

2014

# Change in developmental quotient in toddlers assessed for Autism Spectrum Disorder

Lindsey Willis Williams

*Louisiana State University and Agricultural and Mechanical College*

Follow this and additional works at: [https://digitalcommons.lsu.edu/gradschool\\_theses](https://digitalcommons.lsu.edu/gradschool_theses)



Part of the [Psychology Commons](#)

---

## Recommended Citation

Williams, Lindsey Willis, "Change in developmental quotient in toddlers assessed for Autism Spectrum Disorder" (2014). *LSU Master's Theses*. 4274.

[https://digitalcommons.lsu.edu/gradschool\\_theses/4274](https://digitalcommons.lsu.edu/gradschool_theses/4274)

This Thesis is brought to you for free and open access by the Graduate School at LSU Digital Commons. It has been accepted for inclusion in LSU Master's Theses by an authorized graduate school editor of LSU Digital Commons. For more information, please contact [gradetd@lsu.edu](mailto:gradetd@lsu.edu).

CHANGE IN DEVELOPMENTAL QUOTIENT IN TODDLERS  
ASSESSED FOR AUTISM SPECTRUM DISORDER

A Thesis

Submitted to the Graduate Faculty of the  
Louisiana State University and  
Agricultural and Mechanical College  
in partial fulfillment of the  
requirements for the degree of  
Master of Arts

in

The Department of Psychology

by  
Lindsey Willis Williams  
B.A., Erskine College, 2006  
M.S., University of North Carolina at Chapel Hill, 2009  
May 2014

# Table of Contents

Abstract .....	iii
Introduction.....	1
Autism Spectrum Disorders .....	3
History .....	3
<i>DSM-IV-TR</i> Diagnostic Criteria.....	9
Current Diagnostic Criteria .....	12
Prevalence.....	12
Early Detection .....	14
Early Childhood Development.....	17
Typical Development.....	17
Atypical Development.....	18
Developmental Delays in Autistic Disorder and PDD-NOS .....	19
Developmental Testing .....	20
History and Purpose .....	20
Challenges of Testing Young Children .....	20
Developmental Differences in ASD.....	22
Changes in Developmental Scores and ASD Symptoms .....	24
Louisiana's EarlySteps Program .....	30
Eligibility and Service Provision.....	30
Outcomes Measurement.....	32
Purpose.....	33
Method .....	35
Participants.....	35
Measures .....	36
Procedure.....	38
Statistical Analyses .....	39
Results.....	43
Discussion .....	45
References .....	48
Vita .....	61

## Abstract

ASD is marked by significant delays in social and language development, while development in other areas, such as cognitive functioning, can be highly variable from person to person. Though preschool-aged children with ASD often exhibit a profile of developmental delays similar to children with other developmental disorders at a discrete moment in time, few studies have investigated possible differences in rate of skill acquisition in developmental domains in children with different disorders. Sensitive periods of development are marked by less stability in performance of developmental skills. Results of prior studies suggest that if appropriate early interventions are applied during sensitive stages of development or periods in which individuals with ASD exhibit particular responsivity to interventions in certain domains (e.g. motor or language development), rate of skill acquisition increases. This study uses repeat administrations of the *Battelle Developmental Inventory, Second edition (BDI-2)* to investigate developmental profile and rate of skill development of atypically developing toddlers with and without Autism Spectrum Disorder who are receiving individualized early intervention services through Louisiana's EarlySteps program. Additionally, the relationship between severity of autistic symptomatology and development as measured by the *Baby and Infant Screen for Children with aUtism Traits (BISCUIT)* and subsequent *BDI-2* scores is explored.

## Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that is present from birth and typically diagnosed in the first few years of life. Until the recent publication of the 5<sup>th</sup> edition of the *Diagnostic and Statistical Manual of Mental Disorders* in May 2013 (*DSM-5*; American Psychiatric Association [APA], 2013), ASD as a *DSM* category included five neurodevelopmental disorders. As outlined in the previous version of this widely used manual, the *DSM-IV-TR* (APA, 2000), these disorders included Autistic Disorder, Asperger's Disorder, Rett's Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). These disorders as defined in the *DSM-IV-TR* are marked by varying degrees of deficiencies in social skills and communication as well as restricted interests, activities, and behaviors, e.g., hand flapping, preoccupation with objects, and rocking.

Ever since Kanner and Asperger first described ASD in the 1940s, it has been noted that individuals with ASD have widely discrepant skills in various developmental domains. In 1944, Asperger noted that the individuals he studied had unequal cognitive development, with some possessing above average intelligence but also severe deficits in other areas such as communication or social skills. Over the ensuing decades, it has become apparent that early intervention provides a great advantage for future prognosis in persons with ASD; accordingly, great effort has been put forth in developing measures and techniques to aide in early diagnosis. However, some early intervention programs are more successful than others, and sheer number of hours does not account for variability in the effect of intervention programs. Numerous studies have compared developmental delays in early childhood between children with ASD and other developmental delays, with mixed findings concerning the presence of significant differences in developmental domains including communication, language, cognitive, motor, and adaptive skills at discrete points in time. However, relatively few studies have investigated the possibility that differences may exist among groups in the relative rate of development across domains. Early intervention has proven beneficial for children with developmental delays due to ASD or other disabilities. Identifying differences in developmental trajectory or sensitive periods of skill acquisition in ASD versus non-ASD related disorders may warrant future research into refining early interventions to be more efficacious for children with individuals with ASD-related delays. The purpose of this study is to add to the body of knowledge regarding potentially significant differences in the developmental process of toddlers with and without

ASD. Additionally, as all children in this study received services from a statewide intervention program, this study will investigate potential differences in rate of developmental skill acquisition and ASD symptom severity after receiving individualized services through Louisiana's EarlySteps intervention program. Prior to this study, a brief description of ASD will be presented along with current research in the area of early childhood development in children with and without ASD.

# Autism Spectrum Disorders

## History

**Leo Kanner.** In 1943, Leo Kanner published an article in the journal *The Nervous Child* detailing a unique pattern of behavior observed in 11 children (Kanner, 1943). Kanner described these children as having “extreme autistic aloneness that, whenever possible, disregards, ignores, shuts out anything that comes to the child from outside,” with a fundamental inability to relate “in the ordinary way. . . from the beginning of life” (Kanner, 1943, p. 248). The children did not respond when spoken to, and thus were thought to be hearing impaired. The children exhibited pronoun reversal, literal interpretation of language, and echolalia, and insistence in the invariable execution of various daily habits. Kanner categorized food, loud noises, moving objects, and other sensory stimuli as unwelcome intrusions from the outside, frequently “reacted to with horror . . . even the wind could on occasion bring about a major panic” (Kanner, 1943, p. 245). Each child’s behavior was marked by an obsessive insistence on sameness and extremely limited spontaneous activity. Kanner concluded that the common behaviors constituted “a unique ‘syndrome’ not heretofore reported, which seems to be rare enough, yet is probably more frequent than is indicated (Kanner, 1943, p. 242).

Kanner used the word “autism” to characterize the self-centered, idiosyncratic nature of the disordered behavior of the children in his observations. Kanner acknowledged the similarities between “the combination of extreme autism, obsessiveness, stereotypy, and echolalia” and “some of the basic schizophrenic phenomena” (Kanner, 1943, p. 248), noting that some of the children had been diagnosed with schizophrenia. Kanner was not the first to use the term “autism” in relation to schizophrenia; Eugene Bleuler, the Swiss psychiatrist who coined the term “schizophrenia,” also coined the word “autism” in reference to the social withdrawal common in cases of schizophrenia (Rutter, 1968). In fact, the symptoms that eventually became considered characteristic of today’s Autism Spectrum Disorder were initially thought to represent childhood schizophrenia. During the course of his observations, Kanner set about outlining ways in which the conditions differ, including differences in onset, course, and family history. The children he examined came from families with little history of mental illness of any sort, and exhibited symptoms from infancy, rather than the later onset of symptoms common in schizophrenia. Additionally, while the children were unable to relate to people, they maintained “excellent, purposeful, and ‘intelligent’ relation to objects” (Kanner, 1943, p. 249). These behaviors differ from those typical of schizophrenia, in

which individuals typically exhibit a withdrawal from a world with which they were previously in touch. In Kanner's sample, the children "gradually compromise" with "a world in which they have been total strangers from the beginning" (Kanner, 1943, p. 249). Accordingly, the children are able to gradually learn language that is more communicative; accept wider varieties of food, noise, and other stimuli; and accept interactions with certain other people to the extent that doing so will satisfy their own needs. In conclusion, Kanner assumes that the children exhibited "inborn autistic disturbances of affective contact" (Kanner, 1943, p. 250). A year after this publication, the term early infantile autism entered psychiatric nomenclature in the United States, though the term was not included in the 1968 *Diagnostic and Statistical Manual of Mental Disorders-II* (Kanner, 1944, 1971).

Many of the core features Kanner originally observed remain part of the diagnostic criteria (APA, 2000), including a lack of social awareness and inability to form appropriate social relationships, peculiarities of language acquisition and use, and perseverative, stereotyped patterns of behavior or routines. Additional observations are still considered key symptoms of autism, including lack of eye contact or social reciprocity, but good relation to objects in the environment. Kanner also noted that each of the original 11 children were generally physically normal and healthy, had excellent fine motor skills, and exhibited good to exceptional cognitive ability (Kanner, 1943).

By the 1950s Kanner had diagnosed over 100 cases of early infantile autism (Kanner, 1951). Gender differences emerged with a higher prevalence rate in males versus females (4:1). Kanner also observed that males were often referred for evaluation at 2 to 6 years of age, while females were typically referred later between 6 and 8 years of age (Kanner, 1958; Kanner, 1971). Symptoms were first noted earlier in his sample, however, and included a preponderance of feeding problems sometimes as early as difficulty nursing in infancy. Most commonly the first signs of autism occurred during the first two years of life, including a lack of anticipatory reaction to being picked up by a primary caregiver (Eisenberg & Kanner, 1956).

Beginning with the original eleven cases, Kanner observed several commonalities in parental background. All of the parents in the first cases were highly educated, with all of the fathers holding advanced degrees and having successful careers. Most of the mothers were also college graduates, and held a variety of prestigious occupations (Kanner, 1943). Even after making over 100 additional diagnoses, Kanner continued to report a similar pattern, asserting "to this day, we have not encountered any one

autistic child who came of unintelligent parents” (Kanner, 1954). Although Bender (1959) refuted this with her observation that many children with autism were born of parents with “defective” intelligence, Kanner continued to report a correlation between highly intelligent parents and autism. Researchers continued to cite the affluent and intelligent background of children with autism even into the 1980s; in a study of 50 sets of parents, Dor-Shav and Horowitz (1984) concluded that fathers of autistic children “were significantly above average in intelligence.”

In hypothesizing possible causes for early infantile autism, Kanner detected no pattern regarding the child’s physical state, birth circumstances, or hereditary pattern (e.g. he noted scarcely any family history of schizophrenia) (Kanner, 1954). However, he often noted what he considered autistic-like traits in the parents of children diagnosed with early infantile autism. For example he noted the extraordinarily detailed, even obsessive histories provided by the parents of his original 11 cases, as well as a general lack of affection towards their children (Kanner, 1943, 1954). Kanner hypothesized that emotional deprivation in infancy was a key component in autism etiology. This notion was supported by Bakwin’s (1949) description of “hospitalism,” in which infants subject to emotional deprivation exhibited traits including lack of interest in the environment, lack of crying or emotional response to others, and feeding problems. Soon, a consensus emerged among researchers regarding the contribution of the cold indifference of “refrigerator mothers” towards the child as a primary factor in the development of early infantile autism (Bettelheim, 1967; Eisenberg & Kanner, 1956; Eveloff, 1960). However, eventually Kanner himself began to question this view stating in 1965 that varying viewpoints existed concerning the role of parent interaction in the development of autism (Kanner, 1965). Additional research supported the idea that parental emotional deprivation was not the cause of autism (DeMyer, Hingtgen, & Jackson, 1981; Rimland, 1964; Rutter, 1968). Kanner was in favor of further research into the etiology of the disorder and supported Rimland’s 1964 book, which proposed additional theories of autism causation including neurologic or metabolic abnormalities.

Although Kanner’s initial views of autism etiology were misguided and often distressing to family members (Schopler, 1971) by the 1970s Kanner posited that “biochemical explorations, pursued vigorously in the very recent past, may open a new vista about the fundamental nature of the autistic syndrome” and that parents should be “dealt with from the point of view of mutuality . . . they have of late been included in the therapeutic efforts, not as etiological culprits, nor merely as recipients of drug prescriptions and of thou-

shalt and thou-shalt-not rules, but as actively contributing cotherapists” (Kanner, 1971). Ultimately Kanner’s research proved fundamental to the discovery of autism and provided the groundwork for furthering understanding of the disorder. Meanwhile, across the Atlantic, an Austrian doctoral student from Kanner’s hometown was making similar discoveries.

**Hans Asperger (1944).** Though Kanner and Asperger published their groundbreaking articles on “autistic” children at around the same time, Kanner received international acclaim while Asperger’s work went largely unnoticed for decades (Van Krevelen, 1971; Wing, 1981) and his first description of the disorder, published in 1944, was not translated into English until 1991 (Asperger & Frith, 1991). Indeed, though Kanner and Asperger were from the same city and used the same terminology (autism) to describe clinical cases, it is generally assumed that the two never met and remained unaware of the other’s work (Frith, 2004; Van Krevelen, 1971). However, others assert that Kanner may have been aware of one of Asperger’s lectures published in 1938 (Fitzgerald, 2008).

Asperger’s first published case studies shared many commonalities with Kanner’s first studies with both researchers noting social impairment as the core feature of the disorder (Asperger, 1944; Kanner, 1943). Like Kanner, Asperger observed impaired nonverbal communication, idiosyncratic speech such as pronoun reversal and echolalia, intense interest in circumscribed activities, repetitive behaviors, resistance to change, unequal cognitive development (e.g. excelling in specific areas despite learning problems in other subjects), and behavioral problems. Asperger also noted that autistic traits were also observed in parents and other family members of affected children (Klin, McPartland, & Volkmar, 2005). He noted that while some girls had “disturbances reminiscent of autism,” none had the “fully fledged” disorder apparent in the boys (Asperger, 1944).

Asperger acknowledged Bleuler’s contribution to establishing the term “autism” to convey the withdrawal from the outside world, a common symptom of schizophrenia (Asperger, 1944). Bleuler defined autism as a detachment from the external world: “Schizophrenics who have no more contact with the outside world live in a world of their own. They have encased themselves with their desires and wishes [...]; they have cut themselves off as much as possible from any contact with the external world. This detachment from reality with the relative and absolute predominance of the inner life, we term autism” (Bleuler, 1911, as cited in Parnas, Bovet, & Zahavi, 2002). Asperger noted the similarity between autism and schizophrenia in the “shutting off of relations between the self and the outside world” (Asperger &

Frith, 1991). Asperger, like Kanner, was careful to differentiate schizophrenia and the syndrome he was investigating, noting that the disconnect between the self and the outside world is present from infancy in autism but is progressive in schizophrenia (Asperger, 1944; Asperger & Frith, 1991). Asperger also noted the lack of psychosis in autism (Asperger, 1944).

**Etiology.** Though early hypotheses of etiology centered around parental characteristics such as the “refrigerator mother” (Bettelheim, 1967), this theory was challenged even by Kanner, who initially proposed the theory (Kanner, 1971; Rimland, 1964; Schopler, 1971). To date, no satisfactory causal explanation for ASD has been determined; evidence suggests ASD is probably multifactorial, with strong evidence for a genetic component (Kaplan, 2012; Kozlowski, Matson, & Worley, 2012; Matson & Minshawi, 2006). Other theories include neurochemical, environmental, dietary, and pre/postnatal influences (“Autism spectrum disorders revisited,” 2011; Johnson & Carter, 2011; Matson & Minshawi, 2006). Unfortunately, although theories implicating vaccines or vaccine preservatives have been disproven with empirical research (Immunization Review Committee, 2004; Price et al., 2010), these misguided theories have persisted in the popular media and continue to influence some parents who choose to avoid vaccination or pursue dangerous and ineffective treatments (Bazzano, Zeldin, Schuster, Barrett, & Lehrer, 2012; Poland, 2011; Stokstad, 2008). Autism etiology continues to be the focus of much research in the field (US Department of Health and Human Services, 2011).

**Historical Nosology.** An extensive body of literature on ASD has amassed since Kanner and Asperger’s original reports. Early debates in the field concerned discerning between schizophrenia and autism, and whether there was a distinction between Kanner’s “early infantile autism” and Asperger’s “autistic psychopathology.” The overlap of symptoms (e.g. social withdrawal) and terminology used to describe autism led many early clinicians to suggest the disorder was an early form of schizophrenia (Bender, 1946). Early autism literature contains many references to Bleuler’s “childhood schizophrenia,” often described as *early childhood psychosis*. Early childhood psychosis was characterized by: 1) impairments in social relationships; 2) lack of awareness of personal identity as indicated by self-injurious behavior, pronoun confusion, and unusual body posturing; 3) strange preoccupation with object characteristics or components rather than function; 4) resistance to change; 5) abnormal response to sensory stimuli such as insensitivity or hypersensitivity; 6) acute anxiety in the face of environmental change; 7) loss of or failure to develop language, or abnormal speech patterns including echolalia; 8) abnormal motor

movements, posturing, or gait; and 9) frequent intellectual impairment, although some children were noted to have average or exceptional intellectual functioning (Creak, 1961). Many of these characteristics are still associated with autism and have even become key components of assessment measures (Matson & Minshawi, 2006). Asperger and Kanner asserted in their initial case studies (Asperger, 1944; Kanner, 1943) that schizophrenia and autism differed substantially in onset, course, family history and characteristics, and presence of hallucinations. Subsequent research has established that autism can be distinguished from both schizophrenia and intellectual disability (Rutter, 1968, 1972, 1999), though that is not to say that these conditions cannot co-occur (Matson & Neal, 2009; Matson & Shoemaker, 2009).

As knowledge of Asperger's observations spread, researchers began debating the similarities and differences between his "autistic psychopathy" and Kanner's "early infantile autism" (Van Krevelen, 1963, 1971). While Kanner's (1943) and Asperger's (1944) accounts had many similarities, Asperger's description differed in that symptoms were not evident until age 3 or later, speech and language acquisition were less frequently delayed, symptoms were not associated with intellectual deficits, and children exhibiting Asperger's symptoms generally had a better prognosis. Van Krevelen (1971) first asserted the notion that autism was a psychotic process, while Asperger's was a personality trait. Rutter (1978) proposed that Asperger's syndrome might be a milder form of Kanner's autism. Wing (1981) pointed out a number of differences in the two disorders, noting that children with autism seemed "aloof and indifferent" towards others, while those with Asperger's Disorder were "passive or inappropriate." While there is a paucity of gestures in children with autism, those with Asperger's Disorder often exhibit an excess of gestural behaviors which seem purposeless; those with Asperger's also tend to have satisfactory grammar and vocabulary while those with autism may be mute or have delayed speech (Wing, 1981). While both disorders are characterized by circumscribed interests, in children with autism this often manifests in stereotyped movements, rules, or routines (e.g. hand flapping, arranging objects in a particular way or insisting on performing rituals or routines exactly). Children with Asperger's are more likely to exhibit obsessive fascinations with facts or specialized topics (e.g. botany or maps). Whereas children with autism commonly have atypical sensory sensitivities, these symptoms are not as common with Asperger's; however, those with Asperger's may be more likely to have problems with motor coordination. Ultimately, however, Wing (1981) concluded that the two disorders are more similar than dissimilar. Research into the relationship between disorders included in the Autism Spectrum has continued (Frith, 2004; Leekam,

Libby, Wing, Gould, & Gillberg, 2000; Matson & Wilkins, 2008; Schopler, Mesibov, & Kuncze, 1998; Volkmar & Klin, 2005), and at present, Autistic Disorder and Asperger's Disorder are two of the five recognized types of Autism Spectrum Disorders (also called Pervasive Developmental Disorders), with the other three categories including Rett's Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder, Not Otherwise Specified (PDD-NOS) (APA, 2000). It appears likely that these distinctions will be removed under the forthcoming *DSM-5* (APA, 2012). The currently proposed revisions include removing Rett's syndrome and collapsing all four remaining diagnoses into one category, Autism Spectrum Disorder (ASD) (APA, 2012). These proposed changes have already prompted considerable debate (e.g., Frances, 2010; Mattila et al., 2011; Ozonoff, 2012). Even after nearly 70 years, autism nosology continues to be the focus of a great deal of debate and research.

### ***DSM-IV-TR* Diagnostic Criteria**

Debate regarding ASD diagnostic criteria has continued since it was first introduced to the *Diagnostic and Statistical Manual, Third Edition (DSM-III)* (APA, 1980) and continues to be an important area of research and debate (Matson, Belva, Horovitz, & Bamburg, 2012; Matson, Kozlowski, Hattier, Horovitz, & Sipes, 2012; McPartland, Reichow, & Volkmar, 2012; Worley & Matson, 2012). Accurate diagnosis and classification is critical for ensuring accurate communication among clinicians, researchers, and educators, as well as providing a consistent framework for researchers investigating the disorder (Volkmar, 1998). Furthermore, diagnostic criteria often determine the provision of resources and benefits (Volkmar, Klin, & Cohen, 1997). The goal is to determine diagnostic criteria stringent enough to avoid false diagnoses, but which at the same do not exclude those who may have a milder expression of the disorder. Diagnostic criteria for autism spectrum disorders have undergone many permutations in the search for this balance as research has expanded understanding of the disorders.

The core symptoms noted by Kanner (1944) have remained hallmarks of the disorder as outlined in the two most widely used sets of criteria, found in the *International Classification of Diseases, 10<sup>th</sup> edition (ICD-10)* (World Health Organisation, 1993) and the *Diagnostic and Statistical Manual of Mental Disorders, fourth edition-text revision (DSM-IV-TR)* (American Psychiatric Association, 2000). The diagnostic criteria in these two manuals are intentionally similar (Volkmar, Klin, & Cohen, 1997). Due to the two systems sharing many similarities, the more widely used *DSM-IV-TR* will be used in the discussion on diagnosis. The Pervasive Developmental Disorders which comprised the ASD spectrum as defined in

the *DSM-IV-TR* included five categories: Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder, Asperger's Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). A brief but comprehensive summary of the *DSM-IV-TR* diagnostic criteria for Autistic Disorder and PDD-NOS will follow as these are the two diagnoses relevant to the current study.

**Autistic Disorder.** It was not until the *Diagnostic and Statistical Manual, Third Edition (DSM-III; APA, 1980)* that the term autism was first introduced as a diagnosis under the category of Pervasive Developmental Disorders. Referred to as Infantile Autism, a diagnosis required the presence of Kanner's three main observations (i.e. socialization deficits, communication difficulties, and other odd behaviors) as well as an onset by the age of 30 months. First, deficits in receptive or expressive social communication, the development of social relationships and reciprocal interaction, or functional/symbolic play must have become apparent before the age of three years. Second, the individual must meet at least six symptoms across three subdivisions. The first subdivision includes impairment in social interaction as evidenced by at least two of the following: 1) poor eye contact, facial expression, posture, or gestures related to social interaction; 2) failure to develop developmentally appropriate, mutual peer relationships; 3) impaired or unusual responses to others' emotions, or lack of behavior adjustment relative to social context; and 4) lack of spontaneous efforts to share enjoyment, interests or achievements. The second subdivision involves at least one communication deficit such as 1) delayed development or absence of spoken language without compensatory gesturing; 2) difficulty initiating or sustaining reciprocal language; 3) idiosyncratic, stereotyped or repetitive use of language; and 4) absence of spontaneous imaginative or socially imitative play when young. The third subdivision concerns restricted, repetitive, and stereotyped patterns of behavior, as manifested in at least one of the following: 1) preoccupation with one or more stereotyped patterns of interest which are abnormal in content or intensity, 2) compulsive adherence to nonfunctional routines; 3) stereotyped mannerism such as hand flapping or twisting; or 4) preoccupation with non-functional characteristics or specific parts of materials (including texture, smell, noise, or other sensory stimuli created by an object). Finally, the third criterion specifies that the symptoms are not due to any other developmental, language, emotional, or intellectual disorder, or due to Rett's Syndrome.

Though using almost identical criteria as the *IDC-10*, the *DSM-IV* (APA, 1994) changed the label of the disorder to "Autistic Disorder," a term which was retained in the *DSM-IV-TR* (APA, 2000). In one change from the *DSM-III-R* (APA, 1987), the *DSM-IV* included age of onset as a criterion, in keeping with

Kanner's (1943) definition (Volkmar, Klin, & Cohen, 1997). The *DSM-IV-TR* set forth three major diagnostic criteria for Autistic Disorder. The first requires at least six symptoms across three subdivisions: 1) two impairments in social interaction as manifested by impairment in nonverbal communication, failure to develop developmentally appropriate peer relationships, lack of spontaneous sharing of enjoyment, interests, or achievements, or lack of social reciprocity; 2) qualitative impairments in communication as evinced by delay in or lack of development of spoken language and absence of compensatory gestures; marked impairment in initiating or maintaining conversation (if speech is present); stereotyped, idiosyncratic, or repetitive use of language; or lack of spontaneous imaginative or socially imitative play; and 3) restricted repetitive and stereotyped patterns of behavior, interests, and activities manifested by encompassing preoccupation with restricted interests that is abnormal in intensity or focus; inflexible adherence to specific, nonfunctional routines; stereotyped and repetitive motor mannerisms such as hand flapping; or persistent preoccupation with parts of objects. *DSM-IV-TR* criteria also require delayed or abnormal functioning in social interaction, social communication, or symbolic play before the age of three years. Finally, the disorder cannot be accounted for by Rett's Disorder or Childhood Disintegration Disorder.

**PDD-NOS.** PDD-NOS is often seen as a disorder between the autism and Asperger's Disorder with regard to IQ, measures of adaptive behavior, and communication skills (Walker et al., 2004). Also known as "atypical autism" (Inglese & Elder, 2009), PDD-NOS includes individuals with some of the impairments common to ASDs including reciprocal social interactions, verbal or nonverbal communication, or presence of stereotyped behaviors, interest, or activities, but who do not meet full criteria for another ASD (APA, 2000). Examples that may warrant a PDD-NOS diagnosis include autistic symptomatology with a later age of onset, clinically significant symptoms that fail to meet the threshold for another ASD, or a constellation of symptoms that fail to meet all criteria for another ASD. The diagnosis of PDD-NOS is thus dependent on ruling out other *DSM-IV-TR* ASDs, yet ruling out other ASD diagnoses is not an objective process as no concrete cutoff points or operational definitions have been established for differentiating between diagnoses. Accordingly, distinguishing between Autistic Disorder and PDD-NOS can be affected by subjective criteria (Wing, 1997). Additionally, individuals with ASD exhibit heterogeneous symptom presentations with symptoms that may not fit precisely into diagnostic criteria. Examples of individuals with autistic symptoms but do not fully meet criteria for another disorder include

those whose symptoms largely overlap with that of Asperger syndrome but who differ in terms of having a lag in language development or mild cognitive impairment, or who did not exhibit at least six of the required symptoms under criterion A for Autistic Disorder, but nonetheless exhibits significant impairment due to the present symptoms. This may cause the majority of individuals with an ASD to be classified as having PDD-NOS (Towbin, 1997), and research indicates that PDD-NOS is the most frequently diagnosed ASD using *DSM-IV-TR* criteria (Buitelarr & Van der Gaag, 1998; Fombonne, 2009).

### **Current Diagnostic Criteria**

The most notable changes in ASD diagnostic criteria in the *DSM-5* (APA, 2013) with include a complete removal of Rett's Disorder and the collapse of the remaining four diagnoses into one ASD diagnosis. Socialization and communication deficits have been combined into one domain in which an individual must meet all three symptoms (i.e., deficits in nonverbal communication during social interactions, lack of social reciprocity, and deficits in developing and maintaining developmentally appropriate relationships) in order to receive an ASD diagnosis.

Individuals must also meet two of the following restricted and repetitive behaviors, interests, and activities criteria: stereotyped or repetitive speech, motor movements, or object use; adherence to routines or ritualized patterns of behavior; highly restricted interests which are abnormal in intensity or focus; or abnormal hypo- or hyper-reactivity to sensory input. The age of onset criterion has been expanded to include early childhood in general, noting that some symptoms may not become fully evident until social demands exceed the individual's level of functioning. Taken together, these symptoms must negatively impact the individual's ability to function in activities of daily living. Finally, those who received an ASD diagnosis under the *DSM-IV-TR* are permitted to retain their ASD diagnosis as the *DSM-5* is adopted. In the future, however, these changes will undoubtedly impact the prevalence of ASD.

### **Prevalence**

Early estimates of the prevalence of ASD identified less than 10 in 10,000 individuals as having some form of ASD (Sevin, Knight, & Braud, 2007). More than five decades ago, Kanner and Eisenberg (1956) estimated that autism occurred four times more frequently in males than females, and this statistic remains true in current estimates (Bertoglio & Hendren, 2009; Dawson, Mottron, & Gernsbacher, 2008; Rice et al., 2010). However, the prevalence rate of the disorder has increased significantly from these early estimates, leading researchers to question whether the overall prevalence of ASD is increasing, or whether

more frequent diagnoses are an artifact of other factors (Matson & Kozlowski, 2011). By the 1980s, prevalence estimates had risen to approximately 30 to 60 per 10,000 children, leading some to worry about an emerging “autism epidemic” (Inglese & Elder, 2009). Hypotheses for the increased rate of diagnoses have included changes in diagnostic criteria, new assessment measurements, inaccurate diagnoses, use of different research methodologies to identify prevalence estimates, cultural differences, and increases in autism awareness (Matson & Kozlowski, 2011). Additionally, some researchers cite the increased awareness amongst researchers, clinicians, primary care physicians, and parents as well as increased efforts to diagnose at an early age as likely factors in the increase in diagnoses (Rice, 2009).

Revised criteria in between editions of the *DSM* have directly affected the rates of ASD between publications of each edition (Fombonne, Quirke, & Hagan, 2009; Matson & Kozlowski, 2011; Shattuck, 2006). Alterations between editions leads to diagnostic substitution, meaning when the diagnostic criteria for disorders change, even if the changes are minor, many individuals are may shift from one diagnostic category to another leading to apparent increases and decreases in prevalence rates (Fombonne et al., 2009). As diagnostic criteria change, the specificity and sensitivity of diagnostic categories become more inclusive or exclusive; for example, changes between editions have led to concurrent increases in ASD rates and decreases in intellectual disability rates over the same time periods.

The Centers for Disease Control and Prevention (CDC) have established the Autism and Developmental Disorders Monitoring Network (ADDM) to investigate the increasing prevalence of ASD both in the United States and worldwide. Recent estimates by the ADDM report a prevalence of roughly 1% or about 1 in 110 children have an ASD (CDC, 2010). With regard to Autistic Disorder specifically, the *DSM-IV-TR* reported a prevalence rate of AD as 5 per 10,000 individuals (APA, 2000) while Howlin (2006) found higher rates of 19 per 10,000 persons. As discussed in the section on PDD-NOS diagnostic criteria, a PDD-NOS diagnosis according to the *DSM-IV-TR* was given when an individual presents with symptoms consistent with ASD but did not fully meet criteria for any other specific ASD (APA, 2000). Accordingly it is no surprise that this was the most frequently given diagnosis of the *DSM-IV-TR* ASDs with rates estimated between prevalence rates ranging from 31.4 to 36.1 per 10,000 individuals (Chakrabarti & Fombonne, 2001, 2005; Howlin, 2006).

The adoption of the *DSM-5* is likely to cause a reversal of the recent trend in increasing diagnosis, with many individuals not meeting the more stringent *DSM-5* criteria despite exhibiting significant

impairments that would have previously warranted an ASD diagnosis. Recent studies have estimated approximately 30% to 45% of individuals diagnosed with an ASD according to *DSM-IV-TR* criteria would not meet criteria under the *DSM 5*, with those who would have been diagnosed with PDD-NOS according to the *DSM-IV-TR* most likely to be affected (Matson, Belva, Horovitz, & Bamburg, 2012; Matson, Kozlowski, Hattier, Horovitz, & Sipes, 2012; McPartland, Reichow, & Volkmar, 2012; Worley & Matson, 2012). The full extent of the impact remains to be seen.

### **Early Detection**

ASD is neurodevelopmental in origin, so it is unsurprising that symptoms of the more severe *DSM-IV-TR* categories of Autistic Disorder and PDD-NOS typically become apparent early in development. Kishore and Bashu (2011) found many parents reported concerns before one year of age, and another recent study found 76.2% of parents were concerned about their child's development before three years of age (Jónsdóttir, Saemundsen, Antonsdóttir, Sigurdardóttir, & Ólason, 2011). Chakrabarti (2009) found that parents of children with Autistic Disorder began to recognize problems when their child was 23.4 months old on average, and would seek professional help approximately 4 months later. However, the mean time from first evaluation to diagnosis was approximately 32 months after the problem was first recognized by parents, indicating a gap of about 2.5 years (Chakrabarti, 2009). Many factors can contribute to delays in diagnosis, including parental denial or lack of knowledge regarding the normal course of child development, symptoms which overlap with other disorders or which may be attributed to transient problems or other developmental delays (DeGiacomo & Fombonne, 1998). Such delays in diagnosis and treatment of developmental problems increase parental stress (Baron-Cohen & Bolton, 1993).

Early identification, diagnosis, and treatment can reduce the impact of delays on later functioning of children with an ASD, improving including social skills, communication skills, adaptive behaviors, and perhaps even IQ (American Academy of Pediatrics Committee on Children with Disabilities, 2001; Committee on Educational Interventions for Children with Autism, 2001; Manning-Courtney et al., 2003; Martinez-Pedraza & Cater, 2009; Matson, 2007). Researchers and practitioners generally agree that the earlier a child with ASD is diagnosed and begins receiving services, the better the overall prognosis will be (Matson, Wilkins, & Gonzalez, 2008).

Most of the interventions successfully applied to young children with an ASD are derived from Skinnerian Behavior Analysis. Applied Behavioral Analysis (ABA) is just one example of such intervention. There are four main varieties of ABA including discrete trial training (DTT), pivotal response training (PRT), incidental training (IT), and applied verbal behavior (AVB) (Leach, 2010). The techniques used in ABA and related treatments include teaching new skills, shaping, chaining, repetitive practice of these skills, learning discrimination, modeling, and direct and clear instruction (Leach, 2010; Martinez-Pedraza & Carter, 2009; Weis, Fiske, & Ferraioli, 2008). In a review of studies of early behaviorally-based interventions for young children with ASD, Ben-Itzhak and Zachor (2007) noted that approximately half of participants were able to perform significantly better on standardized tests and function effectively in mainstream classes, and some even became difficult to differentiate from their typically developing peers. The focus on the value of early intervention has led to an increased focus on early detection (Gutierrez et al., 2009; Hayward, Gale, & Eikeseth, 2009) and an increase in programs to make early intervention services accessible. For example, recognizing the importance of early intervention, Louisiana's statewide program, EarlySteps, is aimed at making screening, diagnostic, and early intervention services accessible to toddlers with developmental delays including ASD irrespective of socioeconomic status.

Even when developmental problems are recognized early on, obtaining an accurate diagnosis may take months, highlighting the importance of periodic re-evaluation as symptoms may change. The increased interest in early diagnosis has led to a greater general awareness of early symptoms among parents and health care professionals, and has spurred greater research and development of scales designed for this purpose thus leading to a decrease in the average age of diagnosis in the past decade (Charman & Baird, 2002). Assessment instruments have been designed to measure the functioning of these young children usually between 18 months and 3 years of age, with varying levels of stability in diagnosis. Lord and Luyster (2006) reviewed two prospective studies one of which resulted in a very high stability (84%) of diagnoses at 2 years of age; however, other studies have been inconclusive and early diagnosis remains difficult. In a review of many studies regarding early diagnosis, Landa (2008) noted that approximately a third of toddlers thought to have an ASD around age 12 months are likely to display instability of ASD-related behavior, with diagnostic impressions shifting from the presence of an ASD at the time of the first birthday to non-ASD by the third birthday using *DSM-IV-TR* criteria. Research by Turner and Stone (2007) indicated that 68% of 2-year-olds who meet the diagnostic criteria for an ASD failed to meet criteria at 4

years of age. Turner and Stone (2007) and Sautera et al. (2007) found similar results regarding diagnostic instability in preschool-aged children thought to have an ASD, in which the majority of children exhibiting unstable diagnoses of ASD had higher cognitive functioning than those with a stable diagnosis, were younger than 30 months of age at the time of the first diagnosis, or both.

In some children, clear signs of ASD are not present until late in the second or third year of life (Landa, Holman, & Garrett-Mayer, 2007; Werner & Dawson, 2005; Werner, Dawson, Munson, & Osterling, 2005). These children might have mild developmental delays at younger ages or may even appear to be developing normally, but gradually become less socially engaged after 14 months of age (Landa et al., 2007). The pattern of onset of ASD symptoms is variable, but regardless of the pattern of onset, any child with an ASD can show regression in which existing skills such as spoken language (Cox et al., 2003; Landa et al., 2007; Luyster et al., 2005) and socioemotional reciprocity (Landa et al., 2007; Luyster et al., 2005) recede or are lost altogether, and atypical patterns of behaviors congruent with ASD might emerge (Bryson et al., 2007).

## Early Childhood Development

### Typical Development

While the developmental process unfolds over the lifespan, childhood development is typically considered to extend from fetal development through adolescence or early adulthood, with early childhood development generally conceptualized as development occurring between the ages of two to five years (Lipkin & Allen, 2005). Development typically occurs very rapidly in early childhood, and developmental processes are multidimensional (American Psychiatric Association Task Force on Evidence-Based Practice for Children and Adolescents, 2008), though these processes are often viewed as interdependent developmental domains for the purposes of assessment (Grantham-McGregor et al., 2007; Petermann & Macha, 2008). Typical early development in young children consists of an organic, orderly, and predictable pattern of increasingly refined skill development across neurological, sensorimotor, and cognitive areas (Bagnato, 2007; Batshaw, Pellegrino, & Roizen, 2007). This process is a combination of biological, social, and psychological processes that occur similarly across cultures (Batshaw, Pellegrino, & Roizen, 2007).

Although typical development is a fairly predictable process, variability exists; children may learn how to stand, walk, and talk in their native language in the same sequence but at different ages (American Psychiatric Association Task Force on Evidence-Based Practice for Children and Adolescents, 2008; Petermann & Macha, 2008). Even within healthy, typically developing children, large variations occur in the age and manner with which children develop different abilities (Petermann & Macha, 2008; Rydz, Shevell, Majnemer, & Oskoui, 2005). Nonetheless, enough consistency exists in the developmental process that a typical timeline of early milestone development is commonly referenced to determine whether a child is on track to develop the skills and behaviors to enable them to adapt to changes and function in their environment at an age-appropriate level (Masten & Coatworth, 1998). For example, the Centers for Disease Control ([CDC], 2012) state that children are expected to have achieved the following developmental milestones by 18 months of age: likes to hand things to others during play, may be afraid of strangers but shows affection to familiar people, points to show others something interesting, says several single words, shakes head to indicate “no,” points to indicate what he or she wants, can follow 1 step verbal commands, knows what ordinary household objects are, walks alone, drinks from a cup, eats from a spoon, and can help undress him or herself. Parents who note their children have not achieved the developmental

milestones published by the CDC are recommended to seek evaluation for developmental delays (CDC, 2012).

### **Atypical Development**

Compared to their typically developing peers and even after allowing for the broad range of variability considered within normal limits (Rydz, Shevell, Majnemer, & Oskoui, 2005) young children with developmental delays do not attain developmental milestones within the expected age range (Bagnato, 2007; Batshaw, Pellegrino, & Roizen, 2007). As measured by standardized, norm-referenced assessments such as the *Battelle Developmental Inventory*, 2<sup>nd</sup> ed. (*BDI-2*; Newborg, 2005a), *Vineland Adaptive Behavior Scales*, 2<sup>nd</sup> ed. (Sparrow, Cicchetti, & Balla, 2005), and *Bayley Scales of Infant and Toddler Development*, 3<sup>rd</sup> ed. (Bayley, 2005), children with developmental delay are performing 1.5 to 2.0 standard deviations below the mean of typically developing peers in two or more domains of development (Shevell, Majnemer, Platt, Webster, & Birnbaum, 2005). Accardo & Capute (2008) note that developmental disabilities may become apparent in three ways: the child may develop skills necessary to reach established developmental milestones at a slower than expected rate, a child may demonstrate an uneven pattern of skill development in various domains, or a child may deviate from the expected developmental course by exhibiting behaviors different from those of a typical child of any age. Self-injurious or repetitive, stereotypic self-stimulatory behaviors are examples of behaviors which may deviate entirely from the expected developmental trajectory, and these behaviors are common in ASD (Matson, Dempsey, & Fodstad, 2009). Developmental delay is present in many developmental disabilities including ASD, although developmental delay does not always indicate the presence of a disability as many young children with delays eventually catch up with their peers (McCormick, Litt, Smith, & Zupancic, 2011).

A recent study found that caregivers of both atypically developing toddlers with and without an ASD were more likely to cite delays in the communication domain than any other developmental domain (Kozlowski, Matson, Horovitz, Worley, & Neal, 2011). In this same sample, caregivers of atypically developing children without ASD were more likely to report delayed motor development as a first concern than caregivers of children with ASD (Kozlowski et al., 2011). Other recent research using the *BDI-2* to compare individuals with Down syndrome, global developmental delay, and premature birth found no significant difference among groups in the adaptive, communication, or cognitive domain, but did note differences in the motor and social domains (Matson, Hess, Sipes, & Horovitz, 2010). The toddlers who

were premature in this study had significantly higher scores in the social and motor development domains (Matson, Hess et al., 2010). Future studies in this area may allow researchers to determine whether differences in development emerge during sensitive time periods in various disorders.

### **Developmental Delays in Autistic Disorder and PDD-NOS**

Intellectual disability is highly comorbid with Autistic Disorder and PDD-NOS, so it is unsurprising that researchers have noted that children diagnosed with Autistic Disorder or PDD-NOS tend to exhibit similar cognitive, behavioral, and communication deficits with individuals whose only diagnosis is moderate to severe ID (Lord, 1995). Children diagnosed with PDD-NOS also have developmental delays in common with those who are language-delayed (Lord, 1995). In concordance with these findings, parents of children later diagnosed with Autistic Disorder or PDD-NOS frequently report being first concerned with their child's communication problems (Howlin & Asgharian, 1999; Kozlowski, Matson, Horovitz, Worley, & Neal, 2011), though other concerns are noted as well. Other developmental problems frequently cited as caregivers' first or primary concern include deficits in socio-emotional development, including social withdrawal, abnormal gaze or eye contact, and poor social interaction (Guinchat et al., 2012).

Guinchat et al. (2012) reported a mean age of 19 months when parents first noted concern and 27 months when parents first sought professional advice for children later diagnosed with autism. Most studies that have examined parents' first concerns in samples do not differentiate by ASD subtype, but there is some evidence that types of behaviors causing initial concern may vary by subtype of ASD. Noterdaeme and Hutzelmeyer-Nickels (2010) found that parents of children with autism were first concerned about language problems (48.5%) and social problems (25.7%), while social problems (41.2%) and general behavioral problems (32.5%) were most likely to be first noted for children later diagnosed with Asperger's syndrome. Young children later diagnosed with atypical autism using the *ICD-10* criteria (which would fall under the PDD-NOS category in the *DSM-IV-TR*) were also likely to have parents first concerned with language problems (51.4%) and social problems (22.9%), but these parents also reported more concern with general behavior problems than those with classic Autistic Disorder (11.4% and 5.6%, respectively) (Noterdaeme & Hutzelmeyer-Nickles, 2010). A recent study also found challenging behaviors and attention problems to be more frequently noted in children with ASD compared to atypically developing children (Kozlowski, Matson, Horovitz, Worley, & Neal, 2011).

# Developmental Testing

## History and Purpose

The American child psychologist and pediatrician Arnold Gesell is often considered the father of the field of child development in the United States and was one of the first to apply the rigorous methods of scientific inquiry to the issues of growth and development in children (Petermann, 2008). Like Piaget, Gesell assumed that behavior patterns in infants followed neurodevelopmental processes which unfold in an orderly sequence and Gesell recognized the need to provide an accurate evaluation of standard growth patterns for each age, which could then be used to identify children with developmental delays.

Gesell systematically studied the behaviors of infants and preschool children for forty years, establishing behavioral norms for early human development in the *Gesell Developmental Schedules* (Gesell, 1925). The *Gesell Developmental Schedules* measured individual differences and provided ranges of normal behavior in young children's observable behaviors (Petermann, 2008). The measure was designed to yield a Developmental Quotient (DQ) for children ages 1 to 60 months by assessing motor, adaptive, verbal and social aspects of behavior (Petermann, 2008).

Many of the items included on current developmental assessments can be traced to Gesell's meticulous study of child behavior and skill acquisition (Kamphaus, 2001). Today developmental testing is commonly used in educational settings for many reasons, including screening children suspected of developmental disabilities (DD), making decisions regarding eligibility for special education or other services, planning and evaluating intervention programs, and monitoring progress (Bradley-Johnson, 2001; Brassard & Boehm, 2007). Generally, interpretation includes determining a developmental status based on contrasting scores between typical and atypical development (Petermann & Macha, 2008). In young children, the emphasis of developmental testing often includes identifying those at risk for DD and re-evaluating those children as they transition from intervention programs to preschool or preschool to kindergarten (Brassard & Boehm, 2007). Multiple administrations of developmental tests are valuable at young ages since skills develop and change rapidly during this period (Petermann, 2008).

## Challenges of Testing Young Children

There are challenges to standardized testing of individuals in early childhood, even in healthy, typically developing children. An important limiting factor has been the difficulty in establishing norms and evaluating individual differences in a reliable and precise manner. This problem is magnified for tests

that measure only one general area of development, e.g. intelligence, without accounting for variability in other domains. Historically, however, the study of developmental delay and disabilities in young children focused primarily on cognition (Flanagan & Harrison, 2011). While some standardized and commonly used measures of intelligence are marketed as suitable for use in children as young as 24 months (e.g., *Stanford-Binet Intelligence Scales, 5th edition*; Roid, 2003), performance may be sharply affected by individual differences in early childhood such as variability in motor, social, and communication developmental domains. For example, young children vary greatly in terms of level of discomfort and performance with unfamiliar adults or settings, and may not respond to verbal items or engage fully in test procedures that do not incorporate play (Feldman et al., 2005; Gilliam, Meisels, & Mayes, 2005). Other concerns are associated with the instability of the construct of early intelligence, inadequate reliability and predictive validity, and insufficient floors hampering the ability to adequately assess lower functioning (Lichtenberger, 2005).

Testing children with developmental delays may present greater difficulty because accommodations may lower test validity. Testing young children with autism presents challenges, particularly with those who are nonverbal, seen for evaluation at a young age, or exhibiting more severe ASD symptomatology (National Research Council, 2001). Accordingly, standardized measures such as commonly used intelligence tests may not always be valid, providing more information regarding motivation or attention than information regarding cognitive or language ability (Koegel, Koegel, & Smith, 1997). Thus, any diagnostic evaluation of a child with a disability that is limited to cognitively based evaluations may seriously misjudge the child's competence as well as the foundation of the child's functional impairments (Achenbach, McConaughy, & Howell, 1987). Evaluations that incorporate input from a variety of settings and informants also can lead to more informed decision making (Achenbach et al., 1987).

Measures that provide a standardized, normed developmental quotient can often be more flexibly administered than standardized intelligence tests like the *Stanford Binet* or *Wechsler Intelligence Scale for Children*. For example, measures like the *Psychoeducational Profile-Revised (PEP-R*; Schopler, Reichler, Bashford, Lansing, & Marcus, 1990) *Bayley Scales of Infant and Toddler Development, 3<sup>rd</sup> ed.* (Bayley, 2005), or *Battelle Developmental Inventory, 2<sup>nd</sup> edition (BDI-2*; Newborg, 2005a) may be more uniquely suited to assessing children with ASD due to factors such as flexible administration, concrete and

interesting materials, lack of timed items, and less dependence on receptive and expressive language to measure domains which are not inherently dependent on language. Additionally, developmental tests such as the *BDI-2* (Newborg, 2005a) allow for gathering information from multiple sources. On the *BDI-2*, interview items are used to obtain parent, teacher or caregiver information about the child using an open-ended question format. Each interview item is scripted to help ensure administration consistency, but also allows the examiner to query further as necessary to ensure sufficient information is gathered. More than one-third of the *BDI-2* items may be administered using multiple sources of information (Newborg, 2005b).

### **Developmental Differences in ASD**

Ozonoff et al. (2010) measured the developmental trajectories of infants from birth to 36 months of age, looking at social behaviors such as eye gaze, social smiles, and directed vocalizations coded from video recordings. Ozonoff and colleagues (2010) were able to distinguish infants with and without autism based on differences in prosocial behaviors at age 12 months. Unfortunately this method is extremely cost and labor intensive due to the time associated with collecting and coding the data. However, this study demonstrates that early developmental differences can be observed. If it is possible that differences in developmental trajectory can be detected at a young age, perhaps identifying these differences can contribute to early diagnosis of autism, intellectual or other disabilities. Different disabilities may warrant separate treatments, and this information may in turn be used to determine treatments most suited to type of disability.

It appears that estimates of DQ can provide clinically useful information regarding young children with ASD. A recent study found that while atypically developing toddlers tend to exhibit less challenging behaviors and higher adaptive scores with higher total DQ, toddlers with Autistic Disorder or PDD-NOS evinced a positive correlation between total DQ and challenging behaviors (Medeiros, Kozlowski, Beighley, Rojahn, & Matson, 2012). Medeiros and colleagues (2012) found that toddlers with PDD-NOS showed higher rates of aggressive and stereotypic challenging behaviors with higher *BDI-2* motor and communication domain scores, while the inverse was true for atypically developing toddlers. In a similar study, researchers found DQ was not correlated with symptoms of impulsivity in children with ASD (Matson, Mahan, Hess, & Fodstad, 2010). Bhave, Bhargava, and Kumar (2011) found that low social quotient was predictive of low overall DQ in a study of children ages 6-24 months, but the reverse was not true. The sample in the Bhave et al. (2011) study was comprised of children visiting a pediatrician's office

for routine appointments, so it is unclear whether these results would be found in individuals with ASD. Due to the nature of symptoms and diagnostic criteria discussed earlier, individuals with ASD are likely to have lower social scores than their peers but may have widely varying cognitive abilities.

Studies comparing motor delays in atypically developing young children with and without ASD have produced mixed results. It is possible that this might be a factor of at what point in development these studies were conducted. Ming, Brimacombe, and Wagner (2007) noted gross motor delay in 9% of 154 young children with ASD; however, these delays were no longer apparent by school age. The authors noted children with ASD and motor deficits were not more likely to receive service than those without the motor deficits, and reduced prevalence of motor deficits in older children suggest improvement over time whether through natural development, interventional therapy, or the combination of both (Ming et al., 2007).

Kuroda and Kato (1995) found significant differences in the developmental trajectory of children with ASD compared to those with Down syndrome. While the nature of intervention services received in the Kuroda and Kato (1995) study was unclear, all individuals (both those in the autism group and those in the Down syndrome group) attended the same day program and received similar services. Kuroda and Kato (1995) found that 20% of children with autism showed a change in DQ score of approximately half a standard deviation within only one year. By comparison, no significant difference was found in the scores of children with Down syndrome within a one year period and scores for children with Down syndrome tended to decline after a two to three year period. The children with autism who experienced the most dramatic increase in DQ improved the most in linguistic development (Kuroda & Kato, 1995).

Recent studies of motor skills in developmentally delayed toddlers with and without ASD have been mixed, with some studies showing no difference (Matson, Mahan, Fodstad, Hess, & Neal, 2010; Noterdaeme, Mildenerger, Minow, & Amorosa, 2002; Provost, Lopez, & Heimerl, 2007), and others showing more impaired gross motor functioning in young children with Autistic Disorder and PDD-NOS than atypically developing peers with a variety of diagnoses (Dewey, Cantell, & Crawford, 2007; Matson, Mahan, Kozlowski, & Shoemaker, 2010). In a study of toddlers with Autistic Disorder, PDD-NOS, and atypical development without an ASD, researchers found that overall participants in the Autistic Disorder group had the greatest percentage of failure to meet developmental milestones, followed by the PDD-NOS group and then the atypically developing group (Matson, Mahan, Fodstad et al., 2010). This same trend

was found for milestones including crawling, walking, and first word; however, the trend was reversed for first phrase, with the atypical (non-ASD) group in this sample having the greatest percentage of delay and the Autism Disorder group least likely to be delayed in meeting this milestone (Matson, Mahan, Fodstad et al., 2010).

Studies of regression as reviewed by Landa (2008) suggest regression occurs in 10–50% of children with autism, at a mean age of 19 months, and usually involves a loss of language skills. However, loss of language skills in autism does not negate the possibility of the acquiring language skills later in development; nor does it predict a more severe impairment in language skills (Goldberg et al., 2003). While IQ and speech level are important predictors of positive outcomes for children with ASD, studies of the impact of intervention on these variables have been mixed (Darrou et al., 2010).

Some studies have indicated that low versus high DQ scores may provide information regarding the degree or rapidity that preschool aged children with ASD are likely to benefit from early intervention services (Ogiwara & Takahashi, 2005; Kuroda & Kato, 1995) with those with higher initial scores showing the greatest degree of improvement in developmental scores following intervention. Further study comparing different types of services would be a beneficial follow up to these findings.

### **Change in Developmental Scores and ASD Symptoms**

**Stability of scores.** Measures of intellectual development are considered generally stable over time, although significant changes in early childhood are not entirely uncommon. In a study of over 1000 children ages 7 to 13 years, Moffitt, Caspi, Harkness, and Silva (1993) found that at each two year interval approximately 10% of children evinced a change of 15 points or more on the *Wechsler Intelligence Scale for Children, Revised (WISC-R; Wechsler, 1974)*, but that for the vast majority of children changes over 2 years were negligible. Factors such as environmental changes, low socioeconomic status, or disability may contribute to changes in IQ scores over time (Breslau et al., 2001).

Studies comparing estimates of cognitive functioning obtained from measures providing DQ scores with those obtained from standardized intelligence tests have indicated DQ measures provide a reasonable estimate of cognitive ability in children with ASD, with some administrative advantages over commonly used intelligence tests (Delmolino, 2006; Kurita, Osada, Shimizu, and Tachimori, 2003). In a study of children with Autistic Disorder ages 2.8 to 12.3 years, Kurita, Osada, Shimizu, and Tachimori (2003) found total DQ was significantly correlated with later estimates of IQ using the Japanese version of

the *Stanford Binet* ( $r = 0.68$ ), with the communication domain being the most highly correlated with IQ estimates ( $r = 0.85$ ).

Several researchers have found stability of DQ and IQ scores in atypically developing children and children diagnosed with ASD. While children with ASD do present with considerable developmental heterogeneity within individuals, some researchers have found that when studied in groups IQ scores remain relatively stable over time even in young children (Baghdadi et al., 2007; Sigman et al., 2009). In a measure of stability of DQ and IQ scores in young, language-impaired children with and without ASD, Lord and Schopler (1989) found that IQ/DQ scores changed little in either group with no significant differences noted in patterns of change, absolute difference scores, or group means. More recently, Szatmari et al. (2000) found that IQ score correlations were high over two years in children with ASD between 4 and 6 years of age. Szatmari and colleagues (2000) did note, however, that the few individuals with Autistic Disorder who did make significant gains in scores had gained oral fluency between first and second administration, leading the researchers to suggest that once children with autism develop a certain level of language fluency, they resemble children with Asperger's syndrome but at an earlier stage of overall development. This conclusion is in line with other studies showing that both speech and overall intellectual functioning predict more positive outcomes in children with ASD (Darrou et al., 2010). Baghdadi et al. (2007) found significant heterogeneity in the developmental trajectories in studies of young children with autism, with some children improving rapidly, while the trajectories of others were both slower and less likely to reveal significant improvement over time (Baghdadi et al., 2007). Baghdadi and colleagues (2007) found particular variability in developmental gains in social and communication domains. However, studies of the rate of developmental gains in children with ASD and other disorders are complicated by the effects of a wide variety of interventions.

**Intervention effects.** Several studies have looked at stability of DQ in young children receiving intervention for developmental delays. Moyal (2010) conducted one such study with toddlers exhibiting developmental delays related to a variety of etiologies. Moyal (2010) looked at developmental scores using multiple administrations of the *BDI-2*. Moyal (2010) found greater stability in Total DQ scores than in Cognitive DQ scores across administrations, with the Cognitive DQ scores only weakly correlated across administrations and inconsistent relationships between DQ stability and length of time receiving special education services. Of note, this study did not look at stability across different diagnostic groups. The

Society for Research in Child Development (2008) found a significant relationship between increased scores on the *BDI* social domain and length of time participating in a social skills / relational intervention program for orphaned children with a variety of DDs, with a smaller positive correlation between the treatment and increases in cognitive and communication subscales. Other studies have found 15 point increases common in early childhood in atypically or developmentally delayed children following various types of intervention (Bhakoo, 1977; Moyal, 2010).

Researchers studying the effect of intervention for young children with ASD have reported varying degrees of gains in DQ or IQ scores. Interestingly, Ogiwara and Takahashi (2005) found that children with autism and DQ below 50 showed no significant improvement in DQ three years later, while those with DQ over 50 evinced significant improvement in scores following three years of early intervention. Regarding children receiving instruction in special education classrooms over a one year span, Sheinkopf and Siegel (1998) found a two point increase in IQ for children, while Eikeseth et al. (2002) reported a four point improvement in IQ in a classroom using 1:1 classroom techniques and a variety of other interventions. Eikeseth et al. (2002) reported no improvement in adaptive behavior scores as measured by the *Vineland Adaptive Behavior Scales* (Sparrow, Balla, & Gicchetti, 1984). Gabriels and colleagues (2001) found a five point IQ score increase after a three-year study of children with ASD receiving an eclectic approach to intervention. Reed, Osborne and Corness (2007) found that children with ASD in special nursery placements in the United Kingdom achieved a 10 point gain in IQ and smaller but statistically significant gains on measures of adaptive behavior over 10 months. On the other hand, several studies have found little improvement in functioning for children with ASD attending special preschools. Studies which showed no improvement in standardized measures of developmental functioning in children with ASD following intervention include studies of children receiving either intensive or non-intensive eclectic special education intervention (Howard, Sparkman, Cohen, Green, & Stanislaw, 2005), “generic” educational interventions over a three year period (Lovaas, 1987), and a two year follow up of preschoolers receiving community-based ABA or special nursery school programs (Magiati, Charman, & Howlin, 2007). Salt et al. (2002) followed toddlers receiving 4 hours per week of behaviorally based intervention per week over 11 months, and found no increase in adaptive scores, indicating that while the toddlers gained some skills, they gained no more than would be expected due to changes in chronological age. Sallows and Graupner (1999) found an eight-point decrease in developmental scores over a three year

period in toddlers (36 months and younger) receiving a treatment combination of special education, occupational therapy, and speech therapy. However, in this same study the researchers also reported significant IQ and adaptive gains for toddlers receiving at least 13 hours per week of intensive behavioral-based therapy (Sallows & Graupner, 1999).

Baghdadli and colleagues (2007) conducted a study of 219 preschoolers with ASD receiving services from 51 developmental centers in France, Switzerland, and Luxembourg. In this study intervention services differed by center but were generally administered via multidisciplinary programs that included psychotherapy, special education, behavioral and unstructured activities aimed at developing social, communicative, and adaptive skills. Services also included speech therapy, psychomotor rehabilitation, and alternative communication systems using pictures or icons (Baghdadli et al., 2007). Children were aged 2-7 at first assessment, and were reassessed three years later. The results of this study showed that although development differed significantly among some individuals, as a group the average tendency was stability in developmental domains including communication and socialization, but regression in adaptive skills that were gained at a rate slower than chronological age (Baghdadli et al., 2007). The observed changes were not linked to gender, diagnostic category (atypical autism or infantile autism according to the ICD-10), or the presence of perinatal antecedents (Baghdadli et al., 2007). The extent of regression in the Baghdadli et al. (2007) study was related both to the child's developmental delay and autistic severity at time of first assessment. These observations partly correspond with result attained by Liss et al. (2001), who studied the relationship between IQ, speech, and adaptive development and found that variance in adaptive development was essentially explained by IQ. However, these variables explained only 30% of the developmental variance observed in the Baghdadli et al. (2007) sample. Thus it seems that variability in development is impacted by variables other than those typically used for predicting autism prognosis and raise the issue of how to identify and measure the variables most sensitive to developmental changes in autism, especially with respect to intervention service provision.

Behavioral intervention programs have been shown to positively affect development in young children with ASD, and to be more effective with increased hours (Eldevik, Eikeseth, Jahr, & Smith, 2006; Smith, Eikeseth, Levstrand, & Lovaas, 1997). Intensity of intervention as measured by number of hours of therapy is not necessarily a predictor of efficacy, however. Intensive, eclectic interventions are not as effective as behavioral interventions despite increased hours (Gabriels, Hill, Pierce, Rogers, & Wehner,

2001; Jonsdottir et al., 2006). The amount of intervention is not the only factor related to outcome; the type of intervention received is an important factor in improving prognosis. Reed, Osborne, & Corness (2007) investigated three types of early intervention programs for toddlers aged 2.5 to 4 years of age, including a 1:1 ABA program, a special preschool program, and home-based interventions offered by local educational districts over a 10 month period. The intensity of these interventions varied across groups, with the ABA recipients averaging 30.4 hours per week, compared to 12.7 hours per week for the preschool students and 8.5 hours for those receiving school-provided, home-based intervention (Reed, Osborne, & Corness, 2007). Reed, Osborne, and Corness (2007) found no significant effect on overall severity of autistic symptoms in any treatment groups as measured by the *Gilliam Autism Severity Rating Scale* (Gilliam, 1995), but medium to large effects on adaptive skills and developmental domains as measured by the *Vineland Adaptive Behavior Scales* (Sparrow, Balla, & Cicchetti, 1984) and *PEP-R* (Schopler, Reichler, Bashford, Lansing, & Marcus, 1990). The ABA intervention had the greatest impact on developmental domains, while the preschool setting had the greatest impact on the toddlers' overall adaptive skills. The toddlers receiving school-provided, home-based educational intervention showed significant improvement only in measures of nonverbal communication using picture-matching models.

In a study of 208 children assessed at 5 and then 8 years of age, Darrou et al. (2010) that the amount of intervention in terms of number of hours was not related to outcome when the children received eclectic therapies (the study took place in a number of developmental centers in France, where the authors note behavioral programs are not often used, with intervention programs generally comprised of a combination of special education, rehabilitation, and psychotherapy). Regardless of types or amounts of interventions received in this study, the majority of children had stable developmental profiles in which the children continued to progress but at a slower pace than typical children, causing an increasing gap between chronological and developmental age (Darrou et al., 2010).

That is not to say that intervention is ineffective. In some cases interventions can significantly impact development in young children with ASD. Reed, Osborne, and Corness (2010) followed toddlers ages 2:6 to 4:0 years old for ten months after the toddlers received one of three types of early interventions. In one group, parents attended a 5 day training session and then provided 1:1 intervention in the home ( $M = 12.6$  hours/week) with occasional supervision and support from a supervising psychologist. Other toddlers attended either a preschool for children with a variety of special needs ( $M = 11.5$  hours/week), or a

preschool specifically for toddler with ASD ( $M = 16.3$  hours/week). None of the groups exhibited significant change in autistic symptomatology after 10 months of intervention as measured by the *Gilliam Autism Rating Scale* (Reed, Osborne & Corness, 2010). Although the sample size of this study ( $N = 33$ ) limited the statistical significance of findings, Reed, Osborne and Corness (2010) reported intriguing results. Effect sizes for the parent-provided interventions were mixed, ranging from -0.1 to 0.5. The toddlers receiving general special needs or ASD-specific interventions, however, obtained gains of moderate effect size in overall developmental functioning as measured by the *PEP-R* (0.63 and 0.70, respectively), and large effect sizes in adaptive behavior as measured by the *Vineland Adaptive Behavior Scales* composite score (0.8 and 1.2, respectively). Unfortunately, this study did not provide much detail on the components of each treatment, although 1:1 intervention and behaviorally based components were included in each treatment (e.g. individualized reinforcement-based activities, discrete trial training).

In a review of studies of developmental and behavioral stability in children with ASD, Fisch (2012) noted that more recent studies are more likely to report improvement in symptomology and developmental scores, perhaps suggesting that as children with Autistic Disorder or PDD are identified at an early age, intensive treatment regimens are having a positive impact on future functioning. As noted above, some early intervention programs have resulted in significant developmental gains in toddlers with ASD, though number of hours is not the most important factor in effecting positive change (e.g., Baghdadli et al., 2007; Eldevik, Eikeseth, Jahr, & Smith, 2006; Gabriels, Hill, Pierce, Rogers, & Wehner, 2001; Jonsdottir et al., 2006; Sallows & Graupner, 1999; Smith, Eikeseth, Levstrand, & Lovaas, 1997).

## Louisiana's EarlySteps Program

### Eligibility and Service Provision

Louisiana Part C Early Intervention is Louisiana's early intervention system for infants and toddlers from ages birth to three years (36 months) who have a mental or physical condition with a high probability of resulting in a developmental delay. EarlySteps also provides services to infants and toddlers who do not have an established medical condition but are determined to be delayed in cognitive, physical, communication, social/emotional or adaptive development as evinced by a score of 78 in one of the developmental domains of the *BDI-2* (Newborg, 2005a). According to the *BDI-2* Examiner's Manual (Newborg, 2005b), children with standard scores below 80 (but above the 78 cut off for EarlySteps) fall into a mild developmental delay category. A statewide autism-screening program was implemented beginning July 1, 2008. Funding for EarlySteps interventions comes from a variety of sources, including federal and state Part C funds, federal Medicaid funds, and state Medicaid matching funds (OCDD, 2012a).

The purpose of EarlySteps services is to improve the family's ability to enhance their child's development during early childhood (Office for Citizens with Developmental Disabilities [OCDD], 2012a). Services are provided in the child's natural environment, such as the child's home, childcare or any other community setting typical for the child. The EarlySteps program provides services in the following areas: assistive technology, audiology, vision, family education, evaluation for medical services, nutrition, occupational and physical therapy, psychological services, social work, individualized special education instruction, speech and language pathology, translation, transportation to services, and service coordination (OCDD, 2012a). The purpose of the program is to use interdisciplinary teams to determine the type and amount of evidence based interventions best suited to each child's needs and family circumstances (OCDD, 2012b). Data for the fiscal year 2010-2011 indicated that 99% of Louisiana's infants and toddlers with Individual Family Service Plans (IFSPs) primarily received early intervention services in the home or in community-based settings. The process for EarlySteps referral, assessment, and service provision is outlined in Figure 1.

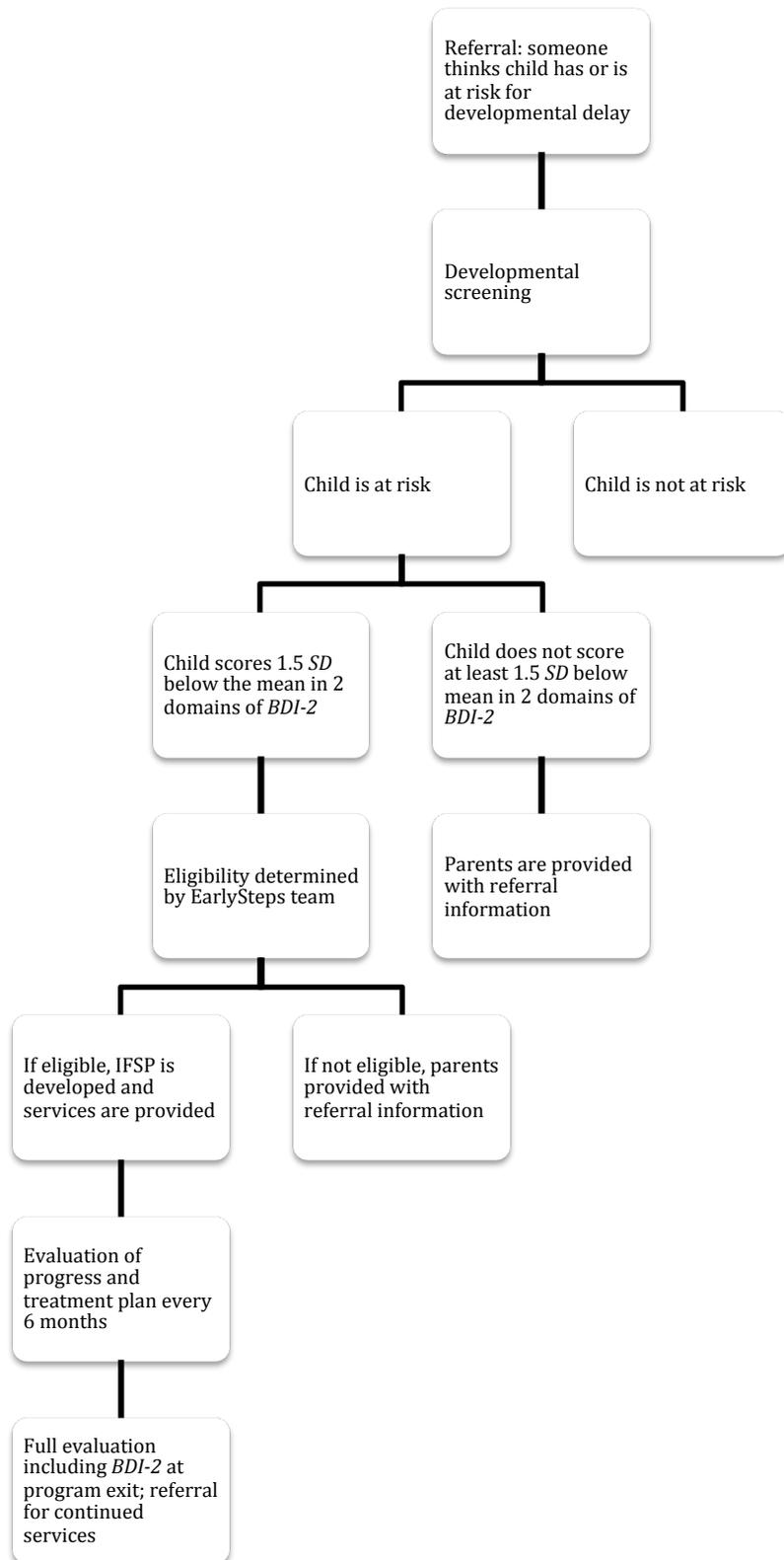


Figure 1  
EarlySteps Referral and Service Provision Process

## **Outcomes Measurement**

The *BDI-2* (Newborg, 2005a) is the developmental assessment tool used statewide for EarlySteps eligibility determination and is also used to collect child outcome data when the child exits the program. The *BDI-2* has been used for collecting and reporting EarlySteps outcomes data since 2007. Outcome data is based on change in positive social-emotional skills (including social relationships), acquisition and use of knowledge and skills (including early language/communication), and use of appropriate behaviors to meet the child's needs (OCDD, 2012b). *BDI-2* domain scores are used to determine change in the areas included in the outcome data. The Personal-Social domain is used to report "positive social-emotional skills, including social relationships." The Communication domain is used to report "acquisition and use of knowledge and skills (including early language/communication)." The Motor domain is used to report the "use of appropriate behavior to meet needs" (OCDD, 2012b).

Children performing 1.5 standard deviations below what would be expected given the child's age are considered performing significantly below expectations (OCDD, 2012b). Aggregate outcome data for fiscal years 2007-2010 included children who had received intervention for a minimum of six months. The data show that of those who entered the program performing significantly below age expectations in social skills, 20.5% significantly increased their rate of development. For the same time period, 34% of children performing below age expectations in language and communication at entrance showed significant improvement in rate of development by time of exit. Finally, for the third outcome criterion, 23.4% showed significant improvement in rate of development for use of appropriate behaviors to meet needs (OCDD, 2012b).

## Purpose

The possibility that developmental skill development as measured by developmental quotient (DQ) can vary significantly in early childhood may provide a strong rationale for the need for targeted early intervention. Widespread screening and efforts to make early intervention services more accessible stand to have a significant impact on the developmental trajectory and prognosis of young children with ASD and other disabilities. However, it is unclear whether those with ASD are benefitting from early intervention services more or less than other atypically developing children.

Additionally, there are few studies examining the stability of developmental quotient in early childhood for children with ASD, with several of the published studies using measures not common in the United States (Kurita, Osada, Shimizu, & Tachimori, 2003; Ogiwara & Takahashi, 2005; Seto & Hatano, 1997; Takeda, 2005, 2007). A review of norm-referenced developmental measures commonly used in the United States revealed that the *BDI-2* is one of the most widely used developmental screeners in the country (Brassard & Bohem, 2007). The *BDI-2* Examiner's Manual (Newborg, 2005b) includes presentation of a series of studies on clinical groups in which all total DQ scores for all clinical groups were between one and two standard deviations below the mean. Groups included children with autism, cognitive delays, developmental delays, premature birth, and language delays from ages birth to 8 years. These studies indicate that the *BDI-2* Total DQ score is useful in discriminating between atypically and typically developing children, but domain scores could not differentiate between these groups (Alfonso, Rentz, & Chung, 2010). However, no test-retest studies were conducted on these special groups, and it is possible that differences may exist in the relative rate of development across domains. Although an investigation of domain stability in a sample of children receiving individualized early intervention may be limited by the variable effects of the interventions, significant differences in development would suggest a need for future studies in this area.

Accardo and Capute (2008) noted that children with developmental delays may exhibit global delays or may demonstrate an uneven pattern of skill development in various domains. Unequal development across domains in children with ASD has been noted since Asperger (1944) first began describing the disorder. By definition, individuals with ASD show developmental delay and often exhibit uneven skill development, as significant social and communication deficits are inherent to the disorder. However, these individuals may otherwise have average or even above average cognitive abilities. In

typically and atypically developing children, early childhood is marked by rapid developmental growth. It is possible that studying the relative change in developmental domains in preschool aged children with ASD may contribute to determining whether differences in development emerge during sensitive time periods in which intervention may have a particularly potent effect.

Although many studies have shown similarities and differences in developmental domains among children with ASD and other disorders at a given age or point in time, relatively few studies have been published regarding stability in scores across time in the preschool demographic. In general, this line of study stands to contribute to our understanding of sensitive periods in developmental domains to add to the body of knowledge regarding developmental differences between children with ASD relative to other atypically developing children. This study also investigates whether atypically developing children with or without ASD have exhibited the greatest developmental gains in various domains during the course of enrollment in Louisiana's EarlySteps program and receipt of individualized intervention programs. The purpose of this study is to investigate possible differences in developmental growth over time between children with and without ASD who were referred to EarlySteps for ASD assessment.

## Method

### Participants

Participants in the current study include 142 toddlers ages 18-36 months and their caregivers recruited through Louisiana's EarlySteps program. Demographics for each group are presented in Table 1.

Table 1  
Overall Sample Demographics

	ASD ( <i>N</i> = 48)	Atypically Developing ( <i>N</i> = 94)	TOTAL ( <i>N</i> = 142)
Age in months			
Time 1	23.3	23.0	23.1
( <i>SD</i> )	(3.9)	(3.6)	(3.7)
Months between Time 1 and Time 2	10.0	9.8	9.8
( <i>SD</i> )	(2.4)	(2.9)	(2.8)
Sex			
Male	74.5%	69.2%	73.8%
Female	25.5%	30.8%	26.2%
Race			
Caucasian	54.2%	48.9%	48.6%
African-Am.	41.7%	42.6%	42.3%
Hispanic	0.0%	5.3%	3.5%
Not specified	10.4%	3.2%	5.6%

Note: *SD* = Standard Deviation.

Under the Individuals with Disabilities Education Act, Part C, this early intervention program provides services throughout the state to children with a suspected developmental delay from birth to 36 months in age. Though anyone may make a referral for assessment, physicians and other healthcare professionals in the state are mandated to refer any toddlers with a suspected developmental delay. Because the EarlySteps screening program and related services are provided statewide, it is believed that this sample is representative of toddlers with developmental disabilities in the state of Louisiana. The participants were selected from a pre-existing database that includes demographic, diagnostic, and evaluation information gathered through the EarlySteps program. Participants were selected on the basis of whether necessary data were included; for the current study, participants needed to have *BISCUIT-Part 1* scores and two administrations of the *BDI-2* in the database as well as birthdate, dates of test administration, and *DSM-IV-TR* diagnosis. Most of the caregivers who participated and provided information regarding their child's development included biological parents, foster or adoptive parents, grandparents, or other relatives. Diagnoses included in the sample include, but are not limited to cerebral palsy, Down syndrome, epilepsy, arthrogryposis, microcephaly, mitochondrial disease, deafness, asthma,

hypoplastic left heart syndrome, premature birth, phenylketonuria, developmental delay, Rett's Disorder, Autistic Disorder, and PDD-NOS.

## **Measures**

**Baby and Infant Screen for Children with aUtism Traits: Part 1 (BISCUIT: Part 1).** The *BISCUIT* was designed as a diagnostic tool for assessing Autistic Disorder and PDD-NOS in children ages 17 to 37 months of age (Matson, Wilkins, Sevin, et al., 2009). The *BISCUIT* also assesses comorbid psychopathology (Part 2) and challenging behaviors (Part 3) commonly occurring in individuals with ASD. The section of interest to this analysis, the *BISCUIT-Part 1*, consists of 62 items in which the parents rate their child's impairments in comparison to typically developing children of the same age on symptoms related to core features of ASD. Items are scored on a 3-point scale: "0" indicates "no difference or no impairment," "1" indicates "somewhat different or mild impairment," and "2" indicates "very different or severe impairment" in comparison to the child's peers. A total score below 17 falls into the "No ASD/Atypical Development" range, scores between 18 and 34 indicate "Possible ASD/PDD-NOS," and a score at or above 35 falls into the "Probable ASD/Autistic Disorder" range (Matson, Wilkins, Sharp, et al., 2009). Internal consistency for this section of the *BISCUIT* was found to be high, Cronbach's alpha = .97 (Matson, Wilkins, Sevin, et al., 2008).

Validity studies found that the *BISCUIT-Part 1* was able to effectively distinguish between those with and without ASD (Matson, Wilkins, & Fodstad, 2011). Furthermore, the sensitivity and specificity was established as .84 and .83, respectively, when differentiating between PDD-NOS and autism (Matson, Wilkins, Sharp, et al., 2009). These statistics slightly increased to .85 and .86, respectively, when distinguishing between PDD-NOS and no diagnosis (Matson, Wilkins, Sharp, et al., 2009). Lastly, the overall classification rate was found to be 88.8 for the *BISCUIT-Part 1* (Matson, Wilkins, Sharp, et al., 2009). The major advantage of this measure is that it is designed to be a diagnostic tool rather than a screener. Additionally, its short administration time and use of scaled scoring are other strengths of the *BISCUIT*. However, it does not include a direct observation component characteristic of other assessment tools.

**Modified Checklist for Autism in Toddlers (M-CHAT).** The *M-CHAT* (Robins, Fein, & Barton, 1999) is an informant-based measure developed as a quick screener to be easily administered by pediatricians and other health care professionals. The *M-CHAT* consists of 23 items to which caregivers

respond “yes” or “no” regarding the child’s typical functioning (Robins, Fein, Barton, & Green 2001). If three or more items are failed, then the screen is considered positive, indicating a need for further assessment. The *M-CHAT* also includes six critical items, and a failure of two or more of these items results in a positive screen. An internal reliability of .85 has been reported, with an internal reliability of .83 for the critical items alone (Robins et al., 2001). Sensitivity and specificity were found to be .87 and .99, respectively (Robins et al., 2001).

**Battelle Developmental Inventory, Second Edition (BDI-2).** The *BDI-2* is an individually administered, norm-referenced test developed to assess multiple domains of development in children from birth to 7 years 11 months of age (Newborg, 2005b). The test was designed to measure developmental strengths in children with and without developmental disabilities and to screen those at risk for developmental disabilities. The *BDI-2* is one of the most widely used tests in the United States to measure multiple domains of development (Brassard & Boehm, 2007). Domains assessed include personal/social, adaptive, motor, communication, and cognitive development (Newborg, 2005b). The 450 items are measured using a Likert scale in which “0” indicates “no ability in this skill,” “1” indicates “emerging ability in this skill,” and “2” indicates “ability at this skill.” The measure provides a total DQ with a mean of 100 and a standard deviation of 15; DQ is also calculated for each domain. Studies using the *BDI-2* have indicated that the measure has sound psychometric properties. Test-retest reliability was evaluated based on 2-25 day intervals between first and second assessment in a group of four-year-old and a group of two year old children and was above .80 for all domain and total scores (Alfonso, Rentz, & Chung, 2010). Newborg (2005) also reports a study of 126 two-year-olds who were given the *BDI-2* twice by the same examiner, averaging two weeks between administrations. The test-retest reliability for domain scores in this sample were all above .87, and overall test-retest reliability was excellent: .93 for the two year old group and .94 for the four year old children (Barton & Spiker, 2007). Internal consistency kappa coefficients ranged from .98 to .99 (Newborg, 2005).

In addition to the normative sample, the *BDI-2* was given to 301 children with various disabilities including autism, cognitive delay, developmental delay, motor delay, speech and language delay, and premature birth (Newborg, 2005). These special groups were not included in the normative sample, as they were “not intended to be representative of the U.S. population or of all possible individuals with the indicated condition” (Newborg, 2005, p. 105). However, convergent validity has been established in these

special groups, including those with ASD and other developmental delays (Newborg, 2005). A series of studies was conducted comparing mean differences in *BDI-2* scores and effect sizes of scores in groups of children either with a developmental delay or at risk of developing the above-mentioned disabilities and delays. When these groups were compared to matched children from the standardization sample, sensitivity in detecting those with expected delay or disability at one standard deviation below the mean level ranged from .75 to .91, with sensitivity below .80 for groups with more heterogeneous developmental patterns (i.e., developmental, motor, and speech/language delay groups; Barton & Spiker, 2007). Elbaum, Gattamorta, and Penfield (2010) reported sensitivity and specificity values that matched or exceeded those of many other commonly used developmental screening tests. Overall, the test was found to have adequate psychometric properties and utility in measuring childhood development (Athanasίου, 2007; Bliss, 2007). For this study, cognitive, social, adaptive, motor, and communication domain scores were analyzed in addition to the total DQ score across test administrations.

### **Procedure**

Data collection for the data used in this study was conducted under approval from both the Louisiana State University Institutional Review Board and Louisiana's Office for Citizens with Developmental Disabilities. After obtaining informed consent from informants, all of whom were parents or other legal guardians, trained test administrators provided participants to be included in this study with a comprehensive assessment battery. Measures given via interview with caregivers include the *BISCUIT*, *M-CHAT*, and *BDI-2*; assessments also included direct observation. Assessments in this study were conducted through the EarlySteps program and occurred in the child's home or daycare setting. The first assessment was conducted when the child first entered the EarlySteps program after referral; the second assessments were conducted at termination of services, most often when the child aged out of eligibility for the EarlySteps program. All test administrators had necessary licensures, degrees, or certifications to qualify for service provision, were experienced in assessment and intervention with young children, and were trained and experienced in administering the measures used. Licensures and certifications included the fields of occupational therapy, physical therapy, special education, social work, speech-language pathology, and psychology; degrees ranged from bachelor's degrees to doctoral degrees in closely related fields. Additionally, each tester had experience in assessment and intervention with young children and with the assessment scales previously described.

All diagnoses in this sample were made by a licensed doctoral psychologist with over 30 years of experience in the field of developmental disabilities. The licensed clinician had access to the information gained from the EarlySteps assessment administrations including the *M-CHAT* and *BISCUIT* and also had access to information from record review. Diagnoses were based on clinical judgment using the *DSM-IV-TR* (APA, 2000) criteria for Autistic Disorder and PDD-NOS. Additionally, a second doctoral level clinical psychologist with expertise in assessment and treatment of children with developmental disabilities provided diagnoses for a subset of the sample ( $n = 97$ ) to measure the interrater reliability of diagnoses. The second clinician was provided with the same clinical and background information but was also blind to previous diagnoses. Using this information, diagnoses were assigned according to clinical judgment and *DSM-IV-TR* criteria. Interrater reliability for diagnoses was high with 98.97% agreement (kappa value .98,  $p < .001$ ). Fombonne et al. (2004) studied this method of diagnosis of pervasive developmental disorders via systematic record review with good results. Fombonne et al. (2004) found a diagnosis of PDD confirmed in 92.5% of reviewed cases, with unconfirmed cases having poor quality data that precluded a positive confirmation of diagnosis. Based on this validation study, it is concluded that the Fombonne method of diagnosis is valid and provides positive predictive value of the diagnosis of *DSM-IV-TR* ASD categories or the broad category of pervasive developmental disorders (Fombonne et al., 2004).

### **Statistical Analyses**

Participants were divided into two diagnostic groups: ASD and Atypically Developing. The ASD group included participants diagnosed with either Autistic Disorder or PDD-NOS according to the *DSM-IV-TR*. PDD-NOS and Autistic Disorder share a great deal of symptomatology and the thresholds differentiating Autistic Disorder versus PDD-NOS under the age of three are difficult to determine (Kleinman et al., 2008), providing rationale for combining these diagnostic categories for the purpose of this study. Furthermore, these diagnostic categories have been collapsed into Autism Spectrum Disorder in the *DSM-5*. Participant membership in these groups was based on the diagnosis received by a licensed clinical psychologist.

G\*Power 3, a power analysis computer program (Faul, Erdfelder, Lang, & Buchner, 2007), was used *a priori* to determine the sample size needed. A medium effect size of .25, power of .80, and alpha of .05 were used. These are conventional and accepted levels for use in psychological research for alpha and power (Cohen, 1988; Hinkle, Wiersma, & Jurs, 2003). A medium effect size was also chosen due to the

widely varying results of studies investigating change in various developmental domains in atypically developing children with and without ASD (e.g. Kuroda & Kato, 1995; Lord & Schopler, 1989; Matson, Mahan, Fodstad, Hess, & Neal, 2010; Ogiwara & Takahashi, 2005). According to the results of the power analysis, a minimum of 122 participants were required to find differences if they existed.

An exploratory data analysis was conducted to investigate errors or missing data for the variables used in this study. Little's Missing Completely at Random test (MCAR; Little, 1988) was conducted to examine whether missing data occurred at random or was related to other variables. The results of the MCAR were insignificant, thus indicating missing data were random (Tabachnick & Fidell, 2013). Donner (1982) recommends retaining a participant if fewer than 10% of individual items are missing, provided those items are replaced with the mean for that item. Accordingly, if a child's *BISCUIT-Part 1* data were missing 6 or fewer items, the missing items were replaced with the overall diagnostic group mean for that item, affecting four participants. The data were taken from an initial sample of 151 participants, with 142 participants remaining after accounting for missing or improperly coded data.

Differences in sex and ethnicity were analyzed using Chi-square, with age further examined using an Analysis of Variance (ANOVA) to compare group differences in age. All analyses were conducted by using SPSS 21.0 with the exception of effect sizes, which were calculated by hand. Group differences were not significant for sex,  $\chi^2(1) = 0.02, p = .89$ , or ethnicity,  $\chi^2(4) = 7.50, p = .111$ . Analyses of variance (ANOVAs) were conducted to examine other possible differences among diagnostic groups. Levene's test was not significant when examining possible group differences in age at first *BDI-2* administration ( $p = .873$ ), indicating the ANOVA assumption of homogeneity of variance was upheld. Results of the ANOVA were also insignificant,  $F(1,140) = 0.21, p = .648$ , indicating no significant difference in age at first administration between diagnostic groups. Similarly, Levene's test was not significant when examining possible group differences in months between first and second *BDI-2* administrations ( $p = .223$ ), indicating the assumption of homogeneity of variance was upheld, and results of this ANOVA were also insignificant,  $F(1,140) = 0.29, p = .593$ . This indicated no significant difference in the number of months separating *BDI-2* administrations between diagnostic groups. Because there were no differences in the above-mentioned variables, they were not added as covariates in subsequent analyses.

Individual developmental quotients for *BDI-2* domain scores across the entire sample ranged from 12-142. The lowest overall mean observed was Communication at Time 1 ( $M = 72.5$ ) and the greatest observed standard deviation observed was Communication at Time 2 ( $SD = 17.40$ ). Normal distribution of data is one assumption for conducting multivariate tests. Domain scores by diagnostic group were tested for normality using the Kolmogorov-Smirnov test. The results of the Kolmogorov-Smirnov test were significant for some dependent variables. Non-normal distributions were found for Motor domain scores at both administrations for both diagnostic groups; Communication scores at both administrations for the ASD group; Social domain scores for the Atypically Developing group at both administrations and at Time 2 for the ASD group; and Adaptive, Communication, and Cognitive scores for the Atypically Developing group at Time 1. However, MANOVAs are generally fairly robust to multivariate non-normality when groups consist of ten or more participants (Tabachnick & Fidell, 2013).

Inspections of skewness and kurtosis revealed all values were within acceptable limits for psychometric purposes (Field, 2005). Box's M value was significant ( $M = 98.89$ ;  $p = .002$ ). According to Tabachnick and Fidell (2013), if sample sizes are unequal then a significant Box's M test should be followed with further analysis of variance ratios. If sample sizes are relatively equal (with a ratio of 4:1 or less), Tabachnick and Fidel (2013) suggest a variance ratio of up to 10:1 is acceptable. In the current analysis, the ratio of variance for all domain scores by diagnostic group remained within acceptable limits ( $F_{\max} = 3.14$ ). Researchers have suggested using Pillai's trace to interpret MANOVA results when groups differ in size and along more than one dimension (Field, 2009); accordingly, Pillai's trace was used in subsequent analysis.

After the *a priori* tests and the tests for assumptions, the main analysis compared *BDI-2* domain scores (i.e., Adaptive, Social, Communication, Motor, and Cognitive) between the two groups using a multivariate analysis of variance (MANOVA). Conducting a MANOVA before conducting follow-up ANOVAs helps protect against the chance of Type I error due to inflated alpha values. Results of the MANOVA were insignificant for effect of administration time on domains by diagnostic group; accordingly, no follow-up ANOVAs were conducted. However, the MANOVA revealed a significant effect of administration time across domain variables across the sample as a whole.

To investigate the significance of score change across *BDI-2* administration for the sample as a whole, paired-samples *t*-tests were conducted. Prior to the analysis, skewness and kurtosis of difference

scores for each domain were inspected for normality of distribution across the entire sample, with all difference scores falling within the expected range with the exception of Communication ( $S = 1.12$ ;  $K = 3.22$ ). However, with larger sample sizes, the impact of departure from zero skew or kurtosis diminishes, and skew and kurtosis for the Communication difference scores are within acceptable limits for this sample size (Tabachnick & Fidell, 2013).

Following significant  $t$ -tests for the Social and Communication domains and near-significant results for the Cognitive domain after Bonferroni corrections, Pearson's correlations were used to investigate relationships between ASD symptom severity according to the *BISCUIT-Part 1* total scores and the degree of change in *BDI-2* administrations. First, a change variable was computed by subtracting domain scores at first administration from domain scores at second administration. The absolute difference value was retained for analysis. Visual inspection of *BISCUIT-Part 1* score scatterplot revealed a positively skewed distribution. Accordingly, *BISCUIT-Part 1* scores were transformed using  $\log(10)$  to normalize distribution before Pearson's correlations were conducted.

## Results

A repeated measures MANOVA was conducted to examine the differences in *BDI-2* domain scores (i.e., Adaptive, Social, Communication, Motor, and Cognitive) between groups between first and second administration. Using Pillai's Trace, no significant difference was found between diagnostic groups,  $V = 0.02$ ,  $F(4, 137) = 0.57$ ,  $p = .683$ ,  $partial \eta^2 = .02$ . Due to these insignificant results, no follow-up ANOVAs were conducted. However, results did reveal a significant difference in the overall sample across the two *BDI-2* administrations,  $V = 0.49$ ,  $F(4, 137) = 32.36$ ,  $p = .000$ ,  $partial \eta^2 = .49$ .

Paired-sample *t*-tests were conducted to investigate the overall sample differences in domain scores between first and second *BDI-2* administration. Scores at first and second administration are presented in Table 2.

Table 2  
Summary of *BDI-2* Domain Scores and Change

<i>BDI-2</i> Domain	Time 1 M(SD)	Time 2 M(SD)	DQΔ M(SD)	<i>r</i>
ASD ( <i>N</i> = 48)				
Adaptive	79.44(15.52)	80.58(11.82)	1.15(12.42)	.62
Social	87.64(12.16)	81.72(11.62)	-6.21(11.78)	.55
Communication	67.67(13.51)	74.89(16.00)	7.23(11.32)	.46
Motor	89.71(14.50)	91.02(16.39)	1.31(17.16)	.55
Cognitive	77.44(11.70)	74.17(11.42)	-3.27(10.38)	.60
Atypically Dev. ( <i>N</i> = 94)				
Adaptive	86.53(12.48)	87.90(11.12)	1.143(13.52)	.62
Social	94.70(11.29)	90.05(11.60)	-4.72(12.55)	.55
Communication	75.40(16.23)	84.52(17.26)	9.47(15.70)	.46
Motor	100.11(14.50)	99.40(12.31)	-0.53(10.99)	.55
Cognitive	84.92(9.70)	83.64(12.14)	-1.34(11.33)	.60
Total ( <i>N</i> = 142)				
Adaptive	84.10(14.01)	85.43(11.84)	1.33(13.12)	.5
Social	92.37(12.13)	87.14(12.26)	-5.23(12.28)**	.49
Communication	72.56(15.79)	81.27(17.40)	8.71(14.37)**	.63
Motor	96.48(17.08)	96.57(14.33)	0.09(13.36)	.65
Cognitive	82.44(11.00)	80.45(12.68)	-1.99(11.02)*	.58

Note: \*  $p < .05$ . \*\*  $p < .01$  in paired samples *t*-test

Bonferroni corrections with an alpha level of .01 were applied to account for multiple tests ( $N = 5$ ). Significant differences were found for change in Social domain scores between *BDI-2* first administration ( $M = 92.37, SD = 12.13$ ) and second administration ( $M = 87.14, SD = 12.26$ );  $t(141) = 5.07, p < .001, d = -0.43$ . Significant differences were also found in the Communication domain scores between *BDI-2* first administration ( $M = 72.55, SD = 15.79$ ) and second administration ( $M = 81.27, SD = 17.39$ );  $t(141) = -7.22, p < .001, d = 0.53$ . Additionally, results approached significance for the Cognitive domain scores between *BDI-2* first administration ( $M = 82.44, SD = 11.00$ ) and second administration ( $M = 80.45, SD = 12.69$ );  $t(141) = 2.15, p = 0.033, d = -0.17$ . These results are presented above in Table 2. A comparative view of scores at first and second administration is presented in Figure 2.

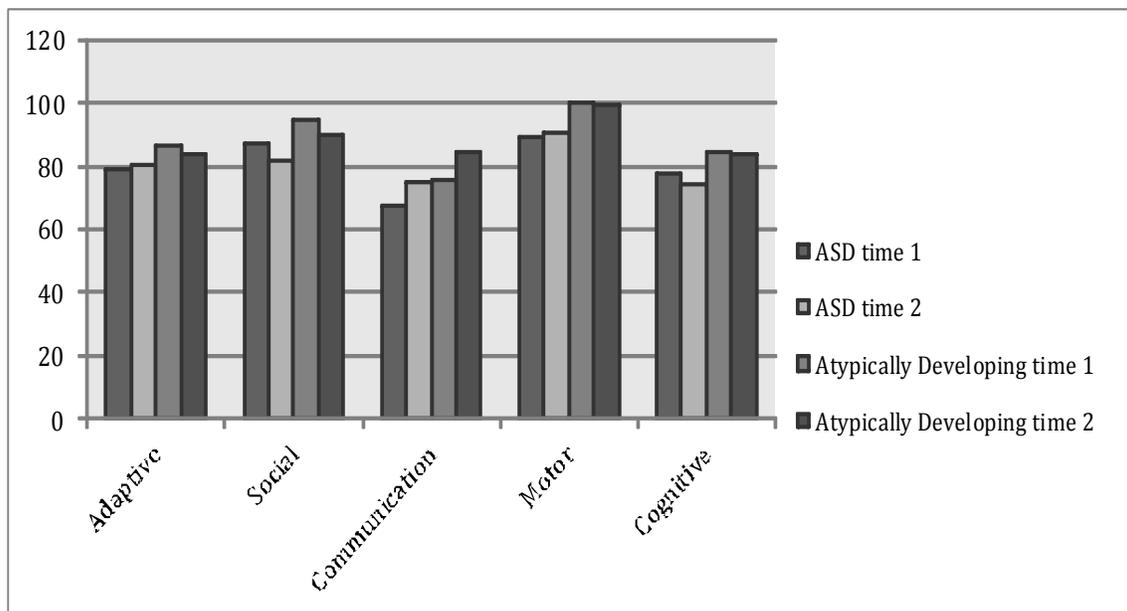


Figure 2  
*BDI-2* Scores by Diagnostic Group and Administration

To investigate the variability in scores between administrations and ASD symptom severity as measured by the *BISCUIT-Part 1* scores at first assessment, Pearson's correlations were computed for the Communication, Social, and Cognitive domains of the *BDI-2*. Results revealed no significant correlation between *BISCUIT-Part 1* scores and change in Social domain scores,  $r = -0.08, N = 142, p = 0.312$ ; Communication domain scores,  $r = -0.116, N = 142, p = 0.168$ ; or Cognitive domain scores,  $r = -0.109, N = 142, p = 0.196$ .

## Discussion

In the current study, Communication domain scores at first administration represented the lowest mean domain scores for both groups, indicating a relatively greater degree of impairment across the entire sample. Further, communication scores were lower for the ASD group than the Atypically Developing group, suggesting more severe impairment in this category. These results are in line with previous research findings that communication is a common first concern among caregivers of atypically developing toddlers with and without an ASD. However, age of first concern tends to be younger in children eventually diagnosed with ASD, thus suggesting more severe impairment (Kozlowski, Matson, Horovitz, Worley, & Neal, 2011). Interestingly, this study did not find a significant difference in age at first assessment between those children who were and were not diagnosed with ASD, despite the fact that those with ASD did exhibit greater impairment as evinced by lower Social and Communication domain scores. Social and communication deficits are critical components of Autistic Disorder (Matson, Fodstad, Hess, & Neal, 2009), and it has also been shown that overall, children with autism exhibit more impairment in the area of communication when compared to children with other developmental delays (Anderson et al., 2007; Matson, Fodstad, Hess & Neal, 2009).

Toddlers with ASD and those with atypical development without ASD have been shown to reach major developmental milestones at different rates (Matson, Mahan, Fodstad, Hess, & Neal, 2010), so it was hypothesized that the above analyses would reveal a significant difference in change in scores over time in some *BDI-2* domains. This hypothesis was upheld, as significant changes in scores were found for the whole sample. The lowest correlations in domain scores between administrations in this sample were: Adaptive,  $r = .495$ ; Social,  $r = .493$ ; and Cognitive,  $r = .575$ . There are no comparable studies over a similar period of time for *BDI-2* domain correlations in typically developing children, so comparisons between this atypically developing sample and typically developing toddlers cannot be made. Nonetheless, these low correlations suggest a great degree of variability in developmental trajectory for the toddlers in this study. Specifically, these scores indicate a great deal of improvement for most children in adaptive and communication skills, with mean domain scores improving over time. However, overall Social domain scores declined over time, indicating that the participants in this study had a tendency to lag further and further behind their typically developing peers in this domain. It is possible that participants in this study received a greater proportion of services targeting other domains, thus widening the gap between

performance in the Social versus other domains. It is also possible that the cumulative effect of delays across several domains may have an additive effect in preventing typical same-age peer interactions and thus typical social development in children with delays in multiple domains. Future studies could investigate whether more targeted interventions for social skills might have a significant impact in reducing the social skills deficits in toddlers receiving EarlySteps services.

Contrary to hypothesized patterns, no significant differences were found between mean change scores or direction of change scores between diagnostic groups. Many different disorders or causes for developmental delay were present in the Atypically Developing group, likely contributing to variability in expected developmental trajectory. Similarly it is possible that the heterogeneity of ASD symptom development, expression, and severity masked differences in developmental trajectory among this group.

It has been estimated that between 10-50% of young children with ASD show regression of communication and/or social skills (Cox et al., 2003; Landa, Holman, & Garrett-Meyer, 2007; Luyster et al., 2005). Estimates of the mean age of regression vary, with most estimates ranging from 18-24 months of age (Davidovitch, Glick, Holtzman, Tirosh, & Safir, 2000; Goldberg et al., 2003; Landa, 2008; Richler et al., 2006). Given the age range at first assessment of the ASD group in this study (18-32 months;  $M = 23$  months), it is likely that some of the participants in this group exhibited regression of skills. Not all children with ASD show regression of skills, however, and many intervention services including those commonly provided by EarlySteps (OCDD, 2012a) target these identified deficits. It was therefore hypothesized that those children with an ASD diagnosis would exhibit greater degrees of variation in change over time in communication and socialization domain scores relative to those individuals with non-ASD related developmental delay. However, this hypothesis was not supported; although the children in this sample with ASD had lower overall Social and Communication scores at both *BDI-2* administrations than the atypically developing children, at both times, their standard deviation of scores was less than the atypically developing group, and the analyses revealed no significant differences in change scores between groups. It is possible that the decision to combine the children with Autistic disorder and the less severely affected children with PDD-NOS into one ASD group may have masked differences between diagnostic groups.

By definition, core symptoms of ASD (e.g., impaired socialization and communication skills) significantly interfere with daily functioning. It is also noted that ASD symptoms occur along a continuum,

with heterogeneous expression and often a great deal of symptom overlap with other disorders. Those who are less severely affected have been shown to benefit from intervention more quickly and to a greater degree (Ogiwara & Takahashi, 2005; Kuroda & Kato, 1995). Acknowledging the continuum of symptom development, diagnostic groups were collapsed to investigate the correlation between ASD symptom severity and change in *BDI-2* domain scores. Surprisingly, no significant correlation existed between ASD symptom severity and *BDI-2* domain score changes. If present, correlations between ASD severity and development may have been more apparent if ASD symptom severity at second *BDI-2* administration was taken into account.

To further investigate the relationship between *BDI-2* domain score change over time and ASD symptom severity, future studies could use ASD severity scores from a second administration of the *BISCUIT-Part 1*, which is typically given at the same time as the second *BDI-2* assessment, but these existing scores were not available at the time of this study. Studies using both *BISCUIT-Part 1* scores could investigate whether ASD symptoms worsened over time for some children, and if change in ASD symptom severity correlated with *BDI-2* domain score changes. Future studies could also investigate differences in *BDI-2* scores and ASD symptoms over time as related to types and amounts of intervention provided through the EarlySteps program, further investigating a possible relationship between treatment provision and the overall trend of increase in Communication domain scores but decrease in Social domain scores for this sample.

With changes to the *DSM-5* diagnostic criteria, the population of children receiving an ASD diagnosis will be more severely impaired than those receiving an ASD diagnosis under the *DSM-IV-TR* (Matson, Belva, Horovitz, & Bamburg, 2012; Matson, Kozlowski, Hattier, Horovitz, & Sipes, 2012; McPartland, Reichow, & Volkmar, 2012; Worley & Matson, 2012), thus differences in developmental trajectory may be more evident in a more homogeneous sample in future studies. Overall, child developmental trajectories are widely varying and largely individual during the first few years of life. Person-centered analytic models, such as cluster analysis or latent class analysis, may be better suited to examining data from this developmental period to account for the uniqueness in developmental trajectory for individual children. This may be particularly true when investigating differences in development among children who are already atypically developing.

## References

- Accardo, P. J., & Capute, A. J. (2008). *Capute & Accardo's neurodevelopmental disabilities in infancy and childhood (3<sup>rd</sup> ed.)*. Baltimore: Paul H. Brookes.
- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, *101*, 213-232.
- Alfonso, V. C., Rentz, E. A., & Chung, S. (2010). Review of the Battelle Developmental Inventory, Second Edition. *Journal of Early Childhood and Infant Psychology*, *6*, 21-40.
- American Academy of Pediatrics Committee on Children with Disabilities (2001). The pediatrician's role in the diagnosis and management of autistic spectrum disorder in children. *Pediatrics*, *107*, 1221-1226.
- American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders (3rd ed.)*. Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders (3rd ed. rev.)*. Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (4th ed., text revision)*. Washington: Author.
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Washington, DC: Author.
- American Psychological Association Task Force on Evidence-Based Practices for Young Children and Adolescents. (2008). *Disseminating evidence based practice for children and adolescents: A systems approach to enhancing care*. Washington, DC: Author.
- Anderson, D. K., Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., . . . Pickles, A. (2007). Patterns of growth in verbal abilities among children with Autism Spectrum Disorders. *Journal of Counseling and Clinical Psychology*, *75*, 594 – 604.
- Asperger, H. (1944). Die "Autistischen Psychophaten" im Kindesalter (Vol. 117, pp. 76-136).
- Asperger, H., & Frith, U. (1991). 'Autistic psychopathy' in childhood. In U. Frith (Ed.), *Autism and Asperger syndrome*. (pp. 37-92). New York, NY: Cambridge University Press.
- Athanasiou, M. (2007). Review of the Battelle Developmental Inventory, 2<sup>nd</sup> Edition, by J. Newborg. In *The seventeenth mental measurements yearbook*. Available from <http://buros.unl.edu/buros/>.
- Autism spectrum disorders revisited. (2011). *Harvard Mental Health Letter*, *28*(4), 1-3.
- Baghdadli, A., Picot, M. C., Michelon, C., Bodet, J., Pernon, E., Burstejn, C., . . . Aussilloux, C. (2007). What happens to children with PDD when they grow up? Prospective follow-up of 219 children from preschool age to mid-childhood. *Acta psychiatrica scandinavica*, *115*(5), 403-412.
- Bagnato, S. J. (2007). *Authentic assessment for early childhood intervention: Best practices*. New York: The Guilford Press.
- Bakwin, H. (1949). Emotional deprivation in infants. *Journal of Pediatrics*, *35*, 512-521.

- Baron-Cohen, S., & Bolton, P. (1993). *Autism: The facts*. Oxford: Oxford University Press.
- Barton, L. R., & Spiker, D. (2007). Review of the Battelle Developmental Inventory, 2<sup>nd</sup> edition, by J. Newborg. In *The seventeenth mental measurements yearbook*. Available from <http://buros.unl.edu/buros/>.
- Batshaw, M. L., Pellegrino, L., & Roizen, N. J. (2007). *Children with disabilities (6<sup>th</sup> ed.)*. Baltimore: Paul H. Brookes Publishing Co.
- Bayley, N. (2005). *Bayley scales of infant and toddler development (3<sup>rd</sup> ed.)*. San Antonio, TX: Pearson.
- Bazzano, A., Zeldin, A., Schuster, E., Barrett, C., & Lehrer, D. (2012). Vaccine-related beliefs and practices of parents of children with autism spectrum disorders. *American Journal on Intellectual and Developmental Disabilities, 117*(3), 233-242.
- Bender, L. (1946). Childhood schizophrenia. *American Journal of Orthopsychiatry, 17*, 40-56.
- Bender, L. (1959). Autism in children with mental deficiency. *American Journal of Mental Deficiency, 64*, 81-86.
- Ben-Itzhak, E., & Zachor, D. A. (2007). The effects of intellectual functioning and autism severity on outcome of early behavioral intervention for children with autism. *Research in Developmental Disabilities, 28*, 287 – 303.
- Bertoglio, K., & Hendren, R. L. (2009). New developments in Autism. *Psychiatric Clinics of North America, 32*, 1 – 14.
- Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.
- Bhakoo, O. N., Kaur, Satwant, Narang, A., & Verma, S. K. (1977). A longitudinal study of variations in development quotients (DQ) in the first 30 months of life. *Indian Journal of Clinical Psychology, 4*, 59-68.
- Bhave, A., Bhargava, R., & Kumar, R. (2011). Correlation between developmental quotients (DASII) and social quotient (Malin's VSMS) in Indian children aged 6 months to 2 years. *Journal of Paediatrics & Child Health, 47*(3), 87-91.
- Bliss, S. L. (2007). Test reviews: Newborg, J. (2005). Battelle Developmental Inventory, Second Edition. *Journal of Psychoeducational Assessment, 25*, 409-415.
- Bradley-Johnson, S. (2001). Cognitive assessment for the youngest children: A critical review of tests. *Journal of Psychoeducational Assessment, 19*, 19-44.
- Brassard, M. R., & Boehm, A. E. (2007). *Preschool assessment*. New York: The Guilford Press.
- Breslau, N., Chilcoat, H. D., Susser, E. S., Matte, T., Liang, K. Y., & Peterson, E. L. (2001). Stability and change in children's intelligence quotient scores: a comparison of two socioeconomically disparate communities. *American Journal of Epidemiology, 154*(8), 711-717.
- 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.
- Buitelaar, J. K., & van der Gaag, R. J. (1998). Diagnostic rules for children with PDD-NOS and multiple complex developmental disorder. *Journal of Child Psychology and Psychiatry, 39*, 911–919.
- Centers for Disease Control. (2012). *Developmental milestones*. Retrieved November 12, 2012 from <http://www.cdc.gov/ncbddd/actearly/milestones/index.html>.

- Centers for Disease Control and Prevention (2010). *Prevalence of Autism Spectrum Disorders (ASDs)*. Retrieved November 1, 2012, from <http://www.cdc.gov/ncbddd/autism/data.html>.
- Chakrabarti, S. (2009). Early identification of autism. *Indian Pediatrics*, *46*, 412-414.
- Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. *Journal of the American Medical Association*, *285*, 3093-3099.
- Chakrabarti, S., & Fombonne, E. (2005). Pervasive developmental disorders in preschool children: Confirmation of high prevalence. *American Journal of Psychiatry*, *162*, 1133-1141.
- Charman, T., & Baird, G. (2002). Practitioner Review: Diagnosis of autism spectrum disorder in 2- and 3-year-old children. *Journal of Child Psychology & Psychiatry & Allied Disciplines*, *43*, 289.
- Charman, T., & Baird, G. (2008). Practitioner review: Diagnosis of autism spectrum disorder in 2-and 3-year-old children. *Journal of Child Psychology and Psychiatry*, *43*(3), 289-305.
- Cohen, J. (1988). *Statistical power analysis for the behavior sciences* (2<sup>nd</sup> ed.). Hillsdale, NJ: Erlbaum.
- Committee on Educational Interventions for Children with Autism. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Cox, A., Klein, K., Charman, T., Baird, G., Baron-Cohen, S., Swettenham, J., . . . Wheelwright, S. (2003). 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.
- Creak, M. (1961). Schizophrenia syndrome in childhood: Progress report of a working party. *Cerebral Palsy Bulletin*, *3*, 501-504.
- Darrou, C., Pry, R., Pernon, E., Michelon, C., Aussilloux, C., & Baghdadli, A. (2010). Outcome of young children with autism: Does the amount of intervention influence developmental trajectories? *Autism*, *14*(6), 663-677.
- Dattalo, P. (2008). *Determining sample size: Balancing power, precision, and practicality*. Oxford University Press, USA.
- Davidovitch, M., Glick, L., Holtzman, G., Tirosh, E., & Safir, M. (2000). Developmental regression in autism: Maternal perception. *Journal of Autism and Developmental Disorders*, *30*, 113-119.
- Dawson, M., Mottron, L., & Gernsbacher, M. A. (2008). Learning in Autism. *Learning and Memory: A Comprehensive Reference*, 759 – 772.
- DeGiacomo, A., & Fombonne, H. (1998). Parental recognition of developmental abnormalities in autism. *European Child and Adolescent Psychiatry*, *7*, 131-136.
- Delmolino, L. M. (2006). Brief Report: Use of DQ for estimating cognitive ability in young children with autism. *Journal of Autism and Developmental Disorders*, *36*, 959-963.
- DeMyer, M. K., Hingtgen, J. N., & Jackson, R. K. (1981). Infantile autism reviewed: A decade of research. *Schizophrenia Bulletin*, *7*(3), 388-451.
- Dewey, D., Cantell, M., & Crawford, S. G. (2007). Motor and gestural performance in children with autism spectrum disorders, developmental coordination disorder, and/or attention deficit hyperactivity disorder. *Journal of the International Neuropsychological Society*, *13*(02), 246-256.
- 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.

- Dor-Shav, N. K., & Horowitz, Z. (1984). Intelligence and personality variables of parents of autistic children. *Journal of Genetic Psychology, 144*, 39-50.
- Eikeseth, S., Smith, T., Jahr, E. & Eldevik, E. (2002). Intensive behavioral treatment at school for 4 to 7 year old children with Autism: A one-Year comparison controlled study. *Behavior Modification 26*, 49–68.
- Eisenberg, L. (1956). The autistic child in adolescence. *The American Journal of Psychiatry, 112*, 607 – 612.
- Eisenberg, L., & Kanner, L. (1956). Early infantile autism, 1943-55. *American Journal of Orthopsychiatry, 26*, 556-566.
- Elbaum, B., Gattamorta, K. A., & Penfield, R. D. (2010). Evaluation of the Battelle Developmental Inventory, 2<sup>nd</sup> edition, screening test for use in states' child outcomes measurement systems under the Individuals with Disabilities Education Act. *Journal of Early Intervention, 12*, 255-273.
- Eldevik, S., Eikeseth, S., Jahr, E. & Smith, T. ( 2006). Effects of low-intensity behavioral treatment for children with autism and mental retardation. *Journal of Autism and Developmental Disorders 36*(2): 211-224.
- Eveloff, H. H. (1960). The autistic child. *Archives of General Psychiatry, 3*, 66-81.
- Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G\*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191.
- Feldman, H. M., Dale, P. S., Campbell, T. F., Colburn, D. K., Kurs-Lasky, M., . . . Paradise, J. L. (2005). Concurrent and predictive validity of parent reports of child language at ages 2 and 3 years. *Child Development, 76*, 856-868.
- Field, A. (2005). *Discovering statistics using SPSS*. London: Sage Publications.
- Fisch, G. S. (2012). Autism and epistemology III: Child development, behavioral stability, and reliability of measurement. *American Journal of Medical Genetics Part A*.
- Fitzgerald, M. (2008). Autism: Asperger's syndrome--history and first descriptions. In J. Rausch, M. Johnson, and M. Casanova (Eds.), *Asperger's Disorder* (pp. 1-6), New York, NY: Informa Health Care.
- Flanagan, D. P., & Harrison, P. L. (Eds.). (2011). *Contemporary intellectual assessment: Theories, tests, and issues*. Guilford Press.
- Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research, 65*, 591–598.
- Fombonne, E., Heavey, L., Smeeth, L., Rodrigues, L. C., Cook, C., . . . Hall, A. J. (2004). Validation of the diagnosis of autism in general practitioner records. *BMC Public Health, 4*(5), Retrieved from <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC394332/>.
- Fombonne, E., Quirke, S., & Hagen, A. (2009). Prevalence and interpretation of recent trends in rates of pervasive developmental disorders. *McGill Journal of Medicine: MJM: An International Forum for the Advancement of Medical Sciences By Students, 12*, 73-73.
- Frances, A. (2010). Opening Pandora's box: The 19 worst suggestions for DSM5. *Psychiatric Times, 27*(2).
- Frith, U. (2004). Emanuel Miller lecture: confusions and controversies about Asperger syndrome. *Journal of Child Psychology & Psychiatry, 45*(4), 672-686.

- Gabriels, R.L., Hill, D.E., Pierce, R.A., Rogers, S.J. & Wehner, B. (2001). Predictors of treatment outcome in young children with autism: A retrospective study. *Autism, 5*, 407-429.
- Gesell, A. (1925). *The mental growth of the pre-school child*. New York, NY: Macmillan.
- Gilliam, J. E. (1995). *Gilliam Autism Rating Scale*. Austin, TX: Pro-Ed.
- Gilliam, W. S., Meisels, S. J., & Mayes, L. C. (2005). Screening and surveillance in early intervention systems. In M. J. Guralnick (Ed.), *The developmental systems approach to early intervention* (pp. 73-89). Baltimore, MA: Paul Brookes.
- 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.
- Grantham-McGregor, S., Cheung, Y. B., Cueto, S., Glewwe, P., Richter, L., . . . Strupp, B. (2007). Developmental potential in the first 5 years for children in developing countries. *The Lancet, 369*, 60-70.
- Guinchat, V., Chamak, B., Bonniau, B., Bodeau, N., Perisse, D., Cohen, D., & Danion, A. (2012). Very early signs of autism reported by parents include many concerns not specific to autism criteria. *Research in Autism Spectrum Disorders, 6*, 589-601.
- Gutierrez, A., Jr., 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*, 630-638.
- Hayward, D. W., Gale, C. M., & Eikeseth, S. (2009). Intensive behavioral intervention for young children with autism: A research-based service model. *Research in Autism Spectrum Disorders, 3*, 571-580.
- Hinkle, D. E., Wiersma, W., & Jurs, S. G. (2003). *Applied statistics for the behavioral sciences*. Boston, MA: Houghton Mifflin Company.
- 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*, 359-83.
- Howlin, P. (2006). Autism spectrum disorders. *Psychiatry, 5*, 320-324.
- Howlin, P., & Asgharian, A. (1999). The diagnosis of autism and Asperger syndrome: findings from a survey of 770 families. *Developmental Medicine & Child Neurology, 41*(12), 834-839.
- Immunization Review Committee (2004). *Immunization Safety Review: Vaccines and Autism*. Washington, DC: National Academies Press.
- Inglese, M. D., & Elder, J. H. (2009). Caring for children with Autism Spectrum Disorder, part I: Prevalence, etiology, and core features. *Journal of Pediatric Nursing, 24*, 41 – 48.
- Johnson, M. K., & Carter, M. R. (2011). Autism Spectrum Disorders: A review of the literature for health educators. *American Journal of Health Education, 42*(5), 311-318.
- Jónsdóttir, S. L., Saemundsen, E., Antonsdóttir, I. S., Sigurdardóttir, S., & Ólason, D. (2011). Children diagnosed with autism spectrum disorder before or after the age of 6 years. *Research in Autism Spectrum Disorders, 5*, 175-184.

- Kamphaus, R. W. (2001). *Clinical assessment of child and adolescent intelligence (2<sup>nd</sup> ed.)*. Needham Heights, MA: Allyn and Bacon.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217-250.
- Kanner, L. (1944). Early infantile autism. *The Journal of Pediatrics*, 25, 211-217.
- Kanner, L. (1951). The conception of wholes and parts in early infantile autism. *The American Journal of Psychiatry*, 108, 23-26.
- Kanner, L. (1954). To what extent is early infantile autism determined by constitutional inadequacies? *Research publications - Association for Research in Nervous and Mental Disease*, 33, 378-385.
- Kanner, L. (1958). The specificity of early infantile autism. *Zeitschrift für Kinderpsychiatrie*, 25, 108-113.
- Kanner, L. (1965). Infantile autism and the schizophrenias. *Behavioral Science*, 10(4), 412-420.
- Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Developmental Disorders*, 1(2), 119-145.
- Kaplan, A. (2012). Research points to shared environmental factors for autism and ASD. *Psychiatric Times*, 29(3), 1-6.
- Kishore, M. T., & Basu, A. (2011). Early concerns of mothers of children later diagnosed with autism: Implications for early identification. *Research in Autism Spectrum Disorders*, 5, 157-163.
- Kleinman, J. M., Ventola, P. E., Pandey, J., Verbalis, A. D., Barton, M., Hodgson, S., ... & Fein, D. (2008). Diagnostic stability in very young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 38(4), 606-615.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, A. Klin, & D. Cohen (Ed.), *Handbook of autism and pervasive developmental disorders, Vol. 1: Diagnosis, development, neurobiology, and behavior* (3 ed., pp. 772-798). Hoboken, NJ: John Wiley & Sons Inc.
- Koegel, L. K., Koegel, R. L., & Smith, R. L. (1997). Variables related to differences in standardized test outcomes for children with autism. *Journal of Autism and Developmental Disorders*, 27, 233-241.
- 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.
- Kozlowski, A. M., Matson, J. L., & Worley, J. A. (2012). The impact of familial autism diagnoses on autism symptomatology in infants and toddlers. *Research in Autism Spectrum Disorders*, 6(1), 151-157.
- Kurita, H., Osada, H., Shimizu, K., & Tachimori, H. (2003). Validity of DQ as an estimate of IQ in children with autistic disorder. *Psychiatry and Clinical Neurosciences*, 57, 231-233.
- Kuroda, Y., & Kato, Y. (1995). Developmental quotient scores: Stability and predictability in very young children with autism. *Japanese Journal of Special Education*, 33(3), 39-45.
- Landa, R. J. (2008). Diagnosis of autism spectrum disorders in the first 3 years of life. *Nature Clinical Practice Neurology*, 4(3), 138-147.
- 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.

- Leach, D. (2010). Bringing ABA into your inclusive classroom: A guide to improving outcomes for students with autism spectrum disorders. Baltimore, MA: Paul H. Brooks.
- Leekam, S., Libby, S., Wing, L., Gould, J., & Gillberg, C. (2000). Comparison of ICD-10 and Gillberg's criteria for Asperger's syndrome. *Autism, 4*, 11-28.
- Lichtenberger, E. O. (2005). General measures of cognition for the preschool child. *Mental Retardation and Developmental Disabilities, 11*, 197-208.
- Lipkin, P. H., & Allen, M. C. (2005). Introduction: Developmental assessment of the young child. *Mental Retardation and Developmental Disabilities Research Reviews, 11*, 171-172.
- Liss, M., Harel, B., Fein, D., Allen, D., Dunn, M., Feinstein, C., ... & Rapin, I. (2001). Predictors and correlates of adaptive functioning in children with developmental disorders. *Journal of Autism and Developmental Disorders, 31*(2), 219-230.
- 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.
- Lord, C. (1995). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry, 36*, 1365-1382
- Lord, C., & Luyster, R. (2006). Early diagnosis of children with Autism Spectrum Disorders. *Clinical Neuroscience Research, 6*, 189 - 194.
- Lord, C., & Schopler, E. (2006). Stability of assessment results of autistic and non-autistic language-impaired children from preschool years to early school age. *Journal of Child Psychology and Psychiatry, 30*(4), 575-590.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology, 55*(1), 3.
- Luyster, R., Richler, J., Risi, S., Hsu, W. L., Dawson, G., Bernier, R., . . . Lord, C. (2005). Early regression in social communication in autism spectrum disorders: a CPEA Study. *Developmental Neuropsychology, 27*(3), 311-336.
- Magiati, I., Charman, T., & Howlin, P. (2007). A two-year prospective follow-up study of community-based early intensive behavioural intervention and specialist nursery provision for children with autism spectrum disorders. *Journal of Child Psychology and Psychiatry, 48*(8), 803-812.
- Manning-Courtney, P., Brown, J., Molloy, C. A., Reinhold, J., Murray, D., Sorensen-Burnworth, R., ... Kent, B. (2003). Diagnosis and treatment of Autism Spectrum Disorders. *Current Problems in Pediatric and Adolescent Health Care, 23*, 283 - 304.
- Martinez-Pedraza, F. L., Carter, A. S. (2009). Autism spectrum disorders in young children. *Child and Adolescent Psychiatric Clinics of North America, 18*, 645 - 663.
- Masten, A., & Coatsworth, J. (1998). The development of competence in favorable and unfavorable environments. *American Psychologist, 53*, 205-220.
- Matson, J. L. (2007). 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*, 207 - 218.
- Matson, J. L., Belva, B. C., Horovitz, M., & Bamburg, J. (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*, 403-414.

- 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., Fodstad, J. C., Hess, J., & Neal, D. (2009). Social and communication behaviors in infants and toddlers with autism and pervasive developmental disorder not otherwise specified. *Developmental Neurorehabilitation, 12*, 152–157.
- Matson, J. L., Hess, J., Sipes, M., & Horovitz, M. (2010). Developmental profiles from the Battelle Developmental Inventory: a comparison of toddlers diagnosed with Down syndrome, global developmental delay and premature birth. *Developmental Neurorehabilitation, 13*(4), 234-238.
- Matson, J. L., & Kozlowski, A. M. (2011). The increasing prevalence of autism spectrum disorders. *Research in Autism Spectrum Disorders, 5*(1), 418-425.
- Matson, J. L., Kozlowski, A. M., Hattier, M. A., Horovitz, M., & Sipes, M. (2012). DSM-IV vs DSM-5 diagnostic criteria for toddlers with autism. *Developmental Neurorehabilitation, 15*(3), 185-190.
- Matson, J. L., Mahan, S., Fodstad, J. C., Hess, J. A., & Neal, D. (2010). Motor skill abilities in toddlers with autistic disorder, pervasive developmental disorder-not otherwise specified, and atypical development. *Research in Autism Spectrum Disorders, 4*(3), 444-449.
- Matson, J. L., Mahan, S., Hess, J. A., & Fodstad, J. C. (2010). Effect of Developmental Quotient on Symptoms of Inattention and Impulsivity among Toddlers with Autism Spectrum Disorders. *Research in Developmental Disabilities: A Multidisciplinary Journal, 31*(2), 464-469.
- Matson, J. L., Mahan, S., Kozlowski, A. M., & Shoemaker, M. (2010). Developmental milestones in toddlers with autistic disorder, pervasive developmental disorder-not otherwise specified and atypical development. *Developmental Neurorehabilitation, 13*(4), 239-247.
- Matson, J. L., & Minshawi, N. F. (2006). *Early intervention for autism spectrum disorders: A critical analysis*. Oxford, England: Elsevier Science, Inc.
- Matson, J. L., & Neal, D. (2009). Diagnosing comorbid psychiatric conditions: autism spectrum disorders. *Psychiatric Times, 26*(4), 38.
- 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. & Wilkins, J. (2008). Nosology and diagnosis of Asperger's syndrome. *Research in Autism Spectrum Disorders, 2*(2), 288-300.
- 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*, 75-84.
- Matson, J. L., Wilkins, J., Sevin, J. A., Knight, C., Boisjoli, J. A., & Sharp, B. (2009). Reliability and item content of the Baby and Infant Screen for Children with aUtism Traits (BISCUIT): Parts 1-3. *Research in Autism Spectrum Disorders, 3*, 336-344.
- Matson, J. L., Wilkins, J., Sharp, B., Knight, C., Sevin, J. A., & Boisjoli, J. A. (2009). 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*, 924 – 930.

- 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 & Adolescent Psychiatry*, 50(6), 583-592.
- McCormick, M. C., Litt, J. S., Smith, V. C., & Zupancic, J. A. (2011). Prematurity: An overview and public health implications. *Annual Review of Public Health*, 32.
- McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for Autism Spectrum Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51, 368-383.
- Medeiros, K., Kozlowski, A. M., Beighley, J. S., Rojahn, J., & Matson, J. L. (2012). The effects of developmental quotient and diagnostic criteria on challenging behaviors in toddlers with developmental disabilities. *Research in Developmental Disabilities*, 33, 1110-1116.
- Ming, X., Brimacombe, M., & Wagner, G. C. (2007). Prevalence of motor impairment in autism spectrum disorders. *Brain and Development-English Edition*, 29(9), 565-570.
- Moffitt, T. E., Caspi, A., Harkness, A. R., & Silva, P. A. (1993). The natural history of change to intellectual performance: Who changes? How much? Is it meaningful? *Journal of Child Psychology and Psychiatry*, 34(4), 455-506.
- Moyal, N. (2010). *The Battelle Developmental Inventory: A study of concurrent validity and stability in young children with known disabilities* (Doctoral dissertation). Farleigh Dickinson University, Hackensack, NJ.
- National Research Council. (2001). *Educating children with autism*. Washington, DC: National Academy Press.
- Newborg, J. (2005a). *Battelle Developmental Inventory, 2<sup>nd</sup> edition*. Itasca, IL: Riverside.
- Newborg, J. (2005b). *Battelle Developmental Inventory, 2<sup>nd</sup> edition, Examiner's manual*. Itasca, IL: Riverside.
- Noterdaeme, M., & Hutzelmeyer-Nickels, A. (2010). Early symptoms and recognition of pervasive developmental disorders in Germany. *Autism*, 14(6), 575-588.
- Noterdaeme, M., Mildemberger, K., Minow, F., & Amorosa, H. (2002). Evaluation of neuromotor deficits in children with autism and children with a specific speech and language disorder. *European Child & Adolescent Psychiatry*, 11(5), 219-225.
- Office for Citizens with Developmental Disabilities [OCDD], Louisiana Department of Health and Hospitals (2012a). *About EarlySteps*. Retrieved from <http://new.dhh.louisiana.gov/index.cfm/page/139/n/139>.
- Office for Citizens with Developmental Disabilities [OCDD], Louisiana Department of Health and Hospitals (2012b). *EarlySteps Part C Annual Performance Report for 2010-2011*. Retrieved from EarlySteps website: <http://new.dhh.louisiana.gov/index.cfm/page/139/n/139>.
- Ogiwara, H., & Takahashi, O. (2005). Changes in developmental and intelligence quotients in children with autism. *Japanese Journal of Child and Adolescent Psychiatry*, 46(4), 439-448.
- Ozonoff, S. (2012). Editorial: DSM-5 and autism spectrum disorders—two decades of perspectives from the JCPP. *Journal of Child Psychology and Psychiatry*.

- Ozonoff, S., Iosif, A.-M., Baguio, F., Cook, I., Hill, M., Hutman, T., . . . Young, G. S. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*, 256–266.
- Parnas, J., Bovet, P., & Zahavi, D. (2002). Schizophrenic autism: Clinical phenomenology and pathogenetic implication. *World Psychiatry, 1*(3), 131-136.
- Petermann, F. (2008). Developmental assessment. *Journal of Psychology; Special Issue: Developmental Assessment, 216*, 125-126.
- Petermann, F., & Macha, T. (2008). Developmental assessment: A general framework. *Journal of Psychology, 216*, 127-134.
- Poland, G. A. (2011). MMR vaccine and autism: Vaccine nihilism and postmodern science. *Mayo Clinic Proceedings, 86*(9), 869-871.
- Price, C. S., Thompson, W. W., Goodson, B., Weintraub, E. S., Croen, L. A., Hinrichsen, V. L., . . . DeStefano, F. (2010). Prenatal and infant exposure to thimerosal from vaccines and immunoglobulins and risk of autism. *Pediatrics, 126*, 656-664.
- Provost, B., Lopez, B. R., & Heimerl, S. (2007). A comparison of motor delays in young children: Autism spectrum disorder, developmental delay, and developmental concerns. *Journal of Autism and Developmental Disorders, 37*(2), 321-328.
- Pry, R., Petersen, A., & Baghdadli, A. (2005). The relationship between expressive language level and psychological development in children with autism 5 years of age. *Autism, 9*(2), 179-189.
- Reed, P., Osborne, L. A., & Corness, M. (2007). The real-world effectiveness of early teaching interventions for children with autism spectrum disorder. *Exceptional Children, 73*(4), 417-433.
- Reed, P., Osborne, L. A., & Corness, M. (2010). Effectiveness of special nursery provision for children with autism spectrum disorders. *Autism, 14*(1), 67-82.
- Rice, C. (2009). *Prevalence of autism spectrum disorders--Autism and developmental disabilities monitoring network, United States, 2006* (pp. 1-21). Department of Health and Human Services, Centers for Disease Control and Prevention.
- Rice, C., Nicholas, J., Baio, J., Pettygrove, S., Lee, L., Van Naarden Braun, K., . . . Yeargin-Allsopp, M. (2010). Changes in autism spectrum disorder prevalence in 4 areas of the United States. *Disability and Health Journal, 3*, 186 – 201.
- Richler, J., Loyster, R., Risi, S., Hsu, W., Dawson, G., Bernier, R., . . . Lord, C. Is there a 'regressive phenotype' of Autism Spectrum Disorder associated with the measles-mumps-rubella vaccine? A CPEA Study. *Journal of Autism and Developmental Disorders, 36*, 299-316.
- Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior*. East Norwalk, CT US: Appleton-Century-Crofts.
- Robins, D. L., Fein, D., & Barton, M. L. (1999). *The modified checklist for autism in toddlers (M-CHAT)*. Storrs, CT: Self-published.
- 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*, 131-144.
- Roid, G. H. (2003). *Stanford-Binet Intelligence Scales, 5<sup>th</sup> edition (SB5)*. Rolling Meadows, IL: Riverside.

- Rutter, M. (1968). Concepts of autism: A review of research. *Journal of Child Psychology and Psychiatry*, 9(1), 1-25.
- Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Developmental Disorders*, 2(3), 315-337.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8, 139-161.
- Rutter, M. (1978). Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, 8, 139-161.
- Rutter, M. (1999). Autism: Two-way interplay between research and clinical work. *Journal of Child Psychology and Psychiatry*, 40(2), 169-188.
- Rydz, D., Shevell, M. I., Majnemer, A., & Oskoui, M. (2005). Developmental screening. *Journal of Child Neurology*, 20, 4-21.
- Sallows, G. O., & Graupner, T. D. (1999, June). Replicating Lovaas' treatment and findings: Preliminary results. In *PEACH conference, London, England*. Retrieved from <http://www.wiautism.com/pdf/ReplicatingLovaas1999.pdf>.
- Salt, J., Shemilt, J., Sellars, V., Boyd, S., Coulson, T., & McCool, S. (2002). The Scottish Centre for autism preschool treatment programme II: The results of a controlled treatment outcome study. *Autism*, 6(1), 33-46.
- Schopler, E. (1971). Parents of psychotic children as scapegoats. *Journal of Contemporary Psychotherapy*, 4(1), 17-22.
- Schopler, E., Mesibov, G. B., & Kunce, L. J. (1998). *Asperger syndrome or high-functioning autism?* New York, NY: Plenum Press.
- Schopler, E., Reichler, R., Bashford, A., Lansing, M., & Marcus, L. (1990). *Psychoeducational profile (Rev. ed.)*. Austin, TX: Pro-Ed.
- Seto, J., & Hatano, E. (1997). A study of variation in the developmental quotient (DQ) of mentally retarded preschool children. *Japanese Journal of Developmental Psychology*, 8(1), 53-64.
- Sevin, B. M., Knight, C. L., & Braud, S. A. (2007). Autism and pervasive developmental disorders. In J. L. Matson (Ed.), *Handbook of assessment in persons with intellectual disability* (pp. 163-195). London: Academic Press.
- Shattuck, P. T. (2006). The contribution of diagnostic substitution to the growing administrative prevalence of autism in US special education [corrected] [published erratum appears in PEDIATRICS 2006 Aug; 118(2): 852]. *Pediatrics*, 117, 1028-1037.
- Sheinkopf, S. J., & Siegel, B. (1998). Home-based behavioral treatment of young children with autism. *Journal of Autism and Developmental Disorders*, 28, 15-23.
- Shevell, M. S., Majnemer, A., Platt, R. W., Webster, R., & Birnbaum, R. (2005). Developmental and functional outcomes at school age of preschool children with global developmental delays. *Journal of Child Neurology*, 20, 648-654.
- Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with Autism, Down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development*, 64, 256.

- Smith, T., Eikeseth, S., Klevstrand, M., & Lovaas, O. I. (1997). Intensive behavioral treatment for preschoolers with severe mental retardation and pervasive developmental disorder. *American Journal on Mental Retardation*, 102(3), 238-249.
- Society for Research in Child Development. (2008). The effects of early social-emotional and relationship experience on the development of young orphanage children: IX. The effects of the intervention on children's general behavioral development (Battelle Developmental Inventory). *Monographs of the Society for Research in Child Development*, 73(3), 142-166.
- Sparrow, S., Balla, D., & Cicchetti, D. (1984). *Vineland Adaptive Behavior Scales*. Circle Pines, MN: American Guidance Service.
- Sparrow, S., Cicchetti, D. V., & Balla, D. A. (2005). *Vineland adaptive behavior scales (2<sup>nd</sup> ed.)*. San Antonio, TX: Pearson.
- Stokstad, E. (2008). Stalled trial for autism highlights dilemma of alternative treatments. *Science (New York, N.Y.)*, 321(5887), 326-326.
- Sutera, S., Pandey, J., Esser, E. L., Rosenthal, M. A., Wilson, L. B., Barton, M., . . . Fein, D. (2007). Predictors of optimal outcome in toddlers diagnosed with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 37(1), 98-107.
- Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics, 6<sup>th</sup> ed.* New York: Pearson.
- Takeda, T., Koyama, T., Kanai, C., & Kurita, H. (2005). Clinical variables at age 2 predictive of mental retardation at age 5 in children with pervasive developmental disorder. *Psychiatry and Clinical Neurosciences*, 59(6), 717-722.
- Takeda, T., Koyama, T., & Kurita, H. (2007). Comparison of developmental/intellectual changes between autistic disorder and pervasive developmental disorder not otherwise specified in preschool years. *Psychiatry and Clinical Neurosciences*, 61(6), 684-686.
- Towbin, K. (1997). Pervasive developmental disorders not otherwise specified. In D. J. Cohen (Ed.), *Handbook of autism and pervasive developmental disorders* (pp. 123-147). New York: John Wiley & Sons, Inc.
- Turner, L. M., & Stone, W. L. (2007). Variability in outcome for children with an ASD diagnosis at age 2. *Journal of Child Psychology and Psychiatry*, 48, 793-802.
- US Department of Health and Human Services. (2011). 2011 Strategic Plan for Autism Spectrum Disorder Research: Interagency Autism Coordinating Committee.
- Van Krevelen, D. A. (1963). On the relationship between early infantile autism and autistic psychopathy. *Acta Paedopsychiatrica*, 30, 303-323.
- Van Krevelen, D. A. (1971). Early infantile autism and autistic psychopathy. *Journal of Autism and Child Schizophrenia*, 1, 82-86.
- Volkmar, F. R. (1998). Categorical approaches to the diagnosis of autism: An overview of the DSM-IV and ICD-10. *Autism*, 2, 45-59.
- 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, third edition, volume 1: Diagnosis, development, neurobiology, and behavior*, (pp 5 – 41). New York: Wiley.

- Volkmar, F. R., Klin, A., & Cohen, D. J. (1997). Diagnosis and classification of autism and related conditions: Consensus and issues. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders, second edition* (pp. 5-40). New York: John Wiley & Sons, Inc.
- 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.
- Wechsler, D. (1974). *WISC-R, Wechsler intelligence scale for children, revised*. San Antonio, TX: Psychological Corporation.
- Weis, M. J., Fiske, K., & Ferraioli, S. (2008). Evidence-based practice for autism spectrum disorders. In J. L. Matson (Ed.), *Clinical assessment and intervention for autism spectrum disorders* (pp. 91-129). Burlington, MA: Elsevier.
- Werner, E., & Dawson, G. (2005). Validation of the phenomenon of autistic regression using home videotapes. *Archives of General Psychiatry, 62*(8), 889.
- Werner, E., Dawson, G., Munson, J., & Osterling, J. (2005). Variation in early developmental course in autism and its relation with behavioral outcome at 3–4 years of age. *Journal of Autism and Developmental Disorders, 35*(3), 337-350.
- Wing, L. (1981). Asperger's syndrome: a clinical account. *Psychological Medicine, 11*(1), 115-129.
- Wing, L. (1997). The autism spectrum. *Lancet, 350*, 1761-1766.
- World Health Organisation. (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research (10th ed.)*. Geneva: Author.
- 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-5 diagnostic criteria. *Research in Autism Spectrum Disorders, 6*, 965-970.

## **Vita**

Lindsey Willis Williams, a native of Prosperity, South Carolina, received her bachelor's degree at Erskine College in 2006 and then a master's degree in Rehabilitation Counseling and Psychology from the University of North Carolina at Chapel Hill in 2009. Thereafter, she worked providing employment support for adults with autism at University of North Carolina's Division TEACCH. As her interest in developmental disabilities grew, she made the decision to enter Louisiana State University's Clinical Psychology Doctoral Program under the supervision of Dr. Johnny L. Matson. Her clinical and research interests include the assessment and treatment of autism spectrum disorders and other developmental disabilities across the lifespan.