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Neurocognitive Resilience in Children Born Late and Moderate Preterm (LAMP): Predictors of  
Outcomes in Attention, Working Memory, and Executive Functioning

by

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Submitted to the Department of Psychology

Eastern Michigan University

in partial fulfillment of the requirements for the degree of

DOCTORATE OF PHILOSOPHY

in

Clinical Psychology

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July 30, 2021

Ypsilanti, Michigan

### Acknowledgments

As I reach this milestone, I am filled with an extraordinary sense of gratitude to the family and friends that got me here. Thank you to my mom and my dad for unconditionally loving, supporting, and believing in me. Thank you for teaching me to work hard, be persistent, and live by my values. You make my dreams possible. Thank you to my incredible friends: Tristin, Tim, Bre'Anna, Sarah, Patrick, Bryce, Margaret, Tiffany, and so many others. You fill my life with a kind of love that makes all good things seem possible. And to my partner Shashwat: thank you for supporting me even when I was a nightmare human who wanted to fight you about the location of the toothpaste. You have been patient and kind and loving and supportive through the most difficult two years of my graduate career. A relationship that survives internship and a dissertation is a relationship with teeth. There is so much adventure ahead of us.

I would like to thank my advisor and chair, Dr. Carol Freedman-Doan. Thank you for sticking out the last 7 years with me. I have benefitted from your mentorship as a researcher, clinician, and human. Thank you to my committee members, Dr. Lajiness-O'Neill, Dr. Gindlesperger, and Dr. Breza. I have sincerely appreciated your questions, guidance, and feedback. Thank you to those additional neuropsychologists who have helped shape the course of my training: Dr. Brooklier, Dr. Rothermel, and Dr. Tan. Thank you for helping me believe in myself and find a community where I feel like I belong.

Lastly, thank you to all the tiny humans who were brave coming to the hospital and working with a new person, and who worked hard to complete a full battery of neuropsychological tests. I hope this research helps to advocate for access to services so you can shine bright and chase big dreams.

## Abstract

This study sought to broaden the findings of the current research on the relationship between late and moderate preterm (LAMP) birth and long-term neurocognitive outcomes—specifically those related to attention-deficit/hyperactivity disorder (ADHD). The purpose of this study was to better understand the relationship between gestational age (GA) and ADHD by (a) comparing prevalence of diagnosis between term-born and LAMP children, (b) comparing ADHD behavioral symptom severity between term-born and LAMP children, (c) and by examining neurocognitive status between term-born children and LAMP children (with and without ADHD diagnoses). The study also examined other factors that contribute to the relationship between GA and ADHD, including consideration of other risk factors and potential symptom-mitigating factors. The final sample for this study included 169 patients between the ages of 8 and 12 years who had completed an outpatient neuropsychological evaluation. GA was not related to ADHD diagnostic status, but lower GA predicted high ADHD-related symptom severity as indicated by caregiver report on the Conners-3. Though LAMP children did not differ from their term-born peers across measures of neurocognitive functioning, lower GA showed a marginally significant negative relationship with overall neurocognitive functioning (WISC-V FSIQ). Risk factors such as family history of ADHD and in-utero exposure to substances were consistently significantly related to ADHD symptom severity, and cumulative risk negatively impacted overall cognitive functioning, attention, working memory, and executive functioning. Adaptive skills and social skills were found to mitigate ADHD symptomatology as indicated by caregiver report on the BASC-3, though total symptom mitigating factors did not influence cognitive outcomes. Lastly, moderation analyses showed that gestational age interacts with birthweight at a marginally significant level in predicting overall cognitive functioning.

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## Chapter 1: Introduction

Attention-deficit/hyperactivity disorder (ADHD) is the most common disorder of childhood, affecting 10.2% of children in the United States (US; Carbray, 2018; Xu et al., 2018). One well known risk factor for ADHD is preterm birth (D'Onofrio et al., 2013). As preterm birth and ADHD share several risk factors, it is difficult to discern what variance in ADHD presentation (e.g., symptom severity, subtype, associated neurocognitive deficits) is accounted for by preterm birth, specifically, and not those shared risk factors. Furthermore, many extremely preterm and very preterm infants (at/before 25 weeks and 26 to 32 weeks, respectively) are born with significant medical complications that impact developmental outcomes; these additional medical complications substantially increase the variance in neurocognitive development, and therefore obscure the relationship between preterm birth status and ADHD.

By studying ADHD in late and moderate preterm (LAMP) infants (34 to 36 weeks, and 32 to 34 weeks, respectively), it may be possible to gain a clearer understanding of these infants' course of development and risk for ADHD. Though preterm infants are at substantially increased risk for developing ADHD, many do not grow up to be diagnosed with ADHD (multifinality) and many with ADHD do not have a history of preterm birth (equifinality). Therefore, we must examine risk factors that predict ADHD/ADHD-symptoms following LAMP to better understand the etiology of ADHD from a biopsychosocial perspective and consequently develop better interventions. The relationship between LAMP birth and ADHD can best be understood in the larger context of developmental psychopathology. Therefore, the present review will begin with a broad discussion of how developmental factors impact psychopathology, before discussing the specifics of ADHD as an outcome, and LAMP birth as a predisposing risk factor.

### **Theory of Developmental Psychopathology**

The principle of equifinality refers to nonlinear epigenesis, wherein multiple pathways can lead to the same outcome (Cicchetti & Rogosch, 1996). Stated another way, children with different risk and protective factors may arrive at a diagnosis of ADHD. The principle of multifinality suggests that the effect of an adverse event is dependent on the system in which the organism lives. The same pathway can result in different patterns of adaption or maladaptation (Cicchetti & Rogosch, 1996). Therefore, LAMP birth may or may not result in ADHD; the pathway from predictor/risk factor to outcome is an opportunity for intervention.

The concepts of equifinality and multifinality highlight behavioral and biological plasticity. Biological and psychosocial factors must be considered in the etiology of a particular outcome, in this case ADHD. The presence of one factor, such as preterm birth, is not in itself inextricably linked to the outcome of ADHD, and it may be possible to shift the course of development through intervention. By examining both ADHD and neuropsychological status as outcomes, research may be able to identify the totality of attributes associated with risk and other processes (i.e., symptom-mitigating factors) that shift the pathway between LAMP birth and childhood psychopathology (i.e., functional impairment)

### ***Psychopathology of Attention-Deficit/Hyperactivity Disorder***

ADHD is a high base rate neurodevelopmental disorder of childhood, affecting more than one in ten children in the US (Carbray, 2018; Xu et al., 2018). There has been a substantial increase in prevalence within the last two decades (Carbray, 2018) from 6.1% between 1997 and 1998, to 10.2% in 2015-2016 (Xu et al., 2018). The diagnosis of ADHD is based on a child's behavioral presentation across two or more settings. Though evaluation of underlying neurocognitive functioning is not required to make the diagnosis, neuropsychological assessment

is useful (if not essential) in better understanding a child's behavioral symptoms and informing appropriate supports. ADHD-related behaviors may be driven by external factors (environment), by internal factors (neurocognitive deficits in attention or related systems), or both, and an understanding of which behaviors can be attributed to which factors informs intervention strategies.

There are two main symptom dimensions along which a child's behaviors are assessed: (a) inattention and (b) hyperactivity/impulsivity (Boada et al., 2014). Meta-analytic research supports these two separate dimensions (Willcutt et al., 2012). Symptoms of inattention are characterized by disruptions in sustained attention and failure to attend to stimuli, whereas hyperactive/impulsive symptoms are characterized by behavioral dysregulation including disinhibition and motoric overflow. The full diagnostic criteria and examples of symptoms in each dimension, as taken from the *Diagnostic and Statistical Manual of Mental Disorders* (5<sup>th</sup> Ed.; DSM-5; American Psychiatric Association, 2013) is available in Appendix A. Based on these symptoms, the ADHD diagnosis is further specified into one of three subtypes: (a) combined type, (b) predominantly inattentive presentation, and (c) predominantly hyperactive/impulsive presentation.

There are observed differences among these subtypes. ADHD predominantly hyperactive/impulsive type has less genetic heritability and a lower prevalence of academic and cognitive impairments. ADHD combined presentation and predominantly inattentive presentation have comparable neuropsychological impairment, etiological influences, and intervention outcomes (Willcutt et al., 2012). Despite the differentiation of subtypes observed in childhood, they do not have longitudinal stability (Boada et al., 2014). Therefore, when

considering ADHD as an outcome across development, it may be more helpful to look at ADHD as a broad diagnostic indicator, rather than attempting to use subtypes.

**Long-Term Outcomes.** Children with ADHD are more likely to exhibit underachievement in academics, occupational outcomes, and social functioning (Barkley et al., 2006; Biederman et al., 2006; Franke et al., 2018; Galéra et al., 2009; Klein et al., 2012). There are certain prognostic factors that affect the course of ADHD, including long-term outcomes, symptom severity, and functional impairment. Female children are less likely to be diagnosed with ADHD, with the ratio of male to female diagnoses around 4:1 (Taylor et al., 2016). Research has also found gender and age differences in symptom dimensions, with males more commonly presenting as predominantly hyperactive or combined and females more commonly presenting as predominantly inattentive (DuPaul et al., 2016). Stress (e.g., family adversity) and parent disciplinary strategies (e.g., inconsistent parenting) both contribute to long-term outcomes (Sasser et al., 2016).

**Neural Mechanisms of ADHD.** There are many different theories of attention in typically developing individuals. The three main attentional systems proposed by Petersen and Posner (2012) include the alerting network, the orienting network, and an executive network. The alerting network includes brain stem arousal systems and right hemisphere systems related to sustained vigilance (Petersen & Posner, 2012). The orienting network includes the frontal and posterior areas that help an individual orient toward a prioritized sensory input in the presence of competing stimuli (winner take all); it also includes the parietal cortex involved in related processing and distinguishing between sensory versus motor processing (Petersen & Posner, 2012). The executive network involves the frontoparietal and cinguloopercular network. Though

the orienting network and executive network are distinct, they arise from the same origin in earlier development (Petersen & Posner, 2012).

In addition to these three systems, distinguished by their function, research supports another two anatomically and functionally distinct attention systems. There is the dorsal frontoparietal system, also referred to as the dorsal attention system, and the ventral frontoparietal system, referred to as the ventral attention system (Corbetta & Shulman, 2002). The dorsal attention system mediates top-down processes, with voluntary direction of attention to locations or features. The ventral attention system detects unattended or unexpected stimuli, helping the individual shift their attention toward the new stimuli. These two systems flexibly interact to enable a more dynamic control of attention for the purpose of both top-down goals and attention to bottom-up sensory stimulation (Vossel et al., 2014).

In the case of ADHD, the attentional systems are disrupted. These disruptions are evident in several regions and networks, most notably in the regions/systems associated with the prefrontal cortex (PFC). The PFC is the last area of the brain to reach maturation in early adulthood. However, the infrastructure and networks connected to and communicating with the PFC begin to develop in-utero. Thus, disruption of the gestational period in LAMP birth impacts early development of the PFC networks (Willcutt, 2010), and provide a neural foundational understanding of how preterm birth may operate as a risk factor for and predictor of ADHD.

The frontal-striatal networks, which include the thalamus, basal ganglia, and dorsolateral and ventrolateral regions of the PFC have also been widely studied in the ADHD literature (Willcutt, 2010). This dysfunction can be observed in reduced volume and activation among individuals with ADHD (Castellanos & Proal, 2012; Cubillo et al., 2012; Durston et al., 2011; Friedman & Rapoport, 2015; Makris et al., 2007; Ortiz et al., 2015; Seidman et al., 2005;



Seidman et al., 2006). Dysfunction of the frontal-striatal networks affects response selection, inhibition, maintenance, manipulation of information, and planning and organization of behavior.

Further, researchers have found that the orbitofrontal cortex may be implicated in the development of ADHD. More specifically, the orbitofrontal cortex includes those feedback loops of the ventromedial PFC, limbic structures, and other areas of PFC that play an important role in decision-making processes (Willcutt, 2010). Primate studies have shown that the ventromedial PFC neurons activated in prediction/response to reward impact learning and establishment of behavioral performance (Schultz, 2000). Studies of those with traumatic brain injury have found that damage to this region results in difficulty associating emotional valence to behavior based on feedback. Reduced left orbital PFC volume has also been observed among those with ADHD (Hesslinger et al., 2002). The orbitofrontal cortex and related feedback loops coordinate the interface between motivation/emotion and cognition, and therefore, damage to this region affects aversion to delay, learning from mistakes, and monitoring shifts in reward and punishment.

There is also evidence of dysfunction in related neural systems outside of the PFC. It is important to reiterate that a brain region does not work in isolation; rather, all the regions are interconnected and work together for functional processing. Persons with ADHD have been found to exhibit neurological differences in the anterior cingulate cortex, as functional magnetic resonance imaging (fMRI) studies indicate under-activation (Makris et al., 2007; Ortiz et al., 2015; Seidman et al., 2006). Functional magnetic resonance imaging (fMRI) is a non-invasive neuroimaging technique that utilizes changes in blood oxygenation level-dependent contrast as a measure of neural activity (Huettel, 2017), and provides some evidence for regional patterns of activity in the brain (Mather et al., 2013). These differences in the anterior cingulate cortex affect response selection and disrupt the central relay station for top-down and bottom-up processing.

The dorsal attention system is connected to the PFC and parietal region, and disruptions here affect associated cognitive processes such as attention and inhibition. The ventral attention system is connected to limbic structures, and disruptions here affect emotional processes.

The cerebellum has also been implicated as a mediator. Research studies evidence reduced volume and activation in those with ADHD (Cubillo et al., 2012; Durston et al., 2011; Makris et al., 2007; Ortiz et al., 2015; Schulz et al., 2004; Stoodley, 2014, 2016). Cerebellar under-activation affects temporal processes, such as processing speed, response to stimulus, and reaction time.

Lastly, caudate under-activation has been associated with childhood ADHD (Szekely et al., 2017). Studies using fMRI have found this deficit particularly pronounced for the right caudate, and more severe in ADHD combined type than inattentive subtype (Rubia, 2018). Performance differences have been observed between those with ADHD and controls and are evident on go/no go tasks (i.e., tests of response inhibition), suggesting that response inhibition is affected by hypoarousal of the caudate.

**Neuropsychological Constructs.** There are several neuropsychological constructs implicated in ADHD. The executive functions, which refer to higher order cognitive processes, are the most frequently identified neurocognitive deficits (Lezak et al., 2012; Mueller et al., 2010). The executive functions can be subdivided into more distinct and measurable constructs, including attention, cognitive flexibility, working memory, and inhibition. Attention, as a neurocognitive domain, is often subdivided into selective and sustained attention. Selective attention refers to attending to target stimulus over non-target stimulus (i.e., distractors); this neuropsychological construct corresponds to the ventral attention system. Sustained attention refers to continuous performance over time, without significant degradation of performance; it

corresponds to the dorsal attention system. Sustained attention is different from vigilance, which implies sustained attention that is specific to threats/dangers. Cognitive flexibility refers to the ability to switch between tasks without loss of performance. Working memory refers to the ability to hold and manipulate information in mind. Lastly, inhibition refers to the ability to suppress actions, including those that are automatic or overlearned, in favor of the desired response.

In addition to the executive functions, several other neurocognitive domains are impaired in children with ADHD. Aversion to delay reflects an individual's desire to decrease the amount of time spent waiting; it can be conceptualized as an inhibitory control problem or regulative deficit (Sonuga-Barke et al., 1992; Willcutt, 2010). It reflects the ADHD symptom dimension of impulsivity/hyperactivity. Functional analyses of hyperactive children's impulsivity during neuropsychological assessment can be understood as a function of delay aversion, both to pre-reward and post-reward delay, without regard for other economic constraints and reward conditions (Sonuga-Barke et al., 1992). In this regard, children with ADHD exhibit a diminished capacity to modulate their behavior in response to reward and punishment (Willcutt, 2010). Processing speed has also been found to be diminished in children with ADHD (Shanahan et al., 2006). Specifically, children with ADHD show greater response precision variability and deficits in short-duration temporal processing (Mueller et al., 2017; Willcutt, 2010).

**Neuropsychological Assessment of ADHD.** In keeping with best practice standards published by the American Academy of Pediatrics (AAP) regarding diagnosis and treatment of ADHD, a thorough evaluation is a necessary first step toward diagnosing ADHD. Though the AAP leaves evaluation open to clinical interpretation, cognitive testing is an extremely informative part of the evaluation, and often necessary to rule out other conditions that may

better account for attention-related symptoms (e.g., intellectual disability; Achenbach & Rescorla, 2000). Cognitive testing generally includes an estimate for overall intellectual quotient (IQ or *g*), and such measures become stable once children reach school-age (Bartels et al., 2002; Hoekstra et al., 2007). Notably, attention problems may interfere with a child's performance on IQ testing (Schweizer & Moosbrugger, 2004), in which case a clinician must be mindful of the attentional demands of a particular test, and may have to flexibly administer another test to ascertain overall IQ when that is necessary for drawing diagnostic conclusions.

In addition to accounting for a child's IQ, evaluations should include assessment of those neurocognitive domains associated with ADHD, including attention, working memory, executive functioning, and processing speed (Mahone & Schneider, 2012). Attention is a complex neurocognitive construct. In assessment, it is typically measured via selective and sustained attention tasks, such as continuous performance tasks that measure attentional performance over time. Scores on continuous performance tasks usually include omissions and commissions, which reportedly indicate inattention and impulsivity, respectively. However, other subtests included in the core subtest of the cognitive batteries also provide some indication of attentional problems. Attentional capacity can be determined by any test with a fleeting stimulus (either visual or auditory) that is presented once before the examinee is asked recall it.

Working memory is the ability to hold and manipulate information in the mind (Cowan & Alloway, 2009). Many working memory tests have been designed to assess components of Baddeley's multi-component model including the visuo-spatial sketch pad, the central executive system, the phonological loop, and the episodic buffer (A. Baddeley, 2000, 2003; Baddeley et al., 2011). Common measures of working memory require the individual to hold onto verbally presented information and reorder it (e.g., WISC Digit Span Backwards and Sequencing). When

asked to reorder backwards, the task requires spatial revisualization. When asked to sequence the stimulus by a familiar organizational framework (e.g., first in alphabetical and then numerical order), auditory acoustic memory is required.

Executive functioning broadly refers to higher order cognitive processes involved in formulating goals, planning, organization, and performance maintenance (Lezak et al., 2012). There are lower-level executive functions (e.g., sequencing and shifting/cognitive flexibility, inhibition, inhibition with switching) and higher-level executive functions (e.g., complex problem solving, ability to shift problem solving strategies based on real-time feedback). Lower level executive functions develop earlier, while higher-level executive functions continue to develop through adolescence (Best & Miller, 2010). Therefore, children with ADHD exhibit lower-level executive dysfunction.

Speed of information processing refers to the ability to process and respond to stimulus accurately. Measures of processing speed are different from tests of simple reaction time or visual discrimination because they require some component of cognitive decision making and allow for learning over time. Low speed of information processing may be indicative visual discrimination problems, distractibility, slowed decision making, motor difficulties, and/or generally slow cognitive speed (Wechsler, 2014).

**Risk Factors.** There are numerous etiological risk factors for ADHD, in addition to stress, especially among higher risk populations (e.g., racial/ethnic minority children, low socioeconomic status, religious minorities, sexual minorities). Genetic heritability is the strongest predictor of ADHD (Biederman, 2005; Du Rietz et al., 2018; Faraone et al., 2005; Franke et al., 2012; Leung & Hon, 2016). Following this risk factor, within the prenatal environment, intrauterine exposure to substances such as alcohol is a significant risk factor for

ADHD. Additionally, there is substantial substance use in the USA during pregnancy (Franke et al., 2018; Knopik et al., 2018; Spiers et al., 2015). Tobacco, alcohol, cannabis, and other illicit substances are the most common substances (Forray & Foster, 2015; Knopik et al., 2018).

Tobacco use during pregnancy and prenatal alcohol exposure (PAE), which may lead to Fetal Alcohol Syndrome (FASD), are strong predictors of psychiatric and emotional problems, school disruptions, legal difficulties, and behavioral problems (Williams & Smith, 2015). In-utero exposure to substances substantially increases the risk for negative neurocognitive outcomes and behavioral problems including ADHD (Tsang et al., 2016).

Furthermore, it is crucial to consider postnatal factors such as environmental exposures. Lower socioeconomic status (SES) poses a risk for negative outcomes and may be associated with increased likelihood of exposure to teratogens (e.g., lead), infections (e.g., encephalitis), abuse/neglect, an unstable home environment, maternal smoking, nutritional factors, and parental psychopathology (American Psychiatric Association, 2013; Braun et al., 2006; Humphreys et al., 2018; Russell et al., 2014).

One of the strongest perinatal risk factors for ADHD is preterm birth. Those who are born prematurely show a two- to three-fold risk of developing ADHD (American Psychiatric Association, 2013). Research has demonstrated a dose-response relationship between early gestation and psychiatric morbidity, specifically ADHD (D'Onofrio et al., 2013). This relationship was independent of familial confounds (e.g., age at first parenthood, marital status, social welfare recipient) and child (e.g., sex, birth order, year of birth) and parent (e.g., age at index child's birth, education level, history of criminal conviction) covariates consistent with causal inference (D'Onofrio et al., 2013). Prematurity is a unique risk factor for ADHD even after accounting for other risk factors.

**Symptom-Mitigating Factors.** Children with ADHD present with a sequela of symptoms. In addition to behavioral symptoms (inattention, hyperactivity, impulsivity) and neurocognitive deficits in attention (including attention, working memory, executive functioning, and processing speed), children with ADHD are more likely to present with social skills deficits and adaptive behavior deficits (Deboo & Prins, 2007; Lindblad et al., 2013; Staikova et al., 2013). Social skills and adaptive behaviors can be learned naturalistically, as well as through evidence-based interventions. Therefore, it is worth considering that bolstering these skills areas may help to reduce the overall ADHD-related symptom burden and may be conceptualized as symptom-mitigating factors. It may be the case that those children who have ADHD-related neurocognitive deficits remain subthreshold for a diagnosis because they are able to utilize peer relationships and adaptive behavioral strategies to minimize functional impairment caused by their ADHD.

**Diversity Issues.** There are numerous diversity issues related to ADHD. Firstly, the literature shows that stress negatively impacts cognition (Mueller et al., 2010). Secondly, greater life stress is associated with a higher ADHD symptom report, which suggests that stress exacerbates ADHD symptomatology (Sasser et al., 2016). Given that minorities experience a greater amount of stress (S. Cohen & Janicki-Deverts, 2012), it logically follows that minorities are at an increased risk for decrements to their cognitive performance and attentional interference. Research has shown that African American children receive higher teacher ratings of attention problems and are also less likely to receive treatment (DuPaul et al., 2016; Willcutt, 2010). It may be the case that minority children with ADHD experience symptom exacerbation as a function of increased life stress, leading to higher ratings of attention problems. It is also possible that minority children are more likely to be pathologized. This is a very significant

problem when using informant reports of behavior as the foundation of evidence for a diagnosis, such as in the case of ADHD.

### ***Psychopathology of Late and Moderate Preterm (LAMP) Birth***

Late and moderate preterm birth are high base rate occurrences, and preterm status is a known risk factor for ADHD. Preterm birth is a significant problem, with LAMP occurring in nearly 10% of babies born in the US (7.99% and 1.48% late and moderate preterm, respectively); these rates are lower among singletons (6.99% and 1.18% late and moderate preterm, respectively; Martin et al., 2015). Approximately 71% of all preterm births (GA < 37 weeks) fall in the late preterm category (Martin et al., 2015), and greater than 80% of all preterm births are LAMP (GA 32 to 36 weeks; Howson et al., 2012). LAMP infants represent a significant population among the general population and the vast majority within the preterm population. There has been a more recent wave of literature devoted to LAMP infants, who represent most of all preterm births. The proposed study seeks to better understand the risk and symptom-mitigating factors that determine the pathogenesis from LAMP birth to ADHD; extremely preterm and very preterm infants (those born before 32 weeks' gestation) will be excluded owing to the high prevalence of medical comorbidity and complexity in these groups.

**Definitions.** Preterm birth refers to babies born before 37 weeks of gestation; it can be further subdivided into extremely preterm (at/before 25 weeks), very preterm (less than 32 weeks), moderately preterm (32 to 33 weeks), and late preterm (34 to 36 weeks; Mayo Foundation for Medical Education and Research, 2017). Late and moderate preterm are often grouped together and may collectively be referred to as LAMP infants (Howson et al., 2012; Menon, 2008). Full-term babies are also subdivided into early term (37 to 38 weeks), full term



(39 to 40 weeks), late term (41 weeks), and post term (at or after 42 weeks; March of Dimes, 2018).

Gestational age (GA) and birth weight are closely related, and birth weight is sometimes used as a proxy for GA (Taylor, 2010). Essentially, low birth weight (LBW) suggests prematurity. LBW is less than 2500g (World Health Organization, 2014). Much like preterm birth can be categorized into subgroups, so too can low birth weight: very low birth weight (VLBW) is less than 1,500 grams and extremely low birth weight (ELBW) is less than 1,000 grams. However, LBW can be indicative of preterm birth, intrauterine growth restriction/fetal growth restriction, or both (Cutland et al., 2017). LBW may represent a construct other than GA, and consequently, this study is most interest in LAMP status determined by GA rather than as indicated by birth weight. Nevertheless, this study examined the interactive effect of LBW on GA in determining ADHD status.

**Outcomes Following LAMP Birth.** Though LAMP infant survival rates have improved over the last decade with medical advances, late preterm death still accounts for 10-15% of global neonatal deaths (Osrin, 2010). The incidence of early and late neonatal death is 2.8 per 1000 among LAMP compared to 0.4 per 1000 in term-born controls (Bonnevier et al., 2018). LAMP infants are at higher risk for respiratory distress, transient tachypnea, intraventricular hemorrhage, bacterial sepsis, feeding problems, neonatal intensive care unit (NICU) admission, and death (Shapiro-Mendoza & Lackritz, 2012). Hypoglycemia, jaundice, temperature instability, apnea, and the need for intravenous infusion are also common (Engle et al., 2007). The brain develops in a caudal-to-rostral direction, and therefore, frontal (especially prefrontal cortex) regions and networks connected to these regions, which are last to develop, are negatively impacted by a shortened gestational period (Kolb et al., 2012). LAMP infants that

survive infancy go on to show differences in their neurodevelopment compared to full term infants. These children show higher rates of neuromotor/sensory impairment, including hearing, vision, and gross motor impairments. They also have higher rates of neurodevelopmental disability status (Johnson et al., 2015). By school age, there are differences in LAMP children's academic achievement. They show slower language acquisition and social-emotional development (Johnson et al., 2018) compared to their same-aged peers.

During the course of development, neuropsychological differences become evident, and these deficits become measurable. The development of attention networks is disrupted in preterm birth (Ball et al., 2014; Rommel et al., 2017; van den Heuvel et al., 2015). These children show a higher prevalence of cognitive impairments, including nonverbal and expressive language deficits (Johnson et al., 2015). They exhibit statistically significant differences on performance-based measures (administered at age 7-years) of intellectual functioning, visuospatial reasoning, attention control, and inhibition (Cserjesi et al., 2012; Kerstjens, 2013). In addition to differences in neuropsychological status, there are also differences in behavioral functioning. The parents of LAMP children report increased problems with executive functioning and behavioral regulation compared to parents of term-born children (Cserjesi et al., 2012; Kerstjens, 2013). Taken together, there are consistent brain-behavior deficits associated with LAMP status that would predispose children to ADHD symptomatology (e.g., problems with attention and inhibition).

**Risk Factors.** There are numerous risk factors for LAMP, many of which have an impact on outcome after birth. Prenatal risk factors for spontaneous preterm birth include maternal age, pregnancy spacing (e.g., short interval and long interval between births), multiple pregnancy, infection (e.g., STIs, HIV/AIDS, Rubella), underlying maternal chronic health condition (e.g., diabetes, hypertension, anemia), nutrition (e.g., folic acid and iron deficiencies), stress (e.g.,

working more than 5-days/week, heavy lifting), maternal psychological health (especially depression), trauma exposure (e.g., intimate partner violence), substance use (e.g., tobacco, alcohol, illicit substances), and genetic heritability (Howson et al., 2012; Shapiro-Mendoza & Lackritz, 2012). The most common causes of late preterm births are preterm premature rupture of the membranes (PPROM), hypertensive diseases, pre-gestational diabetes, and placental disorders (Bonnevier et al., 2018). Notably, many of the prenatal risk factors for LAMP birth reflect the health disparities prevalent among low socioeconomic status (SES) and ethnic/racial minorities in the US.

While two-thirds of preterm births are spontaneous, one-third are medically indicated (Shapiro-Mendoza & Lackritz, 2012). Perinatal risk factors observed in provider-initiated preterm birth include obstetric indication (labor arrest, multiple gestation, fetal malposition), fetal indication (fetal heart rate, suspected fetal macrosomia), or another not medically indicated reason, such as elective caesarian (Barber et al., 2011; Howson et al., 2012). The latter—elective cesarean—is becoming an increasingly common practice (Davis-Floyd, 2007). The culture in the US is particularly problematic, as the rate of physician-initiated elective caesarians are higher than other developed nations, often resulting in LAMP birth (Morris, 2016; Rosenberg & Trevathan, 2018).

Once a child is born LAMP, there are certain prognostic factors that would predict severity of consequent development. Predictors of a very preterm phenotype (characterized by early delays and school-aged deficits in cognitive processing, attention, social/emotional functioning, and autism spectrum disorder-like symptoms) in LAMP children include preeclampsia during pregnancy and being male (Johnson et al., 2018). Other studies show that by age 7-years, preterm boys have regressed to the mean, catching up to their full term birth peers,

whereas preterm girls lag behind their peers (Cserjesi et al., 2012; Kerstjens, 2013). Therefore, the role of gender remains unclear, though existing research suggests that its role is dynamic and changes over the course of development. SES is another prognostic factor, contributing to an increased likelihood of developmental delay above and beyond the risk accounted for by moderate preterm birth itself (Potijk et al., 2013).

**Diversity Issues.** There are numerous diversity issues in both the occurrence of LAMP birth and its long-term outcomes. As previously mentioned, many of the prenatal risk factors for LAMP birth are disproportionately present among low income and racial/ethnic minorities. Consequently, a significant racial disparity exists in the rates of preterm birth; African Americans have two- to three-fold risk compared to Caucasians (Menon, 2008). This disparity is not fully attributable to factors such as SES, maternal behavior (e.g., in-utero exposure to tobacco, alcohol, drugs), age, gravidity, marital status, education, or income (Menon, 2008). Rather, the etiology of LAMP birth is likely multifactorial, with contributions from biological (e.g., genetic, nutrition) and psychosocial factors (e.g., environmental risk factors, SES), that interact in a complex system. SES, as previously mentioned, is both a risk for LAMP and impacts the prognosis of LAMP born children. This is yet another reflection of the ways in which decades of institutional racism have resulted in health disparity and differential access and quality of care and intervention services.

Notably, gestational age cutoffs used to define prematurity are arbitrary. There is substantial evidence to suggest that the 37-week cutoff should be raised; children born close to term, as it is currently defined, show poorer outcomes than do children born closer to 40 weeks' gestation (Goldenberg et al., 2012). There is significant evidence to suggest that studying the long-term outcomes of LAMP infants is crucial, as this group accounts for the highest percentage

of preterm births and their long-term outcomes shed light on the role of late gestation on development. Furthermore, full-term subjects should be assigned more discrete group membership. Since the variables of interest in the present study revolve around the last weeks of gestation, the late and moderate preterm born children will be examined as the two experimental groups, and early and full term born children will be examined as the control groups.

## Chapter 2: LAMP and ADHD Research

Though recent research has focused on LAMP infants, findings have been mixed regarding the relationship between LAMP status and longer-term outcomes such as ADHD and associated delays and deficits. A closer examination of the literature highlights some of the strengths in the existing studies, and the limitations of others, which make it difficult to conclusively remark on the attention capacity of LAMP born infants by school-age.

### **Research Concluding No Difference in ADHD Outcomes Among LAMP Children**

Rabie et al. (2015) conducted a retrospective study to examine the neurodevelopmental outcomes (specifically ADHD and/or developmental speech and language disorders, as defined by the ICD-9CM) among late preterm children as compared to early term and term born controls via a review of the Medicaid record among 3,270 late preterm infants and 24,005 term infants. Gurka et al. (2010) conducted a prospective study to compare late preterm infants and full-term controls from the age of 4 years to 15 years on measures of cognition, achievement, socio-emotional, and behavioral outcomes among 1,298 children (53 of which were born late preterm). Both studies concluded that there were no differences between LAMP children and term born controls on outcomes related to ADHD. There are several explanations for these findings outlined below, with suggestions for how the problems will be addressed in the present study.

**Study Designs.** Gurka et al. (2010) observed children from birth through the age of 15 years. Data collection took place at age 54 months, and continued yearly from kindergarten through the sixth grade, with follow-up at the age of 15 years. The strength of longitudinal work such as this is that data is collected across development and can capture the changes associated with development as well as intervention. However, given that it is more difficult to recruit participants who are willing to participate long-term, the Gurka et al. (2010) study only included

53 late preterm participants. Gurka et al. (2010) did not perform power analyses; rather, they chose to provide a range of possible differences between groups based on confidence intervals to conclude whether the suggestion of equivalence between groups was appropriate. While provision of confidence intervals was a valuable redress, overall, the study was underpowered (preterm group  $N = 53$ ). The present study was much better powered and able to detect medium effect sizes.

Rabie et al. (2015) utilized archival data with variables that were not designed for psychological research. Specifically, they determined ADHD by ICD-9 code as an outcome based on review of the Medicaid record. However, diagnostic codes by insurance record are subject to error and bias and may not represent the child's full medical record. That the variables utilized in the study were not designed for research, nor were they comprehensive clinical indicators, is a weakness of this retrospective research design. The present study improves upon this point by utilizing diagnoses formulated following a comprehensive neuropsychological evaluation (rather than diagnostic codes used in billing) and performance-based measures of neurocognition.

Subjects for both studies were sorted into clinical and control groups based on gestational age, but classification of preterm status varied by study. Measurement of and classification based on gestational age can be accomplished several ways, and each way has its own strengths and weaknesses. Rabie et al. (2015) used GA indicated on birth certificates. This methodology is highly accurate, though difficult in retrospective studies of older children who are more likely to receive care in hospitals other than the one they were born in. Gurka et al. (2010) determined GA using birthdate and due date, as reported by parents; this is potentially problematic owing to parental error in report. However, this is a common method, requiring less effort in data

collection. The present study will utilize gestational age based on parent report, but it will also review the electronic medical record (EMR) for the hospital-recorded GA of those children who were born within an identified hospital system in a large metropolitan area as a secondary means for examining accuracy of parent-reported GA.

Many studies look at full-term children without regard for their gestational age. Rather than subsuming “full-term” into one heterogeneous group, it is important to look at smaller, incremental groupings. Rabie et al. (2015) addressed this issue and provided two term groups: early term (37 to 38 weeks’ gestation) and term born (39 to 41 weeks’ gestation), which allowed for multiple control group comparisons to the late preterm group. However, the study failed to include children born moderate preterm. As noted previously, each week of gestation plays an important role in neural development. Consequently, inclusion of discrete groups is an important step in better understanding the long-term outcomes of shortened gestation. The present study will include Moderately preterm (32 to < 34 weeks), Late preterm (34 to < 37 weeks), Early term (37 to < 39 weeks), and Full term (39 to 40 weeks), with the preterm children collectively referred to as LAMP children, and the full-term children collectively referred to as term-born children.

Lastly, the time of diagnostic review changes prevalence rates of ADHD among LAMP children. Rabie et al. (2015) chose to include subjects between the ages of 3 and 5 years. This is extremely problematic as most children in their sample who would later be diagnosed with ADHD were not captured in their analyses. Diagnoses for ADHD are most often made after formal schooling has begun, when the child has had time to build instructional control and adjust to the behavioral routine of full-time schooling, and informants (e.g., parents, teachers) are able to compare the child’s behavior to other similarly-aged children (Evans et al., 2010). The present



study improves upon this by examining children in middle childhood who are more likely to have exhibited ADHD-related deficits relative to their peers.

**Inclusion/Exclusion Criteria.** Rabie et al. (2015) excluded subjects if they were part of multiple births, were small or large for gestational age, and/or had a congenital anomaly. This is problematic as a high proportion of LAMP infants are from multiple births. “Congenital anomaly” is not specifically defined and may have included cosmetic congenital anomalies that would not otherwise impact neurocognitive development or have subsequent impact on neuropsychological status/ADHD as an outcome. Rabie et al. (2015) also failed to exclude participants on the basis of IQ. The amount of variance in neurocognitive presentation accounted for by an extreme departure from the mean IQ of 100, as is the case in intellectual disability (generally indicated by an IQ below a standard score of 70), can significantly skew the results. The present study allowed for multiple birth children and excluded those children with intellectual disability (as diagnosed following comprehensive neuropsychological evaluation).

Gurka et al. (2010) also utilized a somewhat biased sample; the exclusion criteria stated that the family had to live within one hour of the research site, which limited participants to those who lived within metro city limits. Furthermore, it was stipulated that participants had to live in neighborhoods that were sufficiently safe for researchers to visit, though how this was determined is unclear and clearly limits higher risk participant inclusion. Additionally, the families were told not to participate if they planned to move anytime in the next three years. There were additional exclusion criteria, including maternal and infant health factors, largely related to SES and racial health disparity, maternal substance use, significant maternal or infant illness, and greater than seven-day hospital stay at birth. Taken together, the exclusion criteria limited the sample population so that those who were recruited had a higher SES and had higher

educational levels. The sample lacked diversity in racial backgrounds and did not include a rural representation. Therefore, the conclusions drawn from this study have extremely limited external validity. Gurka et al. found that the children born late-preterm showed no differences to the children born full-term in all examined child and family characteristics. This is likely an artifact of the biased sample rather than a reflection of the general population in the USA, in which many of the exclusion criteria are factors disproportionately represented among babies born LAMP and children with ADHD.

Rabie et al. (2015) utilized all Medicaid subjects in the review, but a Medicaid-based population is more likely to have poorer obstetric outcomes, lower SES, and significant drop off in Medicaid enrollment compared to mixed insurance samples (i.e., a sample that would have included private insurance). These types of confounds and biases in the data need to be statistically redressed. Specifically, poorer obstetric outcomes are their own risk factor in the development of ADHD. Lower SES may restrict access to intervention services and enrichment opportunities that could serve as factors that mitigate symptom severity or functional impairment associated with ADHD. Drop-out in Medicaid enrollment leads to attrition bias and limits statistical power. The present study includes both singletons and non-singletons from a highly diverse population.

**Variables.** Regarding measurement error in criterion variables, Gurka et al. (2010) measured cognition and achievement using subtests from the Woodcock-Johnson Psycho-Educational battery (Rev. ed.; Picture Vocabulary, Passage comprehension, Letter-Word Identification, and Applied Problems); however, the vast majority of these are related to academic achievement rather than cognition. Although Picture Vocabulary, which measures expressive fund of word knowledge, provides, at best, an estimate of overall IQ, it cannot

provide an indication of strengths and weaknesses in the cognitive profile. Thus, Gurka et al. (2010) fails to provide information regarding the cognitive domains of interest—those related to ADHD and those which would be measurable in a sample of school-aged children. The present study utilized testing instruments that were better attuned to neurocognitive functioning, specifically those that captured weaknesses associated with ADHD.

Regarding behavioral symptoms, Gurka et al. (2010) used the Child Behavior Checklist (CBCL) scales to generate standardized externalizing, internalizing, aggression, anxiety/depression scores based on parent report (Achenbach & Rescorla, 2001). However, they did not utilize other informants who primarily observed symptoms and functioning in settings outside the home (i.e., teacher-report of symptoms in the school setting), nor did they utilize any attention-specific measures (i.e., those that would be more sensitive to ADHD symptomatology). The present study utilized several behavioral reports, including one which specifically targeted attention-related impairment.

Rabie et al. (2015) controlled for alcohol abuse and preeclampsia as indicated in the child's Medicaid claims file, without regard for other risk factors predictive of both LAMP and ADHD and without capturing factors the parents might have otherwise reported but were not documented in the Medicaid medical record. The present study will report on multiple prenatal and perinatal risk factors queried for in the evaluation (e.g., in-utero exposure to alcohol, tobacco, illicit substances, prescription drugs, gestational diabetes, high blood pressure/toxemia, infections, placenta abruptia, placenta previa, pre-eclampsia, maternal chronic illness, and major life stress).

Additionally, several factors impact the perceived relationship between the predictor and criterion variables. Many studies encounter the third variable problem—failure to consider

moderators and/or mediators—and this may influence observed variables and cause them to covary when in fact there is no direct relationship (Bordens & Abbott, 2018). Neither Rabie et al. (2015) nor Gurka et al. (2010) included any moderators or mediators. The present study looked at early intervention services and symptom-mitigating factors (e.g., social skills and adaptive skills) as potential moderators between LAMP and ADHD/ADHD-related outcomes.

### **Research Finding Differences in ADHD Outcomes Among LAMP Children**

Johnson et al. (2015) conducted a prospective study to examine neurodevelopmental outcomes (specifically neurosensory and cognitive impairment) among LAMP children as compared to term born controls via parent report among 638 LAMP children and 765 term infants. Similarly, Sucksdorff et al. (2015) conducted a prospective study to examine GA as a predictor for ICD ADHD diagnosis as indicated in the Finnish Medical Birth Register among 10,321 children with ADHD and 38,355 controls. Further, Rommel et al. (2017) and James et al. (2018) conducted studies to examine ADHD symptoms and related cognitive impairments between preterm-born children (N = 186), term-born children with ADHD (N = 69), and term controls without ADHD (N = 135) via electroneurodiagnostics (specifically event related potentials, or ERPs), skin conductance levels, and cued performance tasks. These studies all concluded that there were statistically significant differences between LAMP children and term born controls in early neurodevelopmental delays, higher rates and greater severity of ADHD, ADHD associated neurocognitive deficits, and greater functional impairment (James et al., 2018; Johnson et al., 2015; Rommel et al., 2017; Sucksdorff et al., 2015). These study designs, subject selection, and measurement techniques provided a roadmap for the design of the current study.

**Study Design.** The age of study participants impacted outcome data for many of the studies reviewed previously. Rommel et al. (2017) reviewed the differences in prevalence of

ADHD, ADHD-like symptoms, and related neurocognitive deficits among three different groups of adolescents: those born preterm, those born full-term but diagnosed with ADHD, and those born full-term without ADHD (who served as controls). The time of diagnostic review utilized in the Rommel et al. (2017) study is likely accurate, as measurement in adolescence is more likely to capture ADHD prevalence than when prevalence is measured in early childhood. ADHD diagnoses likely have been made by adolescence and the associated neurocognitive deficits can reliably be measured. Notably, the prefrontal cortex continues to develop into early adulthood, and thus it is possible that the neurocognitive presentation and/or deficits associated with ADHD, including functional and adaptive deficits, may further change and present differently later in development. This is one of the reasons that the present study examined outcomes in middle childhood when neurocognitive functioning can be reliably measured, and prevalence estimates are stable.

Sucksdorff et al. (2015) captured ADHD diagnosis over time, tracking subjects through childhood, and required that diagnoses be given based on the International Classification of Diseases (ICD-10; World Health Organization, 1992). Notably, the ICD-10 is a commonly used diagnostic manual favored by European countries, and classifies ADHD as “hyperkinetic disorder,” including symptoms of over activity, poor modulation of behavior, inattention, lack of persistent task involvement that persists across settings and over time and is evident in early childhood (World Health Organization, 1992). Sucksdorff et al.’s (2015) findings would be stronger if they were replicated using *DSM-5* criteria to increase reliability and external validity in the US, which generally favors the *DSM-5*. Notably, differences in prevalence estimates worldwide suggests that the methodological criterion used to determine diagnosis accounts for significant variance (Levy, 2014). More specifically, though the ICD and *DSM* provide similar

lists of symptoms, the ICD has been associated with lower prevalence rates as symptoms must reach a certain threshold of severity in all dimensions whereas the *DSM-5* requires that symptoms be present across two of more settings (American Psychiatric Association, 2013; Levy, 2014). The *DSM-5* criteria for ADHD was used in this research study in order to maximize generalizability of findings within a US population.

Control groups varied for many of the studies examining GA and ADHD. Sucksdorff et al. (2015) matched children with ADHD to four controls groups based on birthdate ( $\pm 30$  days), gender, and child birthplace. Controls were excluded if they had a diagnosis of ADHD, conduct disorder, or severe or profound intellectual disability. By matching children with ADHD to controls on these criteria, those characteristics can be eliminated as potential confounds that could mistakenly lead to group differences in the variables of interest. Rather than utilizing a matched-groups design, other studies have recruited control groups solely based on “full-term” status, defined by a GA above 37 weeks. However, this can be problematic as there is data showing that early term infants, those born 37 to 39 weeks’ GA, are also at higher risk for developing neurological problems compared to those born at 40 weeks’ GA (Johnson et al., 2015). The present study examined discrete groups of preterm and full-term children and utilized control variables, rather than a matched-groups design.

Johnson et al. (2015) and Sucksdorff et al. (2018) were well powered studies (LAMP  $N = 1,130$  and ADHD  $N = 10,321$ , respectively). Rommel et al. (2017) and James et al. (2018) were smaller studies that were also able to detect differences in ADHD-related outcomes among LAMP children. The present study is sufficiently powered and could detect large effect sizes.

Rommel et al. (2017) and James et al. (2018) required medical record verification of GA. Sucksdorff et al. (2015) determined gestational age from last menstrual period and verified the

GA using first trimester ultrasound results. Though the present study relied on parent-reported GA, the medical record was also reviewed as a means of secondary assessment. Gestational age is often used as a continuous variable, with groupings based on recommended classification (Sucksdorff et al., 2015). However, using GA as a continuous variable among preterm children can be problematic. Rommel et al. 's (2017) standard deviation for GA among preterm children was approximately 3 weeks—a significant period for gestational age; tighter grouping/more stringent GA criteria would have been more useful. Groupings can be made using other strategies. For example, Johnson et al. (2015) created a LAMP group (32 to < 37 weeks' gestation), without dividing moderate from late preterm infants. The present study examined discrete groups of preterm and full-term children, as well as compare the overall sample of LAMP to term-born children.

Studies conducted by Rommel et al. (2017) and James et al. (2018) used a primarily White population from England, which is very different from racial composition in the US. A predominantly White sample very much limits the generalizability of findings and is particularly problematic for drawing conclusions regarding US preterm children, many of whom are racially diverse. The present sample successfully gathered data from racially/ethnically diverse patient population by utilizing data from a healthcare system in a major Metropolitan area in the US.

**Inclusion/Exclusion Criteria.** Johnson et al. (2015) excluded subjects with major congenital anomalies, though it was unclear whether these anomalies were those that impact cognitive development. Rommel et al. (2017) excluded subjects with an IQ below a standard score of 70, and those with general learning difficulties, cerebral palsy, epilepsy, medical conditions affecting motor coordination, and brain disorders and genetic or medical disorders that might mimic ADHD. This exclusion criteria (learning difficulties, specifically) limits

variance in neurocognitive outcomes and is problematic as many children with ADHD have comorbid specific learning disorders (Efron et al., 2016). The present study includes exclusion criteria that functions to eliminate biases (e.g., ID) and other conditions that could account for the relationship of interest (e.g., brain disorders and/or medical conditions that mimic ADHD or directly impact attentional capacity), without diminishing expected variance in outcome due to learning disabilities.

**Variables.** Maternal age, maternal substance abuse, maternal psychiatric history, in-utero exposure to tobacco, gravidity, marital status, paternal age, and urbanity of the child's birthplace have all been shown to have an association with preterm birth (GA) and ADHD (Sucksdorff et al., 2015). Sucksdorff et al. (2015) chose to examine confounders in relationship to preterm birth, weight for gestational age, and ADHD, while controlling for factors related to the primary predictor, secondary predictor, and criterion variable. Maternal SES and paternal psychiatric history and immigrant status were additional confounds in predicting ADHD. After adjusting for all confounds, premature birth remained a risk factor for ADHD. Rommel et al. (2017) did not control for risk factors of preterm birth (e.g., malnutrition, low SES), which may covary with associated neurocognitive deficits, or serve as moderators or mediators, especially by adolescence (i.e., cumulative risk over time).

As noted above, there can also be measurement error in the criterion variables. Rommel et al. (2017) used the Diagnostic Interview for ADHD in Adults (DIVA) to determine ADHD status. Notably, this measure was normed for adults, though the study administered it to adolescents, meaning that the tool may not have been appropriately sensitive to the symptoms that present in adolescence. Johnson et al. (2015) utilized measures specific to cognitive ability and, given that the outcome of interest was neurodevelopmental disability in early childhood,



measures of more distinct cognitive domains were not required. The present study utilized tools created for measuring neurocognition (including specific domains of interest) and behavior in the target age-range.

### **Summary of Prior Research**

ADHD and LAMP birth are both commonly occurring conditions. LAMP is a risk factor for ADHD, but there are numerous biopsychosocial factors that contribute to this outcome. There are identifiable biological disruptions in prematurity that relate to the observed disruptions in attentional capacity associated with ADHD. There are also psychosocial factors that relate these two conditions, including shared risk and prognostic factors. Yet there are many children born LAMP who do not go on to develop ADHD. The multiplicity of outcomes among LAMP children is captured in the existing body of literature. Though recent research has focused on LAMP infants, findings have been mixed regarding the relationship between LAMP status and longer-term outcomes such as ADHD and associated delays and deficits. Per review of the recent relevant literature regarding outcomes following LAMP birth, including ADHD and ADHD-related impairment, there is stronger evidence to support higher prevalence, greater symptom severity, and higher functional impairment among those children born LAMP than term-born controls (James et al., 2018; Johnson et al., 2015; Rommel et al., 2017; Sucksdorff et al., 2015).

The research that contradicts this conclusion has significant limitations, most notably procedural timing, elements of bias (e.g., subject population), and poorly selected assessment tools, that threaten validity and interpretive value (Gurka et al., 2010; Rabie et al., 2015). Prior studies have largely examined gestalt measures of ADHD (diagnosis by history or by research determination) or cognitive functioning (IQ). However, this does little to provide information regarding the underlying neurocognitive deficits associated with both LAMP birth and ADHD.

These components are crucial in the understanding of decrements to overall functioning that contribute to the functional impairment that warrants a diagnosis.

Taken together, it is crucial to understand what factors predict resilience in this high risk, high base-rate group of LAMP children, in the hope that behavioral and biological plasticity will prevail, and that these children will adapt and not develop impairments that warrant an ADHD diagnosis. Identifying and understanding the biopsychosocial relationship between ADHD/ADHD-related neurocognitive outcomes and LAMP birth is a crucial first step. The purpose of the present study was to examine (a) ADHD and associated neurocognitive deficits among full-term and LAMP born children, (b) which factors predict improved neurocognition among high-risk LAMP born children, and (c) which factors are the strongest predictor(s) of an ADHD diagnosis among LAMP children. By gaining a better understanding of the risk and symptom-mitigating factors that may alter the diagnostic status (functional impairment) and symptom severity among LAMP children at school-age, early intervention services can be better tailored to this population who are at risk for attentional deficits.

### **Hypotheses**

The study contributes to the existing literature on LAMP and ADHD in several ways. Firstly, it provided updated statistics in prevalence rates among more discrete groups of preterm (LAMP) and full-term born children. Secondly, it examined ADHD as the heterogeneous outcome that it is—a diagnostic label, behavioral presentation (i.e., symptom severity), and distinct neurocognitive deficits. Thirdly, it utilized patient history as well as behavioral reports to ascertain which risk and symptom-mitigating factors further illuminate the relationship between GA and ADHD outcomes.

The present study utilized a school-age population, when neurocognitive processes, including the executive functions, can be measured reliably. De-identified patient data were collected at a major hospital system in a large metropolitan area in the Midwest and included children between the ages of 8 and 12 years. Efforts were made to include an equal number of male and female children in the analyses, with a goal of 200 children total (100 LAMP children with approximately 50 moderate preterm and 50 late preterm, and 100 term-born children with approximately 50 early term and 50 full-term).

Overall neurocognitive functioning ( $g$  or IQ) was measured using the FSIQ of the WISC-V. Discrete measures of neurocognitive functioning were taken across the following domains: sustained attention, specifically brief attention, inattention and impulsivity; working memory, specifically spatial revisualization and auditory acoustic memory; executive functioning, specifically cognitive flexibility and inhibition; processing speed, specifically speeded visuomotor integration and visual attention and matching. These measures capture those neuropsychological deficits associated with ADHD (Mahone & Schneider, 2012). Diagnostic status (i.e., ADHD or other diagnosis) were taken from the medical record. Behavioral reports from parents were utilized as an indication of symptom severity (impulsivity, hyperactivity, inattention). Risk factors (e.g., race, Medicaid status, parent educational level, genetic heritability, and prenatal exposure to substances) were taken from the medical record. Environmental factors that predict better outcomes were also examined. Firstly, history of early intervention services was taken from the patient history form. Secondly, given that ADHD is also related to social skills and adaptive behavior deficits (Deboo & Prins, 2007; Lindblad et al., 2013; Staikova et al., 2013), these skill areas may function as symptom-mitigating factors that reduce functional impairment and thus the likelihood of diagnosis, as well as symptom severity.

Symptom-mitigating factors (e.g., social skills and adaptive skills) were captured in the behavioral reports. Table 1. provides an overview of all predictor and outcome variables. The following text describes the hypothesized relationships between these variables.

**Table 1***Summary of Variables Included in Primary Analyses*

Predictors	Outcomes
Gestational age	ADHD diagnosis (as determined by neuropsychologist)
GA (weeks, by caregiver report)	Symptom severity (by caregiver report on behavior rating forms)
LAMP (yes/no)	Attention/Inattention (BASC-3; Conners-3)
Risk factors (by caregiver report)	Hyperactivity/Impulsivity (BASC-3; Conners-3)
Minority status (yes/no)	DSM ADHD Inattentive Symptoms (Conners-3)
Lack of caregiver higher level education (yes/no)	DSM ADHD Hyperactive/Impulsive Symptoms (Conners-3)
Medicaid status (yes/no)	Neurocognitive functioning (performance-based measures)
Genetic heritability (yes/no)	<i>g</i> /IQ (WISC-V FSIQ)
Prenatal exposure to substances (yes/no)	Attention (CPT2/CPT3; WISC-V DSF)
Symptom mitigating factors (by caregiver report)	Working memory (WISC-V DSB & DSS)

**Table 1** *continued*

Predictors	Outcomes
Participation in early intervention services	Executive functioning (D-KEFS TMT4/Children's Trails B; D-KEFS CWIT3/NEPSY Inhibition; D-KEFS CWIT4/NEPSY Inhibition-Switching)
Social Skills (BASC-3, ABAS-3)	Processing speed (WISC-V PSI)
Adaptive Skills (BASC-3, ABAS-3)	

### *Behavior Rating Scales*

**ADHD Diagnosis and Symptom Severity as Outcome—Hypothesis 1.** The first hypothesis examined the relationship between the child's gestational age (GA) at birth and diagnostic status from the patient data files, specifically prevalence of ADHD and symptom severity. While the continuous GA and categorical GA variables are effectively the same, it was expected that there would be group differences. Therefore, if GA as a continuous variable was found to be significant, then additional analyses were conducted to look at GA group differences. Both diagnostic status (yes/no ADHD) and the symptom severity of ADHD (parent report of inattention, hyperactivity/impulsivity, attentional problems, and/or total ADHD problems) were examined in the analyses.

*1a.* A *t*-test was used to determine whether the average gestational age (GA) was different between those children diagnosed with ADHD and those children who were not diagnosed with ADHD. It was predicted that children with ADHD would have a significantly lower mean GA than children without ADHD.

*1b.* A chi-square test was used to determine whether there would be a significant difference between expected and observed rates of ADHD diagnosis (yes/no) by GA as a categorical variable (late preterm, moderate preterm, early-term, and full-term). It was predicted that LAMP children would exhibit greater than expected rates of ADHD, and term-born children would not.

*1c.* Bivariate correlational analyses were conducted to examine if GA as a continuous variable was related to symptom severity indicated via behavioral report. The specific symptoms to be analyzed were inattention, hyperactivity/impulsivity, attention problems, and/or total ADHD problems. It was predicted that GA would be negatively correlated with symptom

severity: the closer a child is to 40 weeks' gestation at the time of birth, the lower the ADHD symptom severity.

*1d.* An analysis of covariance, ANCOVA, was used to determine if there were significant differences in symptom severity between LAMP and full-term groups, after accounting for any significant covariates (e.g., ADHD medication). It was predicted that the average symptom severity would be highest among moderate preterm children, followed by late preterm children, early term children, and lastly full-term children. The ANCOVA analysis would pinpoint the precise GA that places children at risk of developing ADHD symptoms.

**Risk Factors Impacting Diagnosis and Symptom Severity—Hypothesis 2.** This hypothesis examined the relationship between several risk factors (race, SES, parent educational level, genetic heritability, prenatal exposure) and ADHD in the sample. Both the global diagnosis of ADHD and the severity of ADHD symptoms as reported by the parents were examined in relation to these risk factors.

*2a.* Chi-square tests were run to examine the difference between expected and observed risk factors based on diagnostic status (yes/no ADHD). It was predicted that those with risk factors would exhibit greater than expected rates of ADHD diagnosis (i.e., non-white racial status, lower SES group, lower parental education, presence of the likelihood of genetic heritability for ADHD, and presence of various prenatal environmental risk factors).

*2b.* *T*-tests were run to examine mean score differences in symptom severity by risk factors (race, SES, parent educational level, genetic heritability, prenatal exposure). It was predicted that caregivers would report higher mean symptoms of attention problems/inattention and hyperactivity/impulsivity on behavior rating forms if they also endorsed the following risk factors in their child's history: racial minority status, parents without higher level education,



Medicaid insurance rather than commercial, genetic heritability for ADHD, and/or history of in-utero exposure to substances (alcohol, tobacco, illicit substances).

**Symptom Mitigating Factors—Hypothesis 3.** This hypothesis examined the relationship between several factors hypothesized to mitigate ADHD symptoms. Because early intervention services are likely to have a positive impact on development, it was hypothesized that a history of early intervention services would be negatively related to ADHD-symptom severity. Secondly, since ADHD is often associated with poor social skills and diminished adaptive skills, it was hypothesized that children who have strength in these areas would exhibit diminished symptom severity. Early history of intervention services, social skills, and adaptive skills are collectively referred to as symptom-mitigating factors and represent the hypothesized negative relationship between these factors and ADHD outcomes.

**3a.** Chi-square tests were run to examine the difference between expected and observed symptom-mitigating factors (history of early intervention services, current social skills, and adaptive skills) based on diagnostic status (yes/no ADHD). It was predicted that those without symptom-mitigating factors would exhibit greater than expected rates of ADHD diagnosis, and those with symptom mitigating factors would not.

**3b.** *T*-tests were run to examine the mean difference in symptom severity by symptom-mitigating factors. It was predicted that mean symptom severity would be lower among those children with higher symptom-mitigating factors.

**Additional Factors Impacting Relationship Between GA, ADHD Diagnosis, and Symptom Severity—Hypothesis 4.** We examined the additive predictive power of risk factors and symptom mitigating factors with GA on ADHD symptom severity. This was done to clarify

what amount of variance in ADHD-related symptoms could be accounted for by GA, other known risk factors, and the hypothesized symptom mitigating factors.

*4a.* Of those risk factors found significantly correlated with ADHD symptom severity from Hypothesis 2, a multiple linear regression was conducted to examine which of those risk factors accounted for significant variance in predicting symptom severity, above and beyond what was accounted for by gestational age. It was predicted that the risk factors would account for additional variance in predicting ADHD-related symptom severity.

*4b.* Of those symptom-mitigating factors found significantly correlated with ADHD from Hypothesis 3, a logistic regression was conducted to examine which of those risk factors accounted for significant variance in predicting a diagnosis of ADHD, above and beyond what was accounted for by gestational age. It was predicted that the symptom mitigating factors would account for additional variance in predicting an ADHD diagnosis.

### *Neurocognitive Outcomes*

The first four hypotheses were related to behavioral symptom presentation and diagnostic status; the following hypotheses investigated the underlying neurocognitive dysfunction theorized to underly that behavioral presentation.

**Relationship Between GA and Neurocognitive Functioning—Hypothesis 5.** This hypothesis examined the relationship between GA and neurocognitive functioning in the following domains: overall intelligence ( $g/IQ$ ), attention (selective, sustained, brief), working memory (spatial revisualization, auditory acoustic memory), executive functioning (cognitive flexibility, inhibition, inhibition with switching), and processing speed. Firstly, correlational analyses were conducted with the expectation that across domains, GA would be negatively correlated with neurocognitive functioning. Independent samples  $t$ -tests were conducted to

highlight the differences between LAMP children and term-born infants across neurocognitive domains. ANOVA analyses were used to pinpoint if the specific GA categories were relevant in determining severity of neurocognitive deficits.

**5a.** It was predicted that GA would be negatively related to *g*/IQ, attention, working memory, executive functioning, and processing speed (bivariate correlational analyses).

**5b.** It was predicated that LAMP children would have lower average scores than term-born children on measures of *g*/IQ, attention, working memory, executive functioning, and processing speed (independent samples *t*-tests).

**5c.** It was predicted that lower performance on measures of *g*/IQ, attention, working memory, executive functioning, and processing speed would correspond to GA group (moderately preterm, late prem, early term, and full-term, respectively).

**Additional Factors Impacting Relationship Between GA and Neurocognitive Functioning—Hypothesis 6.** This hypothesis examined the relationship among identified risk factors, symptom-mitigating factors, and the outcome variables of interest (neurocognitive ability in the domains of attention, working memory, executive functioning, and speed of information processing). Although researchers have examined the risk factors for ADHD (American Psychiatric Association, 2013; Biederman, 2005; Braun et al., 2006; D’Onofrio et al., 2013; Du Rietz et al., 2018; Faraone et al., 2005; Forray & Foster, 2015; Franke et al., 2012, 2018; Humphreys et al., 2018; Knopik et al., 2018; Leung & Hon, 2016; Russell et al., 2014; Spiers et al., 2015; Tsang et al., 2016; Williams & Smith, 2015), none have examined how these factors map onto discrete neurocognitive skill deficits. It may be that examining these relationships in LAMP children is particularly critical for understanding how environmental factors affect children who are already at higher risk for developing ADHD. Once the significant risk factors

and symptom mitigating factors were identified in the current sample, this hypothesis examined which of those factors accounted for the greatest variance in predicting domain-specific performance.

**6a.** Bivariate correlational analysis were conducted for all risk factors (race, SES, parent educational level, genetic heritability, prenatal exposure) with all neurocognitive domains (brief and sustained attention, spatial revisualization and auditory acoustic memory, cognitive flexibility, inhibition, inhibition with switching, and processing speed). It was predicted that the risk factors would be significantly correlated with each outcome (positively correlated in the case of higher performance-based scores indicating deficits, and negatively correlated in the case of higher performance-based scores indicating better performance).

**6b.** Bivariate correlational analysis were conducted for all symptom-mitigating factors (history of early intervention services, social skills, and adaptive skills) and all neurocognitive domains (brief and sustained attention, spatial revisualization and auditory acoustic memory, cognitive flexibility, inhibition, inhibition with switching, and processing speed). It was predicted that the symptom-mitigating factors would be significantly correlated with each outcome (negatively correlated in the case of higher performance-based scores indicating deficits, and positively correlated in the case of higher performance-based scores indicating better performance).

**6c.** Of those risk factors found significantly correlated with neurocognitive outcomes, hierarchical regression analyses were conducted to examine whether those risk factors account for significant variance within neurocognitive domain, above and beyond what would be accounted for by gestational age. It was predicted that the risk factors would account for

additional variance in performance-based neurocognitive outcomes above what was accounted for by gestational age.

*6d.* Of those symptom-mitigating factors found significantly correlated with neurocognitive outcomes, hierarchical regression analyses were to be conducted to examine whether those factors account for significant variance within the neurocognitive domains, above and beyond what was accounted for by gestational age. It was predicted that the symptom-mitigating factors would account for additional variance in performance-based neurocognitive outcomes above what was accounted for by gestational age.

### *Experimental Analyses*

**Interaction between GA and Additional Factors in Predicting Neurocognitive Outcomes—Hypothesis 7.** To better understand how environmental factors and history interact with GA in predicting long-term neurocognitive abilities, moderation analyses were conducted. Cumulative risk was derived from the number of endorsed risk factors.

*7a.* It was predicted that there would be a significant interaction between gestational age and cumulative risk in predicting neurocognitive ability by domain.

*7b.* It was predicted that there would be a significant interaction between gestational age and cumulative protection (i.e., total symptom mitigating factors) in predicting neurocognitive ability by domain.

**Interaction between GA and Birthweight in Predicting Neurocognitive Outcomes—Hypothesis 8.** Though the focus of the present analyses were on gestational age rather than birthweight, experimental analyses were conducted to examine the interaction birthweight has with gestational age in predicting long-term neurocognitive outcomes among LAMP children and term-born children. Gestational age (GA) and birthweight are closely related, and

birthweight can be used as a proxy for GA (Taylor, 2010). Essentially, low birthweight (LBW) suggests either prematurity, intrauterine growth restriction/fetal growth restriction, or both (Cutland et al., 2017). LBW is less than 2500g (World Health Organization, 2014). Even marginally LBW has been associated with lower cognitive scores, including lower verbal comprehension IQ, lower visual-motor integration, and lower attention performance by school-age (Starnberg et al., 2018). Therefore, rather than controlling for birthweight, this hypothesis looked at birthweight as a moderator that is related to both gestational age and neurocognitive outcomes.

**8a.** It was predicted that there would be a significant interaction effect between birthweight and gestational age (birthweight was used as a moderator) in predicting symptom severity.

**8b.** It was predicted that there would be a significant interaction effect between birthweight and gestational age (birthweight was used as a moderator) in predicting neurocognitive performance across each domain (attention, working memory, executive functioning, and processing speed).

## Chapter 3: Methods

### Participants

Table 2 provides demographic information about the 169 patients represented in this study. The study utilized de-identified patient data collected between 2014 and 2021 from a major metropolitan health system. Patients ranged from 8.01 to 12.95 years in age, with good representation across years. The final sample included nearly twice as many male identifying subjects as female, which is in keeping with the literature that says males are more likely to be identified as having attention problems and referred for evaluation (American Psychiatric Association, 2013). There was a significant representation of non-white participants, including 25% who identified as being Black/African American, resulting in a highly diverse sample that was consistent with the health system's urban city demographics (US Census Bureau, 2019). Prior to their scheduled neuropsychological evaluations, patients' caregivers were not given specific/standardized instructions regarding ADHD medication administration on the day of evaluation; caregivers were allowed to decide whether they wanted results that would reflect their child's abilities while on ADHD medication or off ADHD medication. Therefore, ADHD medication was captured in two ways: current prescription for ADHD medication and ADHD medication taken on the day of evaluation.

**Table 2***Patient Demographics*

	M	SD	Range	<i>n</i> (%)
Age	10.54	1.34	8.01-12.95	
8 (8.00-8.99)				28 (16.57)
9 (9.00-9.99)				36 (21.30)
10 (10.00-10.99)				33 (19.53)
11 (11.00-11.99)				45 (26.63)
12 (12.00-12.99)				27 (15.98)
LAMP	10.46	1.29	8.05-12.95	
Moderately preterm	10.43	1.41	8.05-12.56	
Late preterm	10.46	1.27	8.29-12.95	
Term born	10.59	1.37	8.01-12.81	
Early term	10.60	1.31	8.01-12.72	
Full term	10.59	1.40	8.17-12.81	
	LAMP	Term Born	Total	
	<i>n</i> (%)	<i>n</i> (%)	Sample	
			<i>n</i> (%)	
Gender				
Female	29 (42.03)	22 (22.00)	51 (30.18)	
Male	40 (57.97)	78 (78.00)	118 (69.82)	
Trans/nonbinary/other	0 (0.00)	0 (0.00)	0 (0.00)	



**Table 2** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
<b>Race/ethnicity</b>			
American Indian/Alaskan Native	0 (0.00)	0 (0.00)	0 (0.00)
Asian	1 (1.45)	2 (2.00)	3 (1.78)
Black/African American	20 (28.99)	23 (23.00)	43 (25.44)
Native Hawaiian/Pacific Islander	0 (0.00)	0 (0.00)	0 (0.00)
White	43 (62.32)	58 (58.00)	101 (59.76)
Latino/Hispanic	2 (2.90)	7 (7.00)	9 (5.33)
Biracial/Mixed	3 (4.35)	6 (6.00)	9 (5.33)
<b>Handedness</b>			
Right	56 (81.16)	84 (84.00)	140 (82.84)
Left	9 (13.04)	10 (10.00)	19 (11.24)
Ambidextrous	4 (5.80)	6 (6.00)	10 (5.92)
<b>Previous psychiatric diagnosis (by history)</b>			
ADHD	33 (47.83)	45 (45.00)	78 (46.15)
Anxiety	15 (21.74)	31 (31.00)	46 (27.22)
Depression	9 (13.04)	15 (15.00)	24 (14.20)
Learning disability	9 (13.04)	6 (6.00)	15 (8.88)
Language disorder	6 (8.70)	2 (2.00)	8 (4.73)

**Table 2** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
Participation in early intervention services	25 (36.23)	21 (21.00)	46 (27.22)
Early On	15 (21.74)	8 (8.00)	23 (13.61)
Speech and language pathology (SLP)	21 (30.44)	15 (15.00)	36 (21.30)
Occupational therapy (OT)	6 (8.70)	7 (7.00)	13 (7.69)
Physical therapy (PT)	4 (5.80)	5 (5.00)	9 (5.33)
ADHD Medication			
Current prescription for ADHD medication	24 (34.78)	39 (39.00)	63 (37.28)
ADHD medication on day of evaluation	17 (24.64)	31 (31.00)	48 (28.40)
Special education services			
IEP	24 (34.78)	23 (23.00)	47 (27.81)
504	8 (11.59)	9 (9.00)	17 (10.06)
Repeated a grade	8 (11.59)	12 (12.00)	20 (11.83)
Caregivers Education Level			
Primary/secondary school	15 (21.74)	22 (22.00)	91 (53.85)
One or more caregivers has higher level education	32 (46.38)	59 (59.00)	37 (21.89)
Unknown	22 (31.88)	19 (19.00)	41 (24.26)

**Table 2** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
<hr/>			
Insurance			
Medicaid	29 (42.03)	33 (33.00)	62 (36.69)
Commercial	39 (56.52)	66 (66.00)	105 (62.13)
Bilingual or secondary language	4 (5.80)	15 (15.00)	19 (11.24)

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***Inclusion Criteria***

The proposed study utilized a representational sample of 8- to 12-year-old children previously seen for a neuropsychological evaluation. This age group was chosen owing to the relative reliability of cognitive findings (Moser et al., 2017). Furthermore, the greatest predictor of an ADHD diagnosis is the child's behavior relative to other children of the same age/grade, often via teacher observation and report (Layton et al., 2018). Children in the identified age range had multiple years of schooling and therefore opportunity for teachers to have raised concerns regarding functional impairment caused by potential ADHD symptoms. However, as previously demonstrated, significant changes by domain, especially in executive functioning including attention and working memory, occur from middle childhood to adulthood. ADHD-related neurocognitive deficits may affect symptom presentation as well as cognitive and academic performance across the school years (Ang & Lee, 2008; Gow et al., 2011; Gur et al., 2012). The goal was to recruit a roughly equivalent number of male and female subjects and controls, with a total recruitment goal of 200 children (100 LAMP born children, and 100 full-

term children); however, there were reductions in outpatient evaluation volume (i.e., furlough) owing to the COVID-19 pandemic and, consequently, there were fewer patient files to review for potential inclusion in the study.

### ***Exclusion Criteria***

Any child with a diagnosis of intellectual disability (ID), autism spectrum disorder (ASD), epilepsy, genetic disorders, other neurodevelopmental disorder or major or mild neurocognitive disorder due to another medical condition (such as stroke or traumatic brain injury), when there was anticipated or observed neurological impact, was excluded so that the relationship between the variables of interest could be observed without significant influence from radical outliers (i.e., aberrant neurocognitive functioning). GA as determined by caregiver report was used to exclude children who were born very or extremely preterm (GA < 32 weeks), or late term (GA  $\geq$  41 weeks). Any child outside of the identified age range (8 to 12 years old) at the time of their neuropsychological evaluation was also excluded. Cases assessed prior to 2014 were not included to ensure that the utilized measures would be current and applicable to clinical practice and research.

### **Institutional Review Board**

An Institutional Review Board (IRB) application was submitted for the present study to the health system where electronic records were to be pulled. An expedited review was completed, and the study was deemed exempt (see Appendix B). The IRB application required CITI training for anyone involved in the research study. The primary investigator (author of the present document) worked within the health system as a research affiliate; in this role, the primary investigator also applied for and was granted remote access to the EMR and secure drive so that work could be conducted in a safe off-site location. A second IRB application was

completed with Eastern Michigan University. This ensured that there was approval for the author of the present document to conduct analyses and complete dissertation work in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Clinical Psychology.

### *Screening*

**Stage 1: Preliminary Screening.** Pediatric neuropsychologists on staff in the Division of Neuropsychology in a major metropolitan healthcare system provided a list of the cases they had supervised/conducted since 2014. There were three such supervisors; in total, they turned over a pool of 9,382 patients to be screened for inclusion. This list included ages and diagnostic status at the time of evaluation. A research assistant screened for eligible participants by sorting those between 8 and 12 years old who were not excluded by diagnostic status (e.g., ID, ASD, epilepsy, genetic disorders, other neurodevelopmental disorder, or major or mild neurocognitive disorder). These potential subjects were put into a file that contained their identifying information, including their medical record number (MRN).

**Stage 2: EMR Initial Screening.** Once subjects were identified in Stage 1 screening, their electronic medical record was reviewed. Subjects whose GA was not reported, who did not complete an intake form, or who did not complete a battery of performance-based neuropsychological tests (including a Wechsler cognitive test) and a behavioral rating scale, were screened out. Any child whose caregiver reported that they were born prior to 32 weeks' gestation, or at/after 41 weeks' gestation, was also excluded from the study. The remaining subjects were therefore screened for inclusion and exclusion criteria and were ready for data entry. The final subject pool included 169 children (69 LAMP and 100 term-born children).

**Stage 3: Final Screening and Data Entry.** Relevant data were pulled from the EMR for data entry (e.g., patient history form, neuropsychological evaluation report, and testing data

summary sheet). Subjects' data were stored in a corresponding folder on a secure shared drive owned by the health system. Each subject's file was reviewed, and relevant variables were entered into the database (also stored on the secure shared drive). After the data were entered, it was double checked to ensure accuracy. After this two-step entry/check procedure, the copy of the patient file was deleted from the secure shared drive. The file that linked the patient via MRN to the database was deleted so that the database no longer contained any identifying information.

### **Measures**

Children presenting for neuropsychological evaluation were accompanied by parents/caregivers who completed paperwork including a patient history questionnaire and a social/emotional/behavioral rating scale(s). Children had completed performance-based neuropsychological testing. Notably, the hospital suspended outpatient neuropsychological evaluations between March 2020 and June 2021 owing to the COVID-19 pandemic (i.e., furlough). Evaluations completed after the furlough, between June 2020 and January 2021, were completed in the context of the ongoing COVID-19 pandemic; as such, both the examiner and the patient wore a face mask throughout testing. Efforts were made to maintain social distancing within the testing environment, and a plexiglass barrier separated examiner and patient. Use of a face mask during administration of in-person measures is an adaptation of standardized administration, which was considered in the interpretation of results. All scores derived from behavior rating scales and performance-based neuropsychological measures are age and gender normed, and, therefore, additional consideration for age and gender as covariates was not necessary. Diagnostic impressions were taken from the completed neuropsychological evaluation reports.

***Prenatal, Perinatal, and Other Background History from Patient History Form***

Parents/caregivers completed a patient history form. This form queried the child's history, including early intervention experiences, prenatal factors, perinatal factors, medical history, and family history. It is also where caregivers reported on demographic factors such as race, Medicaid status, and parent educational level, presented in Table 2. Table 3 presents a summary of relevant medical history and background information taken from the patient history form. Notably, the current sample had a 51.5% hereditary risk for ADHD. Furthermore, nearly 1 in 10 of the patients were born to caregivers experiencing pre-eclampsia, nearly 1 in 20 had in-utero exposure to substances (cigarettes, alcohol, or illegal substances), and 1 in 20 required intensive care at the time of birth. Taken together, the sample was comprised of children with multiple significant risk factors for ADHD and other neurodevelopmental concerns, consistent with what would be expected in a clinical setting where patients are referred by providers and/or pre-authorized for the evaluation by their insurance company owing to established risk/probability.

**Table 3***Patient Prenatal and Perinatal History*

	M	SD	Range	<i>n</i> (%)
Gestational age	37.40	2.27	32-40	
LAMP	35.04	1.32	32-36	69 (40.83)
Moderately preterm	33.00	0.94	32-34	17 (10.06)
Late preterm	35.71	0.46	35-36	52 (30.77)
Term born	39.02	1.03	37-40	100 (59.17)
Early term	37.68	0.48	37-38	31 (18.34)

**Table 3** *continued*

	M	SD	Range	<i>n</i> (%)
Full term	39.62	0.49	39-40	69 (40.83)
Birthweight (ounces)	108.91	22.52	45-162	
LAMP	93.25	21.69	45-145	
Moderately preterm	78.18	20.03	45-111	
Late preterm	98.27	19.97	53-145	
Term born	120.12	15.31	74-162	
Early term	115.10	15.04	74-142	
Full term	122.55	14.95	86-162	
Mother's age at delivery	29.49	6.33	16-48	
LAMP	29.51	6.43	16-45	
Moderately preterm	28.82	7.05	19-43	
Late preterm	29.75	6.27	16-45	
Term born	29.47	6.29	16-48	
Early term	27.94	5.56	20-38	
Full term	30.18	6.52	16-48	
	LAMP		Term Born	Total
	<i>n</i> (%)		<i>n</i> (%)	Sample
				<i>n</i> (%)
Medication during pregnancy				
Anti-seizure	0 (0.00)		1 (1.00)	1 (0.59)
Anti-depressant	6 (8.70)		1 (1.00)	7 (4.14)



**Table 3** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
Anti-anxiety	1 (1.45)	2 (2.00)	3 (1.78)
Thyroid	4 (5.80)	3 (3.00)	7 (4.14)
Other	12 (17.39)	15 (15.00)	27 (15.98)
Complications during pregnancy			
Bleeding	5 (7.25)	4 (4.00)	9 (5.33)
Gestational diabetes	4 (5.80)	8 (8.00)	12 (7.10)
High blood pressure/toxemia	9 (13.04)	0 (0.00)	9 (5.33)
Infection	2 (2.90)	1 (1.00)	3 (1.78)
Seizure	1 (1.45)	1 (1.00)	2 (1.18)
Injury/accident	3 (4.35)	0 (0.00)	3 (1.78)
Hospitalization	8 (11.59)	4 (4.00)	12 (7.10)
German measles/Rubella	0 (0.00)	0 (0.00)	0 (0.00)
Placenta abruptia	2 (2.90)	1 (1.00)	3 (1.78)
Placenta previa	1 (1.45)	2 (2.00)	3 (1.78)
Pre-eclampsia	13 (18.84)	3 (3.00)	16 (9.47)
Rh incompatibility	1 (1.45)	3 (3.00)	4 (2.37)
Chronic illness	9 (13.04)	4 (4.00)	13 (7.69)
Major life stress	2 (2.90)	4 (4.00)	6 (3.55)
Other	14 (20.29)	11 (11.00)	25 (14.79)

**Table 3** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
In-utero exposure to substances	11 (15.94)	11 (11.00)	22 (13.00)
Cigarettes	9 (13.04)	7 (7.00)	16 (9.5)
Alcohol	3 (4.35)	2 (2.00)	5 (3.0)
Illegal substances	4 (5.80)	4 (4.00)	8 (4.73)
Labor complications			
Blue at birth	2 (2.90)	2 (2.00)	4 (2.37)
Breech	1 (1.45)	5 (5.00)	6 (3.55)
Cord around neck	4 (5.80)	12 (12.00)	16 (9.47)
Fetal distress	4 (5.80)	5 (5.00)	9 (5.33)
Group B streptococcus	2 (2.90)	1 (1.00)	3 (1.78)
Induced	20 (30.00)	27 (27.00)	47 (27.81)
Intrauterine growth restriction	2 (2.90)	0 (0.00)	2 (1.18)
Jaundice	18 (26.09)	12 (12.00)	30 (17.75)
Meconium staining	1 (1.45)	2 (2.00)	3 (1.78)
Premature labor	21 (30.43)	0 (0.00)	21 (12.43)
Prolonged labor	1 (1.45)	6 (6.00)	7 (4.14)
Slow heart rate	4 (5.80)	4 (4.00)	8 (4.73)
Other	9 (13.04)	7 (7.00)	16 (9.47)

**Table 3** *continued*

	LAMP	Term Born	Total
	<i>n</i> (%)	<i>n</i> (%)	Sample <i>n</i> (%)
<b>Delivery</b>			
Vaginal	41 (59.42)	68 (68.00)	109 (64.5)
Caesarean section	27 (39.13)	32 (32.00)	59 (34.91)
<b>Intensive care</b>			
Incubator	11 (15.94)	4 (4.00)	15 (8.88)
Bili lights	17 (24.64)	8 (8.00)	25 (14.79)
Oxygen	8 (11.59)	8 (8.00)	16 (9.47)
Transfusion	0 (0.00)	0 (0.00)	0 (0.00)
Other	2 (2.90)	2 (2.00)	4 (2.37)
<b>Family history</b>			
ADHD	36 (52.20)	51 (51.00)	87 (51.50)
Anxiety	31 (44.90)	49 (49.00)	80 (47.34)
Depression	41 (59.40)	68 (68.00)	109 (64.50)
Intellectual disability	11 (15.90)	7 (7.00)	18 (10.65)
Language disorder	8 (11.60)	3 (3.00)	11 (6.51)
Learning disability	31 (44.90)	23 (23.00)	54 (31.95)
Birth defects	1 (1.40)	5 (5.00)	6 (3.55)
Structural brain abnormality	1 (1.40)	0 (0.00)	1 (0.59)

***ADHD Diagnosis***

Diagnosis of ADHD was determined by diagnostic impressions indicated in the comprehensive neuropsychological evaluation. Neuropsychologists determined ADHD diagnosis using *DSM-5* diagnostic criteria based on all available sources of information (e.g., clinical interview, patient history form, behavior rating scales, behavioral observations during evaluation, performance-based measures of neurocognition, etc.). Therefore, participants in the present study were coded as having an ADHD diagnosis if that diagnosis had been given following comprehensive neuropsychological evaluation; if a caregiver reported a history of ADHD, but the results of the evaluation did not support this diagnosis (i.e., no ADHD diagnosis given), the participant was coded as having a history of ADHD but not a current diagnosis. Table 4 shows prevalence of ADHD diagnosis in the present sample, following comprehensive neuropsychological evaluation, including rates of ADHD diagnosis among the LAMP and term-born groups.

**Table 4***ADHD and Other DSM-5 Diagnostic Outcomes*

	Total sample	LAMP	Term-born
	n (%)	n (%)	n (%)
ADHD diagnosis	130 (76.9)	55 (79.7)	75 (75)
Predominantly inattentive presentation	35 (20.7)		
Predominantly hyperactive/impulsive presentation	1 (0.6)		
Combined presentation	94 (55.6)		

*ADHD-Related Symptom Severity as indicated by Behavior Rating Scales*

Behavioral reports are also a critical part of school-aged assessment for ADHD, especially considering that the diagnosis is made based on observed behaviors. ADHD subtype and severity are also captured by different scales included within measures. Caregivers of all the children in the sample were administered the school-age form of one or both of the following measures: The Conners (3<sup>rd</sup> Ed.; Conners-3; Conners, 2008) and the Behavior Assessment System for Children (3<sup>rd</sup> Ed.; BASC-3; Reynolds & Kamphaus, 2015). For purposes of this study, across behavioral reports, caregivers report on the Attention Problems, Inattention, Hyperactivity/Impulsivity, and *DSM* ADHD subscales (Conners-3 only) were used to represent ADHD symptom severity in the analyses. For both measures, across behavioral reports, raw scores were converted to *t*-scores based on the norms from the standardization sample.

The BASC-3 is a behavior rating form that assesses a broad range of emotions and behaviors via caregiver completion of the child form (ages 6-11 years, 175 items) or adolescent form (ages 12-21, 173 items). Caregivers are instructed to rate the frequency of their child's behavior over the course of the last several months scale (never, sometimes, often, almost always). In the present study, 118 caregivers completed the BASC-3 behavior rating form. The Conners-3 is a behavior rating form for children between the ages of 6 and 18 years and was developed specifically to assess for attentional deficits and symptoms related to ADHD. It comes in two forms: the full-length form has 110 items and yields all content scales and symptom scales that capture *DSM* symptomatic criteria (this includes the *DSM* ADHD Inattentive subscale and the *DSM* ADHD Hyperactive/Impulsive subscale), while the short form has 45 items and yields content scales but does not include those clinical subscales aligned with the *DSM* criteria. Caregivers are instructed to rate the frequency of their child's behavior over the course of the last

month on a Likert scale ranging from 0 = *not true at all* (never, seldom), to 3 = *very much true* (very often/very frequently). In the present study, 111 caregivers completed the Conners-3 behavior rating form.

Neuropsychologists chose whether to administer one or more of these rating forms (and may have administered other behavior rating forms not utilized for the purpose for this study); as such, 74 caregivers in the current sample completed both the BASC-3 and the Conners-3 behavior rating scales. The BASC-2 Attention problems subscale was previously found to be strongly correlated with the Conners-3 Inattention subscale ( $.72, p < .01$ ), and the BASC-2 Hyperactivity subscale was also strongly correlated with the Conners-3 Hyperactivity/Impulsivity subscale ( $.77, p < .01$ ). As such, to maximize the number of participants with caregiver-rated symptom severity, a variable called Inattention Combined Measures was created using the Conners-3 Inattention score and, in the case that the Conners-3 had not been administered, the BASC-3 Attention Problems score. Similarly, a variable called Hyperactivity Combined Measures was created using the Conners-3 Hyperactivity/Impulsivity score, in the case that the Conners-3 had not been administered, the BASC-3 Hyperactivity subscale.

Using a threshold of  $\alpha = .70$  (Santos, 1999), all measures utilized had an acceptable internal consistency per values reported in their manuals ( $\alpha = .84-.99$ ). In larger samples involving more than 30–40 participants, research supports that the sampling distribution has a tendency toward normalcy regardless of the shape of the data (Ghasemi & Zahediasl, 2012) and therefore, parametric tests can be justified even when the data deviate from a normal distribution (Field, 2009). A range of alpha coefficients is provided from the manuals of respective measures; this range shows purported alpha coefficients for children between the ages of 8 and 12 years, on

those subscales that were utilized in the present study (Conners, 2008; Reynolds & Kamphaus, 2015); alpha coefficients were not calculated using study data because only standard scores from behavior rating scales were available for analyses (i.e., individual items on behavior rating scales were not available). Descriptive statistics for this sample on all behavioral rating scale indicators of symptom severity appear in Table 5.

**Table 5***Descriptive Statistics for Symptom Severity as Indicated by Behavior Rating Scales*

Construct	Subscale	N	M	SD	Range	$\alpha$
<b>Problems with Attention</b>						
	Attention problems (BASC-3)	118	64.53	10.08	33-84	.88-.91
	Inattention (Conners-3)	111	77.37	11.62	35-90	.91-.94
	Inattention Combined Measures	155	73.93	12.48	35-90	
<b>Behavioral Hyperactivity</b>						
	Hyperactivity (BASC-3)	118	63.00	13.88	35-92	.84-.89
	Hyperactivity/Impulsivity (Conners-3)	111	73.32	15.59	38-90	.93-.95
	Hyperactivity Combined Measures	155	70.39	15.75	35-92	
<b>DSM Indicators for ADHD</b>						
	DSM ADHD Inattentive (Conners-3)	98	75.60	12.48	40-90	.92-.93
	DSM ADHD Hyperactive/Impulsive (Conners-3)	98	71.94	15.64	38-90	.89-.93

**Attention Problems.** On the BASC-3 Parent Rating Scales, patients' mean attention problems fell in the "At-Risk" range, with scores ranging from "Acceptable"/within normal

limits (WNL) to “Clinically Significant.” Mean problems with inattention, as reported on the Conners-3 Parent Form, fell in the “Very Elevated” range, and ranged from “Average or Below”/WNL to “Very Elevated.” In addition, 33.1% (56 of 169) of the patients included in this sample were identified as having clinically significant attention problems by one or both measures ( $T \geq 70$ ).

**Hyperactivity/Impulsivity.** On the BASC-3 Parent Rating Scales, patients’ mean hyperactivity problems fell in the “At-Risk” range, with scores ranging from “Acceptable”/within normal limits (WNL) to “Clinically Significant.” Mean problems with hyperactivity and/or impulsivity, as reported on the Conners-3 Parent Form, fell in the “Very Elevated” range, and ranged from “Average or Below”/WNL to “Very Elevated.” 27.2% (46 of 169) of the patients included in this sample were identified as having clinically significant problems with hyperactivity/impulsivity by one or both measures ( $T \geq 70$ ).

**DSM ADHD Problems.** The two symptom scales utilized in the present study include the *DSM* ADHD Inattentive subscale and the *DSM* ADHD Hyperactive/Impulsive subscale. Both of these scales were originally created to correspond with the ADHD diagnostic criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (4<sup>th</sup> ed.; Text rev.; *DSM-IV-TR*). After the *DSM-5* was published, a manual update was provided to scoring and interpretation of these clinical subscales; the only change that pertains to the present sample was that the subscale names were updated to reflect the reclassification of subtype presentations (e.g., predominantly inattentive presentation, predominantly hyperactive/impulsive presentation, and combined presentation). Items included on these subscales approximate symptom-level criteria from the *DSM*, but do not capture full diagnostic criteria (Conners, 2014a). Caregivers report at the item level determined whether a specific symptom was indicated (2 = *pretty much true*, 3 = *very much*



*true*), may be indicated (2 = *pretty much true*), or not indicated (1 = *just a little true*, 0 = *not true at all*).

Scores on the *DSM-5* ADHD Inattentive scale ranged from “Average or Below”/WNL to “Very Elevated,” with a mean score in the “Very Elevated” range. Furthermore, 40.24% (68/169) of the sample was identified as having clinically significant risk for ADHD using the *DSM-5* ADHD Inattentive scale. Scores on the *DSM-5* ADHD Combined scale ranged from “Average or Below”/WNL to “Very Elevated,” with a mean score in the “Very Elevated” range. About one third (33.14%; 56/169) of the sample were identified as having clinically significant risk for ADHD using the *DSM-5* ADHD Combined scale. Though the BASC-3 has a similar clinical subscale of DSM-based ADHD Probability, this score was not available for review in this sample.

### ***Neuropsychological Measures***

To maximize the number of participants and minimize missing data, neuropsychological tests that measure the same construct and are highly intercorrelated were used interchangeably (i.e., multiple measures used as singular variable for construct of interest). Descriptive statistics for neuropsychological measures appear in Table 6.

**Table 6***Descriptive Statistics for Neuropsychological Measures*

Construct	Measure	N	M	SD	Range
g/IQ	WISC-V FSIQ	169	93.37	12.20	64-146
Attention					
Sustained attention	CPT-2/CPT 3 Omissions	167	60.15	14.83	40-109
Impulsivity	CPT-2/CPT 3 Commissions	167	53.19	7.83	31-70
Brief attention	WISC-V DSF	168	8.34	2.46	3-14
Working memory					
Spatial revisualization	WISC-V DSB	168	8.45	2.96	1-17
Auditory memory	WISC-V DSS	168	8.81	3.03	1-18
Executive functioning					
Cognitive flexibility		140	8.39	3.84	1-16
	D-KEFS TMT-4	112	8.10	3.80	1-16
	Trails B	28	9.00	4.15	1-14
Inhibition		127	8.80	3.40	1-16
	D-KEFS CWIT-3	101	9.23	3.11	1-15
	NEPSY Inhibition	26	7.12	3.95	1-16
Inhibition with switching		126	9.58	3.07	1-16
	D-KEFS CWIT-4	101	9.98	2.87	1-16
	NEPSY Inhibition-Switching	25	7.96	3.37	1-15
Processing speed	WISC-V PSI	168	94.64	14.80	56-132

**g/IQ.** The Wechsler Intelligence Scale for Children (5<sup>th</sup> ed.; WISC-V; Wechsler, 2014) provides an estimate of overall cognitive functioning: the Full-Scale IQ (FSIQ). The FSIQ is a composite derived from performance across domains of verbal comprehension, visual spatial processing, fluid reasoning, working memory, and processing speed. It is considered the most reliable score and most representative of general intellectual functioning (g) (Wechsler, 2014). See Appendix C further information on psychometric properties. In the present sample, the mean g/IQ, estimated by the WISC-V FSIQ, was in the average range, although slightly below the population mean of 100 and SD of 15. Patients' performance ranged from impaired to very superior. The FSIQ was interpreted by the attending clinician in conjunction with other test data and background information to determine diagnostic status (e.g., intellectual disability), which was used to determine if the patient could be included in the present study.

**Attention.** Attention is a complex neurocognitive construct. In assessment, it is typically measured via selective and sustained attention as well as brief attentional capacity. Regarding formal measures of attention, the Conners Continuous Performance Test (2<sup>nd</sup> ed. and 3<sup>rd</sup> ed.; CPT-2 and CPT-3) are commonly used diagnostic tools in ADHD (Conners, 2014; Dupaul et al., 1992; Nichols & Waschbusch, 2004; Rapport et al., 2000). This test measures sustained attention to visual stimuli and response inhibition over time. Respondents sit at a computer, watch the screen, and press the space bar for every letter that flashes on the screen except for the letter x. Scores on the CPT-2/CPT-3 include omissions and commissions, which reportedly indicate inattention and impulsivity, respectively. These two scores are represented by *t*-scores with a mean of 50 and SD of 10. Both DS-F and the Conners CPT-2/CPT-3 demonstrate excellent psychometric properties (see Appendix D further information on psychometric properties) and

will be used as a measure of sustained attention as indicated by omissions and commissions. The Digit Span Forward (DS-F) subtest of the WISC-V provides some indication of brief attentional capacity. The DS-F requires the subject to repeat back increasingly longer strings of numbers presented to them. The subtest has a mean scaled score of 10 and SD of 3. DS-F will be used as a measure of brief attention.

In the present sample, patients' mean performance on the CPT-2/CPT-3 was characterized by a high-average rate of omission errors; analysis of patients' performance shows that 41.92% had an elevated or very elevated rate of omission errors. Patients' mean performance on a measure of impulsivity was characterized by an average rate of commission errors; analysis of patients' performance shows that 20.96% had an elevated or very elevated rate of commission errors. Patients' brief attention as measured by performance on the WISC-V Digit Span Forward subtest fell in the average range. Analysis of patients' performance found that 35.1% of the sample performed below the average range (impaired, borderline impaired, or low average).

**Working Memory.** Working memory is the ability to hold and manipulate information in the mind (Cowan & Alloway, 2009). The WISC-V includes a Working Memory Index (WMI) that was designed in accordance with Baddeley's multi-component model of memory (A. Baddeley, 2000, 2003; Baddeley et al., 2011). The WMI on the WISC-V is composed of two subtests: Digit Span (consisting of Digit Span Forward, Backward, and Sequencing) and Picture Span. Digit Span Forward is a measure of brief auditory attention, while Backwards and Sequencing are measures of working memory (notably backwards includes spatial revisualization, and sequencing requires auditory acoustic memory). Picture Span requires the child to remember visually presented stimuli (i.e., familiar objects) and identify them in sequence among non-targets. The child can receive partial credit for correctly identifying targets from non-

targets, despite incorrect sequence in identification. Therefore, the strongest measure of working memory from either WMI, as it is classically defined, is Digit Span Backwards (DS-B) and Sequencing (DS-S). On DS-B, the respondent is read increasingly longer strings of numbers and asked to recite them backwards. DS-S requires the respondent to put the presented numbers in sequence, from smallest to largest. These subtests have a mean scaled score of 10 and SD of 3. DS-B and DS-S demonstrate good psychometric properties and were used as a measure of working memory with spatial revisualization and auditory acoustic memory (see Appendix D further information on psychometric properties).

In this sample, patients' mean score on measures of working memory (WISC-V DSB and DSS) fell in the average range. On a measure of working memory and spatial revisualization, 36.9% of patients performed below the average range (impaired, borderline impaired, or low average). On a measure of working memory and acoustic memory, 31.5% of patients performed below the Average range.

**Executive Functioning.** Executive functioning broadly refers to higher order cognitive processes involved in formulating goals, planning, organization, and performance maintenance (Lezak et al., 2012). The most comprehensive battery for measuring executive functioning is the Delis-Kaplan Executive Function System (D-KEFS; Shunk et al., 2006). It includes several subtests that measure discrete aspects of executive functions. Commonly used subtests included in neuropsychological assessment of school-aged children presenting for concerns related to ADHD include the Trail Making Test Condition 4 (TMT 4), which measures sequencing and shifting/cognitive flexibility. The Trail Making Test Part B (TMT B) is an alternate version, and it is interchangeable with D-KEFS TMT 4. The main difference between these two versions is the normative data. TMT B utilizes normative data from 1997, while TMT 4 utilizes normative

data from 2006. Both trail-making tests require the respondent to alternate connecting numbers and letters in sequential order by making pencil lines between the encircled stimuli on a page. TMT 4 is a subtest with a mean score of 10 and SD of 3. TMT B yields a z-score with a mean score of 0 and SD of 1. For ease of comparison, TMT B was transformed into a scaled score with a mean of 10 and SD of 3 so that it could easily be compared to TMT 4. D-KEFS TMT and TMT B demonstrate good psychometric properties and were used as measures of cognitive flexibility (see Appendix D further information on psychometric properties).

The Color Word Interference Test (CWIT) is another D-KEFS subtest used to assess neurocognitive concerns related to ADHD, and it measures inhibition and cognitive flexibility. There are two parts to CWIT. On the CWIT inhibition (CWIT I) task, the respondent is presented with color names printed in a different colored ink, and they must inhibit reading the word and instead name the dissonant ink color in which the word is printed. In CWIT inhibition with switching (CWIT IS), the respondent must switch back and forth between naming the dissonant ink color and reading the word. CWIT I and CWIT IS yield scaled scores with a mean of 10 and SD of 3 (see Appendix D further information on psychometric properties).

Alternatively, a clinician may choose to measure inhibition and inhibition with cognitive flexibility using the NEPSY Inhibition test, which includes an Inhibition Condition and Switching Condition parallel to the D-KEFS Inhibition and Inhibition with Switching. However, the NEPSY Inhibition may be more appropriate for children with dyslexia or reading difficulties, or who have color blindness, as the stimuli are white and black arrows rather than words. In the Inhibition condition, the respondent must say “up” when the arrow is pointing down and say “down” when the arrow is pointing up. In the Switching Condition, the respondent must switch between saying the dissonant direction and saying the correct direction depending on whether the

arrow is black or white. The NEPSY Inhibition test yields scaled scores with a mean of 10 and SD of 3 for both conditions. Per the manual, the NEPSY Inhibition test shows a consistent moderate relationship with the D-KEFS CWIT; CWIT I and NEPSY Inhibition Condition showed a .43 correlation, and CWIT IS and NEPSY Inhibition Switching Condition have a .57 correlation (Delis et al., 2001; Homack et al., 2005; Korkman et al., 2007). The D-KEFS CWIT and NEPSY Inhibition test demonstrate good psychometric properties and were used as measures of inhibition with cognitive flexibility (see Appendix D further information on psychometric properties).

In this sample, patients' mean performance was in the average range on all subtests from the DKEFS Trail Making Test (TMT) and the Color-Word Interference Test (CWIT). Mean performance was in the low-average range on a measure of cognitive flexibility (NEPSY Inhibition) and was in the average range on a measure of inhibition with switching (NEPSY Inhibition with Switching). As previously mentioned, neuropsychological tests that measure the same construct and are highly intercorrelated were used interchangeably. As such, certain subtests were collapsed into the following single variables: Cognitive Flexibility (D-KEFS TMT 4 or Trails B), Inhibition (D-KEFS CWIT 3 or NEPSY Inhibition), and Inhibition with Switching (D-KEFS CWIT 4 or NEPSY Inhibition with Switching). Mean overall cognitive flexibility, inhibition, and inhibition with switching all fell in the average range.

**Processing Speed.** Processing speed is a component of executive functioning that refers to the speed with which an individual can process information and react to it meaningfully. The WISC-V includes a Processing Speed Index (PSI) composite score derived from performance on two subtests: Coding and Symbol Search. Speed of information processing is the target variable measured by each of these subtests. However, notable non-target factors can account for variance

in performance. Coding requires graphomotor dexterity in drawing symbols into boxes based on a key, and performance on this test can be significantly negatively impacted by fine motor deficits. Symbol Search requires visual scanning in finding and crossing out targets among non-targets, and performance on this test can be significantly negatively impacted by visual conditions. Since subjects with neurodevelopmental disorders that include extreme dexterity issues or visual impairment were screened out of the subject pool, the PSI of the WISC-V was considered a valid and reliable measure of the target variable (see Appendix D further information on psychometric properties). The PSI has a mean standard score of 100 with a SD of 15.

In this sample, the mean processing speed as estimated by the WISC-V PSI was in the average range. Patients' performance ranged from impaired to very superior. Analysis of patients' performance found that 36.9% of the sample performed below the average range (impaired, borderline impaired, or low-average).

### ***Risk Factors***

The present study examined risk factors hypothesized to impact developmental outcomes including identification as a racial minority, parents' lack of higher-level education, lower SES as indicated by Medicaid insurance, genetic heritability as indicated by family history of ADHD, and any prenatal exposure to substance (including tobacco, alcohol, or other illicit substances). Each of these risk factors were coded based on caregiver endorsement on the patient history questionnaire and coded 1 = *yes* if they endorsed a particular risk factor, and 0 = *no* if they denied the presence of that risk factor. See Tables 2 and 3 for additional information. Regarding parent education level, if the respondent indicated that neither caregiver had higher level education (defined as anything above a high school diploma, certificate, or GED), that



participant was coded as having the risk factor (yes) of parents' lacking higher level education; if either caregiver was reported to have higher level education, then the participant was coded as not having the risk factor (no) of parents' lacking higher level education.

**Total Risk Factors.** In keeping with Sameroff (2009), a cumulative risk variable was created from the sum of endorsed risk factors, including identification as a racial minority, parents' lack of higher-level education, Medicaid insurance, genetic heritability (family history of ADHD), and in-utero exposure to substances, for a possible score ranging from 0 to 5. In the current sample, this cumulative risk score was able to be calculated for  $n = 128$ , with a mean of 1.58, and an SD of 1.15. There was not a significant difference in mean cumulative risk between the LAMP ( $M = 1.66$ ,  $SD = 1.09$ ) and term-born groups ( $M = 1.53$ ,  $SD = 1.18$ ),  $t(126) = -.61$ ,  $p = .54$ ,  $d = 1.15$ .

### ***Symptom Mitigating Factors***

Symptom mitigating factors were included as individual and combined covariates in the analyses. Firstly, caregiver-completed behavior rating scales, including the Behavior Assessment System for Children (3<sup>rd</sup> ed.; BASC-3) and Adaptive Behavior Assessment System (3<sup>rd</sup> ed.; ABAS-3), were utilized to look at two additional hypothesized symptom-mitigating factors, specifically social skills and adaptive skills, as measured using specific subscales (see Table 7). Clinicians could have decided to use either the BASC-3, the ABAS-3, or both, in their assessment of the child. The BASC-3 subscales are reported in  $t$ -scores with an average of 50 and SD of 10. ABAS-3 composites are reported in standard scores with a mean of 100 and SD of 15. (See Appendix E for summary of psychometric properties of behavior rating scales). Additionally, information regarding participation in early intervention services was pulled from the patient background and history questionnaire.

**Table 7***Descriptive Statistics for Symptom Mitigating Factors Derived from Behavior Rating Scales*

Construct	Subscale	N	M	SD	Range	$\alpha$
Social Skills						
	Social Skills (BASC-3)	118	44.67	10.86	10-66	.90-.93
	Social Composite (ABAS-3)	137	85.94	14.29	52-120	.94-.97
Adaptive Skills						
	Adaptive Skills (BASC-3)	118	41.21	10.93	10-72	.84-.91
	General Adaptive Composite (ABAS-3)	135	83.92	12.95	46-120	.98-.99

**Social Skills.** The Social Skills subscale of the BASC-3 (derived from caregiver response on 10 items) and the Social Composite from the ABAS-3 (derived from caregiver response on 26 items) were used as indicators of parent-reported social skills. On the BASC-3 Parent Rating Scales, patients' mean social skills fell in the "Acceptable"/WNL range, with scores ranging from "Clinically Significant" deficit to "Acceptable"/WNL. On the ABAS-3, patients' mean Social Composite, which includes leisure and social skills, fell in the "Below Average" range, with scores ranging from "Extremely Low" to "High." For this sample, social skills as measured on the BASC-3 or the ABAS-3 were lower than for the standardized population but still within the acceptable range. Per cutoffs in their respective manuals (Harrison & Oakland, 2015; Reynolds & Kamphaus, 2015), children whose caregivers reported their social skills to be in the average range or higher, on *either* measure, were coded as having Intact Social Skills (i.e., coded 1 = *yes*); anyone with a score below this cutoff was coded as lacking this symptom mitigating factor (i.e., coded 0 = *no*). More specifically, caregivers who rated social skills at/above a *t*-

score of 40 on the BASC-3, or at/above a standard score of 90, were coded as having intact social skills. Only 37.9% (64/169) of the sample were identified as having intact social skills by one or both measures.

**Adaptive Skills.** Adaptive skills were assessed using the General Adaptive Composite (GAC) score from the ABAS-3 (derived from caregiver response across all subscales/211 items) and the Adaptive Skills composite of the BASC-3 (derived from caregiver response across all adaptive scales/46 items). On the BASC-3 Parent Rating Scales, mean adaptive skills fell in the “Acceptable”/WNL range although lower than the population mean, with scores ranging from “Clinically Significant” deficit to “Acceptable”/WNL. On the ABAS, patients’ mean Global Adaptive Composite, which includes conceptual, social, and practical skills, fell in the “Below Average” range, with scores ranging from “Extremely Low” to “High.” Per cutoffs in their respective manuals (Harrison & Oakland, 2015; Reynolds & Kamphaus, 2015), children whose caregivers reported their adaptive skills to be in the WNL/average range or higher, on either measure, were coded as having Intact Adaptive Skills (i.e., coded 1 = *yes*); anyone with a score below this cutoff was coded as lacking this symptom mitigating factor (i.e., coded 0 = *no*). More specifically, caregivers who rated adaptive skills at/above a *t*-score of 40 on the BASC-3, or at/above a standard score of 90, were coded as having intact adaptive skills. Only 30.2% (51/169) of the sample were identified as having intact adaptive skills by one or both measures.

**Participation in Early Intervention.** Participation in an early intervention program was also included as a mitigating factor in these analyses because the research literature suggests that participation in early intervention services positively impacts cognitive development (Blauw-Hospers et al., 2007; Spittle et al., 2015; Vanderveen et al., 2009). Based on caregiver report on the patient history questionnaire, information was gathered regarding participation in Early On

(social, health, and educational intervention services for children, birth to age 3 years, who have developmental delays or are at risk for delays due to certain health conditions), speech therapy (SLP), occupational therapy (OT), and physical therapy (PT), at/before the age of 5 years. In the present sample, 13.6% of participants had participated in Early On, 21.3% had received early SLP, 7.7% had received early OT, and 5.3% had received early PT (see Table 2). A combined variable was created, called Participation in Early Intervention, and was coded yes/no if the patient had received any of those early intervention services.

***Total Symptom Mitigating Factors.*** In keeping with Sameroff (2009), a total symptom mitigating score was created from the sum of endorsed symptom mitigating factors which included participation in early intervention services, intact social skills, intact adaptive skills. Possible scores for Total Symptom Mitigating Factors ranged from 0 to 3. In the current sample, this score was able to be calculated for  $n = 151$ , with a mean of 1.21, and an  $SD$  of 1.16. There was no significant difference in mean total symptom mitigating factors between LAMP ( $M = 1.38$ ,  $SD = 1.24$ ) and term-born groups ( $M = 1.09$ ,  $SD = 1.09$ );  $t(149) = -1.49$ ,  $p = .13$ ,  $d = 1.15$ .

### **Power Analyses**

A priori power analyses were conducted, and it was determined that approximately 100 participants in the subject group and the control group would ensure adequate sample size and statistical power (200 participants). A sample size of approximately 100 participants was deemed appropriate because the methodology of the study entailed running a moderation model that would include up to six predictors (risk factors and symptom mitigating factors, as well as the primary independent variable); the other analyses included an equal number of or fewer predictors and were adequately powered at less than 100 per group.

Regarding post-hoc power analyses, effect sizes were provided for all results. Pearson correlations are described as small if the value of  $r$  ( $|r|$ ) is between .1 and .29, medium if  $|r|$  is between .3 and .49, and large if  $|r|$  is equal to or greater than .5 (J. Cohen, 1988). Cohen's  $d$  was presented for all  $t$ -tests, and partial eta squared for all regression analyses. Eta-square are described using Cohen's (1988) conventions for small, medium, and large effect sizes, which are .01, .06, and .14, respectively.

## Chapter 4: Results

### Preliminary Data Analyses

#### *Covariate Analysis*

Pearson correlations and *t*-tests were calculated to examine the impact of ADHD medication as a potential covariate on outcomes (symptom severity as indicated by caregiver report on behavior rating scales and performance-based measures of neurocognitive functioning). Significant group differences were found in caregiver report on behavior rating scales between those children who were prescribed ADHD medication ( $n = 63$ , 37% of total sample) and those who did not have a prescription for ADHD medication. As such, ADHD medication was accounted through partial correlational analyses and as a control on regression analyses. Owing to the sample size, it was not possible to run separate *t*-test analyses subdividing the sample by whether they had a current prescription for ADHD medication. Regarding performance-based measures of neurocognitive functioning, no significant group differences were found based on whether patients had taken ADHD medication on the day of evaluation.

Additional covariates are considered in the primary and experimental analyses. More specifically, the present study examined the impact of risk factors (e.g., minority status, lack of parental higher-level education, Medicaid status, genetic heritability, and prenatal exposure to substances) and potential symptom mitigating factors (e.g., participation in early intervention services, intact social skills, and intact adaptive skills) on ADHD diagnosis (determined by neuropsychologist), ADHD symptom severity (determined by caregiver report on behavior rating scales), and neurocognitive functioning (child performance on neuropsychological measures).

Regarding risk factors, chi-square tests were run to examine difference between expected and observed risk factors by LAMP status (yes = LAMP, no = term-born) to see if risk factors

were endorsed at a higher rate among LAMP children. In the current sample, none of the risk factors were more likely to be found in the history of children born LAMP than among term-born children. Regarding symptom mitigating factors, chi-square tests were run to examine the relationship between symptom mitigating factors and LAMP status (yes = LAMP, no = term-born) to see if symptom mitigating factors were endorsed at a higher rate among LAMP children. There was a significant relationship between participation in early intervention services and LAMP status,  $X^2(1, N = 169) = 4.78, p = .04$ , Cramer's  $V = .17$ . Rate of participation in early intervention services was significantly different between LAMP children (36.2%) and term-born children (21.0%), in the current sample. Intact social and adaptive skills were not significantly different between LAMP and term-born children.

Regarding Hypotheses 4a and 8b, regression diagnostics were run for all relevant variables in the regression models to evaluate assumptions including linearity, homoscedasticity, normality, and independence. From the Loess curve, scatterplots demonstrated that the relationship of standardized predictors to residuals was roughly linear (around zero) and therefore linearity and homoscedasticity assumptions were satisfied. P-P plots provided visual confirmation of generally normal distributions. Furthermore, skewness and kurtosis analyses were run to check for normality. Notably, the Conners-3 Inattention subscale was slightly negatively skewed (-1.06), and the Conners-3 Hyperactivity/Impulsivity and the Conners-3 *DSM* ADHD Hyperactive/Impulsive subscales showed slight platykurtik (-1.07 and -1.14, respectively). Cook's distance was used to assess for the presence of influential outliers. None were found, all Cook's distance values fell well below the recommended cutoff of 1 (Cook & Weisberg, 1982), and observations were independent.

## Analyses of Primary Study Hypotheses

### *Behavior Rating Scales*

**ADHD Diagnosis and Symptom Severity as Outcome—Hypothesis 1.** It was predicted that there would be a higher prevalence of ADHD and greater ADHD-related symptom severity among lower GA-groups, specifically LAMP, compared to children born at term (Hypothesis 1a-d). *T*-tests, chi-square tests, bivariate Pearson correlation coefficients (Table 7), and ANOVA analyses (Table 8) were used to examine the relationship between GA and diagnostic status (yes/no ADHD as determined by a neuropsychologist), and the symptom severity as indicated by parent report of attention problems/inattention (BASC-3 Attention Problems subscale; Conners-3 Inattention subscale), hyperactivity/impulsivity (BASC-3 Hyperactivity subscale; Conners-3 Hyperactivity/Impulsivity subscale), and ADHD problems (Conners-3 ADHD Inattentive subscale; Conners-3 ADHD Combined subscale). Higher scores on parent rating forms of symptom severity indicate greater problems.

*1a.* A *t*-test was used to determine whether the average gestational age (GA) was different between those children diagnosed with ADHD and those children who were not diagnosed with ADHD. Those who received an ADHD diagnosis ( $M = 37.35$ ,  $SD = 2.29$ ) had a comparable average GA as those who did not receive an ADHD diagnosis ( $M = 37.54$ ,  $SD = 2.26$ ),  $t(167) = .44$ ,  $p = .66$ ,  $d = 2.28$ . These results suggest that, in this sample, GA is not a significant predictor to ADHD diagnosis; children with and without ADHD had comparable mean GAs.

*1b.* A chi-square test was used to examine the difference between expected and observed rates of ADHD diagnosis (yes/no) by GA as a categorical variable (late preterm, moderate preterm, early-term, and full-term). There was not a significant relationship between GA group



and ADHD diagnostic status,  $\chi^2(3, N = 169) = .66, p = .88$ , Cramer's  $V = .06$ . ADHD rates were not significantly different across moderate preterm (76.5%), late preterm (80.8%), early term (74.2%), or term-born groups (76.9%) in the current sample.

*Ic.* Because of observed differences between those with a current prescription for ADHD medication and those without one on parent completed behavior rating forms, a partial correlation was completed to account for the impact of ADHD medication on parents' perceptions of attention difficulties. Results are presented in Table 8.

**Table 8**

*Correlations Between GA and Symptom Severity as indicated by Caregiver Report on Behavior Rating Scales*

Construct	Measure	Bivariate	Partial
Inattention	BASC-3 Attention Problems (n = 118)	-.01	-.03 [-.19, .12]
	Conners-3 Inattention (n = 111)	-.16 <sup>t</sup>	-.18 <sup>t</sup> [-.32, -.03]
	Inattention Combined Measure (n = 155)	-.09	-.10 [-.23, .43]
Hyperactivity	BASC-3 Hyperactivity (n = 118)	.06	.04 [-.12, .19]
	Conners-3 Hyperactivity/Impulsivity (n = 111)	-.15	-.17 <sup>t</sup> [-.32, -.01]
	Hyperactivity Combined Measure (n = 155)	-.09	-.11 [-.23, .28]
DSM ADHD Problems	Conners-3 DSM ADHD Inattentive	-.17 <sup>t</sup>	-.18 <sup>t</sup> [-.37, .02]
	Conners-3 DSM ADHD Hyperactive/Impulsive	-.15	-.17 <sup>t</sup> [-.35, .01]

<sup>t</sup> $p \leq .10$  level (2-tailed), BCa bootstrap 90% CIs reported in brackets

The partial correlations between GA and several behavior rating subscales (Conners-3 Inattention, Conners-3 Hyperactivity/Impulsivity, Conners-3 *DSM* ADHD Inattentive, and Conners-3 *DSM* ADHD Hyperactivity/Impulsivity), including current prescription for ADHD medication as a covariate, were found to be marginally significantly negatively related. After adjusting for the effect of a current prescription for ADHD medication on parent report of ADHD symptom severity, the relationship was slightly strengthened across all Conners-3 subscales. Inattention and hyperactivity/impulsivity, as captured by the Conners-3 Parent rating form, shows a marginally significant relationship with GA, both before and after controlling for the impact of a current prescription for ADHD medication; lower GA is related to higher inattention and hyperactivity/impulsivity, and higher GA was related to lower symptom report. Symptom severity for inattention and hyperactivity on the BASC-3 showed a completely non-significant relationship with GA.

*1d.* Based on the results of Hypothesis 1c, an ANCOVA was used to determine if there were statistically significant differences between LAMP and term-born children in attention problems, specifically those measured by the Conners-3, while controlling for current prescription for ADHD medication. The covariate, current prescription for ADHD medication, was significantly related to parents' report of symptom severity across all subscales of the Conners-3. There was also a marginally significant effect of LAMP history on parents' report of symptom severity as indicated by the Inattention and Hyperactivity/Impulsivity subscales of the Conners-3 (see Table 9). Interestingly, there were no significant effects of GA on parents' report of symptom severity as indicated by the *DSM* ADHD subscales of the Conners-3 after controlling for the effect of current prescription for ADHD medication. Having a current prescription for ADHD medication and a history of LAMP were significant and marginally

significant predictors for greater caregiver report of ADHD-related symptom severity as measured by the Conners-3 Inattention and the Hyperactivity/Impulsivity subscales.

**Table 9***ANCOVA for Conners-3 by GA with Current Prescription for ADHD Medication as Covariate*

Subscale	Source	SS	df	MS	F	$\eta^2$
Inattention	Current prescription for ADHD medication (covariate)	1016.90	1	1016.90	8.17**	.97
	LAMP status	412.82	1	412.82	3.32 <sup>t</sup>	.03
	Error	13439.19	108	124.44		
Hyperactivity/Impulsivity	Current prescription for ADHD medication (covariate)	2183.82	1	2183.82	9.91**	.08
	LAMP status	803.65	1	803.65	3.65 <sup>t</sup>	.03
	Error	23799.62	108	220.37		
<i>DSM</i> ADHD Inattentive	Current prescription for ADHD medication (covariate)	676.66	1	676.66	4.58*	.05
	LAMP status	372.78	1	372.78	2.52	.03
	Error	14038.033	95	147.77		

**Table 9** *continued*

Subscale	Source	SS	df	MS	F	$n^2$
<i>DSM</i> ADHD Hyperactive/Impulsive	Current prescription for ADHD medication (covariate)	2121.75	1	2121.75	9.56**	.09
	LAMP status	498.06	1	498.06	2.24	.02
	Error	21082.82	95	221.92		

<sup>t</sup> $p \leq .10$  level (2-tailed)

\* $p \leq .05$  level (2-tailed)

\*\* $p \leq .01$  level (2-tailed)

**Risk Factors Impacting Diagnosis and Symptom Severity—Hypothesis 2.** This hypothesis examined the relationship between several risk factors (race, Medicaid status, parent educational level, genetic heritability, prenatal exposure to substances) and ADHD in the sample (Hypotheses 2a-b). Both the global diagnosis of ADHD as determined by a neuropsychologist and the severity of ADHD symptoms as reported by the parents were examined in relation to these risk factors. *T*-tests and chi-square tests were used to examine the relationship between GA and diagnostic status (yes/no ADHD), and the symptom severity as indicated by parent report of attention problems/inattention (BASC-3 Attention Problems subscale; Conners-3 Inattention subscale), hyperactivity/impulsivity (BASC-3 Hyperactivity subscale; Conners-3 Hyperactivity/Impulsivity subscale), and ADHD problems (Conners-3 ADHD Inattention subscale; Conners-3 ADHD Combined subscale).

**2a.** Chi-square tests were run to examine difference between expected and observed risk factors (yes/no) based on diagnostic status (yes/no ADHD). As can be seen in Table 10, no significant differences were found between expected and observed rates of ADHD diagnosis by risk factor.

**Table 10***Chi-Square Tests Examining Relationship Between Risk Factors and ADHD Diagnostic Status*

	Racial Minority Status	Parents Lacking Higher-Level Education	Medicaid Insurance	Family History of ADHD	In-utero Exposure to Substances
n (Yes:No)	68:101	37:91	62:105	87:82	22:147
$X^2$	.40	1.82	1.74	2.22	2.19
$p$	.53	.18	.19	.14	.14
Cramer's V	.05	.12	.10	.12	.11

**2b.** *T*-tests were run to examine whether mean symptom severity as indicated by caregivers' report on behavior rating scales (BASC-3 Attention Problems, Conners-3 Inattention, BASC-3 Hyperactivity, Conners-3 Hyperactivity/Impulsivity, Conners-3 *DSM* ADHD Inattention, Conners-3 *DSM* ADHD Hyperactive/Impulsive) differed between those who endorsed risk factors (Yes) and those who did not (No). Risk factors included identification as a racial minority, parent's lacking higher-level education, Medicaid insurance, family history of ADHD, and/or prenatal exposure to substances.

As can be seen in Table 11, those who identified as a racial minority had a marginally significantly higher mean Conners-3 Inattention problems as those who identified as White. Overall, being a racial minority was not related to higher symptom report on measures of attention/inattention, hyperactivity/impulsivity, or ADHD problems; notably, the Conners-3

Inattention measure may have captured marginally significant variance attributable to racial identity compared to other measures/subscales.

**Table 11**

*Comparing Mean Symptom Severity by Minority Status*

Subscale	Yes (n = 68)	No (n = 101)	t	d
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	65.91 (10.33)	63.75 (9.91)	-1.12	-.22
Conners-3 Inattention	79.63 (9.08)	75.58 (13.08)	-1.92 <sup>t</sup>	-.35
BASC-3 Hyperactivity	62.72 (14.65)	63.16 (13.52)	0.17	.03
Conners-3 Hyperactivity/Impulsivity	75.45 (14.41)	71.63 (16.38)	-1.29	-.25
Conners-3 <i>DSM</i> ADHD Inattentive	76.98 (12.34)	74.57 (12.60)	-0.94	-.19
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	74.24 (14.64)	70.21 (16.28)	-1.26	-.26

<sup>t</sup>p < .10 level (2-tailed)

The absence of parental higher-level educational attainment was not related to parents' report of behavior rating scales (see Table 12).



**Table 12***Comparing Mean Symptom Severity by Parents Lack of Higher-Level Education*

Subscale	Yes (n = 37)	No (n = 91)	t	d
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	64.21 (10.30)	63.02 (10.37)	-0.48	-.16
Conners-3 Inattention	76.43 (15.99)	77.74 (10.86)	0.34	.08
BASC-3 Hyperactivity	60.63 (13.41)	60.84 (14.04)	0.07	.02
Conners-3 Hyperactivity/Impulsivity	73.74 (17.09)	71.66 (15.43)	-0.53	-.13
Conners-3 <i>DSM</i> ADHD Inattentive	73.33 (16.58)	76.10 (11.41)	0.70	.21
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	71.81 (16.20)	70.13 (15.51)	-0.41	-.11

Those with Medicaid insurance had marginally significantly higher mean Conners-3 *DSM-5* ADHD Inattention problems as those with commercial insurance. Overall, Medicaid insurance status was not related to parents' report of symptom severity on behavior rating scales measuring attention/inattention, hyperactivity/impulsivity, or ADHD problems; notably the Conners-3 ADHD *DSM-5* Inattention subscale may be more sensitive to socioeconomic factors such as Medicaid status (see Table 13).

**Table 13***Comparing Mean Symptom Severity by Medicaid Insurance Status*

Subscale	Yes (n = 62)	No (n = 105)	t	d
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	64.76 (9.82)	64.33 (10.36)	-0.22	-.04
Conners-3 Inattention	79.29 (10.61)	75.94 (12.32)	-1.48	-.29
BASC-3 Hyperactivity	63.67 (13.94)	62.72 (13.98)	-0.36	-.07
Conners-3 Hyperactivity/Impulsivity	75.22 (14.68)	72.34 (16.24)	-0.95	-.18
Conners-3 <i>DSM</i> ADHD Inattentive	78.02 (11.58)	73.75 (13.02)	-1.67 <sup>t</sup>	-.34
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	74.88 (14.23)	70.25 (16.34)	-1.45	-.30

<sup>t</sup>p < .10 level (2-tailed)

As can be seen in Table 14, those with a family history of ADHD were consistently reported by their caregivers as having higher ADHD-related symptom severity, as indicated by behavior rating scales, all with large effect sizes. Overall, family history of ADHD was a marginally significant predictor of attention/inattention, and a significant predictor of hyperactivity/impulsivity and ADHD problems.

**Table 14***Comparing Mean Symptom Severity by Genetic Heritability for ADHD*

Subscale	Yes (n = 87)	No (n = 82)	t	d
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	66.18 (8.98)	62.83 (10.92)	-1.83 <sup>t</sup>	-.34
Conners-3 Inattention	79.00 (11.70)	75.52 (11.36)	-1.59	-.30
BASC-3 Hyperactivity	67.57 (13.66)	58.28 (12.56)	-3.84 <sup>**</sup>	-.71
Conners-3 Hyperactivity/Impulsivity	76.00 (15.65)	70.27 (15.01)	-1.96 <sup>*</sup>	-.37
Conners-3 <i>DSM</i> ADHD Inattentive	78.62 (12.01)	71.74 (12.13)	-2.80 <sup>**</sup>	-.57
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	74.42 (15.61)	68.77 (15.28)	-1.80 <sup>t</sup>	-.37

<sup>t</sup>p < .10 level (2-tailed)<sup>\*</sup>p < .05 level (2-tailed)<sup>\*\*</sup>p < .01 level (2-tailed)

Those children who were exposed to substances in-utero were consistently reported by their caregivers as having higher ADHD-related symptom severity, as indicated by behavior rating scales, all with large effect sizes. Overall, in-utero exposure to substances was significantly related to problems with attention/inattention, hyperactivity/impulsivity, and ADHD problems (see Table 15).

**Table 15***Comparing Mean Symptom Severity by Prenatal Exposure to Substances*

Subscale	Yes (n = 22)	No (n = 147)	<i>t</i>	<i>d</i>
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	69.08 (8.95)	64.02 (10.11)	-1.66 <sup>t</sup>	-.51
Conners-3 Inattention	82.94 (8.71)	76.43 (11.82)	-2.10*	-.57
BASC-3 Hyperactivity	67.92 (15.84)	62.44 (13.62)	-1.30	-.40
Conners-3 Hyperactivity/Impulsivity	84.00 (10.77)	71.52 (15.60)	-3.99**	-.83
Conners-3 <i>DSM</i> ADHD Inattentive	81.93 (9.89)	74.55 (12.60)	-2.08*	-.60
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	81.93 (11.66)	70.27 (15.66)	-3.28**	-.77

<sup>t</sup>*p* < .10 level (2-tailed)\**p* < .05 level (2-tailed)\*\**p* < .01 level (2-tailed)

**Symptom Mitigating Factors—Hypothesis 3.** This hypothesis examined the relationship between several factors hypothesized to mitigate ADHD symptoms (e.g., history of intervention services, intact social skills, and intact adaptive skills) and symptom severity (Hypotheses 3a-b). Both the global diagnosis of ADHD and the severity of ADHD symptoms as reported by the parents were examined in relation to these symptom mitigating factors. *T*-tests and chi-square tests were used to examine the relationship between symptom mitigating factors and diagnostic status (yes/no ADHD), and the symptom severity as indicated by parent report of attention problems/inattention (BASC-3 Attention Problems subscale; Conners-3 Inattention

subscale), hyperactivity/impulsivity (BASC-3 Hyperactivity subscale; Conners-3 Hyperactivity/Impulsivity subscale), and ADHD problems (Conners-3 ADHD Inattentive subscale; Conners-3 ADHD Combined subscale).

**3a.** Chi-square tests were run to examine the difference between expected and observed symptom-mitigating factors (history of early intervention services, current social skills, and adaptive skills) based on diagnostic status (yes/no ADHD). Per results in Table 16, there were no significant differences found between expected and observed rates of symptom mitigating factors by ADHD diagnostic status.

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**Table 16**

*Chi-Square Tests Examining Relationship Between Symptom Mitigating Factors and ADHD*

*Diagnostic Status*

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	Participated in Early Intervention	Intact Social Skills	Intact Adaptive Skills
n (Yes/No)	46:123	64:105	51:118
$X^2$	1.15	.08	.50
$p$	.28	.77	.48
Cramer's V	.08	.02	.05

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**3b.** *T*-tests were run to examine mean symptom severity as indicated by parents' report on behavior rating scales (BASC-3 Attention Problems, Conners-3 Inattention, BASC-3 Hyperactivity, Conners-3 Hyperactivity/Impulsivity, Conners-3 *DSM* ADHD Inattention, Conners-3 *DSM* ADHD Hyperactive/Impulsive) based on symptom mitigating factors, including

history of participation in early intervention services, intact social skills, and intact adaptive skills.

In this sample, participation in early intervention services was not related to symptom severity of attention/inattention, hyperactivity/impulsivity, or ADHD problems (see Table 17, below).

**Table 17**

*Comparing Mean Symptom Severity by History of Early Intervention Services*

Subscale	Yes (n = 46)	No (n = 123)	<i>t</i>	<i>d</i>
	Mean (SD)	Mean (SD)		
BASC-3 Attention Problems	65.76 (9.73)	64.04 (10.23)	-0.84	-.17
Conners-3 Inattention	78.91 (9.66)	76.72 (12.35)	-0.91	-.19
BASC-3 Hyperactivity	65.50 (13.13)	61.99 (14.13)	-1.25	-.25
Conners-3 Hyperactivity/Impulsivity	76.21 (15.33)	72.09 (15.64)	-1.28	-.27
Conners-3 <i>DSM</i> ADHD Inattentive	78.03 (10.41)	74.58 (13.19)	-1.38	-.28
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	74.59 (15.65)	70.83 (15.62)	-11.09	-.24

Those whose caregivers rated them as having intact social skills had significantly lower mean BASC-3 Attention problems to those with below average social skills (see Table 18).

Those whose caregivers rated them as having intact social skills had significantly lower mean BASC-3 Hyperactivity problems to those with below average social skills. Lower reports of attention problems and hyperactivity, specifically on the BASC-3, were reported among those children whose caregivers indicated that they had intact (average or better) social skills. Taken

together, there is some evidence to suggest that intact social skills act as a buffer to caregivers' reports of attention problems and hyperactivity, as indicated on the BASC-3; this same relationship was not observed using the Conners-3 subscales.

**Table 18**

*Comparing Mean Symptom Severity by Intact Social Skills*

Subscale	Yes (n = 64)		No (n = 105)	
	Mean (SD)	Mean (SD)	t	d
BASC-3 Attention Problems	62.58 (10.29)	66.85 (9.39)	2.34*	.43
Conners-3 Inattention	77.32 (12.76)	77.40 (11.07)	0.04	.01
BASC-3 Hyperactivity	60.09 (13.41)	66.44 (13.76)	2.53*	.47
Conners-3 Hyperactivity/Impulsivity	72.97 (16.24)	73.49 (15.35)	0.17	.03
Conners-3 <i>DSM</i> ADHD Inattentive	74.78 (12.59)	76.10 (12.49)	0.50	.11
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	72.14 (15.99)	71.82 (15.57)	-0.10	-.02

\*p < .05 level (2-tailed)

Those whose caregivers rated them as having intact adaptive skills had significantly lower mean BASC-3 Attention problems to those whose adaptive skills were below expectation (see Table 19). Those whose caregivers rated them as having intact adaptive skills had significantly lower mean BASC-3 Hyperactivity problems to those whose adaptive skills were below expectation. Lower reports of attention problems and hyperactivity, specifically on the BASC-3, were reported among those children whose caregivers indicated that they had intact (average or better) adaptive skills.

**Table 19***Comparing Mean Symptom Severity by Intact Adaptive Skills*

Subscale	Yes (n = 51)		No (n = 118)	
	Mean (SD)	Mean (SD)	t	d
BASC-3 Attention Problems	61.43 (9.70)	66.90 (9.78)	3.02**	.56
Conners-3 Inattention	75.74 (12.55)	77.80 (11.40)	0.75	.18
BASC-3 Hyperactivity	60.04 (13.46)	65.25 (13.87)	2.05*	.38
Conners-3 Hyperactivity/Impulsivity	71.65 (15.86)	73.75 (15.58)	0.57	.13
Conners-3 <i>DSM</i> ADHD Inattentive	72.83 (11.88)	76.45 (12.61)	1.22	.29
Conners-3 <i>DSM</i> ADHD Hyperactive/Impulsive	70.87 (16.20)	72.27 (15.57)	0.37	.09

\*p &lt; .05 level (2-tailed)

\*\*p &lt; .01 level (2-tailed)

**Additional Factors Impacting Relationship Between GA, Cumulative Risk and Total Symptom Mitigating Factors, and Symptom Severity—Hypothesis 4.** This study also examined the additive predictive power of risk factors and symptom mitigating factors with GA on ADHD symptom severity to clarify what amount of variance could be accounted for by each, while controlling for current prescription for ADHD medication. Findings in Hypothesis 1 indicated that GA was marginally significantly related to symptom severity as measured by the Conners-3. Findings in Hypothesis 2 indicated a relationship between several risk factors and parent reported symptom severity: being a racial minority was marginally significantly related to Inattention, Medicaid insurance status was marginally significantly related to *DSM* ADHD Inattentive symptoms, family history of ADHD (i.e., genetic heritability) was marginally



significantly related to parents' report of Hyperactivity, and in-utero exposure was significantly related to parents' report across all subscales of the Conners-3. Findings in Hypothesis 3 indicated a significant relationship between symptom mitigating factors and symptom severity only when using the BASC-3 as a measure. Taken together, multiple linear regression was appropriate to examine the relationship between GA and risk factors on symptom severity as measured by subscales on the Conners-3.

*4a.* A stepwise linear regression was conducted predicting Conners-3 Inattention problems from current prescription for ADHD medication (control), GA, and racial minority status. In Step 1, current medication accounted for 7% of the variance. In Step 2, adding GA accounted for an additional 3%. In Step 3, being a racial minority accounted for an additional 3%. Overall, the regression was significant (see Table 20). Of the predictors investigated, current prescription for ADHD medication and racial minority status were significant, and GA was marginally significant. The total model accounted for 12.6% of the variance observed in caregiver report of Inattention (Conners-3).

**Table 20***Predicting Conners-3 Inattention by GA and Racial Minority Status (n = 111)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					0.07**
Current Prescription for ADHD Medication	6.55	2.34	0.26**	2.80	
Step 2					0.03 <sup>t</sup>
Current Prescription for ADHD Medication	6.65	2.32	0.26**	2.87	
	-				
GA	0.86	0.47	-0.17 <sup>t</sup>	-1.84	
Step 3					0.03*
Current Prescription for ADHD Medication	6.75	2.29	0.27**	2.95	
GA	-.84	.46	-.16 <sup>t</sup>	-1.82	
Racial Minority Status	4.10	2.11	.18*	1.95	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*p ≤ .05 level (2-tailed)

\*\*p ≤ .01 level (2-tailed)

A stepwise linear regression was conducted predicting Conners-3 *DSM* ADHD Inattentive problems from current prescription for ADHD medication (control), GA, and Medicaid status. In Step 1, current medication accounted for 6% of the variance. In Step 2, adding GA accounted for an additional 3%. In Step 3, having Medicaid insurance accounted for an additional 1%. Overall, the regression was significant (see Table 21). Of the predictors investigated in the full model, only current prescription for ADHD medication was significant.

Though GA and Medicaid status were marginally significantly related to inattention in bivariate correlational analyses, it was current prescription for ADHD medication *rather* than GA or Medicaid status that was found to account for significant variance in predicating Conners-3 *DSM* ADHD Inattentive problems in the regression model. The total model accounted for 8.9% of the variance observed in caregiver report of *DSM* ADHD Inattentive problems (Conners-3).

**Table 21**

*Predicting Conners-3 DSM ADHD Inattentive Problems by GA and Medicaid Status (n = 96)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.06*
Current Prescription for ADHD Medication	6.29	2.70	.23*	2.33	
Step 2					.03*
Current Prescription for ADHD Medication	6.24	2.68	.23*	2.33	
GA	-.87	.54	-.16	-1.60	
Step 3					.01*
Current Prescription for ADHD Medication	5.74	2.73	.21*	2.10	
GA	-.75	.56	-.14	-1.36	
Medicaid Status	2.51	2.62	.10	.96	

\* $p \leq .05$  level (2-tailed)

A stepwise linear regression was conducted predicting Conners-3 Hyperactivity from current prescription for ADHD medication (control), GA, and family history of ADHD. Overall, the regression was significant (see Table 22). Of the predictors investigated in the full model, current prescription for ADHD medication and GA were significant. Though family history of

ADHD was significantly related to parents' report of hyperactivity on the Conner-3 in bivariate correlational analyses, it was current prescription for ADHD medication and GA that accounted for significant variance in the combined regression model. The total model accounted for 12.7% of the variance observed in caregiver report of Hyperactivity (Conners-3).

**Table 22**

*Predicting Conners-3 Hyperactivity/Impulsivity by GA and Family History of ADHD (n = 111)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.08**
Current Prescription for ADHD Medication	9.60	3.12	.28**	3.08	
Step 2					.03**
Current Prescription for ADHD Medication	9.73	3.09	.29**	3.15	
GA	-1.09	.62	-.16 <sup>t</sup>	-1.74	
Step 3					.02**
Current Prescription for ADHD Medication	9.04	3.10	.27**	2.92	
GA	-1.09	.62	-.16 <sup>t</sup>	-1.76	
Family History of ADHD	4.62	2.84	.15	1.63	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*\*p ≤ .01 level (2-tailed)

Four regressions were run to examine how much variance was accounted for by in-utero exposure to substances above that which was accounted for by current prescription for ADHD medication (control) and GA, on symptom severity as measured by the subscales of the Conners-3. All the regression models were significant (see Tables 23-26).

A stepwise linear regression was conducted predicting Inattention from current prescription for ADHD medication (control), GA, and in-utero exposure to any substances. In step 1, current medication accounted for 7% of the variance. In step 2, adding GA accounted for an additional 3%. In step 3, in-utero exposure to substances accounted for an additional 2%. Overall, the regression was significant (see Table 23). Interestingly, GA was the only marginally significant predictor of Inattention (in-utero exposure was not a significant predictor) after controlling for current prescription for ADHD medication; in contrast, GA was not a significant predictor in the full models predicting other dimensions of symptom severity such as Hyperactivity or *DSM* ADHD problems (see Tables 24-26). The total model accounted for 11.1% of the variance observed in caregiver report of Inattention (Conners-3).

**Table 23***Predicting Conners-3 Inattention by GA and In-Utero Exposure to Substances (n = 111)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.07**
Current Prescription for ADHD Medication	6.55	2.34	.26**	2.80	
Step 2					.03**
Current Prescription for ADHD Medication	6.65	2.32	.26**	2.87	
GA	-.86	.47	-.17 <sup>t</sup>	-1.84	
Step 3					.02**
Current Prescription for ADHD Medication	5.87	2.38	.23*	2.47	
GA	-.80	.47	-.16 <sup>t</sup>	-1.71	
In-utero Exposure to Substances	4.22	3.11	.13	1.36	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*p ≤ .05 level (2-tailed)

\*\*p ≤ .01 level (2-tailed)

In-utero exposure to substances accounted for significant variance in Conners-3 Hyperactivity/Impulsivity and *DSM* ADHD Hyperactive/Impulsive problems, above and beyond what was accounted for by current prescription for ADHD medication. As previously mentioned, GA was not a significant predictor of hyperactive symptom severity. Current prescription medication and in-utero exposure were significant predictors of hyperactive symptom severity in the full models, accounting for 14.8% and 14.5% of the variance in caregiver report of

Hyperactivity/Impulsivity and *DSM* ADHD Hyperactive/Impulsive problems (see Tables 24 and 26, respectively).

**Table 24**

*Predicting Conners-3 Hyperactivity/Impulsivity by GA and In-Utero Exposure to Substances (n = 111)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.08**
Current Prescription for ADHD Medication	9.60	3.12	.28**	3.08	
Step 2					.03**
Current Prescription for ADHD Medication	9.73	3.09	.29**	3.15	
GA	-1.09	.62	-.16 <sup>t</sup>	-1.74	
Step 3					.04**
Current Prescription for ADHD Medication	7.98	3.12	.24*	2.56	
GA	-.94	.61	-.14	-1.54	
In-utero Exposure to Substances	9.46	4.08	.21*	2.32	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*p ≤ .05 level (2-tailed)

\*\*p ≤ .01 level (2-tailed)

**Table 25**

*Predicting Conners-3 DSM ADHD Inattentive Problems by GA and In-Utero Exposure to Substances (n = 98)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.05*
Current Prescription for ADHD Medication	5.73	2.66	.21*	2.15	
Step 2					.03*
Current Prescription for ADHD Medication	5.74	2.63	.22*	2.18	
GA	-.93	.54	-.17 <sup>t</sup>	-1.73	
Step 3					.02*
Current Prescription for ADHD Medication	4.81	2.69	.18 <sup>t</sup>	1.79	
GA	-.86	.54	-.16	-1.60	
In-utero Exposure to Substances	5.48	3.58	.15	1.53	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*p ≤ .05 level (2-tailed)



**Table 26***Predicting Conners-3 DSM ADHD Hyperactive/Impulsive Problems by GA and In-Utero**Exposure to Substances (n = 98)*

	b	SE	$\beta$	t	$\Delta R^2$
Step 1					.09**
Current Prescription for ADHD Medication	10.09	3.26	.30**	3.10	
Step 2					.02**
Current Prescription for ADHD Medication	10.11	3.24	.30**	3.12	
GA	-.93	.66	-.14	-1.41	
Step 3					.04**
Current Prescription for ADHD Medication	8.65	3.28	.26 <sup>t</sup>	2.64	
GA	-.82	.65	-.12	-1.25	
In-utero Exposure to Substances	8.63	4.37	.19*	1.98	

<sup>t</sup>p ≤ .10 level (2-tailed)

\*p ≤ .05 level (2-tailed)

\*\*p ≤ .01 level (2-tailed)

**4b.** Given the findings in Hypotheses 1-3, this hypothesis was no longer indicated, as there was no significant relationship found between GA and symptom severity as measured by the BASC-3 (Hypothesis 1), and symptom mitigating factors were only found to be related to the BASC-3 (Hypothesis 3).

### ***Neurocognitive Outcomes***

The first four hypotheses were related to ADHD diagnostic status (determined by neuropsychologist) and symptom severity (as indicated by caregiver report on behavior rating

scales). The following hypotheses investigated outcomes related to neurocognitive dysfunction theorized to underly the behavioral presentation associated with ADHD.

**Relationship Between GA and Neurocognitive Functioning—Hypothesis 5.** This hypothesis examined the relationship between GA and neurocognitive functioning (Hypotheses 5a-c). *T*-tests, bivariate Pearson correlation coefficients, and ANOVA analyses were used to examine the relationship between GA and overall intelligence *g*/IQ (WISC-V FSIQ), attention (CPT-2/CPT-3 and WISC-V Digit Span Forward), working memory (WISC-V Digit Span Backward and Digit Span Sequencing), executive functioning (D-KEFS TMT 4/Trails B, D-KEFS CWIT 3/NEPSY Inhibition, D-KEFS CWIT 4/NEPSY Inhibition with Switching), and processing speed (WISC-V PSI). Higher scores on measures of neurocognitive functioning indicate greater functioning except for the CPT-2/CPT-3, for which higher scores indicate a higher number of omissions (inattention) or commissions (impulsivity) and suggestive of greater attention-related dysfunction.

**5a.** Bivariate Pearson correlations were run to examine the relationship between GA and *g*/IQ, attention, working memory, executive functioning, and processing speed (Table 27). There was a marginally significant relationship between GA and WISC-V FSIQ,  $r(167) = .14, p \leq .10$ . All other measures of neurocognitive functioning showed a non-significant relationship with GA.

**Table 27***Bivariate Pearson Correlations Between GA and Neurocognitive Functioning Across Domains*

Construct	Measure	GA
<i>g</i> /IQ		
	WISC-V FSIQ	0.14 <sup>†</sup>
Attention		
	CPT-2/CPT-3 Omissions	0.01
	CPT-2/CPT-3 Commissions	-0.02
	WISC-V DSF	0.11
Working Memory (WM)		
	WISC-V DSB	0.08
	WISC-V DSS	0.01
Executive Functioning (EF)		
	Cognitive Flexibility (D-KEFS TMT4/Trails B, combined measures)	0.09
	Inhibition (D-KEFS CWIT3/NEPSY I, combined measures)	0.05
	Inhibition with Switching (D-KEFS CWIT4/NEPSY IS, combined measures)	0.01
Processing Speed		
	WISC-V PSI	0.07

<sup>†</sup> $p \leq .10$  level (2-tailed)

**5b.** An independent samples *t*-test was used to examine mean differences in neurocognitive performance between LAMP children and term-born children (see Table 28). All measures of neurocognitive functioning indicated comparable mean performance between LAMP children and term-born children.

**5c.** ANOVA analyses were planned to examine the relationship between GA group (moderately preterm, late preterm, early term, and full-term) and *g*/IQ, attention, working memory, executive functioning, and processing speed. Owing to the non-significant results in Hypotheses 5a-b, an ANOVA was not indicated.

**Table 28***Comparing Mean Performance Across Neurocognitive Domains Between LAMP and Term-Born Children*

Construct	Measure	LAMP	Term-Born	t
		(n = 69)	(n = 100)	
		Mean (SD)	Mean (SD)	
<i>g</i> /IQ				
	WISC-V FSIQ	91.65 (12.16)	94.56 (13.80)	1.41
Attention				
	CPT-2/CPT-3 Omissions	59.32 (13.91)	60.72 (15.47)	0.60
	CPT-2/CPT-3 Commissions	52.87 (7.99)	53.40 (7.75)	0.43
	WISC-V DSF	8.18 (2.53)	8.45 (2.43)	0.71
Working Memory				
	WISC-V DSB	8.31 (2.55)	8.54 (3.22)	0.52
	WISC-V DSS	8.81 (2.89)	8.81 (3.14)	0.00

**Table 28** *continued*

		LAMP	Term-Born	
		(n = 69)	(n = 100)	
Construct	Measure	Mean (SD)	Mean (SD)	t
Executive Functioning				
	Cognitive Flexibility (D-KEFS TMT4/Trails B, combined measures)	8.28 (3.49)	8.32 (4.11)	0.06
	Inhibition (D-KEFS CWIT3/NEPSY I, combined measures)	8.76 (3.33)	8.82 (3.47)	0.10
	Inhibition with Switching (D-KEFS CWIT4/NEPSY IS, combined measures)	9.30 (3.45)	9.78 (2.77)	0.83
Processing Speed				
	WISC-V PSI	93.16 (14.53)	95.64 (14.97)	1.07

### **Additional Factors Impacting Relationship Between GA and Neurocognitive**

**Functioning—Hypothesis 6.** This hypothesis examined the relationship among identified risk factors, symptom-mitigating factors, and neurocognitive functioning (Hypotheses 6a-6d).

Bivariate Pearson correlations and multiple linear regression analyses were used to analyze the relationship between cumulative risk (sum of endorsed risk factors, including identification as a racial minority, parents' lack of higher level education, SES as indicated by Medicaid Insurance, family history of ADHD, and in-utero exposure to substances) and total symptom mitigating factors (sum of endorsed symptom mitigating factors, including participation in early intervention services, intact social skills, intact adaptive skills) with *g*/IQ (WISC-V FSIQ), attention (CPT-2/CPT-3 Omissions and Commissions, WISC-V DSF), working memory (WISC-V DSB and DSS), executive functioning (D-KEFS TMT 4/Trails B, D-KEFS CWIT 3/NEPSY I, D-KEFS CWIT 4/NEPSY IS), and speed of information processing (WISC-V PSI). In total, 10 bivariate correlations were run to test if cumulative risk is related to neurocognitive functioning, and 10 for total mitigating factors.

**6a.** Bivariate correlational analysis were conducted between cumulative risk (race, SES, parent educational level, genetic heritability, prenatal exposure) and performance across ADHD-related neurocognitive domains (Table 29). There was a significant negative relationship between overall *g*/IQ (WISC-V FSIQ) and cumulative risk, indicating higher endorsement of risk factors was related to lower overall cognitive functioning. Regarding working memory, a marginally significant negative relationship was found between WISC-V DSB and cumulative risk, and a significant negative relationship between WISC-V DSS and cumulative risk (higher endorsement of risk factors related to lower performance on tasks of working memory). Lastly, the combined measure of cognitive flexibility was found to be significantly negatively related to

cumulative risk, indicating higher endorsement of risk factors was related to lower executive functioning. Most neurocognitive domains of interest showed some negative relationship to cumulative risk (the exception being processing speed).

**6b.** Bivariate correlational analysis were conducted for total symptom-mitigating factors (history of early intervention services, intact social skills, and intact adaptive skills) and performance across ADHD-related neurocognitive domains (Table 29). Across all measures of neurocognitive functioning, there was only one significant negative relationship found between WISC-V DSF and total symptom mitigating factors. This could suggest that participation in early intervention services and intact social and adaptive skills are paradoxically related to poorer attentional functioning. Further analyses were needed to understand this relationship; *t*-test and correlational analyses were used to look at the relationship between WISC-V DSF and each symptom mitigating factor as a standalone variable.

A *t*-test was used to determine if average performance on WISC-V DSF was different between those children who had received early intervention services, those with intact social skills, and those with intact adaptive skills. Those who participated in early intervention services had a lower average DSF ( $M = 7.37, SD = 2.30$ ) than those who did not participate in early intervention services ( $M = 8.70, SD = 2.43$ ),  $t(166) = 3.22, p < .005, d = 2.40$ . There were no significant differences in mean DSF based on intact social skills (yes/no) or adaptive skills (yes/no). These results suggest that it is not total symptom mitigating factors that are inversely related to poorer performance on a measure of brief attention (WISC-V DSF), but rather participation in early intervention services that may predict differences in basic attentional capacity.



**Table 29***Bivariate Pearson Correlations Between GA and Neurocognitive Functioning Across Domains*

Construct	Measure	Total Symptom	
		Cumulative Risk Score	Mitigating Factors
<i>g</i> /IQ			
	WISC-V FSIQ	-.24**	-0.05
Attention			
	CPT-2/CPT-3 Omissions	0.11	0.09
	CPT-2/CPT-3 Commissions	0.16 <sup>t</sup>	0.00
	WISC-V DSF	-0.11	-.18*
Working Memory			
	WISC-V DSB	-.18 <sup>t</sup>	-0.05
	WISC-V DSS	-.24 <sup>t</sup>	0.00
Executive Functioning			
	Cognitive Flexibility (D-KEFS TMT4/Trails B, combined measures)	-0.19*	-0.02
	Inhibition (D-KEFS CWIT3/NEPSY I, combined measures)	-0.10	-0.01
	Inhibition with Switching (D-KEFS CWIT4/NEPSY IS, combined measures)	-0.11	-0.01
Processing Speed			
	WISC-V PSI	-0.05	-0.10

**Table 29** *continued*

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<sup>†</sup> $p \leq .10$  level (2-tailed)

\* $p \leq 0.05$  level (2-tailed)

\*\* $p \leq 0.01$  level (2-tailed)

**6c.** GA was not found to be significantly correlated with neurocognitive outcomes (from Hypothesis 5 findings), and, therefore, hierarchical regression analyses to examine the relative contribution of cumulative risk with GA in predicting neurocognitive outcomes was no longer indicated.

**6d.** Similarly, hierarchical regression analyses were no longer indicated to examine the relative contribution of symptom mitigating factors with GA on neurocognitive outcomes.

## **Experimental Analyses**

### ***Moderation Models***

**Interaction Between GA and Additional Factors in Predicting Neurocognitive Outcomes—Hypothesis 7.** Moderation analyses were planned to examine the interaction effect of GA with cumulative risk factors and with total symptom mitigating factors in predicting neurocognitive outcomes. However, because there were no significant relationships between GA and performance across ADHD-related neurocognitive domains, these moderation analyses were no longer indicated.

**Interaction Between GA and Birthweight in Predicting Neurocognitive Outcomes—Hypothesis 8.** Experimental analyses were conducted to examine the interaction effect of birthweight and GA in predicting long-term behavioral and neurocognitive outcomes among LAMP children and term-born children (Hypotheses 8a-b).

**8a.** Moderation analyses were planned using birthweight (moderator) and GA in predicting symptom severity. Results from Hypothesis 1 indicated a marginally significant relationship between GA and symptom severity on the Conners-3 (Inattention subscale, Hyperactivity/Impulsivity subscale, *DSM* ADHD Inattentive subscale, and *DSM* ADHD Hyperactive/Impulsive subscale). Bivariate correlational analyses were run to look at the relationship between birthweight and symptom severity on the Conners-3; results were non-significant, making further moderation analysis unnecessary (see Table 30).

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**Table 30**

*Correlations Between Birthweight and Symptom Severity as Indicated by Caregiver Report on Conners-3, Controlling for Current Prescription for ADHD Medication*

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	Partial Correlation
Conners-3 Inattention	-.08 [-.28, .13]
Conners-3 Hyperactivity/Impulsivity	-.08 [-.29, .13]
Conners-3 <i>DSM</i> ADHD Inattentive	-.10 [-.33, .13]
Conners-3 <i>DSM</i> ADHD Hyperactivity/Impulsivity	-.12 [-.34, .09]

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**8b.** Moderation analyses were planned using birthweight (moderator) and gestational age in neurocognitive performance across each domain (attention, working memory, executive functioning, and processing speed). Results from Hypothesis 5 indicated a marginally significant relationship between GA and *g*/IQ (WISC-V FSIQ). Bivariate correlational analyses were run to look at the relationship between birthweight and FSIQ; there was a significant positive relationship between birthweight and FSIQ,  $r(163) = .20, p \leq .01$ , indicating that moderation analyses would be appropriate to look at the interaction between GA and birthweight in

predicting FSIQ. Incidentally, there was also a marginally significant positive relationship between birthweight and cognitive flexibility,  $r(136) = .16, p = .06$  (Table 31).

**Table 31**

*Bivariate Pearson Correlations Between Birthweight and Neurocognitive Functioning*

Construct	Measure	Birthweight
<i>g/IQ</i>		
	WISC-V FSIQ	.20*
<i>Attention</i>		
	CPT-2/CPT-3 Omissions	-.03
	CPT-2/CPT-3 Commissions	-.01
	WISC-V DSF	.09
<i>Working Memory</i>		
	WISC-V DSB	.05
	WISC-V DSS	.07
<i>Executive Functioning</i>		
	Cognitive Flexibility (D-KEFS TMT4/Trails B, combined measures)	.16 <sup>†</sup>
	Inhibition (D-KEFS CWIT3/NEPSY I, combined measures)	.05
	Inhibition with Switching (D-KEFS CWIT4/NEPSY IS, combined measures)	.05
<i>Processing Speed</i>		
	WISC-V PSI	.12

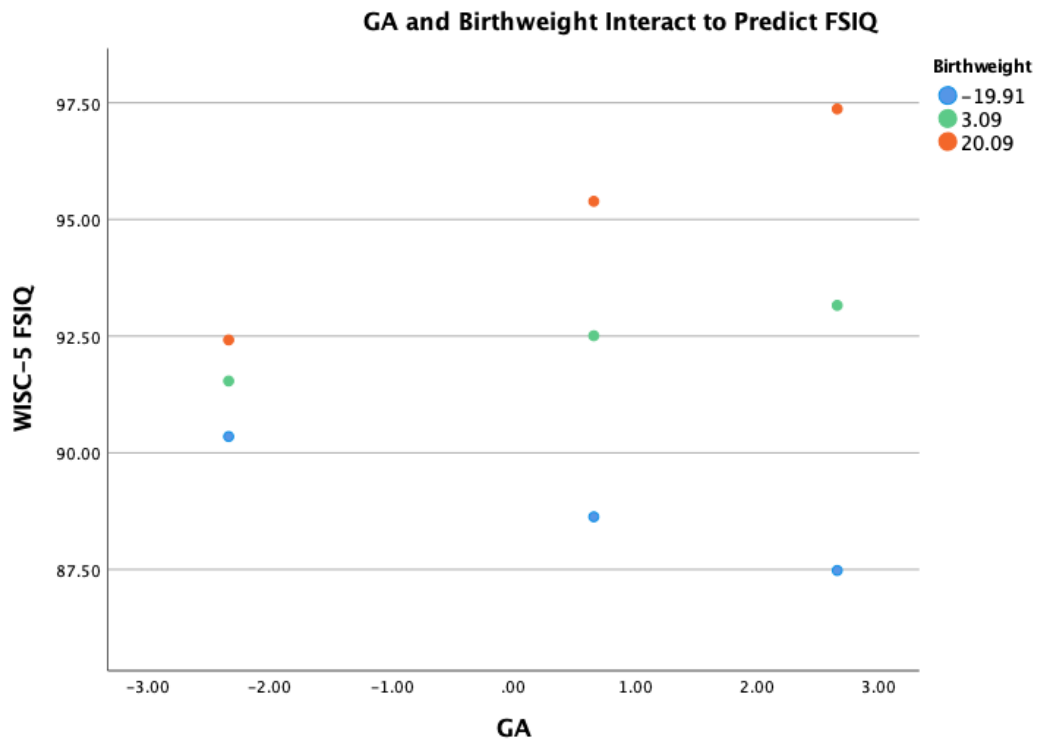
<sup>†</sup> $p \leq .10$  level (2-tailed)

\* $p \leq 0.05$  level (2-tailed)

Using mean-centered predictor variables, which were transformed into  $z$  scores with a mean of 0 and  $SD$  of 1, moderation analysis found a significant interaction effect,  $b = 0.04$ , 95% CI [0.00, 0.08],  $t = 1.96$ ,  $p = .05$ , indicating that the relationship between GA and FSIQ is moderated by birthweight (see Figure 1). Marginally significant transition points within the observed range of the moderator were found using the Johnson-Neyman method; the boundaries of the zone of marginal significance were -58.06 and 41.39. There was a marginally significant negative relationship between birthweight and FSIQ,  $b = -2.29$ , 95% CI [-4.93, .34],  $t = -1.72$ ,  $p = 0.09$ , until the threshold for marginal significance ended at -58.06; as birthweight increased, the relationship between birthweight and FSIQ remained non-significant until the value of birthweight was great enough (41.39),  $b = 1.82$ , 95% CI [-.31, 3.96],  $t = 1.69$ ,  $p = 0.09$ , at which point there was a marginally significant positive relationship between birthweight and FSIQ. Lower GA was related to lower birthweight, and children with this history were more likely to exhibit a similar FSIQ. As GA increases, the impact of birthweight on FSIQ becomes more apparent, with higher GA and higher birthweight predicting better neurocognitive outcomes, and higher GA with lower birthweight predicting poorer outcomes at a marginally significant level.

**Figure 1**

*Birthweight Moderates Overall Neurocognitive Functioning as Measured by the WISC-V FSIQ*



## Chapter 5: Discussion

### Summary and Discussion of Major Findings

Premature birth rates in the United States are among the highest of all developed nations (Howson et al., 2012). Of the estimated 15 million annual preterm births, more than 80% are born LAMP (34-36 weeks and 32-33 weeks; Howson et al., 2012; Mayo Foundation for Education and Research, 2017). It is well established in the literature that children born premature are at increased risk for developmental delays and later neurodevelopmental disorders, among them ADHD (Aylward, 2005, 2014; Vanderveen et al., 2009). However, much of the existing literature has focused on those children who are born very or extremely preterm ( $\leq 31$  weeks' gestation) or examined outcomes among all prematurity groups, even though most premature births are LAMP. Furthermore, by including those children who are born very or extremely premature, who are more likely to have more complex early medical histories and numerous comorbidities, the variance in neurocognitive outcomes includes outliers and medical confounds, making it very difficult to hypothesize about expected neurocognitive outcomes among most preterm children (LAMP). As such, the present study examined the question of whether ADHD prevalence, ADHD symptom severity, and/or ADHD-related neurocognitive deficits were higher among LAMP children than term-born children in a clinical sample of 8- to 12-year-old children. It is imperative that clinicians advise parents/caregivers regarding prevention strategies given risk and intervention strategies at the earliest signs of deviation from normative development; policy should similarly be informed by evidence-based best practice standards for prevention and intervention, as preterm children may be at higher risk for worse medical, academic, and socioemotional outcomes than their term-born peers.

Given the research that has found premature children to be at two- to three-fold risk for ADHD (American Psychiatric Association, 2013), it was hypothesized that this would remain true in the LAMP subset and there would be a higher prevalence of ADHD among children born LAMP than children who were term-born. ADHD diagnoses are given based on reported/observed behavior across multiple settings (see Appendix A for full diagnostic criteria). Behavior rating scales are very frequently used to get a standardized measure of social-emotional behavioral functioning and establish if ADHD-related behaviors deviate from developmental expectation to a clinically significant degree. Therefore, in addition to higher rates of ADHD, it was predicted that LAMP children would also exhibit greater symptom severity in attention problems/inattention, hyperactivity/impulsivity, and *DSM-5* ADHD-related behaviors as reported by their caregivers.

There are certain neuroanatomical and neurocognitive differences that would be expected given those behavior patterns observed in ADHD (i.e., brain-behavior relationships). Brain regions implicated in ADHD include the attention systems. In the case of ADHD, disruptions in the attentional systems are evident in several regions and networks involving the prefrontal cortex (PFC), anterior cingulate cortex, caudate, orbitofrontal cortex, cerebellum, frontal-striatal network, the dorsal frontoparietal system, and the ventral frontoparietal system. Notably, the PFC is the last area of the brain to reach maturation, though the infrastructure and networks connected to and communicating with the PFC begin to develop in-utero; thus, disruption of the gestational period in LAMP birth impacts early development of the PFC networks (Willcutt, 2010), and provides a neural foundational understanding of how preterm birth may operate as a risk factor for and predictor of ADHD. Aligned with those neuroanatomical differences in ADHD, there are expected neurocognitive deficits. In addition to accounting for IQ, ADHD evaluations



should include assessment of attention, working memory, executive functioning, and speed of information processing. As such, it was hypothesized that LAMP children would exhibit lower overall neurocognitive performance (*g*/IQ), diminished attentional capacity, and poorer performance on measures of working memory, executive functioning, and processing speed.

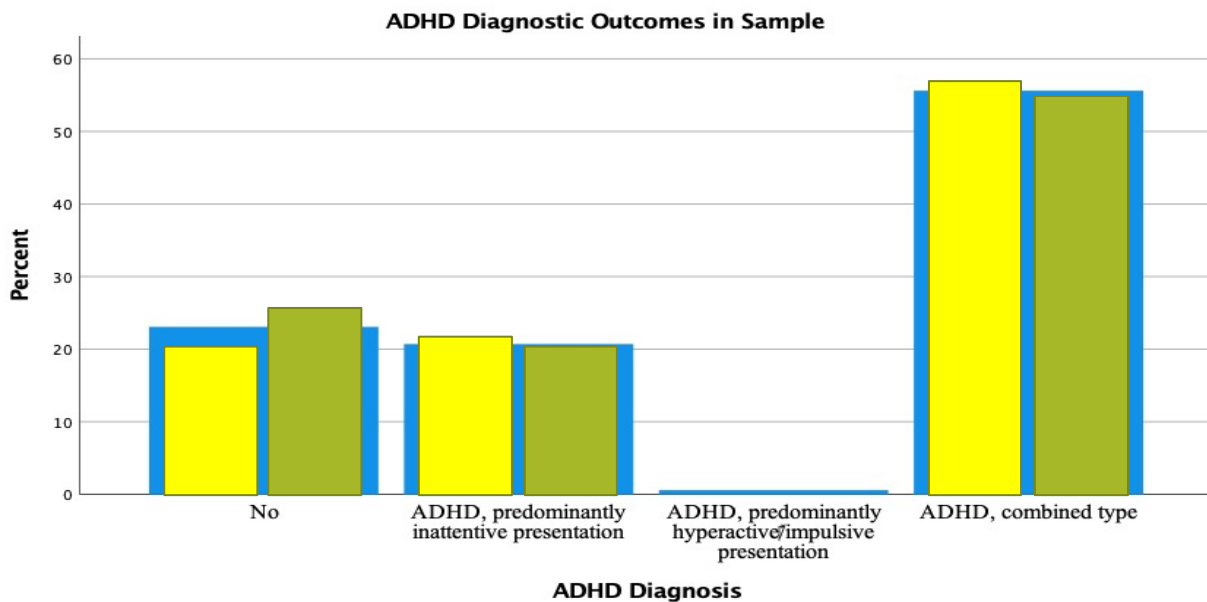
Brain-behavior relationships are further impacted by environmental factors and history (biopsychosocial processes). There are known factors that impact the likelihood of a child developing a neurodevelopmental disorder like ADHD. Though the primary focus of this study was on the relationship between LAMP birth and ADHD-related outcomes, the present study considered the ways in which other factors may impact or predict outcomes (multifinality and equifinality). In addition to preterm birth, genetic heritability is a strong predictor of ADHD. In-utero exposure to substances, SES, lower parental educational achievement, and racial minority status are all factors that also increase the risk for an ADHD diagnosis. It was hypothesized that these risk factors interact with LAMP status resulting in an increased likelihood of developing ADHD-related deficits by school age. In contrast, the literature also supports certain factors that may decrease the likelihood of an ADHD diagnosis, or in other words, reduce the impact of ADHD on day-to-day functioning. Participation in early intervention services is likely to have a positive impact on development. For example, ADHD is often associated with poor social skills and diminished adaptive skills, and children who have strength in these areas or have participated in social skills groups may experience reduced ADHD-burden, including symptom severity, reduced functional impairment, and lower rates of diagnosis. Collectively, participation in early intervention services and intact social and adaptive skills were hypothesized to be symptom mitigating factors that would be related to lower ADHD-related problems.

***ADHD Diagnosis***

Inconsistent with the existing literature (Aylward, 2002; De Kieviet et al., 2012), the current study did not support the hypothesis that there is a higher prevalence of ADHD among those children who were born LAMP compared to children who were term-born; the mean GA among those diagnosed with ADHD and not diagnosed with ADHD was comparable. This finding was supported even when examining the various diagnostic categories for ADHD (see Figure 2).

**Figure 2**

*ADHD Diagnostic Outcomes*



*Note.* LAMP (yellow) and term-born (green) children shown within total sample (blue) in distribution of diagnostic subtypes.

Prevalence of ADHD in the current sample was much higher than in the general population (76.9% versus 10.2%, respectively; Xu et al., 2018). The higher prevalence of ADHD overall in this sample is understandable given that the data were collected in a clinical setting

from a previously completed comprehensive neuropsychological evaluation. Patients in this study were referred by providers and/or pre-authorized for the evaluation by their insurance companies owing to established risk/probability. Were the sample to have been recruited and collected from the general community and/or LAMP children and term-born children had been followed and evaluated in a prospective study design, prevalence rates may have been more consistent with community prevalence or the existing literature on ADHD in prematurity.

Additionally, the higher prevalence rate of ADHD in this sample may be explained by the very high prevalence of other risk factors that were present in the sample and are summarized in Table 6. The multitude of risk factors observed in this sample's prenatal and perinatal history, coupled with the high prevalence of ADHD in this sample, supports an etiology of ADHD that is multifactorial, with contributions from genetic, environmental, and psychosocial factors. This finding is well aligned with the theory of equifinality (Cicchetti & Rogosch, 1996).

There were additional sample characteristics that likely impacted diagnostic outcomes in the present study. The present study's racial demographics were as follows: 1.8% Asian, 25.4% Black/African American, 59.8% White, 5.3% Latino/Hispanic, 5.3% Biracial/Mixed, and 2.4% unreported. There has been a wave of public health research that evidences ways in which Black people living in the United States have worse health outcomes as a direct consequence of systemic and individual level racism. With over one quarter of the sample population identifying as Black, and a cumulative 37.87% identifying as something other than White, this sample was comprised of a significant racial minority population. In such a sample, the deleterious impact of racism (overt, covert, microaggressions, systemic systems, inequality, injustice, within generation and from a multigenerational perspective) and a plethora of associated health risks, increases the overall risk for neurodevelopmental disorders including ADHD (Trent et al., 2019;

Wright et al., 2020). Therefore, when considering race as a risk factor, it is important to understand that identification as a racial minority functions as a proxy for other social conditions (e.g., disparities in access to/quality of healthcare, exposure to racism, minority stress, etc.). Similarly, SES (and proxies for SES such as limited parental educational achievement and Medicaid status) is associated with a plethora of additional risk that can impact development (e.g., disparities in access to/quality of healthcare, quality of education, additional supports/resources, financial stress/housing insecurity/food insecurity, exposure to teratogens and pollution, lower nutrition dietary options and limited food resources, etc.).

### ***ADHD Symptom Severity via Behavior Rating Forms***

The current study partially supported the hypothesis that caregivers of LAMP children would report higher levels of ADHD symptomatology than caregivers of term-born children, regardless of diagnostic status. After controlling for current prescription for ADHD medication, a marginally significant relationship was found between GA and symptom severity as indicated by parent report on the Conners-3, including the Inattention, Hyperactivity/Impulsivity, *DSM* ADHD Inattentive, and the *DSM* ADHD Hyperactive/Impulsive subscales. Furthermore, caregiver report of Inattention and Hyperactivity/Impulsivity (Conners-3) were found to be marginally significantly higher among LAMP children than their term-born peers. Using GA as a continuous variable, there was a clear and consistent relationship with symptom severity on the Conners-3. When using subdivisions of GA, those that define LAMP and term-born children, the relationship between LAMP status and symptom severity on the Conners-3 is somewhat less apparent as it was not-significant for the *DSM* ADHD subscales. This suggests that the relationship between time spent in gestation and behavioral outcomes at school-age may be easier to see when using GA rather than gestational category.

After controlling for current prescription for ADHD medication, there were marginally significant differences observed between LAMP and term-born children on measures of Inattention and Hyperactivity/Impulsivity specifically on the Conners-3, but not the BASC-3. To better understand why that would be, it is worth considering which behaviors are included on these measures. The follow items are included on the Conners-3 Inattention subscale: trouble staying focused on one thing at a time, short attention span, avoids/dislikes things that take a lot of effort and are not fun, trouble concentrating, doesn't pay attention to details/makes careless mistakes, trouble changing from one activity to another, inattentive/easily distracted, gets bored, gives up easily on difficult tasks, and trouble keeping mind on work or play for long (Conners, 2008). No doubt, the items on this subscale capture those behaviors that would be expected in disrupted attention networks. Items on the BASC-3 Attention Problems subscale include: pays attention, organized, short attention span, listens to directions, listens carefully, easily distracted, misses deadlines, pays attention when being spoken to, and has trouble concentrating (Reynolds & Kamphaus, 2015). Examining these two scales qualitatively, the Conners-3 presents all the behaviors as deficits with specific behavioral indicators (e.g., gives up easily on difficult tasks) that directly map onto the *DSM-5* criteria for Inattention (see Appendix A for the diagnostic criteria for ADHD). In contrast, the BASC-3 words some behaviors positively (e.g., pays attention, organized, etc.) that have no context for when these behaviors occur, and there are several items on the BASC-3 Attention Problems subscale related to listening and receptive language functioning, which may or may not reflect a child's specific attentional difficulties. Consequently, it may be the case that the BASC-3 is less sensitive to purely attention difficulties.

Items on the Conners-3 Hyperactivity/Impulsivity subscale include: fidgeting, blurts out answers before the question has been completed, is constantly moving, excitable/impulsive, gets

over-stimulated, acts as if driven by a motor, blurts out the first thing that comes to mind, has difficulty waiting for his/her turn, runs or climbs when he/she is not supposed to, is noisy and loud when playing or using free time, leaves seat when he/she should stay seated, fidgets or squirms in seat, restless or overactive, and interrupts others. Most of these items provide concrete behavioral anchors that can be observed by the reporting caregiver. Items included on the BASC-3 Hyperactivity subscale: acts without thinking, is overly active, interrupts others when they are speaking, has poor self-control, fiddles with things while at meals, is in constant motion, disrupts other children's activities, is unable to slow down, interrupts parents when they are talking on the phone, acts out of control, and cannot wait to take turn (Reynolds & Kamphaus, 2015). There are several items on this subscale that rely on caregivers' interpretation of a child's inner experience or comparison to other children, rather than asking them to report on the presence of specific, observable behaviors.

In sum, results of the present study suggest that the Conners-3 may be a more sensitive measure when it comes to purely attentional constructs. This is consistent with the intention of the Conners-3, which the manual describes as "a thorough and focused assessment of Attention-Deficit/Hyperactivity Disorder (ADHD) and its most common comorbid problems and disorders in children and adolescents" (Conners, 2008, p. 1). The BASC-3, on the other hand, is intended to assess a broad range of behaviors and emotions and was not specifically designed with a focus on ADHD. However, it is also possible that there is questionnaire bias that explains the difference in findings between the Conners-3 and Basc-3; many items on the BASC-3 include more positive behaviors or attributes that may prime the caregiver to report more favorably.

Though the observed differences in symptom severity on the Conners-3 between LAMP and term-born children were only marginally significant from a statistical perspective, the

difference may be qualitatively or clinically meaningful. LAMP children may have subtle differences in their attentional capacity, best captured by measures intended specifically to assess dimensions related to ADHD such as the Conners-3. Without utilizing screening measures specific to attentional capacity, LAMP children may fail to be identified either diagnostically (ADHD) and/or via broad-based behavior and emotional screening, when they are exhibiting attentional difficulties and would benefit from additional supports or intervention services.

An incidental finding of the present study was the impact of a current prescription for ADHD medication on caregivers' report of symptom severity, including aspects of inattention and hyperactivity/impulsivity. There are several possible explanations for the relationship that was found. Firstly, it's possible that children exhibiting significant ADHD behaviors end up being prescribed ADHD medication, and in turn, caregivers continue to report on those behaviors that initially prompted/justified pharmacological management irrespective of the effect medication has on their child's ADHD symptoms. Another possibility is that participants in the sample were recently prescribed medications for ADHD, and the pharmacological benefits were not yet observed or present for the majority of the period in which caregivers were instructed to report frequency of behavior. Accounting for duration of prescription status and giving caregivers additional instruction while completing the behavior rating scale would help speak to these points. Rating scales could explicitly instruct caregivers to rate their child's behavior on or off prescribed medication or allow caregivers to select whether they are rating their child's behavior on or off prescribed medication. It may also be the case that caregivers of children who have been prescribed ADHD medications have been "primed" to report a higher frequency of ADHD related behaviors; this may be to justify continued access to pharmacological management, communicate their own distress in having a more challenging child, and/or

emphasize the need for help to support and advocate for their child. Administering additional measures of satisfaction with medication, parenting stress, or perceived support outside of the home for the child would provide helpful additional information regarding the impact of an ADHD medication prescription on caregiver report on behavior rating scales.

**Risk Factors.** The rating form manuals make recommendations regarding interpretative consideration of other variables that may drive up symptom reporting. The present study also considered the impact of other risk factors and potential symptom mitigating factors on behavioral presentation in symptom severity. As previously mentioned, the Conners-3 Inattention subscale may have captured marginally significant variance attributable to racial identity compared to other measures/subscales, and the DSM ADHD Inattentive problems subscale may have captured marginally significant variance attributable to Medicaid insurance status (proxy for lower SES). Regarding other risk factors, family history of ADHD and in-utero exposure to substances were found to be related to all dimensions of attention, including inattention and hyperactivity/impulsivity. Given that genetic heritability is the single strongest predictor for ADHD, this finding was consistent with the literature (Biederman, 2005; Du Rietz et al., 2018; Faraone et al., 2005; Franke et al., 2012; Leung & Hon, 2016). In the present sample, 13% of children had in-utero exposure to substances (Table 3): 9.5% were exposed to cigarettes, 3% were exposed to alcohol, 0.6% were exposed to narcotics, 1.8% were exposed to stimulants, 1.2% were exposed to cannabis, 0.6% were suspected or exposed to something other, and 0.6% were exposed to something unknown. The strong association between in-utero exposure to substances and neurodevelopmental and behavioral problems that has been observed in the literature (Menon, 2008; Tsang et al., 2016), was similarly found in this sample. Furthermore, the high frequency of family history of ADHD (over 50%) combined with the high



prevalence of in-utero exposure to substances may partially explain the high prevalence of ADHD observed in this sample.

**Symptom Mitigating Factors.** The present study also examined potential symptom mitigating factors. Prior research regarding longer term outcomes of early intervention services have been mixed. Though early intervention has been found to improve early neurodevelopmental skills (Blauw-Hospers et al., 2007), these benefits are no longer apparent in later childhood (Vanderveen et al., 2009); results of the present study were consistent with the latter, and there were no significant differences in ADHD-related symptom severity as indicated by caregiver report on behavior rating scales between those who had and those who had not received early intervention services. Previous studies have found that children with ADHD have lower social and adaptive skills (Deboo & Prins, 2007; Lindblad et al., 2013; Staikova et al., 2013). While there were no differences in social or adaptive skills between those diagnosed with and without ADHD in the present study across rating scales, intact social and adaptive skills were, as hypothesized, negatively related to attention problems and hyperactivity/impulsivity on the BASC-3. As was previously discussed, the BASC-3 assesses a broad range of behaviors and emotions, while the Conners-3 was created specifically to assess for attentional deficit. Therefore, the BASC-3 may be more sensitive to strength-based variables and resilience. Though social and adaptive skills may not differentiate those with ADHD from those without, building these skills through prevention and/or intervention may provide a healthy behavioral outlet (social interactions) and/or compensatory strategies (adaptive skills) that diminish the burden or functional interferences of ADHD symptoms on overall presentation.

It is important to note that ADHD is an incredibly heterogenous neurodevelopmental disorder. Though there are specific behavioral criteria, clinicians must rely on informant report to

capture and/or quantify those behaviors and determine if they are in excess of what would be expected given a child's developmental level. Children may exhibit any combination of behavioral features of ADHD, and/or the associated neurocognitive deficits, and still not receive an ADHD diagnosis if there is no functional impairment. Furthermore, the threshold at which a child's behavior interferes with a child's functioning across settings is highly individualized and dependent on numerous interpersonal and environmental factors.

Informant report can be achieved via clinical interview, behavior rating scales, review of school records that include qualitative descriptions of children's behavior in the classroom, etc. The present study utilized behavior rating forms completed by parents/caregivers and were then able to integrate those standardized measures of behavior into the greater conceptualization and diagnostic impressions. However, owing to the design of the study, and the setting wherein the comprehensive neuropsychological evaluations took place, additional informant report (e.g., teacher) was not consistently provided nor included in analyses, though no doubt was included in the clinician's diagnostic impression and determination.

### *Neurocognitive Outcomes*

The existing literature has found a relationship between LAMP and higher rates of neurodevelopmental disability (Johnson et al., 2015) and cognitive impairments in overall intellectual functioning, attention control, and inhibition (Cserjesi et al., 2012; Johnson et al., 2015; Kerstjens, 2013). Results of the current study were consistent with the existing literature, and a marginally significant relationship between GA and overall intellectual functioning (WISC-V FSIQ) was found. No other measures of neurocognitive functioning were significantly/marginally significantly related to GA. Variance within an individual's neurocognitive profile can be highly variable, especially among those measures that have an

atypical distribution or more variable psychometric properties. The unique strengths and weaknesses that exist in relationship to overall intellectual functioning may be washed out when running analyses in a largescale dataset such as this. It is perhaps this reason that the only marginally significant result between LAMP and neurocognitive abilities was in overall *g*/IQ, and that relative strengths and weaknesses were not evident with the statistical analyses that were utilized.

Children with lower GA are also more likely to have lower birthweight (Taylor, 2010). Low birthweight has its own known associations with negative developmental and cognitive outcomes (Mortensen et al., 2003). Results of the current study were consistent with the existing literature: lower GA was related to lower birthweight, and children with younger GA and lower birthweight were more likely to exhibit a similar FSIQ. As GA increases, the impact of birthweight on FSIQ becomes more apparent, with higher GA and higher birthweight predicting better neurocognitive outcomes, and higher GA with lower birthweight predicting poorer outcomes at a marginally significant level. Taken together, it appears as though birthweight (low and high) differentiates overall cognitive functioning (WISC-V FSIQ) for those children who were born at a later GA and may play a less significant role in predicting outcomes among children born earlier in the pregnancy.

This interaction between birthweight and gestational age among children born at/after the 32<sup>nd</sup> week of gestation in this sample suggests a few things. For those children born earliest within this sample (moderately premature), *g*/IQ was comparable irrespective of birthweight. However, for those children born later within this sample, lower birthweight predicted an FSIQ that was nearly 10 standard score points below those with higher birthweight. Birthweight in the last weeks of gestation differentiates overall neurocognitive outcomes, suggesting that lower

birthweight may be capturing intrauterine growth restriction/fetal growth restriction or poorer overall nutrition, which in turn predicts worse outcomes. For children born early term or term, low birthweight remains a significant risk for later difficulties, despite the child having reached the arbitrary cutoff of 37 weeks that defines term.

**Cumulative Risk and Neurocognitive Outcomes.** LAMP birth and ADHD share risk factors, and many of those risk factors have a negative impact on neurocognitive functioning (Howson et al., 2012; Shapiro-Mendoza & Lackritz, 2012). Therefore, the present study also looked at the relationship between cumulative risk (i.e., number of risk factors endorsed: minority status, lack of parental higher-level education, Medicaid status, genetic heritability, and prenatal exposure to substances) and *g*/IQ, attention, working memory, executive functioning, and processing speed. Consistent with prior literature, overall intellectual functioning, working memory, and EF (specifically cognitive flexibility) were significantly related to cumulative risk.

It may be the case that the observed neurocognitive deficits associated with LAMP birth reflect, at least in part, other risk factors associated with poorer neurodevelopmental outcomes and why the present study did not find a significant relationship between GA and cognitive functioning in specific domains. Though GA among LAMP children may not be a significant predictor for neurocognitive outcomes, it is reasonable to expect that in a population with a higher prevalence of risk factors such as the ones included in the present study (and including both LAMP and term-born children), there would also be a higher rate of associated neurocognitive weaknesses. The present study utilized data from patients presenting for clinical concerns, pre-authorized by their insurance companies owing to identified risk to justify a neuropsychological evaluation, and who reflected the diverse community in which the hospital was located. It follows that this higher risk population would endorse a greater number of known

risk factors, and that those risk factors would predict greater pathology including behavioral challenges and neurocognitive weaknesses (equifinality).

**Total Symptom Mitigating Factors and Neurocognitive Outcomes.** The existing literature has generally found that early intervention services have a positive proximal impact on neurodevelopmental skills but does not exert a measurable impact on cognitive outcomes in later childhood (Blauw-Hospers et al., 2007; Spittle et al., 2015; Vanderveen et al., 2009). Consistent with this literature, results of the current study did not find a significant relationship between total symptom mitigating factors (early intervention and intact social or adaptive skills) and neurocognitive outcomes, with one exception: there was a significant negative relationship between total symptom mitigating factors and a measure of brief attention. Specifically, it was participation in early intervention services that predicted differential performance on this measure (not having intact social or adaptive skills). It may be the case that children with more significant delays warranting participation in Early On are a higher risk group within LAMP children, and differences in attention capacity remain evident in later life, irrespective of intervention efficacy. Additional research is needed to assess attention before and after participation in interventions such as Early On to account for direction of effect.

### **Limitations**

The current study addressed important gaps in the literature, specifically the use of performance-based neuropsychological measures in addition to diagnostic status and behavior rating scales to examine ADHD-related outcomes among a subset of children born premature—LAMP. There were several significant limitations related to the study's design, some of which have been previously alluded to but will be discussed further. First, the study was cross-sectional, and the data were pulled from patients who were seen during a specific window of

time (2014-2020). The direction of influence between GA and behavioral and neurocognitive outcomes by school-age is obscured by chronological variables and interim history. The present study used certain risk and symptom mitigating factors available in the medical record, but very likely missed other variables in the child's history that were related to the measured outcomes; it did not measure factors related to educational experience (e.g., type of school, quality of curriculum, tutoring) or participation in intervention services over the age of 5 years, which could certainly have impacted outcomes in the present study. A longitudinal design that follows both term-born and LAMP children from birth through school-age, and can account for other prevention, intervention, and learning experiences that happen during development, could prospectively examine the relationship between GA and ADHD-related outcomes, while also accounting for other variables that impact those outcomes.

The cross-sectional design also creates a de facto bias in that study participants (term-born and LAMP) were all taken from a clinical sample, where they had been pre-authorized for neuropsychological evaluation owing to known risk. A longitudinal design that followed term-born controls from birth would capture a comparison group free of this type of bias. Specifically, the term-born children would be recruited from birth, and not selected based on existing behavioral or cognitive concerns or other identified risk factors.

The study design was retrospective, and the variables utilized were not selected for research purposes. Risk factors were based on caregiver report on the patient history form and/or during the clinical interview and dependent on caregivers' memory or understanding about their child's early history. For example, the caregiver completing the form/interview may not have been the gestational carrier, and may not have known or remembered substance use, medications taken during pregnancy, or understood the difference between placenta abruptia and placenta

previa, and toxemia and preeclampsia. Had the risk factors been collected via comprehensive review of the medical record for both gestational carrier and the child, as could be incorporated in the design of a longitudinal study, a more accurate and comprehensive picture may have emerged. Additionally, caregivers were not asked questions regarding household income or perceived financial security. Rather, Medicaid insurance coverage and parents' lack of higher-level education were used as proxies for capturing dimensions of SES. Although these proxies are frequently used for SES (Becker & Newsom, 2003; McLaughlin & Sheridan, 2016), perceived financial security is a better measure in that it captures the individual's psychological response to their economic condition.

Symptom severity was measured using caregiver report on behavior rating scales and was not determined by the clinician. As such, the symptom severity only captures one person's perspective, and reporters likely have variable experience working with other children at this age as well as few observations of their child across settings. It would have been ideal to have additional rating scales completed by teachers, who are able to compare a child's attention/inattention and hyperactivity/impulsivity to many other children of the same age and attest to the child's behavior in a structured environment (e.g., school setting outside the home). Alternatively, clinician determination of symptom severity based on integration of parent, teacher, and independent behavioral observation would have been very helpful in gaging presentation across settings. However, the clinician would not provide symptom severity in this way unless the child met criteria for and was given a diagnosis of ADHD. Either a standardized behavior rating form that captures behaviors related to attention and hyperactivity, or coding clinician observation in a clinical setting (e.g., coding behavioral observations), could be useful in future research designs.

There are notable limitations to analyses that use large neurocognitive datasets. Relative strengths and weaknesses that are idiosyncratic to the individual patient are washed out when running analyses that collapse performance across participants. When looking at a whole sample of participants' performance, the comparison is to the general population; within the context of an individual neuropsychological evaluation, comparisons are made to both the general population as well as within the profile to ascertain strengths and weaknesses. Consequently, analyses such as those used in the present study may have failed to capture relative strengths and weaknesses that could further illuminate the relationship between GA and neurocognitive outcomes. Future studies may consider cluster analyses to capture strengths and weaknesses on a larger scale (Allen & Goldstein, 2013).

Lastly, the COVID-19 pandemic impacted the process by which data was to be mined and entered. There were office closures and furloughs, reduced patient volume, and hiring freezes, which prevented the recruitment and training of additional research personnel. As such, all data were pulled from the EMR, entered into the database, and checked by the same researcher. Though the data were double checked and cleaned, the entirety of this process was done by the same researcher, making the dataset vulnerable to inaccuracies. Further, it was hoped the total sample would include 200 participants, with approximately 50 subjects for each of the four LAMP and term-born categories. Given reduced patient volume, there were fewer patient files for review, and the final sample included 17 moderately preterm children, 52 late preterm children, 31 early term children, and 69 full-term children. The smaller N reduced the power in the analyses, hence why a p-value of  $< .10$  was utilized in the interpretation of the data.



### **Clinical Implications and Future Directions**

The current study provided evidence that LAMP children remain at risk for some poorer neurocognitive outcomes, specifically overall intellectual functioning, than their term-born peers. Furthermore, it is birthweight that appears to differentiate children at higher GAs, indicating higher GA and higher birthweight result in better neurocognitive outcomes compared to higher GA with lower birthweight predicting poorer outcomes. Moreover, it was children's cumulative risk (e.g., being a minority, low SES, genetic heritability, and prenatal exposure to substances) that was found to be significantly related to deficits in working memory and executive functioning—not GA. Lastly, two of the hypothesized symptom-mitigating factors (e.g., specifically intact social and adaptive functioning) were related to lower attention problems and hyperactivity (BASC-3); conversely, participation in early intervention services was related to lower performance on a measure of brief attention, suggesting that some delays and/or difficulties in early childhood that warrant participation in Early On may still be evident in later life. Taken together, these findings have implications for clinicians and policy makers who work to improve outcomes for higher risk children.

Though future research is needed to better understand the relationship between GA and ADHD-related behavioral and neuropsychological outcomes, the present study provides preliminary evidence that those last weeks of the gestational period are meaningful in terms of children's neurocognitive development by school-age. However, risk and symptom mitigating factors do appear to exert additional influence on cognitive outcomes, irrespective of GA, suggesting that there is significant opportunity for prevention/intervention. Given the limitations of the study design described above, it is very likely that there were additional variables that impacted outcomes that were not captured. A prospective longitudinal design is necessary to

better understand what factors predict better or worse outcomes for LAMP children, as well as the plethora of risk factors (alone and in combination) that predict ADHD and ADHD-related pathology irrespective of GA.

From a clinical perspective, findings of the present study apply to battery selection in neuropsychological assessment. Results of this study suggest that the BASC-3 may be less susceptible to demographic variables, specifically race and Medicaid status, which is an important consideration especially in diverse settings and among higher risk communities. Furthermore, the BASC-3 was able to capture a negative relationship between social and adaptive skills and attention problems and hyperactivity/impulsivity. The present study provides some evidence that the social skills necessary to create and maintain healthy peer relationships and adaptive behavioral strategies minimize functional impairment of attention problems. The Conners-3 has notable advantages over the BASC-3 in its sensitivity to those attention differences (behavioral and cognitive) that differentiate LAMP children and higher risk children, from term-born and lower risk children. Overall, the Conners-3 was more sensitive to GA, which could be crucial in capturing more subtle deficits that have significant impact on day-to-day functioning, even though these subtleties do not always translate to an ADHD diagnosis. Clinicians should be mindful that the BASC-3 may be better at capturing strength, while the Conners-3 may be better at capturing, specifically, attentional deficits.

Furthermore, clinicians working with higher risk children, including LAMP and those with other known risk factors predictive of neurodevelopmental disorders such as ADHD, should be recommending and advocating for patient access to early intervention, serial monitoring of behavioral and neurocognitive functioning, and bolstering social and adaptive skills to support development through middle childhood and potentially beyond. For example, clinicians working

in NICUs with graduating LAMP children should provide psychoeducation to parents regarding elevated risk and what to monitor for through their child's development; well child visits and other routine pediatric monitoring appointments are the perfect opportunity to provide updated education regarding normative development and expectations, and query for those behaviors and symptoms the child is specifically at risk for exhibiting.

Existing policy requires all states provide services to children from birth through 2 years with documented developmental delay or an established condition documented from another health or mental healthcare provider (Early On Michigan, 2018; IDEA, 1990). Though low birthweight and ADHD are among those established conditions that warrant automatic eligibility to Early On services, preterm birth has not been included (Early On Michigan, 2016). This is problematic as ADHD is not likely to be diagnosed this early in development. Therefore, results of the current study would support policy revision to allow children who are born preterm (including LAMP) and/or low birthweight, be automatically granted access to state-based enrichment services such as Early On.

The public education system provides services to children beginning at the age of 3 years. Individualized education plans (IEPs) are available to children to ensure a free and appropriate education, including school-based intervention services such as speech and language pathology (SLP), occupational therapy (OT), physical therapy (PT), social work (SW), access to a resource room, and additional behavioral supports that will help facilitate optimal development and academic participation (FAPE; Petrovella & Sullivan, 2017). Section 504 of the Rehabilitation Act of 1973, P.L. 93-112, further protects the educational rights of children who have a disability and may be experiencing difficulties despite adequate grades and classroom participation but do not otherwise qualify for services under an IEP. The current educational policy, therefore, serves

to protect children once they exhibit deficits or hold a diagnosis. Results of the present study suggest that LAMP children, who may be at risk for subtle hits to overall neurocognitive functioning, may benefit from a lower threshold of inclusion to help redress this disadvantage. In addition, policy should direct insurance companies to reimburse for auxiliary services, such as those offered in school (OT, PT, SLP), on an outpatient basis so that LAMP children and other high-risk children receive maximal supports as soon as they begin to fall behind their peers or exhibit behavioral and/or cognitive deficits.

Future studies should seek to investigate other variables in a child's history (e.g., events that occur between birth and school-age including educational experiences, traumatic events, participation in prevention/intervention, tutoring, extracurricular enrichment, medical events, etc.) that may impact neurocognitive outcomes in middle childhood. Replication of the present study should use additional measures/perspectives to capture children's behavioral functioning across settings. Being able to better capture a child's own unique strengths and weaknesses (e.g., cluster analysis, coding to capture relative strengths and weaknesses, adjustment for individual baseline), while accounting for other factors that impact neurocognitive development (e.g., risk and protective), will help illuminate which factors best predict ADHD, attention, working memory, EF, processing speed, and related behaviors.

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APPENDICES

Appendix A: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) Criteria for ADHD

<b>DSM-5 Diagnostic Criteria: Attention-Deficit/Hyperactivity Disorder</b>	
A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by six or more of the following symptoms in (1) Inattention or (2) Hyperactivity/Impulsivity that have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities. Several symptoms were present prior to age 12-years and are present in two or more settings. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder.	
<b>(1) Inattention:</b>	<b>(2) Hyperactivity/Impulsivity:</b>
Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities; often has difficulty sustaining attention in tasks or play activities; often does not seem to listen when spoken to directly; often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace; often has difficulty organizing tasks and activities; often avoids, dislikes or is reluctant to engage in tasks that require sustained mental effort; often loses things necessary for tasks or activities; is often easily distracted by extraneous stimuli; is often forgetful in daily activities.	Often fidgets with or taps hands or feet or squirms in seat; often leaves seat in situations when remaining seated is expected; often runs about or climbs in situations where it is inappropriate; often unable to play or engage in leisure activities quietly; is often “on the go,” acting as if “driven by a motor;” often talks excessively; often blurts out an answer before a question has been completed’ often has difficulty waiting his or her turn; often interrupts or intrudes on others.
Specify whether: <b>314.01 (F90.2) Combined presentation:</b> If both Criterion (1) and (2) are met for the past 6 months. <b>314.00 (F90.0) Predominantly inattentive presentation:</b> If Criterion (1) is met but Criterion 2 is not met for the past 6 months. <b>314.01 (F90.1) Predominantly hyperactive/impulsive presentation:</b> If Criterion (2) is met and Criterion (1) is not met for the past 6 months.	
Specify if: <b>In partial remission:</b> When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.	
Specify current severity: <b>Mild:</b> Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or occupational functioning. <b>Moderate:</b> Symptoms or functional impairment between “mild” and “severe” are present. <b>Severe:</b> Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.	

Note. Adapted from the American Psychiatric Association (2013)

## Appendix B: Institutional Review Board Approval Letter

University Human Subjects Review Committee

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Jan 7, 2021 12:23:14 PM EST

Heather Hennrick  
Psychology, Users loaded with unmatched Organization affiliation.

Re: Exempt - Initial - UHSRC-FY19-20-291 Neurocognitive Resilience in Children Born Late and Moderate Preterm: Predictors of Improved Outcomes in Attention, Working Memory, and Executive Functioning

Dear Heather Hennrick:

The Eastern Michigan University Human Subjects Review Committee has rendered the decision below for Neurocognitive Resilience in Children Born Late and Moderate Preterm: Predictors of Improved Outcomes in Attention, Working Memory, and Executive Functioning . You may begin your research.

Decision: Exempt

Selected Category: Category 4. Secondary research for which consent is not required: Secondary research uses of identifiable private information or identifiable biospecimens, if at least one of the following criteria is met:

- (i) The identifiable private information or identifiable biospecimens are publicly available;
- (ii) Information, which may include information about biospecimens, is recorded by the investigator in such a manner that the identity of the human subjects cannot readily be ascertained directly or through identifiers linked to the subjects, the investigator does not contact the subjects, and the investigator will not re-identify subjects;
- (iii) The research involves only information collection and analysis involving the investigator's use of identifiable health information when that use is regulated under 45 CFR parts 160 and 164, subparts A and E, for the purposes of "health care operations" or "research" as those terms are defined at 45 CFR 164.501 or for "public health activities and purposes" as described under 45 CFR 164.512(b); or
- (iv) The research is conducted by, or on behalf of, a Federal department or agency using government-generated or government-collected information obtained for nonresearch activities, if the research generates identifiable private information that is or will be maintained on information technology that is subject to and in compliance with section 208(b) of the E-Government Act of 2002, 44 U.S.C. 3501 note, if all of the identifiable private information collected, used, or generated as part of the activity will be maintained in systems of records subject to the Privacy Act of 1974, 5 U.S.C. 552a, and, if applicable, the information used in the research was collected subject to the Paperwork Reduction Act of 1995, 44 U.S.C. 3501 et seq.

Renewals: Exempt studies do not need to be renewed. When the project is completed, please contact [human.subjects@emich.edu](mailto:human.subjects@emich.edu).

Modifications: Any plan to alter the study design or any study documents must be reviewed to determine if the Exempt decision changes. You must submit a modification request application in [Cayuse IRB](#) and await a decision prior to implementation.

Problems: Any deviations from the study protocol, unanticipated problems, adverse events, subject complaints, or other problems that may affect the risk to human subjects must be reported to the UHSRC. Complete an incident report in [Cayuse IRB](#).

Follow-up: Please contact the [UHSRC](#) when your project is complete.

Please contact [human.subjects@emich.edu](mailto:human.subjects@emich.edu) with any questions or concerns.

Sincerely,

Eastern Michigan University Human Subjects Review Committee

Appendix C: Psychometrics of Measure Capturing *g*

<b>Core Domain</b>	<b>Measure</b>	<b>Core Constructs</b>	<b>Brief Description</b>	<b>Age</b>	<b>Psychometrics</b>
<i>g</i>	WISC-V FSIQ	Intellectual Quotient (IQ)	The FSIQ is composed of 10 subtests that assess abilities across the domains of verbal comprehension, visual spatial processing, fluid reasoning, working memory, and processing speed.	6:0 to 16:11	Reliability (.96 overall, and .96-.97 among 8- to 12-year-olds) and Validity (evidence for validity based on test content, response processes, internal structure, relations with other variables and consequences of testing)

*Note.* Adapted from Wechsler (2014)

Appendix D: Psychometrics of Neuropsychological Measures by Domain

Core Domain	Construct(s)	Measure	Brief Description	Age	Psychometrics
<b>Attention</b>	Sustained attention, visual inattention, impulsivity, vigilance	CPT-2/CPT-3 Omissions and Commissions	14-minute, 360-trial administration, child is required to press the spacebar or wired mouse button when any letter except "X" appears	8:0+	Reliability (median test-retest correlation was $r = .67$ , internal consistency, median split-half reliability $r = .92$ for the norm samples and $r = .94$ for the clinical samples) and Validity (discriminative validity, incremental validity including 88.4% sensitivity, and 87.3% specificity when used with Conners-3 parent rating scale)
	Brief attention	WISC-V Digit Span Forward	The child is read a sequence of numbers and recalls the numbers in the same order, involving auditory rehearsal and temporary storage. Each Digit Span item is composed of two trials with the same span length. There are at least 9 items for each task. Several	6:0 to 16:11	Reliability (.75-.85 across relevant age range, overall mean $r = .81$ , among ADHD sample $r = .82$ , test-retest $r = .82$ )
<b>Working Memory</b>	Working memory, spatial revisualization	WISC-V Digit Span Backward	The child is read a sequence of numbers and recalls the numbers in backwards order, which requires working memory, transformation of information, mental manipulation, and may involve visuospatial imaging. Each Digit Span item is composed of two trials with the same span length. There are at least 9 items for each task.	6:0 to 16:11	Reliability (.75-.83 across relevant age range, overall mean $r = .80$ , among ADHD sample $r = .82$ , test-retest $r = .76$ )
	Working memory, auditory acoustic memory	WISC-V Digit Span Sequencing	The child is read a sequence of numbers and recalls the numbers in sequential order, which requires auditory acoustic memory and numerical re-ordering. Each Digit Span item is composed of two trials with the same span length. There are at least 9 items for each task.	6:0 to 16:11	Reliability (.76-.84 across relevant age range, overall mean $r = .82$ , among ADHD sample $r = .84$ , test-retest $r = .79$ )
<b>Executive Functioning</b>	Cognitive flexibility	D-KEFS TMT 4	The child completes paper and pencil task, connecting encircled numbers and letters in alternating order by drawing connecting line.	8:0 to 89:11	Reliability (internal consistency, stability coefficients, and alternate-form reliability) and Validity (evidence provided in terms of the sensitivity of the tests to measure important areas of higher-level executive functions and correlation studies)

	Cognitive flexibility	Children's Trail Making Test Part B	The child completes paper and pencil task, connecting encircled numbers and letters in alternating order by drawing connecting line.	7:0 to 13:11	Reliability (test-retest reliability coefficient of .56 of children tested a total of three times with 6-month intervals between testing sessions, alternate-form reliability, interrater reliability) and Validity (good sensitivity, ecological validity)
	Inhibition	D-KEFS CWIT Inhibition	The child is presented with color names (e.g., red, green, blue) printed in a different-colored ink and must state the color of the ink rather than reading the word.	8:0 to 89:11	Reliability (internal consistency, stability coefficients, and alternate-form reliability) and Validity (evidence provided in terms of the sensitivity of the tests to measure important areas of higher-level executive functions and correlation studies)
	Inhibition	NEPSY Inhibition Condition	The child is presented with arrows pointing up or down and must state the opposite direction of the arrow rather than stating the indicated direction.	5:0 to 16:11	Reliability ( $r=.86$ average among 7- to 12-year-olds, test-retest reliability $=.64-.87$ , and interscorer agreement $=.98-.99$ ) and Validity (content, construct, concurrent)
	Inhibition and cognitive flexibility	D-KEFS CWIT Inhibition/Switching	The child is presented with color names (e.g., red, green, blue) printed in a different-colored ink and must state the color of the ink rather than reading the word; however, when the word is in a box, they must read the word rather than say the ink color.	8:0 to 89:11	Reliability (internal consistency, stability coefficients, and alternate-form reliability) and Validity (evidence provided in terms of the sensitivity of the tests to measure important areas of higher-level executive functions and correlation studies)
	Inhibition and cognitive flexibility	NEPSY Inhibition Switching Condition	The child is presented with black and white arrows pointing up or down and must state the opposite direction of the white arrows and the indicated direction of the black arrows.	7:0 to 16:0	Reliability ( $r=.87$ average among 7- to 12-year-olds, test-retest reliability $=.73-.94$ , and interscorer agreement $=.98-.99$ ) and Validity (content, construct, concurrent)
<b>Processing Speed</b>	Speed of information processing, graphomotor speed and visual scanning	WISC-V PSI	The PSI is calculated based on performance on two subtests: coding and symbol search. Coding requires a child to use a key to copy symbols that correspond with simple geometric shapes or numbers. Symbol search requires the child to scan search groups and indicate whether target symbols are present. Both are conducted under timed constraints (2 minutes).	6:0 to 16:0	Reliability (.88 overall, and .84-.88 across 8- to 12-year-olds) and Validity ( $r=.58$ indicating moderate correlations between coding and symbol search; evidence for validity based on test content, response processes, internal structure, relations with other variables and consequences of testing)

*Note.* Adapted from Conners (2014), Delis, Kaplan, & Kramer (2001), Korkman, Kirk, & Kemp (2007), Reitan & Wolfson (1985) and Wechsler (2014)

## Appendix E: Psychometrics of Behavioral Reports

Measure	Core Constructs; Relevant Subscales	Brief Description	Age	Psychometrics
<b>BASC School-aged</b>	Hyperactivity, Attention Problems; ADHD Probability Index	Parent (137 items) and teacher (156 items) ratings on adaptive and problem behaviors in the home, community, and school settings.	6:00 to 11:11	Good to Excellent Reliability (alpha coefficients .92-.97 parent and .92-.97 teacher, test-retest $r = .87-.92$ parent and $r = .77-.91$ teacher, interrater reliability $r = .59-.75$ parent and $r = .37-.73$ teacher) and Validity (convergent, construct, content, criterion related)
<b>Conners-3</b>	Inattention, Hyperactivity/ Impulsivity	110-item parents and 115-item teacher rating form to assess youth with characteristics of ADHD	6:0 to 18:11	Reliability (Cronbach's alpha .91 parent and .94 teacher, test-retest $r = .85$ parent and $r = .85$ teacher, interrater $r = .81$ parent and $r = .73$ teacher) and Validity (factorial validity adequate for parents, slightly lower for teachers, convergent, divergent, and discriminative validity)
<b>ABAS-3</b>	Adaptive functioning (General Adaptive Composite); conceptual, social, and practical domains	Comprehensive assessment of adaptive behavior; parent/caregiver completes the measure via self-report regarding the child's abilities across conceptual (communication, functional academics, and self-direction), social (leisure and social skills), and practical (community use, home living, health and safety, and self-care) domains.	5:0 to 21:11	Reliability ( $r = .96-.99$ , good internal consistency, test-retest reliability = .82, interrater reliability = .72) and Validity (evidence based on item content, response process, internal structure, internal consistency, age-group differences, intercorrelations among skill areas, factor structure, correlations with other variables, and sensitivity)

*Note.* Adapted from Achenbach & Rescorla (2000, 2001), Conners (2008), Gioia, Isquith, Guy, & Kenworthy (2015), Harrison & Oakland (2015), and Reynolds & Kamphaus (2015)