.

Yonan, A. L., Alarcon, M., Cheng, R., Magnusson, P. K., Spence, S. J., Palmer, A. A., . . . Gilliam, T. C. (2003). A genomewide screen of 345 families for autism-susceptibility loci. *The American Journal of Human Genetics*, *73*(4), 886-897.

Youden, W. J. (1950). Index for rating diagnostic tests. *Cancer*, *3*(1), 32-35.

Yudofsky, S. C., Silver, J. M., Jackson, W., Endicott, J., & Williams, D. (1986). The Overt Aggression Scale for the objective rating of verbal and physical aggression. *The American Journal of Psychiatry*.

Zoghbi, H. Y. (2003). Postnatal neurodevelopmental disorders: Meeting at the synapse?. *Science*, *302*(5646), 826-830.

Zou, K. H., O'Malley, A. J., & Mauri, L. (2007). Receiver-operating characteristic analysis for evaluating diagnostic tests and predictive models. *Circulation*, *115*(5), 654-657.

Zwaigenbaum, L., & Stone, W. (2006). Early screening for autism spectrum disorders in clinical practice settings. In T. Charman & W. Stone (Eds.), *Social and communication development in autism spectrum disorders* (pp. 88-113). New York, NY: Guilford Press.

Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, *23*(2), 143-152.

Zwaigenbaum, L., Thurm, A., Stone, W., Baranek, G., Bryson, S., Iverson, J., . . . Sigman, M. (2007). Studying the emergence of autism spectrum disorders in high-risk infants: methodological and practical issues. *Journal of Autism and Developmental Disorders*, *37*(3), 466-480.

Appendix

ACTION ON PROTOCOL CONTINUATION REQUEST

Institutional Review Board
Dr. Dennis Landin, Chair
130 David Boyd Hall
Baton Rouge, LA 70803
P: 225.578.8692
F: 225.578.5983
irb@lsu.edu | lsu.edu/irb

TO: Johnny Matson
Psychology

FROM: Dennis Landin
Chair, Institutional Review Board

DATE: June 5, 2014
RE: **IRB#** 2609

TITLE: Developing the Autism Spectrum Disorder (ASD)

New Protocol/Modification/Continuation: Continuation

Review type: Full ___ Expedited _X_ **Review date:** 6/5/2014

Risk Factor: Minimal ___X____ Uncertain _____ Greater Than Minimal_____

Approved_____X_____ **Disapproved**_____

Approval Date: 6/5/2014 **Approval Expiration Date:** 6/4/2015

Re-review frequency: (annual unless otherwise stated)

Number of subjects approved: 2000

LSU Proposal Number (if applicable): _____

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

By: Dennis Landin, Chairman _____

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

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

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

Vita

Matthew Jason Konst, a native of Morganton, North Carolina, is a veteran of the United States Marine Corps, having earned the rank of E-5, Seargent. He received his Master's Degree in Clinical Psychology from Louisiana State University (LSU). His research and professional interests have centered upon serving individuals with developmental disabilities broadly and Autism Spectrum Disorders (ASD) specifically. Specific areas of interest have included early assessment and identification of ASD, comorbid psychopathology, and manifestation of symptoms across the lifespan. He was accepted to the Johns Hopkins University School of Medicine Predoctoral Internship at the Kennedy Krieger Institute. He anticipates graduating with his Doctor of Philosophy Degree in Clinical Psychology from LSU in August 2016.