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# Developmental Functioning of Infants and Toddlers with Autism and Cerebral Palsy

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DEVELOPMENTAL FUNCTIONING OF INFANTS AND TODDLERS WITH AUTISM  
AND CEREBRAL PALSY

A Thesis

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Louisiana State University and  
Agricultural and Mechanical College  
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in

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by

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## TABLE OF CONTENTS

Abstract.....	iii
Introduction.....	1
Purpose.....	16
Method .....	18
Results.....	23
Discussion.....	27
References.....	33
Vita.....	49

## Abstract

Children with Autism Spectrum Disorder (ASD) often have one or more comorbid medical conditions including cerebral palsy (CP). Due to the overlaps between ASD and CP symptomatology (e.g., impairments in speech, repetitive movements, atypical sensory issues), co-occurring CP often leads to delayed diagnoses of ASD interfering with early interventions and subsequently affecting functional outcome. Utilizing the *Baby and Infant Screen for Children with aUtism Traits-Part 1 (BISCUIT-Part 1)* and the *Battelle Developmental Inventory, Second Edition (BDI-2)*, this study assessed and compared ASD symptomatology and developmental functioning in three groups of infants and toddlers, namely, those with ASD only, those with CP only, and those with comorbid ASD and CP. Results of the current study revealed significant group differences were found in both *BISCUIT-Part 1* and *BDI-2* total scores. Significant differences were also found in all *BISCUIT-Part 1* domains and *BDI-2* personal-social and motor domains. Interpretation and implication of the study findings are discussed.

## Introduction

One of the greatest changes in the field of developmental disabilities for the past decades was the emergence of autism spectrum disorder (ASD) as a primary diagnosis. Along with this change was the increase of autism prevalence; next to intellectual disabilities, ASD is now the second most frequent major developmental disability (Newschaffer et al., 2007). Correspondingly, great effort has been put into the development of treatments and services for individuals with autism and early interventions have been identified as a key contributor to better prognosis of children with ASD (Estes et al., 2015; Hampton & Kaiser, 2016; McEachin, Smith, & Lovaas, 1993; Reichow, 2012). While ASD can be diagnosed before the age of 2 (Moore & Goodson, 2003; Valicenti-McDermott, Hottinger, Seijo, & Shulman, 2012), many children are not diagnosed until later delaying their timely entry into services. Various factors have been found to be related with delayed diagnoses including the presence of comorbid conditions (e.g., cerebral palsy [CP], attention-deficit/hyperactivity disorder, fragile X syndrome, tuberous sclerosis, Down syndrome; M. L. Bauman, 2010; Eric Fombonne, Mazaubrun, Cans, & Grandjean, 1997; Gillberg & Coleman, 1996; Miodovnik, Harstad, Sideridis, & Huntington, 2015; Smile, Dupuis, MacArthur, Roberts, & Fehlings, 2013).

ASD is characterized by deficits in social communication and the presence of restrictive, repetitive patterns of behavior or interest (RRB; American Psychiatric Association, 2013), and CP refers to a group of disorders that affect one's movement and posture that develop early in life due to non-progressive disturbances in the brain (Bax et al., 2005). Both ASD and CP are heterogeneous early onset conditions with multiple medical comorbidities and a

wide spectrum of symptomatology (Mannion, Leader, & Healy, 2013; Pruitt & Tsai, 2009; Shevell, Dagenais, Hall, & REPACQ Consortium, 2009; Simonoff et al., 2008). Due to the overlaps in symptom presentations (e.g., impairments in speech, repetitive movements, atypical sensory issues), diagnosis of autism can be complicated by comorbid CP as clinicians may mistakenly contribute all impairments to CP (Smile et al., 2013).

Few studies have inspected the effects of co-occurring CP on the functioning of children with ASD. To further investigate the differences between children with ASD, CP, and comorbid ASD and CP, this study assessed and compared ASD symptomatology and developmental functioning of these three groups. The goal of the current study is to add to the limited research on ASD and CP comorbidity and potentially contribute to more accurate and earlier detection of ASD in individuals with CP. Prior to the study, a brief introduction to ASD and CP will be presented.

### **Autism Spectrum Disorder**

**Leo Kanner.** Autism was first described in its modern sense in 1943 by Leo Kanner in his paper, “Autistic Disturbance of Affective Contact.” Detailed accounts were given on 11 children who exhibited “extreme *autistic aloneness* that, whenever possible, disregards, ignores, shuts out anything that comes to the child from the outside” (Kanner, 1943). While heterogeneity was present among the eight males and three females in several aspects (e.g., level of disturbance, presentation of symptoms, family relationships, and developmental history), they shared a set of core symptoms that could not be accounted for by any existing diagnoses at that time (Kanner, 1943). The most fundamental symptom identified by Kanner (1943) was an “inability to relate” to others. These children showed preference for

objects over people and very minimal eye contact; they were described as showing “no apparent affection.” Deficits in communication (Kanner, 1943) were also observed in all children. Three of them were mute and for those who did develop language, their speech was mainly composed of echolalia (i.e., repetition of words or phrases spoken by others) and was not used to communicate. Another marked shared characteristic was “an anxiously obsessive desire for the maintenance of sameness” (Kanner, 1943). Changes in routines or the environment and anything incomplete or broken would trigger an excessive amount of distress. These symptoms were all present very early in life and in a later paper, he used the term “early infantile autism” to describe the symptom patterns of these children (Kanner, 1944).

**Hans Asperger.** In 1944, another researcher, Hans Asperger also published his work on “autistic” children, however, his work went unnoticed at the time, possibly due to the fact that his paper “Autistic Psychopathy in Children” was originally published in German and was not translated into English until 1991 (Asperger & Frith, 1991). Despite the vast difference in the attention these two studies received at the time of publication, they bore many similarities including the choice of the term “autism.” Like Kanner, Asperger also reported deficits in social interactions (e.g., inability to understand others’ emotions, lack of interest in other individuals), verbal and nonverbal communication (e.g., echolalia, paucity of gestures), and presence of stereotypic interests and behaviors (e.g., collecting objects, lining up objects).

**Childhood Schizophrenia.** The word “autism” was initially coined by Eugen Bleuler, a Swiss psychiatrist, referencing a symptom of schizophrenia described as “an active

turning-away from the external world” (Bleuler, 1951; Kuhn, 2004). For years, confusion remained over whether childhood autism is a subtype of schizophrenia. In their initial case studies, Kanner and Asperger both argued that schizophrenia and autism are two distinct conditions (Asperger & Frith, 1991; Kanner, 1943). Although the disconnection with the outside world is present in both of these conditions, Kanner argued that while the detachment from the outside world is a result of withdrawal from the reality in schizophrenia, individuals with autism exhibit this disconnection due to their innate inability to relate instead of withdrawal (Kanner, 1965). The onset of symptoms was also noted to be different; while schizophrenia manifests itself in a gradual fashion, autism has an earlier onset and is obvious from the beginning of life (Kanner, 1943). In addition to these aspects, Asperger asserted that schizophrenia and autism also exhibit different course, specifically, individuals with schizophrenia often experience a progressive deterioration while autism remains relatively stable over time (Asperger & Frith, 1991).

**Etiology.** During the early years, researchers have taken many different perspectives in an attempt to account for the etiology of this condition. Kanner initially contributed autism to the lack of affection from the parents or “emotional refrigeration” (Eisenberg & Kanner, 1956; Kanner & Eisenberg, 1957). Behavioral theories were also utilized and researchers hypothesized that autism is due to faulty learning and inappropriate conditioning (Ferster, 1961; Lovaas, Freitag, Gold, & Kassorla, 1965). Others have chosen the route of physiological perspective and speculated that autism is a result of brain abnormalities (M. Bauman & Kemper, 1985; Rimland, 1964). While the former two hypotheses have been shown to be unlikely, subsequent studies have suggested a strong neurobiological basis that



individuals with ASD exhibit abnormal neural structure and integrity when compared to those without autism (Baron-Cohen et al., 2000; Currenti, 2010; Trottier, Srivastava, & Walker, 1999). Although genetic factors were initially dismissed by many, more recently, they have proven to play a significant role in the etiology of ASD along side with the environment, supported by the evidence of high concordance rates between twins and siblings (Currenti, 2010; Rutter, 2000; Trottier et al., 1999), and accumulated data on the impact of environmental factors (Herbert, 2010; Hertz-Picciotto et al., 2006).

**Diagnostic Criteria.** Autism made its first appearance as an official diagnosis under the category of Pervasive Developmental Disorders (PDDs) in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III)* in 1980, referred to as Infantile Autism (Rutter & Schopler, 1988). To qualify for a diagnosis of Infantile Autism, one must exhibit early on-set (30 months or earlier) of pervasive deficits in social relationships, language and/or communication, without the presence of delusions or hallucinations as seen in schizophrenia. Also under the label of PDDs, there were also Childhood Onset PDD and Atypical PDD. In 1987, a revision of the *DSM-III*, the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R)*, was published and the label of Infantile Autism was changed to Autistic Disorder while Atypical and Childhood Onset PDDs were changed to Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS; Volkmar, Bregman, Cohen, & Cicchetti, 1988). Further changes were made in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*, where five categories of PDDs were developed, including Autistic Disorder, Asperger’s Disorder, PDD-NOS, Rett’s Disorder, and Childhood Disintegrative Disorder. These categorizations

were then continued to be used in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revised (DSM-IV-TR; Sipes & Matson, 2014)*. To be diagnosed with Autistic Disorder under *DSM-IV-TR*, one must exhibit six or more of the diagnostic items prior to the age of three, and the items endorsed must include at least two or more items from the social domain, one or more from the communication domain, and one or more from the restricted, repetitive, and stereotyped patterns of behavior domain.

**Current Diagnostic Criteria.** Modifications of the diagnostic criteria continued and in 2013, the latest edition of the *DSM*, the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* was published (American Psychiatric Association, 2013). The *DSM-IV-TR* was criticized for its indefinite diagnostic criteria of and vague boundaries between each of the categories of autism (Matson, Belva, Horovitz, Kozlowski, & Bamburg, 2012). To address this concern, the *DSM-5* introduced the concept of spectrum and collapsed Autistic Disorder, Asperger’s Disorder, and PDD-NOS into one diagnosis of ASD, while the other two, Rett’s Disorder and Childhood Disintegrative Disorder, were completely removed.

Another adjustment is that instead of a clear age cut-off, the *DSM-5* adopted a more relaxed standard, specifically, while symptoms must be present in early childhood, they “may not become fully manifest until social demands exceed limited capacities” (American Psychiatric Association, 2013). Changes were also made to the domains of impairments, while the domain of restricted, repetitive patterns of behavior, interests, or activities was preserved, the social and communication domains in the *DSM-IV-TR* were merged into communication and social interaction domain. To qualify for a diagnosis of ASD, one must

endorse all three items in the communication and social interaction domain, namely: (1) impairment in social-emotional reciprocity, (2) deficits in nonverbal communication, and (3) impairment in developing and maintaining relationships. In addition, one is also required to evince at least two of the four items in the restricted, repetitive patterns of behavior, interests, or activities domain, including: (1) stereotyped, repetitive speech, motor movements, or object manipulations, (2) strict adherence to routines, ritualized patterns of verbal or nonverbal behavior, or extreme resistance to change, (3) highly restricted, fixated interests that are unusual in intensity or focus, and (4) over or under reactivity to sensory input or abnormal interest in sensory aspects of the environment (American Psychiatric Association, 2013).

**Prevalence of ASD.** Along with the changes in people's understanding of the etiology of and in the diagnostic criteria for autism, researchers have also noticed a dramatic increase in its prevalence. Autism was once considered a rare condition and early studies mostly reported prevalence rates of 2 to 6 per 10,000 children (E. Fombonne, 1999; C. Gillberg & Wing, 1999; Hoshino, Kumashiro, Yashima, Tachibana, & Watanabe, 1982; Wing, 1993). While the estimates varied across the U.S. and other countries, in recent prevalence studies there have been a constant increase for the past couple of decades (Chakrabarti, 2001; C. Gillberg & Wing, 1999; Honda, Shimizu, Imai, & Nitto, 2005). Starting in 1999, the Centers for Disease Control and Prevention (CDC) began to establish a multi-site surveillance network, referred to as the Autism and Developmental Disabilities Monitoring (ADDM) network, to monitor the prevalence of autism in 8-year-old children across the country (Rice et al., 2007). For the surveillance year of 2006, the CDC reported

approximately 9 out of every 1,000 individuals were affected by autism, in 2008 a rate of 11.3 per 1,000 was reported, which continued to grow and reached 14.7 per 1,000 in 2010 (Baio, 2012; Developmental & Investigators, 2014; Rice, 2009).

More and more children are being diagnosed with autism, while we are not fully certain if this is a genuine increase in incidence, many have hypothesized that multiple factors are at play for the drastic and continued raise in autism diagnoses, including changes in diagnostic criteria, increased awareness and acceptance of this condition, and earlier age of diagnoses (Matson & Kozlowski, 2011; Wing & Potter, 2002). Since Kanner's description in 1943, autism's diagnostic standards and procedures have changed greatly, many have hypothesized and demonstrated that the changes in inclusion criteria for identification can account for most of the increase in prevalence results (Eric Fombonne, 2005; King & Bearman, 2009).

Additionally, with the accumulation of research in autism, also came the increase in numbers of advocacy associations, media exposure, availability of educational and medical resources, and parents' awareness; and these may facilitate parents identifying potential symptoms and requesting assessments for their children, leading to increased diagnoses of autism (Wing & Potter, 2002; Yeargin-Allsopp et al., 2003). Lastly, another noticeable change in the field of autism is the steadily dropping age of diagnoses. At this time, it is generally accepted that children with autism can be reliably diagnosed before the age of two (Moore & Goodson, 2003; Valicenti-McDermott et al., 2012), and this decrease in age of diagnosis might also have contributed to inflated prevalence rates (Leonard et al., 2010).

### **Cerebral Palsy**

**History of CP.** Early 1800s, CP was first extensively studied by William Little, an English surgeon. In a series of lectures entitled “Deformities of the Human Frame,” Little described “a peculiar distortion which affects new born children... the spasmodic tetanus-like rigidity and distortion of the limbs... traced to asphyxia neonatorum, and mechanical injury to the fetus immediately before or during parturition.” For many years that followed, CP was referred to as “Little’s Disease” (Baxter et al., 2007; Schifrin & Longo, 2000).

Although mostly known for his other contributions to the field of psychology and medicine, Sigmund Freud, an Australian neurologist, also made significant contributions to CP and they are still affecting current research in the field. Freud was the first to use CP as a nosographic category (i.e., infantile CP, which encompasses several infantile neurological motor deficits), and his classification system of different types of infantile CP (i.e., general cerebral stiffness, paraplegic stiffness, bilateral hemiplegia, and general chorea and bilateral athetosis) greatly inspired the categorizations used today (Kavcic & Vodusek, 2005).

Continuing to the 21st century, despite the accumulation of new knowledge, development of technology, and numerous changes, the discussion of the definition of CP still goes on without a precise and universally accepted definition. However, from the most frequently cited definition, which refers to CP as “a disorder of posture and movement due to a defect or lesion in the immature brain” (M. C. O. Bax, 1964) to more recent literature, many of them do seem to share some common themes, including: 1) CP is an umbrella term for a group of disorders, 2) it affects one’s movement and posture, 3) these negative effects are permanent but not unchanging, 4) it is due to brain disturbances in the early developmental

stages, 5) these brain interferences are non-progressive (M. Bax et al., 2005; Baxter et al., 2007; Rosenbaum P, Paneth N, Leviton A, Goldstein M, & Bax M, 2007).

**Prevalence of CP.** Along with autism and intellectual disabilities, CP is one of the three most prevalent lifelong developmental disabilities (Sankar & Mundkur, 2005). The ADDM network has also been collecting and monitoring the prevalence rates of CP, which compared to those of ASD, have stayed relatively stable for the past couple of decades at around 1.5 to 3.5 per 1000 (Christensen et al., 2014; Durkin et al., 2016; Kirby et al., 2011). In preterm infants and those with low birthweight, however, different patterns were observed. In the 1980s, there was a sharp increase in CP prevalence in preterm and/or low birth weight infants, which was hypothesized to be related to the emergence and improvement of newborn intensive care, leading to increased survival rates of these infants. In more recent studies, however, there has been a decrease in CP prevalence in this population (Paneth, Hong, & Korzeniewski, 2006; Platt et al., 2007).

**Etiology of CP.** In many cases the causes of CP are unknown, and for the others, numerous factors have been identified as the potential causal factors for CP, including genetic abnormalities, maternal drug use, intrauterine infections, white matter injury often seen in premature infants, placenta abruption, and complications during labor or delivery, etc. A commonly used classification method is grouping these factors into temporal categories based on when these insults happened, specifically, prenatal, perinatal, and postnatal (Mecham, 1996; Odding, Roebroek, & Stam, 2006).

**Classification Systems.** There are three popular classification systems commonly used for CP, and they are each based on motor type, functional severity, and topographical

distribution. Surveillance of Cerebral Palsy in Europe (SCPE) adopted a hierarchical classification of CP with three subtypes based on its motor dysfunctions, including spastic, ataxic, and dyskinetic CP (Cans, 2000). Most cases of CP are spastic CP, characterized by increased muscle tone, including exaggerated reflexes and stiffness (Cans, 2000; Shevell, Dagenais, Hall, & THE REPACQ CONSORTIUM, 2009). Ataxic CP is the rarest form of CP and affected individuals experience loss of muscular coordination and exhibit abnormal force, rhythm and accuracy in their movements (Cans, 2000; Sankar & Mundkur, 2005). Varying degrees of muscle tone and activity levels are the features of dyskinetic/athetoid CP; individuals with reduced activity and increased tone are said to have dystonic CP and those who have increased activity but decreased tone are said to have choreo-athetotic CP (Cans, 2000; O'Shea, 2008).

Another widely utilized classification system is the Gross Motor Function Classification System (GMFCS). The GMFCS provides five age-specific gross motor functioning levels based on one's motor abilities and limitations, from level I being most independent and able (i.e., the child can walk, run, and climb without limitation, however, speed, coordination, and balance are impaired), to level V being least independent and able (i.e., the child does not have voluntary control of movements or posture; Graham, 2005; R. Palisano et al., 1997). The GMFCS grading system has demonstrated adequate reliability and validity; and the different levels have been shown to be predictive of children's developmental trajectory indicating clinical utility (R. J. Palisano et al., 2000; Wood & Rosenbaum, 2000).

From a topographic perspective, CP can also be divided into the different subtypes.

The most commonly used three categories are hemiplegic CP, where the arm and leg of only

one side of the body are affected; diplegic CP, involving all four limbs with the lower limbs more affected; quadriplegic CP, where all four limbs are involved with the upper limbs more or equally affected (Howard et al., 2005). Other categories are also used but not consistently, including monoplegic CP, where only one limb is affected; paraplegic CP, where the lower half of the body including both legs are affected; and triplegic CP, where three limbs are affected, etc. Unlike the GMFCS, this topographical classification system has failed to provide support of sufficient reliability. However, it has been suggested that the combination of the above three systems of classification may be beneficial for the study of CP (Graham, 2005).

### **Cerebral Palsy and Autism**

The ADDM has reported that 7 to 8% of children with CP also exhibit co-occurring ASD (Christensen et al., 2014; Durkin et al., 2016; Kirby et al., 2011). Although other researchers have reported lower comorbid rates, findings remained consistent in that compared to those without CP, higher rates of ASD were found in individuals with CP (Christensen et al., 2014; Kilincaslan & Mukaddes, 2009; Kirby et al., 2011). While it is most widely known by impairments in motor functions, recent research has highlighted that many other aspects of functioning are also frequently affected and should be considered in the study of CP. These include deficits and abnormalities in speech, sensory and perception, cognition, communication, and the presence of behavior problems (Baxter et al., 2007; Rosenbaum P, Paneth N, Leviton A, Goldstein M, & Bax M, 2007; Smile et al., 2013). Many of these domains overlap with the affected areas of autism (e.g., repetitive movements,



communication deficits, cognitive impairments) leading on difficulties identifying and diagnosing ASD in children with CP (Smile et al., 2013).

**Motor Skills.** Obligatory for a diagnosis of CP, varying levels of motor difficulties are experienced by children with CP, including deficits in gross motor functioning (e.g., walking, standing, running) and fine motor skills (e.g., grasping, manipulation, holding of objects; Beckung & Hagberg, 2007). Similarly, the majority of children with ASD were also found to exhibit deficits in motor functioning including both gross and fine motor skills (Lloyd, MacDonald, & Lord, 2013; Provost, Lopez, & Heimerl, 2006). Delays in motor development are frequently reported by parents of children with CP and ASD to be their early concerns, and the detection of motor delays have been found to be related with earlier detection and diagnosis of both conditions (Chawarska, Paul, et al., 2007; Palmer, 2004).

The etiology and presentations of motor difficulties vary across different subtypes of CP. While spastic CP is characterized by persistent and increased muscle tone and reflexes, ataxic CP is accompanied by loss of coordination and control of movement, and many with dyskinetic CP are troubled with involuntary and stereotyped movements (Cans, 2000). While these stereotyped movements differ from the RRBs observed in ASD in that these movements are emitted involuntarily while children with ASD voluntarily engage in repetitive behaviors or actions. However, the topography of these stereotyped movements and RRBs can be very similar (e.g., they may both present as repetitive hand or arm movements) which may lead to confusion during diagnostic processes.

**Communication Skills.** Children with CP may also experience impaired language and communication due to motor difficulties, sensory deficits, intellectual impairments, or often,

a combination of multiple factors (Andersen, Mjøen, & Vik, 2010; Parkes, Hill, Platt, & Donnelly, 2010; Parkes et al., 2010; Rosenbaum P et al., 2007). While motor deficits can lead to difficulties with articulation and gesturing, sensory deficits including vision and hearing impairments can affect the perception and learning of language, and cognitive impairment may hinder processing and understanding of language (Pennington, 2008). Inherent to ASD, affected individuals also exhibit communication deficits including lack of initiation of communication, reduced response to interactions, and delayed verbal language development due to underlying neurodevelopmental disturbances (Chawarska, Klin, Paul, & Volkmar, 2007; Landa, Holman, & Garrett-Mayer, 2007). Targeting different causes of deficits, treatments of communication difficulties in CP and ASD focus on disparate goals, while interventions for CP mostly aim for improvement of intelligibility of existing speech, those for individuals with ASD mainly focus on the increase of use of speech in general, thus the identification and differentiation of ASD and CP are of great significance for treatment planning.

**Cognitive Functioning.** In addition to motor and communication deficits, CP is often accompanied with cognitive impairments. Around 20 to 40% of individuals with CP, varying across different CP subtypes, have some form of cognitive or intellectual impairments with the highest rates among those with epilepsy (Frampton, Yude, & Goodman, 1998; Nakada, 1993; Odding et al., 2006). Similarly, ASD also has a high comorbid rate with intellectual disabilities ranging from 40 to 50% (Kielinen, Rantala, Timonen, Linna, & Moilanen, 2004; La Malfa et al., 2007) and the presence of epilepsy is also correlated with higher rates of intellectual disabilities in ASD (Amiet et al., 2008). While intellectual

disabilities are usually not diagnosed in early childhood, adaptive functioning of infants and toddlers can be indicative of their intellectual functioning and future diagnoses of intellectual disabilities, thus have been the center of focus of many studies assessing early cognitive functioning in infants and toddlers.

## Purpose

Both ASD and CP are usually considered life-long conditions, and much effort has been directed to the study of early diagnoses and interventions for each condition. Recent studies on ASD have indicated that diagnoses can be reliably made as early as at the age of two years, although children are usually not diagnosed until later at around the age of three to four (Matson, Wilkins, & Gonzalez, 2008; Valicenti-McDermott et al., 2012; Zwaigenbaum et al., 2015). Overlapping with this time frame, CP is mostly diagnosed within the first two to three years of life (Ashwal et al., 2004; Smile & Kawamura, 2016). Other overlaps between ASD and CP are also present as discussed above, and these shared elements of symptomatology and developmental functioning deficits, including communication, repetitive movement, and cognitive functioning, may contribute to the difficulties of identifying ASD in the presence of CP due to its masking effect. This is supported by study findings where the presence of comorbid CP was correlated with delayed ASD diagnoses (Smile et al., 2013). Considering these and the raised prevalence of ASD in individuals with CP (Christensen et al., 2014; Kilincaslan & Mukaddes, 2009; Kirby et al., 2011), it is important that researchers and practitioners are able to accurately assess comorbid ASD and CP in young children to allow access to appropriate early interventions. However, very limited research has been conducted on this topic.

Among the existing measures of autism symptomatology, the *BISCUIT* is the only one that has been tested in children with CP (Smile & Kawamura, 2016). Two previous studies have utilized this measurement to assess ASD symptomatology in young children with CP, and found compared to those with CP only, children with comorbid ASD and CP had greater

impairment in communication and RRB (Hattier, Matson, & Kozlowski, 2012; Hattier, Matson, May, & Whiting, 2012). Another study conducted by Smile and colleagues (2013) also assessed the differences in children with CP only and comorbid ASD and CP. They collected and compared these two groups of children in cognitive profiles measured by standardized intelligence tests, age of diagnosis, and behavioral and medical comorbidities based on information collected from developmental and medical history. Their findings indicated greater cognitive impairment, later diagnoses, increased prevalence of asthma and constipation, higher frequency of aggression in children with co-occurring ASD and CP than those with CP only. The current study aims to assess and compare both ASD symptomatology and developmental functioning in infants and toddlers with ASD, CP, and comorbid ASD and CP, in hope to contribute to existing literature and provide directions for future research on this topic revolving around the differentiation of ASD and CP, and early diagnosis of ASD in the presence of CP in young children.

## Method

### Participants

Participants were selected from a pool of infants and toddlers age between 17 and 36 months old ( $Mean=26.44$ ,  $SD=5.05$ ). The subjects were divided into three age and gender matched groups, namely, 1) children with CP and without co-occurring ASD group (CP,  $N=23$ ), 2) those with ASD and no comorbid CP group (ASD,  $N=23$ ), and 3) a group with comorbid ASD and CP (ASD+CP,  $N=23$ ). A breakdown of the demographics by group can be seen in table 1. All participants were recruited through EarlySteps, Louisiana's statewide early intervention program under the Individuals with Disabilities Education Act, Part C. To be eligible for these services, the child must exhibit a developmental delay or a medical condition that is likely to result in a developmental delay, including epilepsy, cerebral palsy, traumatic brain injury, tubular sclerosis, deafness, blindness, and premature birth. ASD diagnoses were assigned by a licensed clinical psychologist with over 30 years of experience in the field of intellectual and developmental disabilities in accordance with a *Diagnostic and Statistical Manual, Fifth Edition (DSM-5)* ASD algorithm. Information about the presence of a CP diagnosis was obtained through caregiver report on the demographic subsection of the *BISCUIT*.

Table 1. Demographic Characteristics of Participants by Group ( $n=69$ )

	<b>CP</b> ( $N = 23$ )	<b>ASD</b> ( $N = 23$ )	<b>ASD+CP</b> ( $N = 23$ )	<b>Total</b> ( $n=69$ )
Age (in months), $M(SD)$	26.22(4.39)	26.13(4.50)	26.13(4.50)	26.16(4.39)
Gender, no. (%)				
Male	18(78.3)	18(78.3)	18(78.3)	54(78.3)
Female	5(21.7)	5(21.7)	5(21.7)	15(21.7)
Race/Ethnicity, no. (%)				
Caucasian	15(65.2)	6(26.1)	7(30.4)	28(40.6)
African American	7(30.4)	15(65.2)	12(52.2)	34(49.3)
Other/Unspecified	1(4.3)	2(8.7)	4(17.3)	7(10.1)

## Measures

### ***Baby and Infant Screen for Children with aUtism Traits- Part 1 (BISCUIT- Part 1).***

The *BISCUIT* (Matson, Boisjoli, & Wilkins, 2007) is the only autism diagnostic measure that has been tested on children with neuromotor disorders (Smile & Kawamura, 2016). It is an informant-report measure made up of three sections each assessing ASD symptomology, comorbid psychopathology, and challenging behaviors among children between the age of 17 and 37 months (Matson, Wilkins, Sevin, et al., 2009). The *BISCUIT* also contains a demographic form which collects data on a range of factors including medical and developmental history (e.g., the presence or absence of CP diagnoses). The *BISCUIT-Part 1* is the diagnostic section of the measure which is made up of 62 items related to three domains (i.e., Socialization/Nonverbal Communication or S/N, Repetitive Behavior/Restricted Interests/RBRI, Communication/COMM; Matson, Boisjoli, Hess, & Wilkins, 2010). Informants rate each item on a 3-point Likert scale (0=“not different; no impairment;” 1=“somewhat different; mild impairment;” 2=“very different; severe impairment”) comparing their child to same-aged peers.

The *BISCUIT-Part 1* was found to have sound psychometrics with an internal reliability of .97 (Matson, Wilkins, Sevin, et al., 2009), an overall correct classification rate of .89, sensitivity of 93.4, and specificity of 86.6 (Matson, Wilkins, Sharp, et al., 2009). It has also demonstrated convergent validity with the *Modified Checklist for Autism in Toddlers (M-CHAT)* and the Personal-Social domain of the *Battelle Developmental Inventory, Second Edition (BDI-2)*, and divergent validity with the *BDI-2*'s Adaptive and Motor domains (Matson, Wilkins, & Fodstad, 2011). The scores of *BISCUIT-Part 1* was used in this study as an assessment of ASD symptom severity with a total score of 17 or above indicating the child is in "at-risk" range for ASD. Using Cronbach's alpha, an internal consistency of .97 was reported for the *BISCUIT-Part 1* in the current study.

***Battelle Developmental Inventory, Second Edition (BDI-2).*** The *BDI-2* (Newborg, 2005) is designed to assess the developmental skills of children from birth to 7 years 11 months old through both informant-report and structured observation. The *BDI-2* is composed of 450 items evaluating five developmental domains: Adaptive (ADP), Personal-Social (P-S), Communication (COM), Motor (MOT), and Cognitive (COG; Bliss, 2007). Each item is scored on a 3-point scale (0="no ability in the skill," 1="emerging ability," 2="ability at the skill") and the sums of item scores result in scores for each domain and a total developmental quotient (DQ) with a mean of 100 and a standard deviation of 15.

The *BDI-2* has been found to be psychometrically sound with test-retest reliability estimates of above .90 for each domain score and total DQ (Newborg, 2005). Internal consistency for both the domain scores and the overall DQ was calculated between .98 and .99 (Newborg, 2005). Its validity was established through comparisons with several



other measures of child development including the *Bayley Scales of Infant Development, Second Edition (BSID-II)* and the *Preschool Language Scales (PLS-4)*; Bliss, 2007). For the current study, total DQ and all five domain scores were used to assess the participants' developmental functioning.

## **Procedure**

The study was approved by the Louisiana State University Institutional Review Board and the State of Louisiana's Department of Health and Hospitals Institutional Review Board. The use of data from EarlySteps was approved by the Office for Citizens with Developmental Disabilities (OCDD) of the State of Louisiana. Before receipt of the database, personal identifiers of each participant, including name and date of birth, were removed by the Department of Health and Hospitals.

Before the initiation of study protocols, informed consent was obtained from guardians of the participants. The *BISCUIT-Part 1* and the *BDI-2* were administered as part of a larger assessment battery that included both parent interviews and child observations. The assessments were conducted in the child's home or daycare by roughly 175 EarlySteps service providers. These providers all held an appropriate degree (e.g., from bachelor's degrees to doctoral degrees) and certifications or licensures in various fields (e.g., occupational therapy, physical therapy, psychology, special education, social work, speech-language pathology) and were proficient in evaluating and treating young children and trained in the administration of the measures used in this study.

## **Statistical Analyses**

Statistical analyses were performed using SPSS Statistics (Version 21). Analyses of variance (ANOVAs) were conducted to explore the relationships between CP and ASD comorbidity and ASD symptom severity and developmental functioning. Group (i.e., CP, ASD, ASD+CP) was selected as the independent variable (IV), and ASD symptom severity, as measured by the total *BISCUIT-Part 1* score, as well as developmental functioning, as measured by the total *BDI-2* score were selected as the dependent variables (DVs). Tukey post hoc analyses were conducted to further examine group differences following each ANOVA.

Further, to explore group differences across the five developmental domains of the *BDI-2* and three *BISCUIT-Part 1* domain scores, two multivariate analyses of variance (MANOVAs) were conducted with group as the IV, scores from the five *BDI-2* developmental domains (i.e., Adaptive, Personal-Social, Communication, Motor, and Cognitive) as the DVs for one MANOVA and *BISCUIT-Part 1* domain scores (i.e., Socialization/Nonverbal Communication, Repetitive Behavior/Restricted Interests, Communication) as the DVs for the other MANOVA. Subsequent ANOVAs were conducted to examine developmental differences more closely with use of Bonferonni corrections (i.e.,  $p$  of less than .01 for *BDI-2* domains and  $p$  of less than .017 for *BISCUIT-Part 1* domains). Tukey post-hoc tests followed the ANOVAs and were used to examine developmental domain differences across groups.

## Results

### Total Scores

One outlier in the CP group was detected by boxplot for the *BISCUIT-Part 1* total score and was kept in the analysis to conserve data. As assessed by Shapiro-Wilk test ( $p > .05$ ), data were normally distributed for both *BISCUIT-Part 1* and *BDI-2* total scores in each group with the exception of the *BISCUIT-Part 1* total score in the CP group and the *BDI-2* total score in the CP+ASD group ( $p < .01$ ). As ANOVA is fairly robust to the violation of the assumption of normality and even more so when sample sizes are equal across groups (Lix, Keselman, & Keselman, 1996), data were left as they were. Homogeneity of variances was achieved for both the *BISCUIT* ( $p = .112$ ) and the *BDI-2* ( $p = .656$ ) as assessed by Levene's test.

Significant group differences were found for both *BISCUIT-Part 1*,  $F(2, 66) = 29.249$ ,  $p < .01$ , and *BDI-2* total scores,  $F(2, 66) = 4.511$ ,  $p < .05$ . Tukey post doc analyses revealed that CP group differed significant from ASD and ASD+CP groups ( $p < .001$ ) in *BISCUIT-Part 1* total score, and ASD+CP group differed significantly from CP and ASD groups ( $p < .05$ ) in *BDI-2* total score. Results are shown in table 2.

Table 2. Mean and standard deviations for *BISCUIT-Part 1* and *BDI-2* ( $n=69$ ).

	CP ( $N=23$ )	ASD ( $N=23$ )	ASD+CP ( $N=23$ )	Total ( $n=69$ )
	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>	<i>M(SD)</i>
<i>BISCUIT-Part 1</i> Total	19.22 (22.29) <sup>c*</sup>	59.43 (21.91) <sup>c*</sup>	64.00 (21.35) <sup>ab*</sup>	47.55 (29.57) <sup>**</sup>
<i>BDI-2</i> Total	65.13 (37.12) <sup>bc**</sup>	63.96 (26.29) <sup>a**</sup>	38.43 (33.93) <sup>a**</sup>	56.17 (34.41) <sup>*</sup>

\* $p < .05$

\*\* $p < .01$

a. Significantly different CP Group

- 
- b. Significantly different from ASD group
  - c. Significantly different from ASD+CP group

*Note.* Higher *BISCUIT-Part 1* scores indicate more deficits while higher *BDI-2* scores indicate better functioning.

### Domain Scores

Using boxplots and Mahalanobis distance, assumption checking revealed several univariate outliers for both *BISCUIT-Part 1* and *BDI-2* domains, one multivariate outlier in *BISCUIT-Part 1* domain scores ( $p < .001$ ), and no outliers in the *BDI-2* domain scores ( $p > .001$ ). Assessed by Shapiro-Wilk test, normality was established in the S/N domain for ASD and ASD+CP groups, in the RBRI domain for the ASD group only, in the COMM domain for the CP group only, ADP domain for the CP and ASD groups, in the P-S domain for all 3 groups, and in the COG domain for only the ASD group ( $p > .05$ ). While *BDI-2* was found to have more outliers, the *BISCUIT-Part 1* only had few, thus adjustment was not made to the data set in an attempt to eliminate these violations due to the need of keeping all participants considering the small sample size and difficulties in interpreting transformed data. No multicollinearity was present for neither measures ( $r < .9$ ), and linear relationships were identified using scatterplots. There was also homogeneity of variance-covariance as assessed by Box's M test ( $p > .05$ ).

Using Wilks' Lambda, significant group effects were found for both *BISCUIT-Part 1* domain scores,  $\Lambda = .419$ ,  $F(6, 128) = 11.623$ ,  $p < .00$ , and *BDI-2* domain scores,  $\Lambda = .519$ ,  $F(10, 124) = 4.812$ ,  $p < .01$ . Follow-up ANOVAs were conducted after each significant result with Bonferroni corrections applied (i.e.,  $p < .017$  for *BISCUIT-Part 1* domains, and  $p < .01$  for *BDI-2* domains). In *BISCUIT-Part 1*, significant group differences were found in the S/N domain,  $F(2) = 30.28$ ,  $p < .01$ , partial  $\eta^2 = .479$ ; the RBRI domain,  $F(2) = 19.98$ ,  $p < .01$ , partial  $\eta^2$

=.377; and the COMM domain,  $F(2)=11.29, p<.01$ , partial  $\eta^2 =.255$ . In *BDI-2*, significant results were reported in group differences in the P-S domain,  $F(2)=5.39, p<.01$ , partial  $\eta^2 =.140$ ; and the MOT domain,  $F(2)=10.95, p<.01$ , partial  $\eta^2 =.249$ ; but not the COM domain,  $F(2)=3.32, p>.05$ , partial  $\eta^2 =.091$ ; the COG domain,  $F(2)=3.58, p>.01$ , partial  $\eta^2 =.098$ ; nor the ADP domain, where  $F(2)=2.51, p>.05$ , partial  $\eta^2 =.071$ . The larger effect sizes of the *BISCUIT-Part 1* is expected as it is a measure of autism symptoms while the *BDI-2* is not. In addition, participants' *BDI-2* scores would cluster towards the lower end as they all had or were at risk for developmental delays.

Results from Tukey post hoc tests revealed statistically significant group differences in the S/N, RBRI ( $p<.01$ ), and COMM ( $p<.05$ ) domains between CP and the other two groups (i.e., ASD, and ASD+CP groups); in the P-S ( $p<.01$ ) domain between CP and ASD+CP groups; and in the MOT domain between ASD group and CP groups (i.e., CP and ASD+CP,  $p<.05$ ). Results are shown in table 3 and graph 1.

Table 3. Mean and standard deviations for *BISCUIT-Part 1* and *BDI-2* domain scores ( $n=69$ ).

	CP ( $N=23$ ) <i>M(SD)</i>	ASD ( $N=23$ ) <i>M(SD)</i>	ASD+CP ( $N=23$ ) <i>M(SD)</i>	Effect Size ( <i>Partial <math>\eta^2</math></i> )
<i>BISCUIT</i> , Socialization	6.96 (10.96) <sup>bc**</sup>	25.43 (11.22) <sup>a**</sup>	31.56 (11.30) <sup>a**</sup>	.479
<i>BISCUIT</i> , RBRI	3.13 (8.20) <sup>bc**</sup>	18.61 (8.47) <sup>a**</sup>	14.78 (9.24) <sup>a**</sup>	.337
<i>BISCUIT</i> , Communication	7.48 (4.43) <sup>b*c**</sup>	10.26 (3.52) <sup>a*</sup>	12.43 (2.39) <sup>a**</sup>	.255
<i>BDI-2</i> , Adaptive	72.87 (18.17)	74.74 (13.86)	65.13 (13.87)	.071
<i>BDI-2</i> , Personal-Social	86.17 (17.50) <sup>c**</sup>	78.00 (10.21)	72.3 (14.57) <sup>a**</sup>	.140
<i>BDI-2</i> ,	72.61	65.43	62.61	.091

Communication	(15.55) <sup>c*</sup>	(11.26)	(13.57) <sup>a*</sup>	
<i>BDI-2</i> ,	76.17	89.13	66.39	.249
Motor	(17.11) <sup>b*</sup>	(14.97) <sup>a*c**</sup>	(17.42) <sup>b**</sup>	
<i>BDI-2</i> ,	75.35	73.04	65.43	.098
Cognitive	(15.51) <sup>c*</sup>	(10.79)	(12.70) <sup>a*</sup>	

\* $p < .05$

\*\* $p < .01$

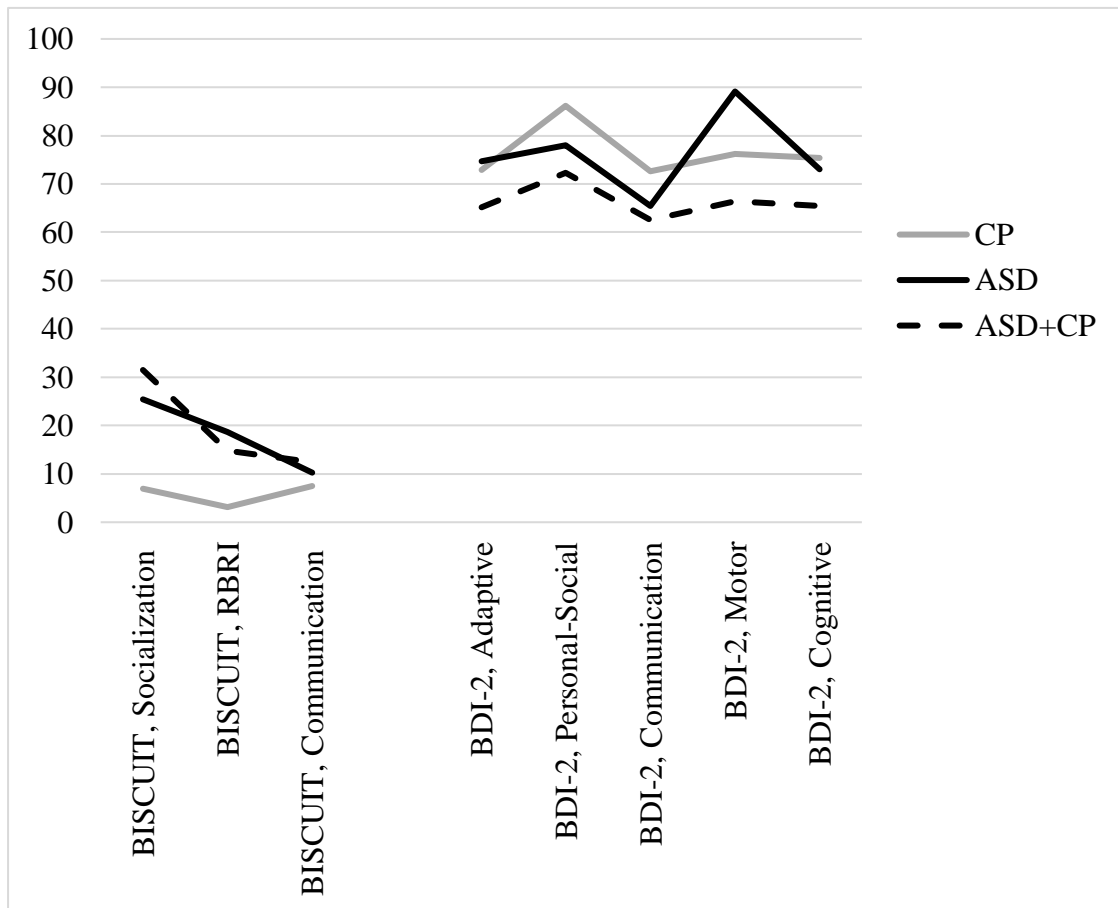
\*\*\*  $p < .017$

a. Significantly different CP Group

b. Significantly different from ASD group

c. Significantly different from ASD+CP group

Note. RBRI= Repetitive Behavior/Restricted Interests. Higher *BISCUIT-Part 1* scores indicate more deficits while higher *BDI-2* scores indicate better functioning.



Graph 1. Mean *BISCUIT-Part 1* and *BDI-2* domain scores for each group ( $n=69$ ).

Note. Higher *BISCUIT-Part 1* scores indicate more deficits while higher *BDI-2* scores indicate better functioning.

## Discussion

Overlaps in the symptomatology of ASD and CP (i.e., impairments in language development, motor abnormalities, and cognitive deficits) may complicate the diagnostic process of autism at the presence of co-occurring CP, which may in turn lead to delays in one's access to early intervention. Considering the detrimental effects of early interventions and increased risk of ASD in those with CP (Christensen et al., 2014; Hampton & Kaiser, 2016; Kilincaslan & Mukaddes, 2009; Kirby et al., 2011; Reichow, 2012; Zwaigenbaum et al., 2015), the amount of research on ASD and CP comorbidity, especially in younger children, is highly limited. To expand the existing literature on ASD and CP comorbidity, and to facilitate early detection of ASD in individuals with CP, this study is designed to assess and compare the similarities and/or differences in ASD symptomatology and developmental functioning in young children with ASD, CP, and comorbid ASD and CP.

As expected, infants and toddlers with CP were rated significantly lower in overall ASD symptom severity than those with ASD (i.e., ASD and ASD+CP groups). When symptoms were examined separately (i.e., grouped into Socialization/Nonverbal Communication, Repetitive Behavior/Restricted Interests, and Communication domains), the same pattern stayed true for all domains. This is consistent with the findings of previous studies which found that compared to those with CP only, the comorbid group (i.e., ASD+CP) had greater impairment in communication and RRB as measured by *BISCUIT-Part 1* (Hattier, Matson, & Kozlowski, 2012; Hattier, Matson, May, et al., 2012). While the same but earlier versions of the database were used in these studies, ASD only group was not included and neither was developmental functioning. With almost twice the sample size and the inclusion of ASD

group and *BDI-2*, the current study was able to provide significantly more information on whether and what types of group differences exist.

Assessed using the *BDI-2*, lower levels of overall developmental functioning were found in participants with both ASD and CP compared to those with ASD only or CP only. With further examinations of the developmental domains (i.e., adaptive skills, personal-social development, communication, motor functioning, and cognition), different patterns were found.

In three out of the five domains, including adaptive skills, communication, and cognitive functioning, participants of different diagnostic groups did not exhibit any significant differences after the application of Bonferroni corrections. This is different from a previous study where greater cognitive impairments were found in children with co-occurring ASD and CP than those with CP only (Smile et al., 2013). A potential explanation may be that the participants of the other study had an older and larger range of age (i.e., 3-18 years), and that cognitive functioning can be harder to assess in infants and toddlers. Considering the limited amount of literature on this topic, less stringent criteria (e.g.,  $\alpha$  of .05) may be used when interpreting the results. Future studies should include a broader age range covering both young and older children so that differences between age groups can be further explored. It is also worth noticing that while the *BDI-2* communication domain did not exhibit any group differences, the communication domain of the *BISCUIT-Part 1* revealed greater impairments in the ASD+CP group when compared to the CP group. Explanations for this difference may be derived from the differences between these two measures. Specifically, while *BISCUIT-Part 1* assesses communication from a functional perspective (e.g., how was



the child's language and gestures used for communicative purposes), *BDI-2* focuses more on the production and decoding of language. Thus, the findings of the current study may suggest that the addition of ASD to CP is related with increased functional deficits in communication instead of the language production and decoding process.

The other two *BDI-2* domains, on the other hand, revealed significant group differences. Specifically, infants and toddlers with co-occurring ASD and CP experienced more impaired personal social functioning than those with CP only, and more deficits in motor functioning were exhibited in CP groups (i.e., CP, ASD+CP) than those with ASD only. These distinctions are expected considering the core characteristics of ASD (i.e., social deficits) and CP (i.e., impaired motor functioning). Interestingly, however, no differences in personal social functioning were found between ASD and CP groups, which is again, different from the results of *BISCUIT-Part 1*. This may suggest that measures targeting social deficits characteristics of autism like the *BISCUIT* are better suited for use when attempting to detect ASD in infants and toddlers with toddlers with CP than more generalized measures like the *BDI-2*. In addition, in the current study, participants with ASD and CP did not differ from those with CP only in their motor functioning, considering the above-mentioned difference in RRB between infants and toddlers with ASD (i.e., ASD and ASD+CP groups) and those with CP only, as assessed by *BISCUIT-Part 1*, results of the current study suggest that the presence of RRB compared to general motor skill functioning, may be a better distinguishing factor to consider when evaluating potential ASD in those with CP.

To the author's knowledge, no previous studies have compared both ASD symptomatology and developmental functioning in relation to ASD and CP comorbidity using samples containing children under the age of 3. As mentioned above, such studies in this population are of great importance as the early diagnosis of ASD in the presence of CP can be complicated by overlaps in symptom presentations of these two conditions, which may lead to delays in one's access to early intervention, affecting the functional outcome of the individuals. If consistent patterns of group differences in ASD symptoms and/or developmental functioning can be found in young children with CP, these results can be utilized to facilitate earlier and more accurate detection of ASD in this population. Based on findings of the current study, it is concluded that with appropriate measures like the *BISCUIT-Part 1*, the differences in communication, socialization deficits, and the presence of RRB may be readily assessed between infants and toddlers with CP and those also with co-occurring ASD. Additionally, developmental functioning may also be utilized to support early detection of comorbid autism in the presence of CP, as those with both conditions were found to function developmentally at a lower level compared to those with CP and no ASD. However, not all areas of developmental functioning exhibit differences between these two groups of young children. As assessed by the *BDI-2*, only social functioning was found to differ significantly between these two groups while no presence of meaningful differences was revealed in the other areas including adaptive skills, communication, motor skills, and cognitive functioning.

While this study can contribute significantly to the current literature and towards early diagnosis of ASD in young children with CP, the following limitations need to be considered

when interpreting the research findings. The power of statistical analyses conducted in this study may be affected by the violations of some of the assumptions and the small sample size. While the assumption violations may be addressed by removing outliers and/or transforming the data, these procedures were not taken due to the limited sample size and difficulties in interpreting transformed data. Future studies with larger sample sizes may provide a better basis for statistical analyses. As pointed out earlier in the text, all participants were receiving services from EarlySteps, thus findings of the current study may not be representative for other populations (e.g., those who are not receiving services or receiving different types of services). Thus, future studies may assess other populations to determine whether service enrollment leads to differential results. Another limitation is that the presence of CP diagnoses was determined based on caregiver report. It is difficult to ensure the reliability of parent reported diagnosis, thus more reliable sources like medical reports should be included or used in future studies.

Although overlaps exist in symptom presentations between ASD and CP, results of the current study indicated that ASD measures can still readily detect differences between infants and toddlers with CP only, ASD only, and comorbid ASD and CP. While lower overall developmental functioning was also detected in those with both ASD and CP compared to those with CP only, this difference seems to be mainly due to greater deficits in personal-social functioning in infants and toddlers with additional ASD diagnoses to CP. Although limited in amount, previous literature combined with the current study provided support for early detection of ASD in young children with CP using ASD measures and other general assessment tools. More studies measuring these ASD symptoms and other related

constructs in young children should be carried out to further explore the similarities and differences between ASD and CP in this population. The selection of different measures and use of multiple assessment tools are also encouraged as different emphases or perspectives may be taken by different measures when assessing the same or similar constructs, thus, the comparison between measures is needed to determine which ones are more appropriate for the purpose of diagnosing ASD in those with CP.

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## Vita

Xinrui Jiang was born in March 1992 in Yangzhou, Jiangsu, China. She was raised in Yangzhou and moved to Shanghai in 2010 for college. In 2012, she was enrolled in the dual degree program hosted by East China Normal University and Colorado State University and moved to the U.S. as an international student. She then graduated with a Bachelor of Science degree in Psychology from both universities in 2014. Jiang is currently a second-year doctoral student in the Clinical Psychology program at Louisiana State University in Baton Rouge, Louisiana, working with Dr. Johnny L. Matson. Her clinical and research interests revolve around the developmental trajectory, assessment, and treatment of intellectual and developmental disabilities.