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# An Investigation of How Previous Diagnoses Affect the Developmental Functioning of Children at Risk for Autism Spectrum Disorder

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**AN INVESTIGATION OF HOW PREVIOUS DIAGNOSES  
AFFECT THE DEVELOPMENTAL FUNCTIONING OF  
CHILDREN AT RISK FOR AUTISM SPECTRUM DISORDER**

A Thesis

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

The Department of Psychology

by  
Michaela Brown  
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## **Abstract**

Research has shown that children with autism spectrum disorder (ASD) often have co-occurring medical and/or psychiatric disorders. This current study aimed to investigate how previous diagnoses, which may become comorbidities of ASD if diagnosed, affect the developmental functioning of children presenting as at risk for ASD compared to those presenting with no risk. Developmental areas such as communication, motor, adaptive, cognitive and social, as well as overall developmental functioning, were considered in the analysis. 11,970 children under the age of three were studied. Results found that the presence of previous diagnoses significantly predicted developmental functioning, particularly in the motor functioning of those not at risk for ASD. The results also demonstrated that the presence of two or more of these conditions significantly impact overall developmental functioning. These results have implications for early assessment and treatment for ASD and its common comorbidities. Limitations and conclusions were then discussed.

## **Introduction to Autism Spectrum Disorder**

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication and restrictive, repetitive behaviors and interests (American Psychiatric Association, 2013). The most contemporary research indicates that ASD occurs in one in 54 children and is 4.3 times more prevalent in boys than in girls (Maenner et al., 2020). In order to receive a diagnosis of ASD, the child must present with three or more deficits in social communication, such as failure to engage in back-and-forth communication, lack of eye contact, improper use of gestures, and/or difficulties creating or maintaining relationships (APA, 2013). The child must also present with at least two symptoms of restricted, repetitive patterns of behavior, such as repetitive motor movements and/or highly restrictive interests with unusual objects (APA, 2013).

Several psychiatric and medical conditions have been found to commonly co-occur within the ASD population (Matson & Nebel-Schwalm, 2007; Mannion & Leader, 2013; Landa et al., 2013). Researchers have evaluated how ASD impacts developmental functioning in young children (Landa et al., 2013). However, they have not assessed how comorbidities in children with ASD, or previous diagnoses in those at risk for ASD, in children at the age of receiving early intervention (EI) services and presenting as at-risk for ASD can impact the child's developmental functioning. The aim of the present study is to further investigate how the presence and quantity of previous diagnoses impact developmental functioning in children already presenting with ASD symptoms and concerns, compared to children presenting with no risk of the disorder. Groups based on the presence of previous diagnoses were compared to see if and how specific areas of development are affected compared to children presenting with no previous diagnoses. Children at risk for ASD were compared to those presenting as no risk.

This current study will expand the extent literature, which has focused primarily on the presence of symptoms of ASD or comorbidities of children with ASD, by investigating the relationship between the two in a specific age group.

## **History of ASD**

The term “autism” was first used in 1911 by German psychiatrist Eugen Bleuler to describe a symptom seen in severe cases of schizophrenia (Evans, 2013). Bleuler believed that autism defined the subject’s symbolic ‘inner life’ and was characterized by “infantile wishes to avoid unsatisfying realities and replace them with fantasies and hallucinations” (p. 4). Piaget continued this symbolic meaning of autism, for he claimed that the pre-verbal stages of children’s thought could be considered ‘autistic’ or ‘symbolic’, and characterized these thoughts as presenting with an absence of logic, visual imagery over conceptual thought, and a lack of awareness that visual perceptions can be connected. However, these Bleuler and Piaget continued to view autism as a symptom of psychosis, not as its own separate disorder.

In 1938, Hans Asperger used the term ‘autistic psychopathology’ to describe the children in his clinical work. Asperger used the term ‘autistic’ to describe the “inward self-absorbed aspect that was reminiscent of schizophrenic withdrawal” (Al Ghazi, 2018, p. 11), and also noted that these children did not demonstrate hallucinations, a typical symptom of schizophrenia.

Symptoms of autism were first discussed in Leo Kanner’s 1943 article “Autistic Disturbances of Affective Contact”. In it, Kanner discusses key symptoms such as an “inability to relate themselves in the ordinary way to people and situations”, “failing to develop the usual amount of social awareness” and a delay in speech that, once it is obtained, tends to present with “delayed echolalia (p. 244- 245). He concludes that these children, despite having “highly

intelligent” and “really warmhearted parents”, “come into the world with innate inability to form the usual, biologically provided affective contact with people” (p. 250).

Bruno Bettelheim had an opposing view of parents of children with autism, believing that the cause of the disorder was due to cold, distant parenting, a parenting style he coined as “refrigerator mom” (Phelps & Fogler, 2018). Bettelheim thought that if a child was presented with this parenting style he or she “would internalize a sense of not being loved by the parent and would thus negate himself or herself by turning inward, not communicating, interaction, and engaging in repetitive self-stimulatory behavior” (p. 6). The concept of refrigerator mom was eventually adopted by Kanner, who continued to study autism with the understanding that parenting style may be an explanation for the etiology of the disorder.

Kanner published a second study in 1971, in which he followed up with the 11 cases he discussed in his previous publication, the infants now adults. He also discussed the transformation of the diagnosis. Initially, in 1943, the syndrome was considered childhood schizophrenia. In 1967, however, Russian investigators decided that autism was to be considered “schizoid psychopathology” (Kanner, 1971, p. 141). The DSM-II in 1968 did not agree, for they used a code system that did not include autism but instead still used the childhood schizophrenia as the only accepted diagnosis. Kanner, however, does not agree with this delineation, believing that the diagnosis led children to be institutionalized who could have fared better in a different setting. Kanner concludes his article with a suggestion that these patterns of behavior and symptoms are due to a “specific syndrome”, with him calling his subjects “autistic children” (p. 145).

Michael Rutter agreed with Kanner’s delineation in his 1968 paper “Concepts of Autism: A Review of Research”, suggesting that infantile autism does not have anything to do with

schizophrenia. Rutter notes that autism is more common in males, whereas schizophrenia presents more equally in both sexes. One's family history is also different in those with schizophrenia compared to those with infantile autism; studies have shown that adult schizophrenia is rare in the parents and siblings of those with autism (Rutter, 1967). Rutter also suggested that autism was not primarily a social relationships disorder, but a language disorder. Rutter supports this hypothesis by pointing out language deficits such as lack of response and/or comprehension to sounds, a tendency of echolalia, and the use of reverse pronouns, such as a confusion of "you" and "I" (Rutter, 1968). These conclusions, along with those of Kanner's, lead to the distinction of the autism disorder, with clear deficits in both social and language functioning.

### **Diagnostic Criteria for ASD**

The *Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III;* American Psychiatric Association, 1980) was the first diagnostic manual to include autism, which at the time was called infantile autism (IA). The DSM-III had created a new group of disorders called the Pervasive Developmental Disorders, which included childhood onset pervasive developmental disorder (COPDD) and atypical pervasive developmental disorder (APDD). IA was defined as an early onset, or before 30 months, disorder characterized by "(1) pervasive lack of responsiveness to other people; (2) gross deficits in language development; (3) peculiar speech patterns, if speech is present at all; (4) bizarre responses to the environment; and (5) an absence of delusions, hallucinations, loosening of associations and incoherence as in schizophrenia" (Volkmar, Cohen & Paul, 1986, p. 190). COPDD differed from IA by age of onset, which was between 30 months and 12 years old, and diagnostic criteria, for COPDD



included characterizations such as resistance to change, inappropriate affect, sudden excessive anxiety, under- or over-sensitivity to sensory stimuli and self-mutilation (Volkmar et al., 1986).

However, the age of onset that differentiated these two diagnoses was not supported by research (Volkmar et al., 1986), so when the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition-Revised (DSM-III-R*; American Psychiatric Association, 1987) came out in 1987, the criteria for autistic disorder was broadened. Sixteen criteria were detailed and grouped to encompass individuals at any age of developmental level (Volkmar, Cicchetti, Bregman & Cohen, 1992). In order to receive a diagnosis of Autistic Disorder in the DSM-III-R, the individual had to present with 8 of the 16 items, with at least two symptoms coming from each of the three groups. Group A included impairments in reciprocal social interaction, Group B included impairments in verbal and nonverbal communication, and Group C included impairments in restricted activities and interests (APA, 1987). A subthreshold category was created, Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS), was created to capture any children who did not meet the diagnostic criteria for autism.

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV*; American Psychiatric Association, 1994) introduced the concept of autism as a spectrum and included three new conditions along this spectrum with autism and PDD-NOS; Asperger's, childhood disintegrative disorder (CDD), and Rett syndrome. Asperger's was considered separate from autistic disorder, for it often presented with a later onset and an interest about the environment surrounding the individual (Woodbury-Smith et al., 2005). It had similar diagnostic criteria as autistic disorder but specified that the child has no history of language, self-help, or adaptive delays (Woodbury-Smith et al., 2005). CDD was defined as a significant loss of skills in at least two developmental areas, such as language, social or adaptive, after the age of two

(Fombonne, 2002). Rett syndrome was a disorder more commonly seen in girls and was characterized by a loss of verbal language and stereotyped hand movements (Neul et al., 2010). Now children needed to only present 6 or more symptoms, with at least two from Group 1 and one each from Groups 2 and 3 (APA, 1994). There was also a hierarchy of diagnosis, with autism taking precedence; the individual would be evaluated for the presence of specific developmental abnormalities and impairments and would receive a diagnosis according to the amount of abnormalities found and when they were first noticed (Woodbury-Smith et al., 2005). When the DSM-IV was revised into the *Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition-Text Edition* (DSM-IV-TR; American Psychiatric Association, 2000), the diagnostic criteria did not change.

However, the diagnostic criteria and the overall definition of the disorder did change in the *Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition* (DSM-V; American Psychiatric Association, 2013). Autistic disorder became autism spectrum disorder (ASD), an umbrella term to capture the range of disorders with varying presentations and severity levels of behavior (Lobar, 2016). All other pervasive developmental disorders, such as Asperger's, PDD-NOS, Rett's and CDD, were included in this spectrum and were no longer standalone diagnoses. The DSM-V also added the social (pragmatic) communication disorder, which can only be diagnosed if ASD is ruled out (Volkmar & Reichow, 2013).

As for the diagnostic criteria, a social communication and social interaction category was created, removing the language/communication category. For the social communication and social interaction category, an individual must display all three of the symptoms; deficits in social-emotional reciprocity, deficits in nonverbal communicative behaviors, and deficits in developing, maintaining and understanding relationships (APA, 2013). For the restricted,

repetitive behavior, interests and activities category, the individual needs to present with at least two of the symptoms. The age of onset in the DSM-V criteria was also updated. In the DSM-IV-R, the abnormal functioning must occur before the age of 3. In the DSM-V, it notes that symptoms must be present in the early developmental period.

### **Prevalence of ASD**

As of 2016, ASD occurs in 1 in 54 of children, and is 4.3 times prevalent in males than females (Maenner et al., 2020). Prevalence in White and non-Hispanic Black children were found to be similar (18.5 and 18.3 per 1,000 children, respectively), with lower ASD rates in Hispanic children (15.4 in 1,000).

## **Common Comorbidities**

Comorbidity is defined as the occurrence of two or more disorders in the same person, whether they be medical or psychopathological in nature (Matson & Nebel-Schwalm, 2007). Studies have shown that children with ASD also have a high prevalence a co-occurring medical and psychiatric disorders, such as gastrointestinal disorders (Samsam, Ahangari & Naser, 2014), epilepsy (Canitano, 2007), intellectual disabilities (Matson & Nebel-Schwalm, 2007), and ADHD (Stevens, Peng & Barnard-Brak, 2016).

### **Epilepsy/Seizures**

Epilepsy and/or seizures have been a commonly reported comorbidity of ASD. Prevalence rates for epilepsy are estimated at 1-2% of children in the general population, however the prevalence rate increases in the ASD population, with estimates ranging from 5% to 38% (Frye, 2016). Saemundsen, Ludvigsson, Hilmarsdottir and Rafnsson (2007) investigated the prevalence rate of ASD in children who experienced seizures within the first year of life and found 7.1% of the sample to have ASD. Clarke et al. (2005) also used an epilepsy-only sample in their study examining ASD symptomology in children and adolescents with the disorder and found that 32% of the children were screened and found to have elevated rates of ASD symptoms. The authors also found a correlation between risk of ASD and a younger mean age of seizure onset of 2 years of age. They surmised that this occurrence correlates with the timing of autistic regression, which often occurs before the age of three (Tuchman & Rapin, 1997).

### **Gastroenterology Disorders**

Gastroenterology (GI) disorders or symptoms have also reported to occur often within the ASD population. A meta-analysis done by McElhanon, McCracken, Karpen and Sharp (2014) found that the ASD population was more prone to experience gastrointestinal symptoms such as

abdominal pain, constipation and diarrhea when compared to a control group. Ming, Brimacombe, Chaaban, Zimmerman-Bier and Wagner (2008) found 59% of their ASD sample presented with gastrointestinal dysfunction. Molloy and Manning-Courtney (2003) found 24% of their ASD sample to have at least one gastrointestinal symptom, with diarrhea being the most common at 17% of the sample reporting it. A number of GI disorders have also been associated with ASD, such as GERD, gastritis, inflammatory bowel disease, celiac disease, and Crohn's disease (Bauman, 2010).

### **Medical Conditions**

Conditions such as premature birth have also been found to be a common co-occurrence with ASD. Limperopoulous et al. (2008) found that 26% of children born before 32 weeks were screened as positive for ASD symptomatic behaviors. Johnson et al. (2010) conducted a longitudinal study following children born before 26 weeks gestation and found that 8% of the sample was diagnosed with ASD. Another longitudinal study found a similar prevalence rate in their sample of children born weighing less than 2000 grams (Pinto-Martin et al., 2011).

Other medical conditions, such as allergies and frequent ear infections, are a common comorbidity for children with ASD. Lyall, Van de Water, Ashwood and Hertz-Picciotto (2015) found that that food allergies were more common in children with ASD. Tan, Thomas and Lee (2019) confirmed these results, finding that parent-reported food allergies were 2.5 times more common in children with ASD compared to the general population. In regard to ear infections, Niehus and Lord (2006) found that children with ASD presented with more ear infections than typically developing children, with a high proportion of children having at least one ear infection before the age of 2. The authors also found that children with ASD were prescribed antibiotics more often than their typical controls, and these antibiotics were almost always associated with

the ear infections. Adams et al. (2016) took the analysis of ear infections in the ASD population one step further by looking at acute otitis media (AOM), an infection characterized by middle ear inflammation and effusion. The authors found that children with ASD are at an increased risk for AOM and other conditions related to the middle ear.

Hearing and vision impairments have also been found to occur often within the ASD population. Diziter et al. (2019) found that hearing impairment, which was defined as any type of deficit in hearing including deafness, abnormal test results, hearing loss and/or hearing complaints, was more common in the ASD population (2.8%) than within a non-ASD population (0.6%). Kancherla, Van Naarden and Yeargin-Allsopp (2013) found that 5.8% of children with hearing loss also had a diagnosis of ASD. The authors also included vision impairment in their analysis and found that 7.2% of those with vision impairment also had ASD. Van Naarden Braun et al. (2015) found a similar comorbidity prevalence rate of 8% of children with both ASD and vision impairment.

### **Neurological/Genetic Disorders**

Neurological and genetic disorders may also be more common in the ASD population. Craig, Savino & Trabacca (2018) found prevalence rates ranging from 3% to 16% of ASD in populations with Cerebral Palsy (CP), suggesting that ASD may occur more often in children with CP. Van Naarden Braun et al. (2015) examined the prevalence of ASD and CP, among other disorders, over a 15-20 year time period and found that 8% of children with CP had co-occurring ASD. Ayoub et al. (2017) found similar results; 8.7% of children with CP also had ASD. Down syndrome has also been reported to co-occur with ASD, with prevalence rates ranging from 3.3% to 15.6% (DiGuseppi et al., 2010). One study found that 18.2% of children

with Down syndrome aged 2 to 11 years also had ASD. Other research has found rates of comorbidity ranging from 16% to 18% (Warner, Howlin, Salomone, Moss & Charman, 2016).

### **Psychiatric Disorders**

Some psychiatric disorders are also common comorbidities with ASD. ADHD has some similar core symptoms of ASD, and until the DSM-5 one could not get an ADHD diagnosis if ASD was present (Stevens, Peng & Barnard-Brak, 2016). Currently ADHD is one of the most common comorbidities in children with ASD, with comorbidity rates ranging from 40% to 70% (Antshel, Zhang-James, Wagner, Ledesma & Faraone, 2016). Stevens et al. (2016) found in their study that 40.6% of their ASD sample also had a confirmed diagnosis of ADHD. Another study found 59.1% of the ASD sample had co-occurring ADHD (Salazar et al., 2015).

One of the most common comorbidities of ASD is intellectual disability (ID). Estimates of prevalence range from 30% to 70% (Tonnsen et al., 2016). The Autism and Developmental Disabilities Monitoring (ADDM) Network found that, of the cases who had IQ information, 33% of children had co-occurring ID and ASD, with it occurring more often in girls than boys (40% versus 32% respectively) (Maenner et al., 2020).

## First Signs of ASD

Though ASD is most often diagnosed around the ages of three or four (Maenner et al. 2020), studies have shown that signs of the disorder can be identified as early as one year. Maestro et al. (2005) found in their study of home videos of children later diagnosed with ASD that 87.5% of their sample had symptoms such as abnormal eye contact and lack of initiative within the first year of life. Werner, Dawson, Osterling and Dinno (2000) also used home videos as the source for their retrospective study and found that children with early onset ASD were less likely to orient to their names when called at 8 to 10 months of age when compared to typically developing children. Clifford and Dissanayake (2008) found in their retrospective study that infants later diagnosed with ASD were reported as having an impairment in their use of smiling and appropriate affect as well as a lack of play behaviors, such as peekaboo.

Zwaigenbaum et al. (2005) further examined these red flag behaviors in children during their first year of life. Infants were followed from 6 to 24 months and assessed using various methods, such as visual orienting task, a temperament measure, and developmental measures, such as the *Mullen Scales of Early Learning* (Mullen, 1995). They compared high-risk infants, children with siblings with ASD, to low-risk infants and found that by the age of 1, children who were later diagnosed with ASD presented with specific behavioral markers, such as abnormal eye contact and visual tracking, a tendency to disengage visual attention, and a specific temperament defined as marked passivity and decreased activity at 6 months of age, followed by extreme distress reactions, decreased positive affect expressions, and a tendency to fixate on particular objects at 12 months of age (Zwaigenbaum et al. 2005).

One's temperament may also be an indicator of ASD. Clifford et al. (2013) conducted a study investigating the temperament of high-risk infants at the ages of 7, 14, and 24 months, with



a diagnostic assessment occurring at 36 months. The authors used the *Infant Behavior Questionnaire-Revised (IBQ-R*; Garstein and Rothbart, 2003), the *Early Childhood Behavior Questionnaire (ECBQ*; Putnam, Gartstein & Rothbart, 2006) and the *Child Behavior Questionnaire (CBQ*; Rothbart, Ahadi, Hershey & Fisher, 2001) to evaluate the infants' temperament using the temperament clusters turned factors of Surgency, Negative Affectivity and Effortful control. Surgency was defined as approach behaviors, which can be observed as smiling and laughter. Negative Affectivity was defined as behaviors demonstrating anger and frustration, then fear-based behaviors as the child gets older. Effortful Control was defined as the ability to regulate attention, behavior and emotions to achieve goals (Clifford et al., 2012).

The authors found that temperaments tend to vary in the first 2 years of life between typically developing infants and infants who would later receive a diagnosis of ASD. In regard to the Surgency factor, parents reported that infants later diagnosed with ASD were more perceptually sensitive to the environment. They also observed that high-risk infants smiled and laughed less than typically developing infants. When analyzing the Effortful Control factor, authors found that infants who went on to develop ASD demonstrated lower levels of Effortful Control during the second year of life. The authors found that the Cuddliness dimension led to this difference in Effortful Control, for high-risk infants were reported as less cuddly as infants when compared to low-risk infants, and high-risk infants continued to be less cuddly throughout the second year of life. In terms of the Negative Affect factor, infants later diagnosed with ASD were found to have higher levels of overall negative affect compared to low-risk infants at 24 months. In summary, Clifford et al. (2012) found that infants later diagnosed with ASD presented with a temperament profile of increased perceptual sensitivity during the first year of life, then increased negative affect and reduced cuddliness during the second year of life.

These studies demonstrate that signs of ASD can be observed during the first two years of life. If red flags can be identified this early in life, services such as early intervention should be able to screen for them and begin the referral process for possible evaluation.

## Early Intervention

The Centers for Disease Control and Prevention (CDC) defines early intervention (EI) as “services and supports that are available to babies and young children with developmental delays and disabilities and their families” (CDC, 2019). More specifically, they are federally mandated services designed to enhance a child’s development and are provided for children with developmental disabilities under the Public Law 105-17, an amendment to Individuals with Disabilities Education Improvement Act (IDEA) (Ramey & Ramey, 1998). Early Intervention, or Part C, is a program responsible for coordinating services within and across community and government agencies to address the needs of children under the age of 3 (Rosenberg, Zhang & Robinson, 2008). Services begin with a comprehensive assessment that looks at the child’s strengths, weaknesses and needs, as well as those of the family. The child is deemed eligible if he or she presented with a developmental delay according to an assessment measure, has a pre-existing condition that leads to automatic eligibility, or is deemed at-risk due to environmental, medical or social circumstances (Ramey & Ramey, 1998). Each state has its own definition of eligibility; for example, in Louisiana, a child needs to either score one and a half standard deviations below the mean in two domains, as measured by the *Battelle Developmental Inventory, 2<sup>nd</sup> edition (BDI-II)* (Newborg, 2005), be deemed as at risk via Informed Clinical Opinion, and/or have an established medical condition (Sharp, 2012).

Once the child is deemed eligible, he or she receives services through a transdisciplinary model, which is defined as the “sharing of roles across disciplinary boundaries, so that communication, interaction and cooperation are maximized among team members” (King et al., 2009, p. 211). Team members may include speech language pathologists, occupational therapists, developmental specialists etc., and these teams implement services in the child’s

natural environment, such as home or daycare, with the child's caregivers involved in the creation of goals and implementation of strategies.

### **Effects of Early Intervention on ASD Population**

For children with ASD, there are two common evidence-based approaches to early intervention; Naturalistic Developmental Behavioral Interventions (NDBI; Schriebman et al., 2015), which use natural cues, models and opportunities to elicit specific behaviors, using natural consequences to reinforce the behaviors, and interventions more focused on operant conditioning, such as applied behavior analysis (ABA) or Early Intensive Behavioral Intervention (EIBI) (Landa, 2018).

Early intervention programs have been shown to be effective for the ASD population. Landa and Kalb (2012) examined the effects a 6-month intervention would have on toddlers, ages 22 to 33 months, with ASD. The interventions included ABA routine-based strategies, such as Pivotal Response Training (Schreibman & Kogel, 1996) and Discrete Trial Training (Lovaas, 1987). The authors found that in those six months the children made significant gains in IQ and communication skills, while severity of autism symptoms significantly decreased.

Estes et al. (2015) assessed the effectiveness of the intensive use of intervention for two years. The children, ages 18 to 30 months, received two years of the Early Start Denver Model (ESDM; Rogers & Dawson, 2010) and were followed until the age of six years old. The authors found that the children with ASD maintained the gains they had made two years previously, including IQ, adaptive behavior, challenging behaviors and symptoms of ASD. When compared to the control group, the children with ASD who received the ESDM demonstrated better adaptive skills and socialization ability as well as fewer severe overall ASD symptoms.

Landa (2018) conducted a meta-analysis of parent-mediated interventions and found that along with improvements in vocabulary and a reduction in symptom severity, parents of children with ASD demonstrated improved parent engagement. The results showed that parents were able to learn models for implementation and acquire new engagement strategies to better support their child's development.

### **Screening for ASD in Early Intervention**

Regular developmental screeners are recommended to occur during pediatric visits to identify potential risks of ASD (King et al. 2010). However, not all pediatricians can effectively monitor for these risks (Ozonoff et al. 2015), which leads the responsibility to the EI services to appropriately screen and evaluate at-risk children. Though regular use of evidence-based screening instruments is recommended for use (CDC, 2020a), they may not be implemented regularly in EI settings. Screening processes may also vary across EI providers, for in order to children to be connected to and receive appropriate services, two elements must occur; “(1) an effective decision rule to determine which children demonstrate sufficient risk to warrant follow-up (i.e. risk stratification) and (2) family engagement with recommended screening, assessment and intervention services” (Sheldrick et al., 2019, p. 2305).

Sheldrick et al. (2019) evaluated these elements in their study evaluating EI's process of screening, EI provider concerns and their perception of parental concern. The authors implemented a three-stage screening protocol in three partner EI agencies. Stage 1 consisted of two screening instruments implemented by the EI provider to the child's parents. If the child scored positive on the screeners, he or she moved on the Stage 2, which consisted of a play-based observational assessment administered by specially trained EI providers. If the child scored positive on this assessment, he or she was referred for a diagnostic assessment, at which

the child was given an DSM-V ASD diagnosis if appropriate. The authors also conducted a qualitative component to their study, in which surveys and in-person semi-structured interviews were conducted with the EI providers participating in the screening protocol as well as the parents involved. Sheldrick et al. (2019) found that parents' and providers' concerns were more predictive of a diagnostic referral completion than were positive scores on the screener. Even if the child did not receive a positive score, providers were given the option of a clinical decision rule, so referrals could be made based on concern rather than scores, and these referrals yielded "more ASD diagnoses per staff hour than referrals based on positive screening results in the absence of clinical concern" (Sheldrick et al., 2019, p. 2316). Overall, referrals and future diagnoses were driven by the presence of concern; the screening process moved quicker and more smoothly when the parents were engaged with the protocol, while a lack of concerns led to a slower process and a continued support towards family engagement.

## **Developmental Functioning and Trajectories**

The assessment process of early intervention looks at all areas of development, from motor skills to adaptive skills. During the ages that early intervention supports, zero to three years old, significant milestones should be obtained. The CDC (2020b) has developmental milestones as follows; by the age of 1, a child should be playing social games, such as “peek-a-boo”, using and imitating simple gestures, and sitting and standing independently. By the age of 2, a child should be copying others, saying sentences with two to four words, following two-step instructions, and running and walking up and down stairs. By the age of three, when the child transitions out of EI, a child should be taking turns in games, carrying on conversations, copying a circle with a writing utensil, and climbing well (CDC, 2020b).

These milestones often present later in children with ASD. Landa, Gross, Stuart and Faherty (2013) conducted a longitudinal study to examine the developmental trajectories of children with and without ASD. Each participant was evaluated every six months starting at six months of age using scales that assessed overall development as well as communication and social skills. The authors found that the children were comparable in their development at 6 months but the ASD and non-ASD groups diverged around 14 months of age, at which time children with ASD demonstrated lower expressive language skills and positive affect scores. At the same age, the children in the non-ASD group demonstrated significant gains in both expressive and receptive language. The authors also found that fine motor skills were less mature in the ASD group compared to the non-ASD group at 14 months of age (Landa et al., 2013).

### **Motor Development**

Other studies have looked at specific domains at development and how they may be impacted by ASD. Flanagan, Landa, Bhat and Bauman (2012) were curious to examine if there was a relationship between poor postural control during infancy and a later diagnosis of ASD in a group of high-risk infants. The authors assessed for postural control by using the pull-to-sit task, in which an adult takes the arms of the infant while in supine position and pulls the infant up to a sitting position. In a child with adequate postural control, one would expect to see the child maintaining head control throughout the task. If the child does not have control, the head would lag backwards. The authors found that within the ASD outcome group, 90% of the children displayed a head lag during the pull-to-sit task at 6 months of age, compared to only 35% of the non-delay group who displayed a head lag.

Kaur, Srinivasan and Bhat (2015) ran a longitudinal study examining the motor milestones of grasping, mouthing, and dropping objects in at-risk infants compared to typically developing infants between the ages of 6 and 15 months. They found that at-risk infants did not have functional grasping and dropping skills at 6 and 9 months old respectively. They also observed that at-risk infants did not mouth objects at 9 months of age but presented with excessive mouthing at 15 months of age, compared to the typically developing infants. When the authors followed up with the at-risk infants at the age of 2 years, 8 of the 14 infants had received an ASD and/or a developmental delay diagnosis.

LeBarton and Iverson (2016) examined the association between gross motor and communication development in infants at risk for ASD and found that children at risk for ASD were more likely to exhibit gross motor delays from 5 to 10 months of age. When associating these motor milestones with communication milestones, the authors found that the ability to sit at 7 months significant when related to when the child began babbling and the emergence of the



“show” gesture, which the authors defined as “the ability to... hold objects and extend them in the direction of the interlocutor to ‘show’ the object to others in a manner that may support coordination with social behaviors” (p. 65). The authors surmised that motor development may create learning environments in which children can experience novel objects and have opportunities to interact with others and develop language skills.

### **Communication Development**

The ability to develop language skills is a known deficit in ASD, with it being a core symptom in the DSM definition of the disorder until the DSM-V. Landa, Holman and Garrett-Mayer (2007) conducted a prospective study analyzing the development course of children diagnosed earlier or later with ASD, specifically looking at communication and social outcomes. The authors defined the early diagnosis group as children receiving a diagnosis of ASD at 14 months and the later diagnosis group as children who received clinical judgements of ASD but no diagnosis at 14 months. However, children in the later ASD diagnosis group did begin to exhibit signs of ASD by their second birthday. They found that children in the early diagnosis group at 14 months of age demonstrated abnormalities in the initiation of communication and the use of vocal and non-vocal forms of communication, and these abnormalities persisted through 24 months of age. These deficits involved skills that typically developing infants gain at 8 to 10 months. The later diagnosis group demonstrated these deficits later on, around 24 months of age. Authors found that this group demonstrated a slowed growth of communication development, such as the use of consonant vowel sounds and words, plateaus of skill acquisition, and decreases behaviors such as the use of gestures. These results showed that ASD may lead one’s communication development to stop, slow, or even regress.

Mitchell et al. (2006) also looked at overall communication development in their prospective study using high and low-risk infants, with the ASD group being high-risk infants who received a diagnosis of ASD at 24 months. The authors found that at 12 months, children with ASD were reported to understand fewer phrases and use fewer gestures than non-ASD children. At 18 months, children with ASD continued to demonstrated delays in understanding phrases as well as in the use and comprehension of single words and gestures.

ASD may also impact specific forms of communication. Watson, Crais, Baranek, Dykstra and Wilson (2013) examined the use of communicative gestures in infants with and without ASD in their retrospective study. The authors used three types of gestures as classified by Bruner (1981) in their analysis; social interaction, or gestures used to direct another's attention to oneself, behavior regulation, or gestures used to control another person's behavior, and joint attention, or gestures used to direct another's attention to an object, event, or person, for the purpose of sharing interest (Watson et al., 2013). The authors found that infants with ASD showed little use of the behavior regulation gestures at 9-12 months, but eventually learned the functionality of these gestures by 15-18 months. The authors surmised this lack of use of this gesture early on could be due to the fact that children on the spectrum struggle with triadic interactions, or coordinating attention between an object and another person, event, or place. The authors also found similar results with the joint attention gestures, with infants with ASD using this type less likely when compared to typically developing and developmentally delayed children in both of the age ranges. In regard to the use of social interaction gestures, the authors found infants with ASD did not significantly differ with typically developing infants. The authors believed this result was due to the parents' overcompensation for their infant's more

limited responsiveness, “giving the child more prompts “in order to maintain interactions with their infants” (p. 33).

### **Cognitive Development**

A child’s cognitive development may also be impacted by ASD. Cognitive skills can include attention, memory, reasoning skills, and perception skills (Newborg, 2005). Pellicano (2010) assessed the development of core cognitive skills such as executive function, or the ability to problem solve and plan out complex actions, theory of mind, or the ability to put oneself in the minds of others, and central coherence, or the ability to focus on global wholes rather than individual elements. The author found that children with ASD demonstrated difficulties in “false-belief understanding, problems with higher order planning and cognitive flexibility together with capabilities in processing local information” (p. 1411). When the children with ASD were assessed three years later, they demonstrated improvements over time in theory of mind and executive function skills, but not central coherence skills.

Estes et al. (2015) also conducted a longitudinal study examining developmental characteristics of high-risk infants, with cognitive development as one of the characteristics. Using the MSEL (Mullen, 1995) to measure overall cognitive ability and delay and found that at 12 months children who were high-risk and eventually received a diagnosis of ASD demonstrated significantly lower Early Learning Composite (ELC) and receptive language scores, then at 24 months the same group had lower scores on the ELC and all of the subscales of the MSEL when compared to the non-ASD high and low-risk groups.

### **Adaptive Development**

Estes et al. (2015) also looked at adaptive development, another area impacted by ASD. *Using the Vineland Adaptive Behavior Scales-II (VABS-II; Sparrow, Balla & Cicchetti, 2005),*

the authors found a similar trajectory pattern as found using the cognitive outcome measure. At 6, 12 and 24 months old, children with ASD had significantly lower Adaptive Behavior Composite (ABC) scores than the other groups, and at 24 months old children with ASD demonstrated significantly lower scores across all subscales when compared to the ASD negative groups.

Bradhsaw, Gillespie, Kaiman, Klin and Saulnier (2019) examined the adaptive developmental trajectory of high-risk infants sorted into three groups; ASD group, in which they received a diagnosis at 24 or 36 months, the “broader autism phenotype” (BAP) group, in which the toddlers reached subthreshold features of ASD, and the unaffected (UA) group, or the toddlers who were determined to be developing typically. The ASD and the BAP groups were considered high-risk in their analysis. Authors found that daily living skills declined significantly between the ages of 12 and 36 months in the high-risk infants while the UA group were stable between 12 and 24 months. The authors concluded that though these infants are making gains in adaptive development, they are “failing to make the large gains that would be expected given their chronological age” (p. 1493).

Franchini et al. (2018) came to a similar conclusion in their study on adaptive functioning trajectories in preschoolers with ASD. When compared to typically developing children, children with ASD had lower adaptive functioning trajectories from ages 18 months to 6 years. When evaluating the subgroups of children with ASD, the authors found that children with higher symptom severity had lower ABC trajectories when compared to the subgroup of lower symptom severity.

## **Purpose**

Many children with ASD also have co-occurring psychiatric or medical concerns, and these previous diagnoses can complicate the screening process in young children entering EI. Although many researchers have investigated the prevalence of common comorbidities in the ASD population, the impact previous diagnoses have on children already at risk for ASD is unknown. The literature does suggest that ASD impacts the developmental trajectories of young children (Bradshaw et al., 2019; Estes et al., 2015) and that several previous diagnoses are more common in the ASD population than in the general population (Diziter et al., 2019; Limperopoulous et al., 2008; McElhanon et al., 2014).

The current study aims to examine the relationship between the presence of previous diagnoses and developmental functioning in children presenting as at-risk for ASD compared to children presenting with no risk. The term “previous diagnosis” was used for this study, for the children included in this study have not yet received a diagnosis of ASD. Although the previous literature suggests that ASD impacts developmental trajectories of children under the age of 3 (Landa et al., 2013), very few studies have included the presence of previous diagnoses as a possible moderator of developmental functioning. Expanding this line of research to younger children may help educate parents and EI providers as to how previous diagnoses may impact scores on assessment measures, and how to differentiate these scores from ASD-specific concerning scores. Therefore, the current study investigates differences in developmental functioning within specific domains (i.e., communication, cognitive, adaptive, motor, and personal-social, total DQ) in infants and toddlers with and without previous diagnoses, comparing children scoring as at-risk for ASD to those who scored as no risk. The quantity of

previous diagnoses and its impact on developmental functioning was also investigated between the two groups.

The literature on ASD and comorbidities provides some evidence that comorbidities are common in the ASD population, and that ASD impacts various areas of development (Mannion & Leader, 2013; Landa et al., 2013). Therefore, it is hypothesized that individuals demonstrating possible symptoms of ASD as well as previous diagnoses may have varying developmental functioning abilities when compared to children at risk for ASD with no previous diagnoses. Research has shown that specific domains, such as the Personal-Social and Cognitive domains, of the *BDI-II* are predictive of ASD (Goldin et al., 2014), but with the addition of previous diagnoses such as premature birth and chronic ear infections, other domains, such as Adaptive and Motor, are expected to demonstrate delays as well. Ultimately, it is expected that the children at risk for ASD and with reported previous diagnoses would present with a wider range of developmental functioning.

## Method

### Participants

All participants are in or were enrolled in and have since aged out of EarlySteps, Louisiana's statewide early intervention program under the Individuals with Disabilities Education Act, Part C. The data for this study has been taken from an existing dataset containing assessment information. In order to be eligible for EarlySteps services, a child must present with or be at risk for a developmental delay or have a qualifying medical condition. The children included in this dataset were referred to EarlySteps due to possible developmental concerns, so the majority of the participants in this study may have some form of atypical development. Only participants between the ages of 17 and 36 months with valid *Baby and Infant Screen for Children with aUtism Traits, Part 1 (BISCUIT: Matson et al., 2007)* and *Battelle Developmental Inventory, 2<sup>nd</sup> edition (BDI-II: Newborg, 2005)* scores were included in the analysis. Also, participants were included if they had been recoded according to comorbidity type in the database, which resulted in 11,970 participants analyzed in this study.

Groups were created based according to the score each participant received on the *BISCUIT*. A 17 and above put the child in the possible ASD range; thus these participants were placed in the "ASD Risk" group. Below a score of 17 placed the child in the atypical development range; thus, these participants were placed in the "No Risk" group. These groups were then analyzed separately, and results were compared. Tables 1 and 2 display the demographic information for each group.

Table 1. Demographic information of No Risk Group

Age in months <i>M(SD)</i>	25.188(4.663)
Sex	
Male <i>n (%)</i>	5061 (65.4%)
Female <i>n (%)</i>	2653 (34.3%)
Missing <i>n (%)</i>	19 (.2%)
Race	
African American	2767 (35.8%)
White	4005 (51.8%)
Hispanic	315 (4.1%)
Other	431 (5.6%)
Missing/Unreported	215 (2.7%)
BDI Scores	
Total DQ <i>M(SD)</i>	87.71 (12.233)
Adaptive DQ <i>M(SD)</i>	88.33 (13.311)
Personal-Social DQ <i>M(SD)</i>	94.83 (11.525)
Communication DQ <i>M(SD)</i>	80.37 (15.930)
Motor DQ <i>M(SD)</i>	99.53 (11.780)
Cognitive DQ <i>M(SD)</i>	84.58 (11.476)
Presence of Previous Diagnosis	
Yes <i>n (%)</i>	3529 (45.6%)
No <i>n (%)</i>	4204 (54.4%)

Table 2. Demographic information of ASD Risk Group

Age in months <i>M(SD)</i>	25.536 (4.668)
Sex	
Male <i>n (%)</i>	3082 (72.7%)
Female <i>n (%)</i>	1144 (27%)
Missing <i>n (%)</i>	11 (.3%)
Race	
African American	1691 (39.9%)
White	2008 (47.4%)
Hispanic	170 (4%)
Other	264 (6.2%)
Missing/Unreported	104 (2.5%)
BDI Scores	
Total DQ <i>M(SD)</i>	72.23 (11.753)
Adaptive DQ <i>M(SD)</i>	75.40 (12.647)
Personal-Social DQ <i>M(SD)</i>	80.95 (11.383)
Communication DQ <i>M(SD)</i>	65.05 (12.589)
Motor DQ <i>M(SD)</i>	90.01 (15.225)
Cognitive DQ <i>M(SD)</i>	74.23 (10.842)
Presence of Previous Diagnosis	



Yes <i>n</i> (%)	1962 (46.3%)
No <i>n</i> (%)	2275 (53.7%)

Conditions found to be commonly comorbid with ASD were included in the analysis, premature birth, global developmental delay, Down syndrome, seizures, hearing concerns, vision concerns, cerebral palsy, ear infections, gastrointestinal disorders, allergies, asthma, and genetic or other medical disorder.

Table 3. Diagnosis type by group

Previous Diagnosis Type	ASD group	No Risk Group
Premature	379	862
General Developmental Delay	110	96
Down's Syndrome	30	102
Seizures	165	135
Hearing Difficulties	80	137
Vision Difficulties	89	92
Cerebral Palsy	62	78
Chronic Ear Infections	412	794
Gastrointestinal Disorders	102	133
Allergies	346	526
Asthma	225	325
Other Genetic Disorder	99	154
Other Medical Diagnosis	674	1272
Total N	4237	7733

## Measures

*Baby and Infant Screen for Children with aUtism Traits, Part 1 (BISCUIT-Part 1: Matson et al., 2007).*

The *BISCUIT* is an informant-based screener that assessed for autistic symptomology and associated features of young children aged 17 to 37 months. It consists of three parts; part 1 evaluates ASD symptomology, part 2 probes for comorbid psychology and part 3 examines possible challenging behaviors. The *BISCUIT-Part 1* assesses core ASD symptoms such as socialization/nonverbal communication, repetitive behavior/restricted interests, and

communication using 62 items. Each of these 62 items are rated using a 3-point Likert scale, prompting the parent to compare the child to same-aged peers and respond with either a “0” (*Not different; no impairment*), “1” (*somewhat different; mild impairment*) or “2” (*very different; severe impairment*). The measure then includes age-based cut-off ranges for the total *BISUIT-Part 1* score; scores of 0-16 land within the “No ASD/Atypical Development” range; scores of 17-38 land within the “Possible ASD” range; and scores of 41-124 land within the “Probable ASD” range. Thus, a score of 17 or higher lands the child in the “at-risk” range.

The *BISUIT-Part 1* has an internal reliability coefficient of .97 (Matson et al., 2009) and a strong convergent validity with the *Modified Checklist for Autism in Toddlers (M-CHAT)* demonstrated by a large positive correlation of  $r = .80$  (Matson, Wilkins & Fodstad, 2011). The *BISUIT Part-1* scores used for this analysis also demonstrated a high internal consistency, as determined by a Cronbach’s alpha of .929. The present study used the *BISUIT-Part 1* total scores as well as the demographic form. The demographic form was used to collect information of the participant’s demographic variables, such as developmental and medical history. The *BISUIT-Part 1* total score was used to assess for risk of ASD.

*Battelle Developmental Inventory, Second Edition (BDI-2; Newborg, 2005)*

The *Battelle Developmental Inventory, Second Edition (BDI-2; Newborg, 2005)* is an assessment instrument used to measure the development of children from birth through 7 years, 11 months. It evaluates five domains of development: Adaptive (ADP), Personal-Social (P-S), Communication (COM), Motor (MOT) and Cognitive (COG). Each of these domains are then broken down into subdomains. The Adaptive domain consists of the Self-Care and Personal Responsibility subdomains; the Personal-Social domain is made up of the Adult Interaction, Peer Interaction, and Self-Concept and Social Role subdomains; the Communication domain consists

of the Receptive Communication and Expressive Communication subdomains; the Motor domain has the Gross Motor, Fine Motor, and Perceptual Motor subdomains; and the Cognitive subdomain is made up of the Attention and Memory, Reasoning and Academic Skills and Perception and Concepts subdomains.

These subdomains make up the 450 items of the measure and are scored on a 3-point Likert scale. A rating of 0 indicates that the child has no ability in this skill; a rating of 1 indicates that child has an emerging ability in the skill; and a score of 2 indicates the child has ability with the skill. These ratings combine for a score in each domain as well as a total developmental quotient (DQ), all of which have a mean of 100 and a standard deviation of 15.

The BDI-II has demonstrated good reliability, with internal consistency reliabilities ranging from .90-.96 across domains and an overall reliability of .99. Individual item scores were not collected on the database used for this analysis, so the internal consistency was not able to be calculated. It was also found to have good convergent validity with many widely used instruments such as the *Bayley Scales of Infant Development, Second edition (BSID-II)*, the *Denver Developmental Screening Test-II (DDST-II)*, the *Preschool Language Scales (PLS-4)*, the *Vineland Social-Screening Test-II (Vineland SEEC)*, the *Comprehensive Test of Phonological Processing (CTOPP)*, the *Wechsler Preschool and Primary Scale of Intelligence-Third Edition (WPPSI-III)*, and the *Woodcock-Johnson III Test of Achievement (WJ III ACH)*, with correlations ranging from .60 to .75 (Newborg, 2005). For the present study, the total scores for five domains as well as the total developmental quotient was used in order to assess for possible developmental delays.

## **Procedure**

The Louisiana State University institutional review board and the State of Louisiana Office for Citizens with Developmental Disabilities approved the study prior to data collection. The *BISCUIT-Part 1* and the *BDI-II* were administered by EarlySteps service providers at the participant's home or daycare center as part of an eligibility assessment that included caregiver interviews and direct observation. All service providers were trained in the administration of the assessment measures and held a degree, certification, and/or licensure in various related fields such as special education, speech-language pathology, occupational therapy, physical therapy, and psychology. Records collected between 2007 and 2018 were included in the analyses.

The data used for the present study will be from a research database of de-identified archival records. The Institutional Review Board determined that the 45 CFR part 46 of the U.S. Department of Health and Human Services regulation does not apply and informed consent was not required.

## **Statistical Analyses**

### *Initially Proposed Analyses*

Initially, univariate and multivariate analyses were proposed to be conducted to investigate the following research question: 1) does the presence of previous diagnoses affect the developmental functioning of children presenting as at risk for ASD. A priori analyses of the differences in the demographic variables of age, gender and race were to be included, and a chi-square analysis was to be conducted to assess differences across groups. Descriptive statistics were conducted for total scores on the *BISCUIT-Part 1* and the five domains of the *BDI-II*.

A one-way multivariate analysis of variance (MANOVA) was proposed to examine the relationship between group and developmental functioning. The independent variable (IV) was group (i.e., with or without previous diagnoses) and the dependent variables (DV) was the

Developmental Quotients of the Adaptive, Personal-Social, Communication, Motor and Cognitive domains of the *BDI-II*. Univariate one-way ANOVAS were to be conducted to further evaluate the developmental differences between the groups and significant ANOVAs were to be followed up with t-tests to examine these results further.

However, when the initially proposed analysis began, several assumptions were violated and were not able to be corrected. After discussion with the primary advisor and the remaining committee members, a revised statistical procedure was created and completed.

#### *Updated Statistical Procedure*

First, the “Previous Diagnoses” category of the *BISCUIT*’s demographic form was recoded into 13 new, dichotomous variables, with “0” representing no presence of the diagnosis and “1” representing the presence of the diagnosis. The demographic variables of “ASD” and “Intellectual Disability” were excluded from the analysis, for the sample included children being assessed for possible ASD risk, and ID cannot be reliably diagnosed within the age range of the sample (APA, 2016). The remaining recoded variables were combined to create three new variables; Presence of Previous diagnosis (PD), Presence of 1 PD and Presence of 2 or More PDs. The variables based off of quantity of PDs were created due to the uneven sample sizes across quantities of PDs. In an attempt to create more evenly distributed groups, one group was made of children with only one reported PD, and another group was made representing children with 2 or more PDs. Analyses were done separately between the two groups of participants; the No Risk and ASD Risk groups. Group assignment was based off the child’s score on the *BISCUIT*; if the child received a score of 17 or above, he or she was determined to be at risk for ASD was placed in the ASD Risk group (Matson et al., 2007). If the child scored below a 17, he or she was placed in the No Risk Group.

An a priori power analysis program, G\*Power, was used to determine the expected number of participants for this study (Erdfelder, Faul & Buchner, 1996). For a simple regression with a medium effect size of .50, the power at .80 and the significant level at  $\alpha = .05$ , a total sample size of 18 would be needed. For a multiple regression with the same effect size, power, and significance level, a sample size of 23 would be needed.

To evaluate the relationship between the presence of PDs and developmental functioning, six simple regressions were run for each of the two groups, resulting in 12 regressions in total. One regression was run for each of the developmental domain scores of the *BDI-II* as well as the Total Developmental Quotient (DQ), with the independent variable of the presence of PDs and the dependent variable each of these scores. Next, to examine the relationship between the number of PDs and developmental functioning, two multiple regressions were run, one for the No Risk group and another for the ASD Risk group, with the independent variables of one PD and two or more PDs. The Total DQ was used as the measure for developmental functioning, thus making it the dependent variable, in these multiple regressions.

## Results

### Preliminary Analysis Results

A priori analyses were conducted to investigate possible differences between the No Risk group and the ASD risk group in regard to the demographic variables of gender, race and age. Results of the chi-square analysis indicated there was a significant difference in gender  $\chi^2(1) = 67.482, p < .0005$ ; both the ASD group (72.9%) and the No Risk group (65.6%) had a higher percentage of males than females. However, the effect size was small, as assessed by Cramer's  $V = .048$ . There was also a significant difference between race and risk status,  $\chi^2(3) = 25.080, p < .0005$ . However, another small effect size was found, with a Cramer's  $V$  value of .046. Although there is an observed difference in the male to female ratio, the gender difference of ASD has been recognized in research (Loomes et al., 2017). An independent samples t-test was then conducted to test for statistically significant differences in age between the risk groups. Homogeneity of variances was found, as assessed by Levene's test for equality of variances ( $p = .089$ ). However, the t-test found a statistically significant difference in age across groups,  $t(11963) = 3.902, p < .0005$ . This result may be due to a mean age (25.311 months) occurring toward the beginning of the age range of the database (17 to 39 months).

### Secondary Analysis

Two linear regressions, one assessing each of the two groups, the ASD Risk group and No Risk group, were then run for the total developmental quotient (DQ) and each of the developmental domains of the BDI-II, resulting in 12 regressions in total. The results from each domain's regression's results are as follows.

#### *Total Developmental Quotient*

First, the relationship of the presence of previous diagnoses to the total DQ score was assessed for the No Risk group. There was linearity, as assessed by visual inspection of a scatterplot. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.803. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. Several outliers were found, with 21 of these outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations. However, these values remained in the analysis due to the atypical nature of the database (i.e., all children in database referred to atypical developmental concerns, received varied scores across domains). The assumption of normality was not violated, as assessed by visual inspection of the histogram and Normal P-P Plot. However, the results were not found to be significant,  $F(1,7731) = .219$ ,  $p = .640$ .

A regression was then run in the ASD Risk group. Linearity was established, as assessed by visual inspection of a scatterplot. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.957. Ten outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis for the reason stated above. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression established that the presence of PDs could predict the total DQ score in children at risk for ASD,  $F(1,4235) = 11.066$ ,  $p < .005$ , and the presence of these conditions accounted for .2% of the explained variability in scores. The regression equation was:  $\text{total DQ} = 72.784 + (-1.203 \times \text{presence of PDs})$ .

#### *Adaptive Domain*



The results of the No Risk group analysis are as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.871. Twenty-six outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression found that the presence of PDs predicted Adaptive domain scores,  $F(1,7731) = 46.328$ ,  $p < .0005$ , and the presence of these conditions accounted for .6% of the explained variability in scores. The regression equation was: Adaptive domain score =  $89.274 + (-2.062 \times \text{presence of PDs})$ .

The ASD Risk group regression results are as follows; a scatterplot established that the assumption of linearity was met. There was an independence of residuals, as assessed by a Durbin-Watson statistic of 1.958. Twelve outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression found that the presence of PDs predicted Adaptive domain scores,  $F(1, 4235) = 37.965$ ,  $p < .0005$ , and the presence of these conditions accounted for .9% of the explained variability in scores. The regression equation was: Adaptive domain score =  $76.510 + (-2.391 \times \text{presence of PDs})$ .

#### *Personal-Social Domain*

The results of the regression of the No Risk group are as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as

assessed by a Durbin-Watson statistic of 1.745. Twenty-six outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression found the presence of PDs could predict Personal-Social domain scores,  $F(1, 7731) = 55.379$ ,  $p < .0005$ , and the presence of these conditions accounted for .7% of the explained variability in scores. The regression equation was: Personal-Social domain score =  $93.935 + (1.9451 \times \text{presence of PDs})$ .

The regression of the ASD Risk group is as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.894. Ten outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression found the presence of PDs could predict the Personal-Social domain scores,  $F(1,4235) = 15.349$ ,  $p < .0005$ , and the presence of these conditions accounted for .4% of the explained variability in scores. The regression equation was: Personal-Social domain score =  $80.311 + (1.372 \times \text{presence of PDs})$ .

#### *Communication DQ*

First a regression was run with the No Risk group. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.874. Thirty-two outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity

was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression found that the presence of PDs predicted Communication domain scores,  $F(1,7731) = 42.549$ ,  $p < .0005$ , and the presence of these conditions accounted for .5% of the explained variability in scores. The regression equation was: Communication domain score =  $79.289 + (2.366 \times \text{presence of PDs})$ .

The regression results for the ASD Risk group are as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.951. Fifty-three outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. However, the assumption of heteroscedasticity was violated, as assessed by visual inspection of scatterplot. Also, the histogram and Normal P-P Plot indicated that the residuals were not normally distributed. The dependent variable, the Communication domain score, was transformed to account for the positively skewed data, first by applying the square root transformation, then the logarithmic transformation, then by applying the inverse transformation. However, each of these transformations did not correct the normality violation. The analysis was discontinued following these violations.

#### *Motor Domain*

The regression results for the No Risk group are as follows. A scatterplot established that the assumption of linearity was met. There was an independence of residuals, as assessed by a Durbin-Watson statistic of 1.868. One hundred and four outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. The histogram and Normal P-P Plot indicated that the residuals

were normally distributed. A linear regression found that the presence of PDS could predict Motor domain scores,  $F(1,7731) = 161.625$ ,  $p < .0005$ , and the presence of these conditions accounted for 2% of the explained variability of scores. The regression equation was: Motor domain score =  $101.072 + (-3.384 \times \text{presence of PDS})$ .

The ASD Risk group was then evaluated via a simple regression, with results as follows. A scatterplot established that the assumption of linearity was met. There was an independence of residuals, as assessed by a Durbin-Watson statistic of 1.870. One outlier with a studentized deleted residual greater than  $\pm 3$  standard deviations was found but kept in the analysis. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. However, the histogram and Normal P-P Plot indicated that the residuals were not normally distributed, demonstrating a negatively skewed distribution. The dependent variable, the Motor domain score, was transformed via three different methods, the square root transformation, then the logarithmic transformation, then the inverse transformation, in an attempt to correct the violation. However, the transformed variables continued to violate the normality assumption. The analysis for this variable was discontinued following these violations.

#### *Cognitive Domain*

The results of the regression of the No Risk group are as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.789. Twenty-nine outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear

regression between the presence of PDs and the Cognitive domain scores in the No Risk of ASD group was not statistically significant,  $F(1,7731) = .772, p = .380$ .

The results of the regression of the ASD risk group is as follows. A scatterplot demonstrated that the assumption of linearity was met. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.890. Fourteen outliers with studentized deleted residuals greater than  $\pm 3$  standard deviations were found but kept in the analysis. The assumption of heteroscedasticity was not violated, as assessed by visual inspection of scatterplot. The histogram and Normal P-P Plot indicated that the residuals were normally distributed. A linear regression established that the presence of PDs could statistically significantly predict Cognitive domain scores,  $F(1,4235) = 5.041, p < .05$ , and the presence of these conditions accounted for .1% of the explained variability of scores. The regression equation was: Cognitive domain score =  $74.576 + (-.750 \times \text{presence of PDs})$ .

### **Multiple Regression**

Two multiple regressions were then run to evaluate how the quantity of PDs can predict overall developmental functioning via the total DQ. First, the No Risk group was evaluated. There was linearity as assessed by partial regression plots and a plot of studentized residuals against the predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.802. There was homoscedasticity, as assessed by visual inspection of scatterplot. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were 14 cases in which the studentized deleted residuals were greater than  $\pm 3$  standard deviations, however these cases were kept in the analysis. There were no leverage values greater than 0.2 and values for Cook's distance above 1. The assumption of normality was not violated, as assessed by a Q-Q plot. The multiple regression established that the number

of PDs could statistically significantly predict total DQ score in the No Risk group,  $F(2, 7729) = 3.248$ ,  $p < .05$ ,  $\text{adj. } R^2 = .001$ . The coefficients demonstrated no statistically significant relationship between the presence of one PD and total DQ score,  $p = .543$ . However, a statistically significant linear relationship was found between the presence of two or more PDs and total DQ; the equation was:  $\text{total DQ} = 87.670 + (-.976 \times \text{presence of 2 or more PDs})$ .

Table 4. Multiple regression for quantity of PDs predicting developmental functioning in No Risk Group

Variable	R <sup>2</sup>	F	B (SE)
	.001	3.248*	
Constant			87.670*** (.189)
1 PD			.186 (.306)
2 or More PDs			-.976* (.439)

Note:  $N = 7732$ , \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$

The ASD risk group was then assessed. There was linearity as assessed by partial regression plots and a plot of studentized residuals against the predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 1.960. There was homoscedasticity, as assessed by visual inspection of scatterplot. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were 10 cases in which the studentized deleted residuals were greater than  $\pm 3$  standard deviations, however these cases were kept in the analysis. There were no leverage values greater than 0.2 and values for Cook's distance above 1. The assumption of normality was not violated, as assessed by a Q-Q plot. The multiple regression demonstrated that the number of PDs could statistically significantly predict total DQ score in the ASD group,  $F(2,4232) = 8.716$ ,  $p < .0005$ . Again, the coefficients demonstrated no statistically significant relationship between the presence of one PD and total DQ score,  $p = .068$ . However, a statistically significant linear relationship was found between

the presence of two or more PDs and total DQ; the equation was: total DQ = 72.784 x (-2.210 x presence of two or more PDs).

Table 5. Multiple regression for quantity of PDs predicting developmental functioning in ASD Risk group

Variable	R <sup>2</sup>	F	B (SE)
	.004	8.716***	
Constant			72.784*** (.246)
1 PD			-.734 (.402)
2 or More PDs			-2.210*** (.537)

Note: N =4235, \* p < .05, \*\* p < .01, \*\*\* p < .001

## DISCUSSION

Across the sample, children in the ASD risk group demonstrated lower developmental functioning overall and across various domains, and the presence of previous diagnoses further impacted these scores. Previously, studies examined the effect of one type of co-occurring diagnosis on children with ASD (Johnson et al., 2010; Diziter et al., 2019). The current study takes into account the developmental functioning of these children with both a previous diagnosis and a risk of ASD as determined by an ASD screener. For the analyzed domains, the presence of previous diagnoses significantly predicted developmental functioning within a developmental domain in children presenting as at-risk for ASD and in those presenting with no risk.

One interesting result found was the significant results of the Motor domain in the No Risk group. This analysis resulted in the largest adjusted R square value found in the analysis as well as the largest slope coefficient, demonstrating the largest difference in developmental functioning between the groups of a presence or absence of previous diagnoses. Though the No Risk group did not present with an at-risk score on the ASD screener, they were being referred to and assessed by an early intervention program for a variety of concerns and may present with overall or specific developmental delays. Studies have shown that early motor delays can be an indicator of later diagnosed neurodevelopmental conditions (Hudry, Chetcuti & Hocking, 2020), such as ADHD (NacNeil & Mostofsky, 2012). and or a specific language impairment (McPhillips et al., 2014). Motor delays, as well as cognitive delays, have also been shown to predict later motor competence and IQ in children born preterm, and if the child also has a neurological impairment, these outcomes are likely to follow them into adulthood (Baumann et al., 2020).



The regression done on cognitive domain also presented with interesting results, for it displayed the presence of previous diagnoses had little impact on the cognitive domain in both groups. Kushner, Bennetto and Yost (2007) found that, both an ASD group and a developmental delay (DD) group, which consisted of diagnoses such as speech delay, genetic disorders and global developmental delay, presented with significant cognitive weaknesses. However, those with ASD demonstrated a division in scores when it came to their nonverbal perceptual and conceptual abilities, performing better than the DD and typically developing (TD) group. Those in the ASD group also displayed specific weaknesses on certain cognitive subtests when compared to the two other groups. This suggests that patterns of cognitive abilities across functioning groups may vary, and that the presence of previous diagnoses may not play as significant a role in cognitive functioning.

Once the presence of previous diagnoses was evaluated, the multiple regressions were run to evaluate whether quantity of these conditions played a role in developmental functioning overall. This analysis found that for both the No Risk and ASD Risk groups, one pre-existing condition did not statistically significantly predict the total DQ score; however, two or more conditions did significantly predict overall developmental functioning. Also, the ASD Risk group also demonstrated a larger coefficient, showing a larger difference between the presence of two or more conditions versus none. This result is in agreement with the current research, for Aldinger et al. (2015) found that the more previous diagnoses the child presents with, the more severe the autism symptoms and/or behavioral impairments are. This increase of symptom severity and impairments can in turn impact functioning in various developmental domains, leading to lower scores on the *BDI-II* total DQ (Goldin et al., 2014).

It should be noted that the fit of the regression models for each of these analyses was not strong; the most variation explained was found in the motor domain, at 2%. The majority of the regressions had percentages below 1%. These results show that, though the presence of previous diagnoses play a significant role in developmental functioning, it is a small role. To further examine the role previous diagnoses play in developmental functioning, the condition type was explored as a possible additional predictor of developmental functioning during the analysis process. The previous diagnoses were grouped according to International Classification of Diseases – 9<sup>th</sup> Revision (ICD-9; 1996) grouping; medical, neurological and genetic. However, several assumptions were violated, and the results were found to be not significant, so the analysis was not continued nor included in this study. This lack of accounting for type of previous diagnosis is considered a limitation of this study. Further research should be done to fully assess whether type or quantity of pre-existing condition plays a role in developmental functioning.

Other limitations were found within this study. The children presenting as at-risk for ASD were not followed up with to confirm an ASD diagnosis, so it is not possible to determine whether these at-risk children met criteria for the disorder. Also, the wide array of previous diagnoses included in the analysis vary based on type, impact on overall functioning, and prevalence in sample and overall population. This wide variety and lack of differentiation from child to child may have impacted the overall analysis. The difference in sample size between the two risk groups is considered a limitation as well. Two separate analyses were run to account for the difference in size, but the comparison of the two should be done with caution. The No Risk group also cannot be considered a true control group, for the group consisted of children being referred to EarlySteps and presenting with various developmental delays other than ASD. Future

studies should be done to truly compare an ASD group to a group of typically developing children.

Though this study did not examine how specific types of previous diagnoses impact development, it can be hypothesized that the three most prevalent previous diagnoses present in the database, chronic ear infections, allergies and asthma, may have played a significant role in developmental functioning due to the results from this study. For example, Konstantareas and Homatidis (1987) found that lower cognitive functioning was significantly associated with ear infections occurring within the age range of the current study, with children with ASD demonstrating a greater number of ear infections when compared to typically developing children. Lyall et al. (2015) found similar results when assessing allergies and asthma, finding that children with these conditions demonstrated lower developmental functioning. It was also discovered that allergies could be related to the repetitive behaviors and/or stereotypies of ASD (Lyall et al., 2015).

Though the current study cannot significantly support these findings in regard to specific diagnoses, previous research has supported this study's findings by demonstrating a relationship between the presence of previous diagnoses and developmental functioning. According to this analysis, the motor domain was impacted most by a previous diagnosis; the cognitive domain the least. In summary, previous diagnoses play a role in developmental functioning in children being assessed by early intervention programs, with its impact varying across children presenting as at risk for ASD and those not at risk for the disorder.

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## **Vita**

Michaela Brown, born in Boston, Massachusetts, received her bachelor's degree from Northeastern University. She began working in the field of early intervention and grew an interest in assessment and treatment of autism spectrum disorder. To pursue her interest she decided to enroll in graduate school, receiving her Master of Arts from Teachers College at Columbia University. She then decided to enter the Department of Psychology at Louisiana State University. Upon completion of her master's degree in May of 2022, she will begin work on her doctorate.