ADULT ADHD AND COGNITIVE DISPERSION:

CHARACTERIZING VARIABILITY IN PERFORMANCE ACROSS COGNITIVE TASKS IN OLDER ADULTS WITH AND WITHOUT ADHD

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A Thesis

Submitted in Partial Fulfillment

of the Requirements for the Degree of

Master of Arts

in Psychological Sciences

Northern Arizona University

May 10, 2023

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ABSTRACT

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Most research on cognitive performance among individuals with Attention Deficit/Hyperactivity Disorder (ADHD) focuses on younger persons and on cognitive variability within speeded response-time tasks. Dispersion (i.e., variability across a range of cognitive domains) is emerging as a promising indicator of age-related and pathological cognitive impairment. There has not yet been an evaluation of differences in dispersion among older adults with and without ADHD to determine if dispersion may be a behavioral marker of ADHD. We address this gap by assessing associations of age, ADHD status, and ADHD severity with dispersion. We hypothesized that older adults would exhibit greater dispersion than comparatively younger adults and explore whether individuals with ADHD and individuals with more ADHD symptoms exhibit greater dispersion than individuals without ADHD and with fewer symptoms. In a sample of 231 adults from the Longitudinal Aging Study Amsterdam (Average age=71.64 years, SD=7.7, Range = 59% female), 23 individuals met DSM-IV criteria for ADHD and 208 were classified as neurotypical. Participants completed 13 tasks spanning domains of attention, fluency, memory, processing speed, and reasoning. Dispersion across the tasks was calculated as an intraindividual standard deviation for each participant. We regressed dispersion on age, ADHD status, and ADHD symptoms and adjusted for sex, education, and depressive symptoms. Older age was significantly associated with greater dispersion (Est =0.06, SE=0.03, p=0.02). However, dispersion profiles did not vary as a function of ADHD status (Est.=-0.90, SE=0.71, p>.05), or

number of ADHD symptoms (Est.=-0.03, SE=0.03, p>.05). Results suggest that dispersion across cognitive tasks may not be a sensitive marker of ADHD in older adults, although statistical power to detect differences was low in the current study. Age was a significant correlate of higher dispersion. Additional research is required to gain more understanding of ADHD in older adulthood. Though our study suggests that within-person fluctuations measured by variability observed across cognitive domains may not be a behavioral marker of ADHD, other indices of variability, such as response time inconsistency, may be relevant for future work on behavioral descriptions of ADHD across the lifespan.

Keywords: Attention Deficit/Hyperactivity Disorder, older adulthood, dispersion, cognition

Table of Contents

Chapter I. Introduction

Overview

Attention-deficit/hyperactivity disorder (ADHD) has been classified as one of the most common neurodevelopmental disorders to occur in childhood (Scandurra, 2019). The disorder has been shown to affect multiple domains of cognition; severe deficits have been observed in dimensions of attention, executive function, memory, inhibitory control, emotional regulation, as well as increased levels of impulsive behavior (Wender, 1998; Kipp, 2005; Carbonneau et al., 2020). Despite the high prevalence, there remains a severe lack of information regarding ADHD, its persistence into adulthood, and the extent of its resulting cognitive deficits throughout the lifespan. A critical gap in knowledge is the role that an ADHD diagnosis has on the cognitive function of older adults. The present thesis aims to investigate this relationship through the use of a dispersion index that measures variability in performance across multiple domains of cognition in older adults with and without ADHD.

Rationale

Estimates of persistence of ADHD into adulthood range widely (Sibley, 2016). Despite this range in estimates, there is significant evidence to suggest that many of the characteristic symptoms associated with adolescent ADHD persist into adulthood and result in cognitive impairment of varying degrees (Faraone, 2006; Kessler, 2005). Given the lack of academic literature explicating the role, diagnostic procedures, and burden of ADHD in adulthood, we often necessarily rely on the vital data gathered concerning ADHD in adolescence, which is markedly more abundant in psychological research, to give us insight into the presentation of ADHD in adulthood. One way in which researchers have characterized ADHD in adolescence is through measures of cognitive task performance. Specifically, research has been conducted that

investigates the effects of an ADHD diagnosis on an individual's levels of response time inconsistency (RTI; Kofler, 2013), defined as trial-to-trial fluctuations within a single speeded response time task. This measure of intraindividual variability (IIV) has been shown to be a behavioral marker of ADHD (Alderson, Rapport, & Kofler, 2007) such that individuals with ADHD exhibit greater levels of RTI than individuals without ADHD.

Though RTI as a measure of IIV provides researchers with a reliable marker of ADHD, this index focuses solely on fluctuations from trial-to-trial within a single speeded response time task and does not provide a widespread representation of functioning in cognitive domains assessed in ways other than speeded performance (e.g., number of words remembered in immediate and delayed recall tasks). Dispersion is a unique measure of cognitive variability that refers to the variability in the individual's cognitive task performance across multiple cognitive domains. Dispersion has been used as a measure of intraindividual variability (IIV) across two or more cognitive dimensions to investigate the effects of cognitive variability on health outcomes and provides information about cognitive health that is distinct from measures of RTI. Greater RTI may reflect greater lapses of attention while completing a single task, whereas greater dispersion may reflect a wider compromise of cognitive functioning across multiple domains of cognitive health (e.g., memory, executive function, processing speed). Increased levels of dispersion are associated with older age and cognitive decline and has emerged as a behavioral marker of certain neurodegenerative diseases such as Alzheimer's disease and related dementias (Hilborn et al., 2009; Halliday et al., 2018).

Chapter II. Literature Review

Theoretical Perspective

The purpose and methods involved in this study are rooted in lifespan developmental theory (Baltes, 1997). Lifespan theory emphasizes the capacity for individuals to develop across the entire lifespan. A theoretical lens that embraces capacity for change in adulthood is important for the examination of variability in cognitive performance among older adults in this thesis. The lifespan development theory posits that adaptive processes that may be more obvious in younger individuals and are often thought to cease in adulthood are present throughout an individual's life, including into older age (Baltes, 1997). The lifespan developmental approach emphasizes the importance of cultivating developmental processes through maximization of gains and the minimization of losses throughout an individual's life (Baltes, 1997). This thesis investigates the association between an ADHD diagnosis and levels of variability that can be observed across cognitive tasks in older adults. Plasticity, or the ability to adapt to changes in the environment and react differently to stimuli, is a driving force of development under this framework and is especially crucial in older adult populations. The presence of gains and losses that accompany aging can help to clarify the role an ADHD diagnosis plays in the amount of dispersion an individual exhibits across cognitive domains. For example, there may be specific gains or strengths associated with aging that equip individuals with an ADHD diagnosis in older age with resources that help to maintain steady cognitive performance (e.g., lower dispersion). Conversely, there may be losses associated with aging that amplify a person with ADHD's variability in cognitive performance (e.g., higher dispersion). Given this capacity for a lifetime of development and change, it stands to reason then that our investigation into dynamic indices in cognitive function and the influence of ADHD diagnosis in older adulthood are valuable to current conceptual paradigms in the aging literature.

Evolution of Diagnostic Criteria for ADHD

The first mention of what we know now as ADHD was first described in the 2nd version of the Diagnostic and Statistical Manual of Mental Disorders published in 1968. At the time, the disorder was termed Hyperkinetic Reaction of Childhood (APA, 1968). This disorder was described in the DSM-II as being, "characterized by overactivity, restlessness, distractibility, and short attention span, especially in young children; the behavior usually diminishes by adolescence' (APA, 1968, p. 50). As the 3rd DSM was being developed, researchers investigating disorders in childhood were shifting their focus away from hyperactivity; instead, investigators began to consider attention deficit and impulse control issues as more significant predictors of the disorder than hyperactivity alone. This hypothesis was supported by a stronger reduction in attention deficit symptoms after treatment than was observed in hyperactivity symptoms. As a result of this shift in focus, in 1980 with the release of the DSM-III, the disorder was renamed attention deficit disorder and included two different types – with hyperactivity and without hyperactivity. With the new naming conventions, the DSM-III was also more specific in its diagnostic criteria by providing three symptom lists for inattention, hyperactivity, and impulsivity. This was also the first time that practitioners and researchers were provided with diagnostic criteria for the disorder that included age cut-offs, and symptom score cutoffs (Lange, 2010).

Though these diagnostic changes were necessary, there remained confusion about the differentiation of the two sub-types of the disorder that were being suggested by the authors of the DSM-III. Specifically, professionals were unsure whether the two subtypes were to be considered two entirely different disorders or if they were qualitatively similar enough to fall under the same psychiatric umbrella. As a way to clarify and hopefully improve the diagnostic process, the revised version of the DSM (DSM-III-R) renamed the disorder Attention Deficit-

Hyperactivity Disorder and dispelled the two-subtype paradigm by combining the symptoms lists into one comprehensive list (Lange, 2010).

The fourth version of the DSM contained many of the concepts and criteria that we use today. It was also in this version of the DSM that the possibility for ADHD symptoms to persist into adulthood was first acknowledged; previous to this, it was widely accepted that symptoms of the disorder abated with time. The fourth version of the DSM also re-introduced the idea of subtypes within the disorder. The subtypes suggested were predominantly inattentive type, predominantly hyperactive/impulsive type, and combined type. The DSM-IV included an 18-item symptom list that was to be used in diagnostic evaluation (e.g., “Often fidgets with hands or feet or squirms in seat”; “is often easily distracted by extraneous stimuli”; APA, 2000). Of these 18 items, 6 were required in either domain (inattention or hyperactivity/impulsivity) to be classified as having ADHD. It is also important to note that the onset criterion specified that symptoms resulting in impairment were required before the age of 7 years. The current DSM version (DSM-5) retains the three subtypes of ADHD and further clarifies adult diagnostic criteria by requiring the presentation of at least 6 items before the age of 12 years instead of 7, and the observance of at least 5 symptoms if above the age of 17. The DSM-5 also specifies the necessity for the symptoms to be present for at least 6 months and for a significant negative impact to be observed before the individual can be diagnosed (APA, 2013).

As previously mentioned, the estimates of the persistence of adolescent ADHD into adulthood range widely in the current literature. In a study investigating ADHD symptom persistence into adulthood, Sibley and colleagues (2016) collected a sample of 579 children with ADHD-combined type and 289 neurotypical classmates (ages 7.0-9.9 years). Of the 579 individuals with ADHD-combined type and 289 neurotypical individuals evaluated in childhood,

476 and 241 were evaluated in younger adulthood (Mean Age=24.7), respectively. Sibley and colleagues found that 60% of the ADHD group presented with a persistence of ADHD symptoms (Sibley et al. 2016). Persistence estimates in other literature range from as low as 5% to over 75% (Barkley, Murphy, & Fischer, 2008; Biederman et al., 2011; Biederman et al., 2012; Halperin et al., 2008; Hinshaw et al., 2012; Klein et al., 2012; Sibley et al., 2012; Weiss & Hechtman, 1993). There are several possible explanations for this range in estimates. Upon close evaluation of the current literature respecting ADHD persistence rates, we see appreciable variability in sources, methods, and symptom thresholds across studies. For example, inclusion criteria for some investigations involving ADHD samples require symptom thresholds that reflect DSM criteria while others utilize a developmentally adjusted norm-based threshold. There is also significant variability in the data collection methodology within these samples; one study might utilize self-report measures while another relies on parent or guardian reports (Sibley et al. 2016). Sibley and colleagues (2016) suggest that persistence rates are higher in studies that utilize parent reports, structured interviews, and a norm-based threshold while lower persistence rates were reported in studies that implemented self-reports, rating scales, and DSM criteria. As a result of this variability in methodology and operationalizations of ADHD, investigations have yielded different results in the estimation of the prevalence of ADHD in adulthood. Despite the range in estimates, there remains sufficient evidence to warrant further investigation, especially in realms of development and aging in clinical populations.

It is valuable to understand the progression of ADHD diagnostic criteria for several reasons. First, recognition of the evolution of the disorder, both in the name and in the elements and characteristics that have been observed in individuals who suffer from it can provide insights into the reasoning for the lack of information regarding ADHD in adulthood and especially older

adulthood. As previously mentioned, the capacity for ADHD symptoms to persist into adulthood was not formally recognized until the publication of the 4th edition of the DSM, less than 30 years ago, even then, the only field trials included in this version were focused exclusively on children and adolescents under the age of 18 (Lahey, 1994). Second, the historical context of ADHD provides evidence for the dynamic nature of ADHD and the need for further clarification. Like many other psychiatric disorders, ADHD can present differently among individuals. There is a great need for further research to clarify cognitive indicators of the disorder that can improve diagnostic procedures through standardization of criteria.

ADHD in Older Adult Populations

Several valuable studies have revealed evidence for the persistence of ADHD symptoms into older adulthood (Michielsen et al., 2018; Michielsen et al., 2012) and the worsening of the overall mental burden of ADHD as age progresses (Brod et al., 2012; Guldberg-Kjär et al., 2013; Guldberg-Kjär & Johansson, 2015; Michielsen et al., 2018). Findings are mixed, however, as other research suggests a decline in the prevalence of ADHD as individuals grow older. One study based on the Australian PATH Through Life Project revealed that in a sample of 3,443 individuals (50% male), when comparing middle-aged adults (48-52 years) and older adults (68-74 years), older adults self-reported lower levels of ADHD symptoms compared to middle-aged adults (Das et al. 2014). Another study investigating the role of ADHD in a Dutch sample of 231 older adults, 23 of which were diagnosed with ADHD, estimated a prevalence rate of 2.8% for syndromic ADHD (the persistence of the diagnostic status) in their older adult sample and a prevalence rate of 4.2% for symptomatic ADHD (the persistence of a partial diagnostic status and cognitive impairment) in the same sample. Overall, younger adults reported significantly more ADHD symptoms than did the older adults in the study (Michielsen et al. 2012). There are

several possible explanations for this decline in ADHD prevalence in older adulthood. Some investigators have suggested the lower life expectancy of individuals with ADHD as being a possible justification for the fewer number of older adults reporting an ADHD diagnosis. This is a valid suggestion given the degree to which individuals with ADHD are at higher risk for accidents, substance abuse, and comorbid psychiatric disorders (Kittel-Schneider, 2019; Torgersen et al., 2016). Torgersen and colleagues (2016) also suggested the lack of adult ADHD diagnostic criteria as a possible explanation for the observed decline in the prevalence of ADHD diagnosis in older adults.

Similar to the conclusion made by Torgersen and colleagues (2016), Simon and colleagues (2009) suggests that adhering closely to the diagnostic criteria presented in the DSM-IV may cause professionals to significantly underestimate the prevalence of ADHD in adulthood. Another factor that may affect the estimated prevalence rates of ADHD in older adults is the age-related health issues that occur in older adult populations. As outlined by Rowe and Kahn's (1997) models of usual vs. successful aging, usual aging is accompanied by the risk of chronic disease; this increase in risk, combined with the limitations of self-report measures that are usually employed in studies investigating the prevalence and burden of ADHD, may be a factor in the possible underestimation of ADHD prevalence rates in older adults. Due to the lack of inclusion of ADHD screening procedures in longitudinal studies related to cognition, ADHD prevalence and its effects in older adults are not well understood. However, one longitudinal study of aging in the Netherlands, the Longitudinal Aging Study Amsterdam (LASA), began to better characterize ADHD in older adulthood with a subsample of participants that underwent diagnostic evaluation for ADHD in 2008.

Several important findings have been presented from LASA that emphasize the value of further investigation into this population. For example, ADHD in older adults has been associated with a higher likelihood of being divorced, fewer family members in the social network, higher levels of emotional loneliness (Michielsen et al., 2013), higher levels of anxiety and depressive symptoms (Michielsen et al., 2013), and lower self-esteem and self-mastery (Michielsen et al., 2014). Though symptoms of ADHD can look much different in adulthood than when they are first observed in childhood, the symptomatic burden can still result in impairment and emotional dysregulation. For example, impulsive behavior resulting from ADHD in adulthood may manifest as anger outbursts, financial carelessness, and reckless driving; while symptoms of inattention in adults may emerge in the form of forgetfulness, disorganization, difficulty with time management, and prioritization of tasks. Further, living longer without having received stimulant treatments for an ADHD diagnosis was associated with a higher likelihood of experiencing comorbid psychiatric disorders (Torgersen et al., 2016). Past research focused on the psychosocial effects of ADHD in older adults is invaluable and gives greater insight into an understudied population. There remains a need, however, for research regarding indicators of cognitive health defined by unique aspects of variability across cognitive tasks that are distinct from average cognitive performance or variability within a single response time task (i.e., RTI). This research holds possible implications for understanding behavioral markers of cognitive impairment in later life.

Past Work Examining Cognitive Function in Older Adults With and Without ADHD

One study has compared cognitive function in older adults with and without ADHD. Like the current project, this study conducted by Semeijn and colleagues (2015) utilized LASA data to investigate associations between ADHD status and cognitive performance in older adults. The

sample used by Semeijn and colleagues comes from a nationally representative sample of older adults (55 – 85 years) in the Netherlands. Three different regions in the Netherlands were identified as areas to gather participants from; these three regions were chosen to represent both rural and urbanized parts of northern and southern areas in the Netherlands. The sample in Semeijn and colleagues' investigation consisted of 231 older adults (age 60 to 94). The aim of the study was to investigate whether older individuals with an ADHD diagnosis performed differently on cognitive tasks when compared to a neurotypical group of older adults. Contrary to hypotheses, the study found that there was no significant association between ADHD diagnosis or ADHD symptoms and performance on the cognitive tasks. One significant result was found between ADHD and domains of working memory and attention such that individuals with ADHD performed worse on working memory and attention tasks. However, this relationship was no longer significant after adjusting for depressive symptoms, suggesting depressive symptoms may partially explain the relationship between ADHD and working memory and attention task performance.

The findings observed by Semeijn and colleagues (2015) provide important implications for the current thesis. First, previous literature investigating the relationship between ADHD and cognitive task performance show poorer performance in individuals who have ADHD (Coghill, 2014, Willcutt, 2005). However, these studies are almost exclusively focused on child and adolescent populations. The results suggested by Semeijn and colleagues may point to important age differences in the occurrence and presentation of ADHD. It is possible that age-related changes in older adults with ADHD are accompanied by protective factors against poorer cognitive function. Further, Semeijn and colleagues (2015) found that cognitive function for neurotypically functioning older adults was not significantly different from adults who had

ADHD with an operationalization of cognitive function as average scores from each task. Moving beyond analyses that compare mean scores alone, it will be valuable to examine the potential unique signal that a more dynamic indicator of variability in cognitive performance may provide. Indeed, mean and variability reflect distinct cognitive processes (Cerino et al., 2021; MacDonald & Stawski, 2020; Jensen, 1992). We will be expanding on the literature regarding associations between ADHD and cognitive function by the utilization of dispersion, an index of an individual's performance variability across multiple domains of cognition.

An Intraindividual Variability Approach to Studying Cognitive Health

Intraindividual variability approaches. Though scientists have been studying the short-term changes that can be observed within individuals for many years (e.g., Nesselroade, 1991), recent empirical advances communicate the value of taking an IIV approach to measure cognitive function. IIV can be defined as short-term dynamic changes in an individual operating on micro timescales (i.e., seconds, minutes, days, or weeks) that can describe people, contexts, or general processes (Cerino & Hooker, 2019). Taking an IIV approach provides investigators with the opportunity to evaluate dynamic fluctuations in cognitive performance (e.g., Cerino et al., 2021; MacDonald & Stawski, 2020; Stawski et al., 2019) beyond the traditionally used measures of central tendency (e.g., mean performance). There are several ways in which methodologies can be designed to observe IIV in a given study. For example, researchers often employ an intensive repeated measurement micro-longitudinal design characterized by collecting repeated measurements of a relevant construct (e.g., stress, affect, cognition) from participants in their daily lives via daily diary entries (Almeida, 2005).

The design of the methods in a given investigation is largely dependent on the way that IIV is operationalized. As mentioned previously, RTI, a measure of the fluctuations in

performance across response times, is one way in which researchers have operationalized IIV in cognitive health. Utilizing IIV methods within domains of cognition and psychosocial functioning provides researchers with an avenue for examining trends in aging within and between individuals across temporal intervals (Cerino & Hooker, 2019). Historically, cognitive aging research predominantly has focused on applying assessments of mean cognitive performance. This approach potentially misses important aspects of variability in performance that may hold unique signals for understanding dynamic characteristics of an individual's cognitive function.

There are important aspects of human functioning and development that may not be adequately represented by estimates of central tendency alone (Spieler et al., 2000; de Ribaupierre, 2018). Measures of IIV may be a more sensitive measure in detecting some of the facets of functioning that are not as well captured by measures of central tendency. For example, two recent studies by MacDonald and Stawski (2020) and Cerino and colleagues (2021) demonstrate how performance variability and mean performance across micro timescales exhibit dissociative patterns in detection of mild cognitive impairment (MCI) in older adults. By using RTI as a measure of IIV, MacDonald and Stawski (2020) found that mean response time (RT) and RTI conveyed different sources of information about cognitive functioning and status. In addition, greater RTI at baseline and steeper increases in RTI over time predicted increased odds of MCI classification above and beyond the influence of mean RT and age. The authors concluded from this study that RTI may be an early behavioral marker of pathological cognitive aging in its predictive utility for MCI. In an ecological momentary assessment design where individuals with and without MCI completed cognitive tests on a smartphone up to six times daily for 16 days, Cerino and colleagues (2021) assessed cognitive status differences in mean

performance and variability in cognitive performance. A dissociative pattern of cognitive status differences in mean performance vs. variability emerged such that variability in processing speed and memory binding performance provided specific detection of MCI (Cerino et al., 2021). The current thesis utilizes an IIV approach to investigate the relationship between ADHD diagnosis and fluctuations in cognitive performance across multiple cognitive tasks. Instead of measuring variability in response time (i.e., RTI, MacDonald & Stawski, 2020) or across repeated assessments (i.e., within-day and day-to-day variability, Cerino et al., 2021), this thesis will measure dispersion as an index of variability across several unique cognitive tasks that were completed during neuropsychological evaluation.

Intraindividual variability and adult ADHD. There has been little research regarding within-individual factors that contribute to differences in levels of multidimensional cognitive function. A neurodevelopmental disorder, like ADHD, constitutes a possible individual factor that could affect a person's cognitive performance and thus provide a representation of their cognitive health. Historically, RTI has been utilized when studying indicators of cognitive health in people with ADHD. Though RTI is usually a unidimensional measure (only representing trial-to-trial fluctuations in one cognitive domain at a time), we can refer to studies investigating RTI trends in individuals with ADHD to provide a foundation on which to build future studies involving other dynamic markers of cognitive performance.

Recent work that takes an IIV approach to understand clinical outcomes has revealed RTI to be a valid indicator of both cognitive impairment and steeper levels of cognitive decline (e.g., Dixon et al., 2007; MacDonald & Stawski, 2020). In a meta-analytic review of 319 studies, Kofler and colleagues (2013) found that both adults and children with ADHD presented with higher levels of RTI than nonclinical control groups. Authors categorized and evaluated age

group differences by examining studies involving child (12 years and below) and adolescent (13 to 18 years) samples, and studies involving adult samples (18 years and above). Child samples exhibited higher effect sizes than adult samples (k = 283 studies, larger effect in child samples as indicated by Hedges g = 0.76 versus medium effect in adult samples as indicated by Hedges g = 0.46; Kofler, 2013). Another example can be found in a study conducted by Buzy and colleagues (2009); participants in this study completed the Visual Serial Addition Task (VSAT) as a measure of working memory and reaction times for each participant were recorded. Researchers concluded that the adolescent participants with ADHD showed significantly higher levels of variability in their performance compared to a control group. Investigators concluded that IIV (operationalized as RTI) was a potential hallmark of ADHD in adolescence. A study by Kuntsi and Klein (2012) suggested similar findings regarding ADHD and higher levels of RTI and also suggested future studies investigate thoroughly the possible neural foundations for the trends in RTI in individuals with ADHD (Kuntsi & Klein, 2012). Additionally, a study conducted using LASA data emphasized the need for further investigation into the cognitive variability that may be present in older adults with ADHD (Semeijn et al., 2015). These findings provide evidence to support the idea that individuals with ADHD present with significantly higher levels of RTI. What is needed now is an examination of cognitive health measurement in ADHD that is not limited to measurements of timing discrepancy alone. The findings from these studies suggest that measuring levels of variability across cognitive domains in people with ADHD may be a valuable way in which a gap in the literature is filled.

Dispersion as a dynamic indicator of age-related cognitive health and pathological cognitive decline. One way in which IIV can be operationalized is through cognitive dispersion, an operationalization of IIV that provides a unique avenue in which ADHD in older adults can be

studied in a way that reflects more than sole task performance variability within a single cognitive dimension as does RTI. In contrast to RTI, a dispersion index gives an estimate of cognitive variability across multiple cognitive domains. This measure, made up through the inclusion of performance scores for two or more cognitive tests, results in a more thorough and inclusive index of cognitive health across multiple domains of functioning. A growing body of literature has utilized dispersion indices as a behavioral marker of age-related cognitive health outcomes (Hilborn et al., 2009) and pathological cognitive decline (e.g., Halliday et al., 2018). For example, greater dispersion has been shown to be associated with a higher likelihood of being classified with Alzheimer's disease (Halliday et al, 2018). This study involved the administration of 11 tasks spanning domains of episodic memory, executive function, and language to a sample of 60 adults aged 65 and older. From the test battery included in the study, Halliday and colleagues (2018) derived a dispersion score that reflected each participant's level of performance variability across the cognitive tasks. These scores were used to make predictions concerning later cognitive impairment. Halliday and colleagues (2018) found that dispersion may be a sensitive marker of cognitive impairment as greater dispersion levels were associated with increased likelihood of being classified with Alzheimer's disease. Regarding other health outcomes, it has been suggested that higher levels of dispersion (i.e., higher levels of between-task, within-person variability) are associated with faster rates of entorhinal and hippocampal atrophy, as well as functional decline after controlling for age, sex, apolipoprotein E genotype, amyloid-β positivity, and mean level of cognitive performance (Bangen et al., 2019).

Higher levels of dispersion have also been found to be associated with older age in general (Hilborn et al., 2009; Hultsch et al., 2002). In one study investigating cognitive function associated with normative aging, researchers administered 15 different cognitive tasks to 197

participants (Schretlen, 2003). Investigators in this study found that higher dispersion was associated with higher ages. From these studies we learn that not only has greater dispersion been shown to be a possible marker of pathological aging, but we also see that greater dispersion is also related to older age. In a sample of 304 older adults (ages 64 – 92 years, M = 74.02) Hilborn and colleagues administered 9 cognitive tasks. Performance scores on these tasks were used to create a dispersion index that indicated the variability in task performance across all tasks. The results of this study revealed that higher levels of dispersion were occurring both in older individuals and in individuals who had experienced cognitive decline (Hilborn et al., 2009).

This evidence has led to the utilization of dispersion as a marker of general cognitive health and cognitive status, in that higher levels may be an indicator of both age-related and pathological cognitive declines and impairment. Given the evidence presented that individuals with ADHD tend to present with higher levels of variability, at least as measured through RTI within a single task, one could hypothesize that individuals diagnosed with ADHD may also present with higher levels of dispersion, thus putting them at higher risk for negative health outcomes in the future. Currently, no work that we know of has investigated age differences in dispersion in a sample of older adults with and without ADHD. This thesis project will also expand on the current knowledge concerning the relationship between age and dispersion by examining age differences in dispersion in a Netherlands sample with clinical relevance (i.e., older adults with and without ADHD).

Chapter III. Study Overview

There are significant gaps in the literature regarding cognitive trends in older adults with ADHD. Currently, the field has very few resources that function as guidelines in the diagnosis and treatment of ADHD in older adults; what is available in the literature is almost exclusively

based on the incidence of ADHD in childhood and adolescence. This study aims to fill these gaps in the literature by investigating the roles that an ADHD diagnosis and age may have on the levels of dispersion observed in older adults. This will provide insight into the general cognitive function of older adults with ADHD and could provide valuable information concerning possible cognitive markers of ADHD.

Purpose, Research Questions, and Hypotheses

The current study aims to characterize dispersion in older adults with and without ADHD by investigating the associations of an ADHD diagnosis and age with cognitive dispersion (i.e., a person's manifested variability in performance across several domains of cognitive function). There are three research questions addressed in this thesis.

The first question relates to levels of dispersion and ADHD diagnosis. (1) What is the relationship between ADHD diagnosis status and levels of dispersion? Given the trends in heightened cognitive variability observed in ADHD populations (Kofler et al., 2013), we hypothesized that adults who have been diagnosed with ADHD will have higher levels of cognitive dispersion when compared to neurotypical adults. We also investigated whether the increase in levels of dispersion were independent of sociodemographic and mental health influences.

The second question that was addressed relates to the quantity of ADHD symptoms reported by an individual and their levels of cognitive dispersion. (2) What is the relationship between ADHD symptoms and levels of dispersion? When considering the cognitive burden of ADHD symptoms, past research has found more ADHD symptoms are related to lapses of attention, memory loss, and slower information processing speed (Gmehlin et al., 2016; Skodzik et al., 2017, Calhoun & Mayes, 2005). These results led us to hypothesize that the more ADHD

symptoms a person possesses (or the worse the severity of ADHD), the higher the levels of cognitive dispersion will be in a given individual.

Our third question evaluates whether previously reported results on higher levels of dispersion occurring in older age extends to a sample of older individuals with and without ADHD in the Netherlands. (3) What is the relationship between age and levels of dispersion? Informed by previous research (Hilborn et al., 2009; Hultsch et al., 2002), we hypothesized that older individuals will exhibit greater dispersion than comparatively younger adults.

Chapter IV. Method

Longitudinal Aging Study Amsterdam (LASA)

The data utilized in this study was drawn from the 2008-2009 wave of LASA (See Flowchart, Figure 1). This longitudinal study was started in 1991 by the Ministry of Health Wellness and Sport to investigate the predictors and outcomes associated with aging in the Netherlands. When the study began, the initial sample represented people ages 55 to 85 years of age. The study focuses on "the determinants, trajectories, and consequences of physical, cognitive, emotional and social functioning" (LASA, https://lasa-vu.nl/en/lasa-main-study/design-lasa/). LASA seeks to evaluate physical, cognitive, emotional, and social functioning through a variety of measures. Topics included in LASA can be subdivided into themes of care (use of care services and perceptions about care, social functioning, cognitive functioning, work, emotional functioning, physical functioning, biomaterial, and demographic aspects).

Data Collection Procedures

Measurement cycles for LASA occur every three years and are conducted by trained interviewers who would visit the participants in their homes. There were two main parts to the

interview session – the main interview and a medical interview which included clinical measurements and an ADHD screening measure in one of the waves of the study (wave G, 2008-2009; See Flowchart, Figure 1). The first cohort began their observation cycles in 1992 and waves continued until an additional cohort of participants was added to the study. This second cohort included participants who ranged in age from 55 to 64 years and was measured in 2003-2004.

The data collection process was divided into 5 different phases. The first phase, called the "preparatory phase", includes the preparation of the testing material and the means of interview. For example, the main and medical interviews were administered by the interviewers asking the participant questions and then entering the respondent's answers directly into a computer. The self-administered questionnaire also required preparation as it was administered through paper and pencil or online, depending on the preference of the participant. These materials required careful evaluation to allow for ease of use and convenience. In preparation for the interviews, the interviewers underwent extensive training on how best to interact with the participants, how to properly use the computer equipment, and became familiar with the interview questions.
The second phase is termed the "data collection" phase and includes 4 main parts. The main interview portion of data collection typically began in September with the medical interview portion of the observation cycle taking place only a few weeks after the main interview. An entire data collection cycle generally took just over a year to complete. Participants were contacted by the interviewers to schedule an appointment for an interview that was estimated to last two hours. All participants were asked to give informed consent and were asked for permission to have the LASA team members contact the participant's medical practitioners in the circumstance of possibly needing more information than was provided by the participant.

Interviewers for the medical interview were recruited specifically for this portion of the data collection process. It was important to the LASA researchers that these interviewers not only have a background in medical contexts (such as nursing or medical assistantships) but also were required to have an affinity for older adults. The medical interviewers were required to contact the participants about scheduling a time for the interview within 3 weeks of the completion of the main interview.

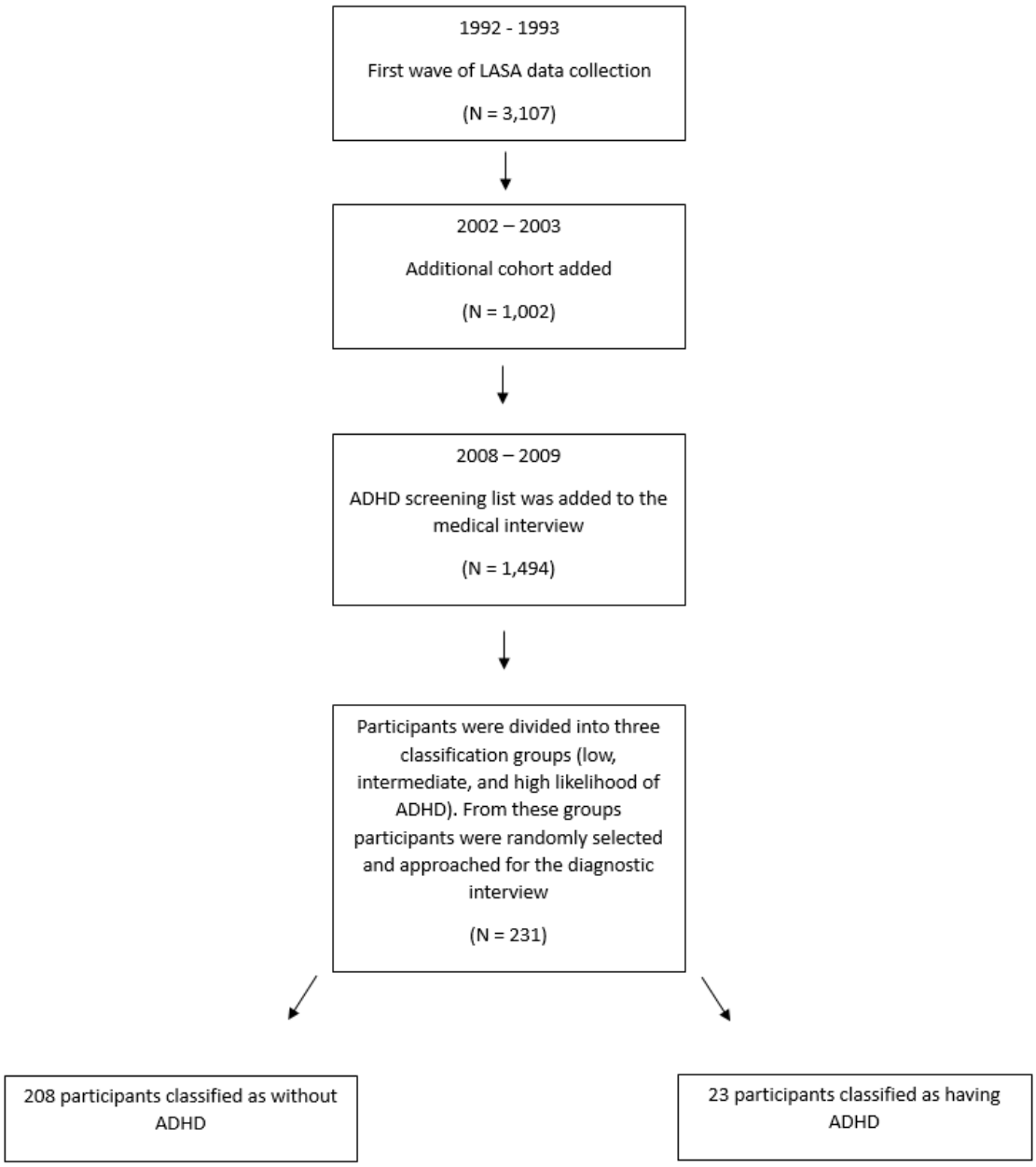
The third and fourth phases of the data collection process were concerned with the processing and transforming of the data files. LASA reports that for all collected data, data entry occurs twice by trained typists who were familiarized with the methods of the study. The two raw data files for all of the data were then compared and inconsistencies were corrected; after which raw SPSS data files were generated. Lastly, the fifth phase is focused on the final steps of data preparation. This last step included basic data cleaning by referring to comments made by the interviewers on report forms that were submitted for specific observations. The resulting data was then checked for internal consistency and validity before being disseminated to researchers.

ADHD Screening Measures in LASA

An ADHD screening list was added to the medical interview portion of the collection cycles in LASA in the 2008/2009 wave of the study. This screening list is based on the ADHD questionnaire established by Barkley et al. (2007) and includes 7 items concerning inattention, one item on hyperactivity, one item on impulsivity, and one item clarifying if the symptoms started before the individual was 16 years old (see appendix C); each item requires a yes/no response. A total ADHD score was generated by summing the scores on the nine items. Based on the results of the ADHD screening measure, the sample was then divided into three groups, these groups were categorized as having a low, intermediate, or high likelihood of an a priori ADHD

diagnosis. After the groups were distinguished, they were randomly sampled for participants who would be approached for the ADHD diagnostic interview. Random samples of the participants in the low and intermediate groups were invited to take part in the diagnostic interview, as were all of the participants in the high likelihood group (N = 271). In the end, 85 (90%) respondents in the low-scoring group, 80 (86%) in the moderate-scoring group, and 69 (82.3%) in the high-scoring group consented to be interviewed (Semeijn et al., 2015). After the exclusion of 6 participants who were unable to complete the interview, there was left a total of 231 participants in the sample ranging in ages between 60 and 94 (Average age=71.64 years, SD=7.7, 59% female). These 231 individuals comprise the analytic sample for this thesis (See Flowchart, Figure 1).

Figure 1. Study Flow Chart



ADHD Diagnostic Interview

The ADHD diagnostic interview that the 231 adults completed includes the use of the second version of the Diagnostic Interview for ADHD in Adults (DIVA). This measure is based on the DSM-IV-TR criteria for ADHD and is commonly used by practitioners in the Netherlands. The DIVA 2.0 utilizes criteria from the DSM-IV-TR by including items about childhood symptomatology as well as items referring to experiences with ADHD in adulthood, thus the measure evaluates both impairments in childhood and current impairment. A participant would be classified as a "case" when they met the following criteria: at least four symptoms of either inattention and/or hyperactivity-impulsivity during six months or longer prior to the interview, and at least six symptoms of either inattention and/or hyperactivity/or impulsivity in childhood (5–12 years of age; Kooij et al., 2005). In addition, the participants were required to present with significant impairment in at least two domains of their daily living (work, education, family, social and relationships, and self-confidence) during at least 6 months prior to the interview and in childhood, as per the DSM-IV-TR diagnostic criteria.

ADHD Symptoms

The sum score of all ADHD symptoms at present time and in childhood collected during the diagnostic interview was used to create a score for the total number of ADHD symptoms for each participant. Scores ranged from 0 to 36 where higher scores indicated more ADHD symptoms.

Data Acquisition and IRB Approval

A data acquisition request was submitted to the LASA research team on April 25th, 2022. This request included the completion of a data sharing agreement document which specified the general purpose of this project, and the submission of a data analysis proposal. This

proposal outlined the objectives, rationale, and research questions in the current thesis and included a list of the variables of interest. On May 29th, 2022 we were informed that the data request submitted in April was approved and we would be granted access to the relevant data files collected through LASA. The data files containing the primary study variables were acquired on June 22nd, 2022 with additional files containing ADHD symptom and diagnostic data being received on July 8th. On June 6th, 2022 the primary investigator of this thesis project submitted the project for review from the Northern Arizona University Institutional Review Board (IRB). On July 21st, 2022, we received IRB approval (Project Number 1933310-1) with designation that this secondary data analysis project did not fall under the classification of research involving human subjects.

Transparency and Openness

Data are available upon submission of a data acquisition request to the LASA research team (https://lasa-vu.nl/en/lasa-main-study/design-lasa/). All analyses were completed using SAS 9.4 (SAS Institute, 2013). All study materials, diagnostic tools, and study analysis code are available below in appendices (Appendix C, Appendix D, Appendix E). To improve transparency in data visualization (e.g., Weissgerber et al., 2019), we have included scatter plots that illustrate relationships between ADHD symptoms and dispersion (Research Question 2) and age and dispersion (Research Question 3) with all observations visible behind the fitted regression line (Appendix B, Figure 4A and 4B). We have also included box plots that illustrate the distribution detail of dispersion differences among individuals with and without ADHD (Research Question 1), as well as the distribution of other study variables and individual cognitive tasks among individuals with and without ADHD (Appendix B, Figures 5 and 6).

Measures of Cognitive Functioning

To create a dispersion index reflecting an individual's cognitive functioning across multiple domains, this study was granted access by LASA to performance score data that came from 13 different outcome scores from 8 cognitive tasks. These tasks covered the cognitive domains of attention, fluency, memory, processing speed, and reasoning. The following section will provide detailed descriptions of the cognitive tasks and outcome scores, the mode of administration to participants, and the methods of scoring and evaluating individual performance results.

Mini-Mental State Examination. The Mini-Mental State Examination (MMSE) was used as part of the main interview to measure general cognitive functioning in LASA participants. The MMSE is a well-known cognitive task that is often used as a way to measure cognitive impairment (Pangman et al. 2000). Administration of the MMSE task generally takes 4-21 minutes and includes 30 items that span domains of orientation, registration, attention, calculation, recall, and language (Tuijl et al. 2011). Given that the measure is scored out of 30, a score of 25 or more is considered normal cognitive functioning based on the MMSE. A score below 24 may indicate abnormal functioning or impairment.

Raven's Progressive Matrices. To measure non-verbal reasoning in LASA participants, the Raven's Coloured Progressive Matrices (RCPM) was administered as part of the main interview. The RCPM is widely considered an effective measure of fluid intelligence and is often included in the clinical assessment of patients with cognitive deficits (Bilker et al. 2012). The task contains 60 items, with levels of difficulty increasing as the participant progresses through the items; the items are designed so that participants are required to choose an element that would complete the pattern of the image presented to them. The version that was utilized by LASA is called The Raven's Coloured Progressive Matrices; this version was specifically

designed to be utilized for children ages 5 through 11, and older adults. The colored progressive matrices are characterized by A and B item sets from the standard RCPM and include an additional set of 12 items. The test was created to include a colored background to make the task more visually appealing and engaging to the participant. The test consists of 36 total items, with a correct response being scored with 1 and an incorrect response being scored with 0. Thus, performance scores can range from 0 to 36, with higher scores indicating better performance (Eissa & Alsayed, 2012). In the case of LASA data collection, the task was shortened to only include parts A and B in order to reduce test burden for the participant; as a result of the exclusion of part ab, performance scores could range from 0-24.

Rey Auditory Verbal Learning Test. Memory was assessed in the main interview using an abbreviated version of the Rey Auditory Verbal Learning Test (AVLT) in LASA participants. The AVLT is recognized to be a useful tool in the assessment of an individual's ability to encode, combine, store, and recover information from immediate memory (Fard et al., 2016). Within the span of 3 trials, participants were asked to recall 15 words that were read to them out loud by the interviewer. A sum score of the three trials was derived to represent the participant's immediate recall, these scores could range from 0 to 45, with higher scores indicating better performance. After a period of 20 minutes, the participants were again asked to recall as many words as possible, this measure was used to derive a delayed recall score ranging in scores from 0 to 15 with higher scores indicating better performance.

Alphabet Coding Task. Information processing speed was evaluated in the main interview portion of LASA data collection using an adapted version of the Alphabet Coding Task (ACT). This coding task, as used in LASA is an adapted version of the ACT-15 that was established by Savage in 1984, which was adapted from a letter substitution task established by

Piccinin and Rabbitt (1999). Participants in this task were asked to view two rows of corresponding characters, they were then asked to complete the letter combinations as best they could within 3 one-minute cycles. The mean score for the three trials was used for analysis with higher scores reflecting better performance.

Stroop Color-word Test. The Stroop Color-word Test (SCWT) was implemented in the medical interview as an additional measure of information processing speed. The test consists of 3 cards that either include color words printed in black and white (card 1), 0.7cm x 2.0cm colored patches (card 2), and color words printed in a different color than what the name reads (e.g. the word red is printed in green ink; card 3; Klein, 1997). For card one, the participants are asked to read the words as fast as possible, participants viewing card two were asked to name the colors of the patches as quickly as possible, and card three required the participant to name the color of the printing ink instead of reading the word. In LASA however, the SCWT was used to assess information processing speed by using cards 1 and 2 only. The time it took for participants to name the words and colors were used to calculate the information processing speed score, with higher scores indicating worse performance. The 3rd card in the SCWT was used to assess executive functioning. An interference score was calculated using performance results on the third card, the interference score was calculated by subtracting the total time taken on card two from the total time taken on card three, with higher scores also indicating worse performance.

Trail Making Task. The trail-making task (TMT) part A and B were used to assess motor speed, working memory, and executive functioning. Part A evaluated motor speed. Part B evaluated executive function and working memory. This task was administered as part of the medical interview portion. The trail making tasks have been shown to be sensitive to detecting cognitive impairment associated with Alzheimer's disease and related dementias. To complete

part A, participants were asked to connect 25 dots on a single page, these dots were in numerical order, and participants were asked to complete the task as quickly as possible. Part B of the TMT also required participants to connect dots by drawing lines, instead of only numbers present in the dots however, letters have been added to complicate the task. This test has been shown to be an effective predictor of cognitive impairment (Partington and Leiter, 1949), the number of seconds that it took to complete the tasks was recorded with lower scores indicating better performance.

Word Fluency Test. Two tasks were implemented as part of the medical interview that were used to assess word fluency, a semantic test, consisting of an animal naming task, and a phonemic test, consisting of word naming that begins with a prespecified letter. The verbal fluency test is an effective measure of cognitive impairment and dementia (Sebaldt et al., 2009; Canning et al., 2004). In LASA, for the phonemic fluency task, participants were asked to name as many words as they could that started with the letter D. For the semantic test, LASA participants were asked to name as many animals as they could. For both tests the time limit was one minute; performance was measured by recording the number of unique and correct words per task. A higher score on these tests indicates better performance.

Digit Span. To examine attention and working memory, LASA participants completed the Digit Span subsection of the Wechsler Adult Intelligence Test as part of the medical interview. This test included the interviewers reading a series of numbers and then asking the participants to recite the numbers in ascending order first (Digit Span Forward), where the numbers ranged from 0 to 16, and then in descending order where the numbers ranged from 0 to 14 (Digit Span Backward). Higher scores on the Digit Span Forward and Digit Span Backward tasks constitute better performance.

Potential Confounding Variables

Several variables may be related to ADHD diagnosis and cognitive function in older adulthood that have the potential to confound the relationship under investigation in this thesis. Sex differences were accounted for by controlling for sex (male, female) in this analysis. Differences in cognitive functioning and learning disorder comorbidity rates have been found to be dependent on sex (Koerner, 2021; Burstein et al., 1980); though the literature has been inconsistent regarding which group tends to perform at higher levels, some work has found that women generally perform better on verbal tests while men perform at higher levels on visual-spatial tasks (Weiss et al., 2003). In addition to these findings, sex differences in children with ADHD are associated with intellectual impairment, levels of hyperactivity, and levels of externalizing behavior.

Although research findings are mixed regarding age differences in cognitive function among those with and without ADHD, we decided to include chronological age (in years) as a covariate based on past work in LASA by Michielsen and colleagues (2012) showing age differences in the cognitive characterization of ADHD such that younger adults reported significantly more ADHD symptoms than older adults. As previously mentioned, age differences have also been found to be associated with differences in levels of cognitive dispersion (Schretlen et al., 2003). These results could imply worse cognitive health outcomes for older individuals with more variability in performance across the cognitive tasks. Level of educational attainment (1=elementary not completed, 2=elementary education, 3=lower vocational education, 4=general intermediate education, 5=intermediate vocational education, 6=general secondary education, 7=higher vocational education, 8=college education, 9=university education) was also included as a covariate in the analysis based on literature that suggests a

relationship between ADHD diagnosis and poorer grades, lower standardized test scores, and relatively low rates of high school graduation (Loe et al., 2007).

Lastly, depressive symptoms were included in our analysis as a covariate due to past work identifying more depressive symptoms were associated with an ADHD diagnosis (Michielsen et al., 2013). Indeed, Semeijn and colleagues (2016) found that the association between ADHD and working memory and attention tasks was partially explained by reported depressive symptoms. In addition to the potential for depressive symptoms to influence the outcome measure (levels of dispersion) as a result of their associations with ADHD, there is evidence to suggest that depressive symptoms are also linked to cognitive performance. A study conducted by McDermott and Ebmeier (2009) revealed that significant correlations were present between depressive severity and cognitive performance in domains of episodic memory, executive function, and processing speed. Depressive symptoms were measured with the 20-item self-report Center for Epidemiological Studies Depression Scale (Radloff, 1977), with items assessing depressive symptomatology in the past week on a 4-point Likert-type scale from 0 (rarely or never) to 3 (mostly or always). A total score of 20 items (ranging from 0 – 60) will be used as a covariate in analyses, with higher scores reflecting more depressive symptoms. In this sample, the CES-D demonstrated adequate internal consistency as indicated by Cronbach's alpha estimate of reliability ($\alpha = 0.90$).

Analytic Plan

Data preparation and calculation of dispersion profiles. To prepare for data analysis, the following measures were taken with the raw datasets provided by LASA. Outliers in the cognitive performance data were assessed and observations that fell 3 standard deviations above or below the mean performance for individuals with and without ADHD were flagged for

removal prior to analysis. Ultimately, 13 observations were removed; two observations from the Stroop total time variable, six observations from the Stroop interference variable, three observations from the trail making task A variable, and two observations from the trail making task B variable were removed due to extreme values that skewed the distribution and were deemed as outliers among the sample. Only two of the observations that were removed were from a participant with ADHD. The study variables were screened for missing data and assumptions of regression methodology. Specifically, linearity between predictor and outcome variables were assessed via scatter plots and showed no evidence of curvilinear relationships (i.e., albeit small, relationships appeared linear). Multivariate normality was assessed via histograms and showed that residuals of the regressions (errors between observed and predicted values) appeared normally distributed. Multicollinearity was assessed via correlation matrices among all predictor variables and showed that no multicollinearity was present (i.e., all correlation coefficients were less than .80). Homoscedasticity was assessed via scatter plots of residuals versus predicted values and showed no clear patterns in distributions (i.e., homoscedasticity met). Due to cell size limitations and lack of coverage of every level of education among people with ADHD, the decision was made to remove the education variable as a covariate from analyses to address the research questions.

Consistent with prior approaches in the dispersion literature (e.g., Halliday et al., 2018), we generated dispersion profiles using regression techniques to obtain an intraindividual standard deviation for each participant. This was achieved by creating a standard deviation for each individual across the 13 outcome scores from the 8 different cognitive tasks using T-scores. The scores used in the dispersion index were the MMSE, RCPM, AVLT Immediate Recall, AVLT Delayed Recall, ACT, SCWT speed score, SCWT interference score, TMT A, TMT B,

Word Fluency Phonemic Test, Word Fluency Semantic Test, Digit Span Forward, and Digit Span Backward. First, the test scores were standardized as T-scores (M = 50, SD = 10) to put all cognitive test scores on a common metric. Next, intraindividual standard deviations were calculated from these standardized T-scores for each participant. The resulting scores represent each individual's level of variability across tasks. Larger dispersion scores indicate more variability in performance across tasks and domains. Lower scores indicate a more consistent cognitive performance across tasks and domains. To further describe the sample, descriptive statistics (e.g., means, standard deviation, range) and bivariate correlations were also assessed for all primary study variables (dispersion score, ADHD diagnosis, number of ADHD symptoms, age, sex, education level, and depressive symptoms). Evaluation of descriptive statistics for primary study variables and each cognitive task were completed for the entire sample, as well as stratified by ADHD status (i.e., for individuals who were diagnosed with ADHD and for individuals without an ADHD diagnosis; see table 1 and table 2).

Statistical Power Considerations. Following approval of our data sharing agreement, the LASA team shared the potential for limited statistical power to detect effects when using the ADHD diagnosis variable. We conducted a power analysis using GPower (Faul et al., 2007) with a power of .80 (i.e., probability that the test correctly rejects null hypothesis when an alternative hypothesis is true), a group of 23 individuals with ADHD compared to a group of 208 individuals without ADHD, and an alpha of .05. We have the power to detect medium-to-large effects for ADHD diagnosis on dispersion (~Cohen's d =.55 or larger). In the approval letter, the LASA team recommended that we add the total ADHD symptom score (range 0-36) as an additional predictor that may help increase statistical power to detect the potential for

comparatively smaller effects. Accordingly, this suggestion aligns to the second research question of this thesis assessing relationships between ADHD symptoms and dispersion.

Analyses to Address Research Questions. SAS 9.4 was used to analyze the data cross-sectionally to assess ADHD and age differences in dispersion scores. An initial independent samples t-test was used to evaluate whether levels of dispersion differed between participants with and without ADHD. Hierarchical linear regression methods were then used to further quantify the impacts of ADHD and age by regressing dispersion on ADHD and age. To address the first research question on the relationship between an ADHD diagnosis and dispersion, an initial regression model was used to regress dispersion scores on ADHD diagnosis. A second regression model added age, sex, and depressive symptoms to determine whether the relationship between ADHD diagnosis and dispersion is robust to the influence of sociodemographic differences and mental health. The same hierarchical regression approach was used to address the second research question on the relationship between ADHD symptoms and dispersion (replacing ADHD diagnosis with number of ADHD symptoms). To address the third research question on the relationship between age and levels of dispersion, an initial regression model was used to regress dispersion scores on age. A second regression model added ADHD status, sex, and depressive symptoms to determine whether the relationship between age and dispersion is robust to the influence of ADHD status, sociodemographic differences, and mental health. In regression analyses, we mean-centered our continuous variables (mean ADHD total symptoms = 10.75, mean age = 71.64 years, and mean depressive symptoms = 9.48) to facilitate meaningful and interpretable parameter estimates. Effect size for the independent samples t-test in research question 1 was assessed with Cohen's d small to medium (.10 to .30), medium to large (.30 to .50) and large (.50 and larger) effect size evaluation. Effect sizes in the regression analyses for

research questions 1, 2, and 3 were assessed with partial eta-squared (η2) small (η2 = 0.01), medium (η2 = 0.06) and large (η2 = 0.14) effect size evaluation.

Chapter V. Results

Descriptive Statistics

Table 1 provides descriptive statistics for primary study variables. Table 2 provides descriptive statistics for the cognitive tests. Box plots by ADHD status (0=No ADHD, 1=ADHD) for dispersion, each cognitive task, and other study variables are provided in Appendix B to provide additional descriptive and distribution detail of cognitive performance differences and other study variable differences among individuals with and without ADHD. The average dispersion score across the whole sample was 9.30 (*SD* = 3.23, Range = 3.33 – 20.72). The average dispersion score for the ADHD group was 8.49 (*SD* = 4.05, Range = 3.33 – 20.72) while the average for the no ADHD group was 9.39 (*SD* = 3.13, Range = 3.36 – 18.55). The no ADHD had a higher mean performance than the ADHD group (*M* = 50.08, *SD* = 3.28, Range = 42.11 – 60.12 versus *M* = 49.09, *SD* = 3.31, Range = 43.72 – 58.87), but this was not a significant difference, *t*(229) = 1.37, *p* = 0.17. Compared to people without ADHD (*M*age = 72.04, *SD* = 7.85, Range = 60.92 - 94.51), people with ADHD were significantly younger (*M* = 68.02, *SD* = 4.87, Range = 61.97 – 79.87, *t*(229) = 2.40, *p* = 0.01) and presented with a significantly higher number of depressive symptoms (*M* = 16.78, *SD* = 7.46, Range = 0 – 37, *t*(229) = -4.57, *p* = <0.001). The only cognitive test that revealed a significant difference between the ADHD group and the no ADHD group was the digit span backward test which is a cognitive test used to measure attention and working memory. Our descriptive analysis revealed that the no ADHD group performed significantly better on the digit span backwards test (*M* = 5.32, *SD* = 1.77, Range = 1 – 12) when compared to the ADHD group (*M* = 4.39, *SD* = 1.59, Range = 2 – 9,

$t(229) = 2.42$, $p = 0.02$). People with and without ADHD had statistically comparable values for education, sex, and all other cognitive tests (all ps > .05).

Given this project's focus on evaluating levels of variability, we wanted to also highlight group level differences in standard deviations in our descriptive statistics. The standard deviations for the ADHD and no ADHD group reported in Table 2 describe the amount of deviation or spread of the data relative to its mean for each group of participants. As communicated in Table 2, there was more spread/variability across the group of participants without ADHD compared to the group of participants with ADHD. The group of participants without ADHD had higher standard deviation values than the group of participants with ADHD for 8 out of the 13 tasks (Trail Making A, Trail Making B, Animal Fluency, Letter Fluency, Digit Span Forward, Digit Span Backward, Ravens Matrices, and Alphabet Coding Task). The group of participants with ADHD had higher standard deviation values in 4 out of the 13 tasks (Stroop Total Time, Stroop Interference, MMSE, and Immediate Recall). Groups of participants had similar standard deviations for Delayed Recall. This helps communicate that the participants without ADHD tend to vary more from each other and their group mean than the participants with ADHD. The index of variability for this thesis, dispersion across cognitive tasks, represents a distinct form of variability from the group level standard deviation as we compute an intraindividual standard deviation for each participant to understand each person's amount of deviation or spread in performance across 13 cognitive tasks.

Bivariate correlations among primary study variables are provided in table 3. The bivariate analysis revealed that there was a significant positive relationship between ADHD status and the number of ADHD symptoms $r(229) = 0.59$, $p < 0.001$, as well as a significant positive relationship between ADHD status and depression $r(229) = 0.29$, $p < .001$. The

correlation analysis also revealed that having an ADHD diagnosis was associated with being younger, $r(229) = -0.16$, $p = 0.01$. Regarding significant correlations with levels of dispersion, the correlation analysis showed that age is associated with higher levels of dispersion $r(229) = 0.15$, $p = 0.02$. Several important correlations were observed that emphasize the role of mental health among relationships between study variables. For example, the correlation analysis revealed a significant positive relationship between depressive symptoms and ADHD, $r(229) = 0.29$, $p < 0.001$, as well as a significant positive relationship between depressive symptoms and the number of ADHD symptoms, $r(229) = 0.32$, $p < 0.001$.

ADHD Status and Dispersion (Research Question 1)

Using an independent samples t-test, we assessed whether there was a significant difference in the levels of dispersion among people with ADHD (n = 23, $M = 8.49$, $SD = 4.05$, range = 3.33 - 20.72) and without ADHD (n = 208, $M = 9.39$, $SD = 3.13$, range = 3.36 – 18.55). Results of the independent samples t-test can be found in table 4. The analysis revealed that this small difference in levels of dispersion (Cohen's $d = 0.25$) was not a statistically significant difference between the two groups, $t(229) = 1.27$, $p = 0.20$. Similarly, the regression analysis revealed that ADHD status was not significantly related to levels of dispersion. Compared to individuals without ADHD, people with ADHD had 0.90 units less dispersion ($b = -0.90$, $SE = 0.71$, $p = 0.20$, 95% CI: -2.30 to 0.49, $\beta = -0.08$; small effect size indicated by partial $\eta 2 = 0.01$). ADHD status continued to not be significantly related to dispersion after adjusting for sociodemographic differences and depressive symptoms ($b = -0.73$, $SE = 0.76$, $p = 0.34$, 95% CI: -2.21 to 0.76; $\beta = -0.07$; small effect size indicated by partial $\eta 2 = 0.01$). See table 5 for full regression results.

ADHD Symptoms and Dispersion (Research Question 2)

To assess the relationship between the number of ADHD symptoms and levels of dispersion, we ran a second set of regression analyses including the ADHD total symptom variable instead of the ADHD status diagnostic variable. The analyses revealed that there was not a significant relationship between the number of ADHD symptoms and levels of dispersion in individuals with ADHD (b = -0.03, SE = 0.03, p = 0.38, 95% CI: -0.08 to 0.03; β = -0.06; small effect size indicated by partial η2 = 0.003). The relationship between ADHD symptoms and dispersion remained non-significant after adjusting for sociodemographic differences and depressive symptoms (b = -0.01, SE = 0.03, p = 0.73, 95% CI: -0.07 to 0.05; β = -0.03; small effect size indicated by partial η2= 0.004). See table 6 for full regression results.

Age Differences in Dispersion (Research Question 3)

The third research question examining a possible relationship between age and dispersion was addressed using regression analyses (see Table 7). Older age was associated with significantly higher levels of dispersion (b = 0.06, SE = 0.03, p = 0.02, 95% CI: 0.01 to 0.12; β = 0.15; small to medium effect size indicated by partial η2= 0.02). A one-year increase in age was associated with a 0.06 unit increase in dispersion. The significant relationship between older age and higher levels of dispersion was robust to statistical adjustment for ADHD status, sex, , and depressive symptoms (b = 0.06, SE = 0.03, p = 0.045, 95% CI: 0.001 to 0.11; β = 0.114); small to medium effect size indicated by partial η2= 0.02). See table 7 for full regression results.

Supplemental Analysis Using Categorical Grouping Variable of ADHD Symptoms

In addition to the primary analyses utilized to assess the aforementioned research questions, we wanted to further describe the possible relationship between ADHD symptoms and dispersion by categorizing and analyzing the ADHD symptoms by 4 groups, (Group 1) those without any ADHD symptoms (n=5), (Group 2) those with ADHD symptoms only in childhood

(*n*=6), (Group 3) those with ADHD symptoms only in adulthood (*n*=27), and (Group 4) those with ADHD symptoms both in childhood and adulthood (*n*=193). When we assessed the difference in levels of dispersion across the 4 groups using a one-way analysis of variance we found that there were no significant differences between the groups of people in their levels of dispersion ($F(3,227) = 0.17$, $p = 0.92$). In recognizing that an omnibus analysis of variance is a crude and overarching assessment which evaluates for any significant difference among the four groups, we wanted to further clarify the relationship between ADHD symptoms and levels of dispersion by running additional comparisons via a post hoc Tukey procedure. The additional tests assessed mean group differences through pairwise contrasts (i.e., Group 1 vs. Group 2, Group 1 vs. Group 3, Group 1 vs. Group 4, Group 2 vs. Group 3, Group 2 vs. Group 4, Group 3 vs. Group 4). The pairwise comparisons further indicated that there were no significant differences between the groups (those without any ADHD symptoms compared to all other groups, those with ADHD symptoms only in childhood compared to all other groups, those with ADHD symptoms only in adulthood compared to all other groups, and those with ADHD symptoms in childhood and adulthood compared to all other groups). All contrasts indicated comparable values among the four groups (all *p*s > .05).

Sensitivity Analysis Using All Available Data

To examine whether removing outliers in initial data cleaning impacted the results, we conducted sensitivity analysis using all available data (including the 13 observations of cognitive task performance we removed for primary analyses). Patterns of association (positive/negative), magnitude of effects (size of parameter estimates), and statistical significance (p-values, 95% CIs) were similar between the primary analyses among cleaned cognitive task variables and the sensitivity analysis using all available data. Using all available data, an independent samples t-

test revealed no significant difference in dispersion between the ADHD group and no ADHD group, $t(229) = 1.39$, $p = 0.17$. In regression analyses among all available data, ADHD status was not significantly related to dispersion ($b = -1.04$, $SE = 0.75$, $p = 0.17$, 95% CI: -2.51 to 0.43, small effect size indicated by $\eta 2 = 0.01$). The amount of ADHD symptoms was also not significantly related to dispersion using all available data ($b = -.04$, $SE = .03$, $p = 0.15$, 95 % CI: -0.10 to 0.02, a small effect size indicated by $\eta 2 = .01$). Using all available data, older age continued to be significantly related to higher levels of dispersion ($b = .08$, $SE = .03$, $p = .01$, 95% CI: 0.03 to 0.14, small to medium effect size indicated by $\eta 2 = 0.03$).

Chapter VI. Discussion

In this investigation, we were interested in evaluating whether there would be differences in levels of dispersion when comparing groups of older adults with and without ADHD in a Dutch sample. We were also interested in assessing whether the evidence of a significant positive relationship found between age and levels of dispersion in past literature remains consistent in this clinically relevant sample of older adults with and without ADHD. Results from an independent samples t-test and linear regression analysis revealed that levels of dispersion, as measured by variability in performance across 13 unique indices of cognitive function, are not significantly different for older adults with ADHD when compared to a group of older adults without ADHD. Additionally, results from the regression analyses showed that there was no significant relationship between the number of ADHD symptoms and levels of cognitive dispersion; this finding held true even when we divided the symptoms variable into four groups in additional analysis representing those without any ADHD symptoms, those with ADHD symptoms in childhood only, those with ADHD symptoms in adulthood only and those with ADHD symptoms both in childhood and adulthood. Finally, among our clinically relevant

sample of older adults with and without ADHD, our regression analyses supported what has been found in current literature suggesting older age is associated with higher levels of cognitive dispersion. The following sections will describe these results further and discuss their theoretical implications for cognitive aging and implications for assessing ADHD in older adult populations.

Theoretical Relevance of Study Findings

This is the first study of which we know that investigates trends in levels of dispersion in domains of cognitive performance among older adults with ADHD in the Netherlands. Our results, showing no significant difference in the levels of dispersion when comparing the ADHD group to the non-ADHD group and showing no significant relationship between the number of ADHD symptoms and levels of dispersion are surprising given current literature focusing on trends in intraindividual variability in ADHD populations. Several studies have reported significantly higher levels of intraindividual variability (as measured through RTI) in groups of people with ADHD compared to groups without (Wada et al., 2000; Barkley et al., 1996; Walker et al., 2000; Murphy et al., 2001). In a psychometric analysis of different parameters of intraindividual variability in ADHD samples, Klein and colleagues (2006) concluded that measures of intra-subject variability (ISV) such as standard deviation or consecutive variance were the strongest influencers of between-group discrimination (Klein et al., 2006). Additionally, past literature has shown that ADHD severity (number of ADHD symptoms) is correlated with levels of intraindividual variability (measured through a response time task) in a sample of 1,156 children (*M*age = 8.79 years, *SD* = 0.66; Kuntsi, 2009). There are several possible explanations for our results showing that when intraindividual variability (as measured using a dispersion index) is compared between older adults with and without ADHD (ADHD status) or across reports of ADHD symptoms, we see no significant differences. Most of the literature presenting

evidence for significant within-person differences in cognitive function in individuals with ADHD utilizes samples of children and adolescents. Our sample represents a much different age demographic which could have relevant implications for outcomes relating to trends in cognitive performance in older adulthood.

According to Paul Baltes's theory of lifespan development (1997) a lifetime of experiences is accompanied by gains and losses that influence an individual's overall development; older adults, unlike children and adolescents, have had a lifetime of development that could result in age-specific adaptations. Perhaps in the case of cognitive performance, older adults with ADHD are equipped throughout their lifespan with resources that function to attenuate the influence of an ADHD diagnosis on variability in cognitive performance. An example of the gains that may be associated with lifespan development is the use of compensatory strategies. Tomaszewski Farias and colleagues (2018) discussed compensatory strategies in older age and defined them as a set of behaviors that help the individual adapt to losses; the authors also emphasize that there is evidence to suggest that many older adults begin to utilize these strategies spontaneously in response to environmental circumstances. Tomaszewski Farias and colleagues collected data from a sample of 125 older adults. Of the 125 older adults, 68 (*M*age = 79.7) were classified as cognitively normal, 31 (*M*age = 82.4) were categorized as having mild cognitive impairment, and 23 (*M*age = 79.5) were classified as having dementia. Through the use of both neuropsychological testing and questionnaires focused on evaluating compensatory strategies in everyday life, Tomaszewski Farias and colleagues found that the use of compensatory strategies by older adult participants, such as managing finances through the use of automatic payment methods or managing medication use through a calendar system, was associated with higher levels of everyday cognitive functioning (Tomaszewski

Farias et al., 2018). Given the results suggesting the positive correlation between compensatory strategies and higher cognitive functioning, it seems plausible to suggest that the older adults with ADHD in LASA who also underwent neuropsychological testing could have developed specific compensatory strategies that helped them adapt to cognitive losses associated with having an ADHD diagnosis.

The perspectives encompassed in this project are largely medical in nature as they focus on the evaluation of a sample of individuals diagnosed with a neurodevelopmental disorder and the ways in which a dynamic index of cognitive function may serve as a behavioral marker of the disorder. However, integrating this medical perspective with a social viewpoint to investigate the presentation of ADHD in older adulthood would be a valuable addition to our field. There are several ways in which viewing ADHD as not only a medical construct but as a social one as well, could enhance our understanding of the incidence of ADHD in older populations. One way to consider ADHD from a social perspective is to consider the social supports that may function to facilitate compensatory strategies in the daily lives of people with ADHD. Perhaps by investigating ADHD through a social lens, researchers could further describe the presentation of ADHD as it manifests in relationships. Within the context of this project, it is possible that individuals with ADHD have maintained or developed specific social supports (e.g., relationships with family and/or friends) that may help to compensate for any increased levels of within-person variability that may be influenced by an ADHD diagnosis.

Another possible explanation for the results showing no significant difference between the two groups could be based on the choice to utilize an index of intraindividual variability that represents scores across multiple different cognitive domains (i.e., memory, executive function, verbal fluency, etc.). As mentioned, most studies focused on assessing variability in cognitive

performance among individuals with ADHD utilize single measures of variability such as RTI (Kofler et al., 2013). Our measure of intraindividual variability was chosen specifically because it is not limited to a single cognitive domain and could be used to represent a more widespread picture of cognitive performance by including multiple dimensions of cognitive functioning. Though dispersion has been shown to be a useful tool for predicting future cognitive impairment (Bangen, 2019; Watermeyer, 2021), perhaps in the case of individuals with a current ADHD diagnosis, a measure of variability across cognitive domains is a bit too expansive and mutes evidence of increased variability that could be identified by examining more subtle, fine-grained fluctuations at the trial-level of individual cognitive measures (i.e., RTI).

Our third research question was focused on investigating the relationship between age and levels of dispersion. As discussed, contemporary literature describing trends in cognitive functioning in older adults have shown that older age is associated with higher levels of dispersion (Hilborn et al., 2009; Hultsch et al., 2002; Schretlen, 2003). The results from this analysis support what is shown in the literature by reflecting a significant positive correlation between age and dispersion. This study expands upon current literature through the utilization of a sample of older adults with and without ADHD in the Netherlands, suggesting that the association between age and dispersion is consistent in other clinically relevant samples as well.

Improving the Characterization of ADHD Across the Adult Lifespan

The idea that dispersion as a measure of cognitive ability may not be a sensitive indicator of the presence of ADHD in older adults has important implications for characterizing ADHD across the adult lifespan. One of the primary goals of this study was to attempt to better characterize ADHD in older adulthood. The current literature regarding the presentation of ADHD is largely focused on child and adolescent populations, resulting in gaps regarding the

incidence of ADHD in younger adulthood, midlife, and older adulthood. Although this study does not directly inform the literature regarding diagnostic classification, this project does have implications for the possible future improvement of ADHD screening tools. Though dispersion does not seem to be a behavioral marker of ADHD in the older adult sample evaluated in this study, future researchers should not rule out measures of cognitive performance, and more specifically, measures of cognitive variability (e.g., response time inconsistency in speeded response-time tasks) as viable tools for detecting ADHD. Future work should continue to evaluate levels of intraindividual variability in older adult populations to gain a greater understanding and clarification of the characterization of ADHD in older individuals.

As researchers further elucidate the nature and presentation of ADHD in older adulthood, screening measures could be improved to better capture the potential for ADHD. For example, the Barkley screening measure used by LASA to screen the participants for a likelihood of ADHD contains only self-report items focusing on commonly reported symptoms of ADHD (2007). Measures such as the Barkley screening tool may benefit from the addition of indices of cognitive functioning that are sensitive to an ADHD diagnosis to complement the self-report items currently used with additional information from behavioral markers as well.

In recognizing that the dispersion profiles provide an extensive measure of variability covering several domains of cognition and that this expansive index may be overshadowing subtle effects on an individual's cognitive performance, it may be beneficial for future studies to create multiple dispersion profiles for each person, each representing a different cognitive domain. For example, results from our analysis revealed that individuals with ADHD performed significantly worse on the digit span backward test which covers domains of attention and working memory; it may be effective to generate a dispersion profile representing an individual's

variability in performance among only cognitive tasks that cover domains of working memory and attention. By limiting a dispersion index to the cognitive domains that current literature specifies as particularly susceptible to the influence of an ADHD diagnosis, perhaps researchers can more precisely and more effectively attempt to detect subtle differences in cognitive functioning between neurotypical and ADHD groups. This precision, provided by a measure of cognitive variability that is sensitive to ADHD status, could help to further characterize the presentation of ADHD across the lifespan.

Current literature emphasizes age differences in both the presentation of ADHD symptoms and in levels of cognitive dispersion. The results from our analysis support these conclusions by suggesting that older adults experience ADHD differently than younger adults and older adults experience more dispersion than younger adults. As researchers continue to study the incidence of ADHD and the role that dispersion may have in cognitive aging, it will be vital to continue to enhance our understanding of the role that age has on levels of dispersion and on ADHD symptoms. In continuing to study the influence of age on certain cognitive markers like dispersion in clinical populations with disorders that tend to be present throughout the lifespan (i.e., ADHD, Autism Spectrum Disorder), we can determine if age continues to be a significant predictor of dispersion as current literature suggests, even in clinical samples. The information provided by studies that focus on these clinical populations may provide other tools by which observational data and screening measures may improve their precision and potential detection of cognitive impairment.

Future work focused on validating behavioral markers of ADHD could be informed by the approach taken in this thesis to evaluate the relevance of age (i.e., examine age differences in dispersion metric) and consider potential confounding variables (e.g., statistically adjust for

comorbid mental health conditions like depressive symptoms) when assessing ADHD status differences in a type of variability metric (i.e., dispersion). Other potential confounding variables may be important to adjust for as well in future research. The role that alcohol use may play as a potential coping mechanism for the symptoms of ADHD or the role of a comorbid diagnosis of alcohol use disorder, for example, may be important to examine when evaluating relationships between ADHD status and dispersion (Luderer et al., 2023). More psychometric work is needed to evaluate aspects of construct validity (e.g., determining how a variability metric could accurately assess the construct of ADHD), discriminant validity (e.g., demonstrating unique signal with ADHD and no relationship with different conditions), and predictive validity (e.g., predicting likelihood of ADHD diagnosis with behavioral markers) of dynamic indices of cognitive performance as possible markers of ADHD.

Researchers using the LASA data should also keep in mind the utility of the DIVA 2.0 in exploring areas of self-reported impairments in daily living. As part of the diagnostic criteria used in the diagnosis of ADHD among LASA participants, individuals were required to present with significant impairment in at least two areas of daily living (work, education, family, social and relationships, and self-confidence) to be classified as a ‘case’. An additional way for investigators to continue to clarify our knowledge regarding the presentation of ADHD in older adulthood is to investigate what the reported domains of impairment tell us about the daily lives of people with ADHD. Qualitative research methods provide researchers with a complementary approach to studying the lived experiences of individuals with ADHD through the co-construction of rich data that could further inform the literature. With current quantitative methods we are able to evaluate important aspects of functioning; qualitative methods, however, provide detailed descriptions of experiences that offer an opportunity for a deeper understanding

of the consequences of an ADHD diagnosis. Interviewing participants and asking about the commonly mentioned areas of impairment reported in the DIVA 2.0 may be an effective way to further characterize ADHD in older adulthood through the use of qualitative methods.

Limitations and Future Directions

There are several limitations to this study that should be considered. Although LASA consists of a nationally representative dataset of Dutch older adults, as discussed earlier, the current study's subsample of adults with and without ADHD may not have enough statistical power to detect small effect sizes due to the small sample size. Future studies should prioritize recruitment efforts to increase sample sizes of people with ADHD to enhance capacity to detect potential small effects from ADHD that may be present, but not emerging in the present study's smaller, underpowered analytic sample. Knowing that there was a potential for our study to be underpowered and under the advisement of the LASA team, we added a variable representing the total number of ADHD symptoms into our plan for regression analyses. Adding this ADHD symptom component would ideally provide us with an opportunity to analyze the data using a larger sample (N = 231, versus n=208 vs. n=23). This analysis, as well as the additional analyses using a categorical grouping of ADHD symptoms, however, provided additional support for no significant difference between ADHD and dispersion in this sample.

An additional limitation is related to the diagnostic criteria for ADHD used to diagnose the older adult participants in LASA. The diagnostic interviews that were conducted during the medical interview portion of the data collection process were based on the DIVA 2.0 which itself was based on DSM 4 criteria. The fourth version of the DSM was the first to acknowledge the possible persistence of ADHD into adulthood and thus may not be an adequate tool by which to measure ADHD symptomatology in adulthood and older adulthood. Simon and colleagues

(2009), in a meta analysis investigating the prevalence and correlates of adult ADHD, reported that most of the studies evaluated communicated similar concerns that the DSM IV criteria for diagnosing ADHD was not sufficient for adult diagnosis. Not only should the DSM criteria for diagnosing ADHD be updated to reflect the presentation of the disorder throughout the lifespan, but future studies using more updated criteria may be better equipped to identify ADHD in adulthood and older adulthood.

In addition to updates to the actual criteria used to diagnose ADHD in adults and older adults, there is also a need for longitudinal data representing individuals with ADHD throughout the lifespan. The data provided by LASA was analyzed in this project cross-sectionally, and though it provided us with important information regarding trends in cognitive function in the sample, it would be valuable to evaluate trends in cognition as they occur across longer time periods. A longitudinal design focused on studying cognition in people with ADHD would not only give investigators a clearer picture of age-related influences on ADHD, but would also provide a way for researchers to better understand the role of aging in levels of cognitive dispersion. Utilizing a measurement burst design with intensive repeated measurements, as one example, where participants are evaluated every day for a week every few years would have the potential to generate a rich data set consisting of multiple indicators of cognitive function across micro (e.g., days) and macro (e.g., years) time periods. In this way, we could better understand what the daily life of these participants look like and ways in which daily processes change across more long-term developmental periods. Longitudinal studies like the one suggested could additionally be utilized as part of a coordinated analysis. Having multiple datasets to evaluate together would not only give researchers a more comprehensive picture of cognitive functioning among older adults with ADHD, it would also give investigators the opportunity to evaluate

possible cultural differences in ADHD symptoms that could be related to predictors of cognitive health outcomes. For example, the Health and Retirement Study in the United States conducted an ancillary cross-sectional study in 2016 on adult ADHD symptoms. It would be informative to assess relationships between ADHD symptoms and dispersion in the HRS to compare and contrast findings in a US sample with the Dutch sample of older adults assessed in this thesis.

Conclusion

Significant gaps in the literature regarding ADHD in adulthood function as obstacles to our progress in developing accurate diagnostic criteria and effective methods of treatment for adult populations. The obstacles resulting from this lack of information are especially evident for clinical populations of older adults. This thesis project provides the field with enhanced characterization of ADHD in older adulthood. The measurement approach in this project provides information on cognitive function through the operationalization of within-person variability in performance across cognitive domains.

The utilization of intraindividual methods for measuring cognitive health has become more prominent in psychological sciences. While most prior research has focused on fluctuations from trial-to-trial within single speed response time tasks (e.g., RTI), the present thesis attempted to fill gaps in our understanding of a more widespread marker of variability (i.e., dispersion) that captures levels of variability across multiple cognitive domains. This is achieved as variability in cognitive task performance is evaluated beyond simple measures of central tendency (e.g., mean performance) and fluctuations within a single-speeded response time task (e.g., RTI). Rather, we have attempted to elucidate the nature and relationship of cognitive variability in adults with ADHD through the utilization of a variability index comprising performance on several primary

cognitive tasks ranging in domains of processing speed, memory, attention, and executive function.

The results of this project emphasize that dispersion, as a measure of within-person variability across multiple cognitive domains, may not be a sensitive behavioral marker of ADHD status or ADHD severity in older adulthood. These results have relevant implications for future research regarding the characterization of ADHD in older adulthood and contribute to our understanding of cognitive trends in older adults with ADHD. Additionally, our results support current theoretical paradigms emphasizing a significant relationship between older age and dispersion. Future research should prioritize investigations focused on describing the prevalence and incidence of ADHD in older adulthood, as well as the characterization of cognitive performance among those with and without ADHD through the use of other intraindividual measures of cognitive variability such as RTI. Future work should also emphasize the evaluation of the role that age, or age-related change has on dispersion. Research on ADHD in older adulthood should continue to explore the utility of dynamic indices of cognitive performance (i.e., variability) that may better clarify the presentation of ADHD across the lifespan.

Appendix A

Tables

Table 1. Descriptive Statistics for Primary Study Variables

	Whole Sample			ADHD			No ADHD			Group Differences	
Variable	N	M(SD)	Range	N	M(SD)	Range	N	M(SD)	Range	t(df)	p
Dispersion	231	9.30(3.23)	3.33, 20.72	23	8.49(4.05)	3.33, 20.72	208	9.39(3.13)	3.36, 18.55	1.27(229)	0.20
ADHD Symptoms	231	10.75(7.47)	0, 35	23	24(4.84)	17, 35	208	9.28(6.15)	0, 28	-11.09(229)	<0.001
Age	231	71.64(7.70)	60.92, 94.51	23	68.02(4.87)	61.97, 79.87	208	72.04(7.85)	60.92, 94.51	2.40(229)	0.02
Female	231	0.59(0.49)	0, 1	23	0.52(0.51)	0, 1	208	0.60(0.49)	0, 1	0.73(229)	0.47
Education	231	3.90(1.96)	1, 9	23	3.52(1.90)	1, 7	208	3.94(1.97)	1, 9	0.97(229)	0.33
Depressive Symptoms	231	9.48(8.43)	0, 47	23	16.78(12.54)	0, 47	208	8.67(7.46)	0, 37	-4.57(229)	<0.001

Table 2. Descriptive Statistics for Individual Cognitive Tasks

	Whole Sample			ADHD			No ADHD			Group Differences	
Variable	N	M(SD)	Range	N	M(SD)	Range	N	M(SD)	Range	t(df)	p
Stroop Total Time	226	46.50(8.96)	30.99, 72.93	23	47.41(9.08)	34.35, 72.71	203	46.39(8.96)	30.99, 72.93	-0.52(224)	0.61
Stroop Interference	221	22.22(13.14)	3.68, 81.91	23	21.31(13.61)	9.53, 70.00	198	22.33(13.12)	3.68, 81.91	0.35(219)	0.72
Trail Making A	225	46.59(16.86)	20.45, 100.16	22	49.36(16.15)	27.74, 100.10	203	46.28(16.94)	20.45, 100.20	-0.81(223)	0.42
Trail Making B	219	121.20(66.19)	40.70, 316	21	101.00(27.35)	63.17, 150.00	198	123.30(68.73)	40.70, 316.00	1.48(217)	0.14
Animal Fluency	230	20.55(5.87)	9, 42	23	21.83(5.77)	9, 39	207	20.41(5.88)	10, 42	-1.10(228)	0.27
Letter Fluency	230	12.86(5.53)	2, 29	23	13.65(5.31)	6, 25	207	12.77(5.56)	2, 29	-0.73(228)	0.47
Digit Span Forward	231	7.74(1.69)	4, 13	23	7.43(1.20)	6, 11	208	7.78(1.73)	4, 13	0.93(229)	0.36
Digit Span Backward	231	5.23(1.77)	1, 12	23	4.39(1.59)	2, 9	208	5.32(1.77)	1, 12	2.42(229)	0.02
Mini Mental State Exam	231	27.93(1.82)	21, 30	23	27.39(2.29)	22, 30	208	27.99(1.75)	21, 30	1.49(229)	0.14
Ravens Matrices	229	19.31(3.44)	8, 24	22	19.05(2.70)	13, 23	207	19.33(3.52)	8, 24	0.37(227)	0.71
Immediate Recall	228	19.83(5.61)	9, 36	23	19.83(5.78)	12, 36	205	19.83(5.61)	9, 35	0.01(226)	0.99
Delayed Recall	229	5.83(2.77)	0, 15	23	5.52(2.78)	2, 12	206	5.86(2.78)	0, 15	0.55(227)	0.58
Alphabet Coding Task	230	27.20(6.57)	8.33, 43.33	23	25.29(6.19)	8.33, 33.33	207	27.41(6.59)	9.67, 43.33	1.47(228)	0.14
Mean Performance	231	49.98(3.29)	42.11, 60.12	23	49.09(3.31)	43.72, 58.87	208	50.08(3.28)	42.11, 60.12	1.37(229)	0.17

Table 3. Bivariate Correlations Among Primary Study Variables

Variable	1	2	3	4	5	6	7
1. ADHD	-						
2. ADHD symptoms	0.59***	-					
3. Dispersion	-0.08	-0.06	-				
4. Age	-0.16*	-0.24***	0.15*	-			
5. Education	-0.06	-0.05	-0.02	0.23**	-		
6. Female	-0.05	-0.05	0.05	0.19*	-0.24***	-	
7. Depressive symptoms	0.29***	0.32***	0.02	-0.09	-0.27***	0.24***	-

Note. *p<.05, **p<.01, ***p<.001.

Table 4. Results of t-test Comparing Levels of Dispersion Between the ADHD and No ADHD Groups

	ADHD (N=23)			No ADHD (N=208)			Group Differences		
Variable	M	SD	Range	M	SD	Range	t(229)	*p*	Cohen's d
Dispersion	8.49	4.05	3.33, 20.72	9.39	3.13	3.36, 18.55	1.27	0.20	0.25

Table 5. Linear Regression with Levels of Dispersion Regressed on ADHD Status

	Model 1: Unadjusted						Model 2: Sociodemographic and Mental Health Adjustment					
Variable	*B*	β	*SE*	*p*	95% CI		*B*	β	*SE*	*p*	95% CI	
					LL	UL					LL	UL
Intercept	9.39	-	0.22	<.001	8.95	9.83	9.29	-	0.36	<.001	8.59	10.00
ADHD	-0.90	-0.08	0.71	0.20	-2.30	0.49	-0.73	-0.07	0.76	0.34	-2.21	0.76
Age							0.06	0.14	0.03	0.045	0.001	0.11
Female							0.13	0.02	0.45	0.77	-0.76	1.03
Depressive symptoms							0.01	0.02	0.03	0.77	-0.05	0.06
R^2		0.01						0.03				

Note. B=unstandardized parameter estimate. β=standardized parameter estimate.

Table 6. Linear Regression with Levels of Dispersion Regressed on ADHD Symptoms

	Model 1: Unadjusted						Model 2: Sociodemographic and Mental Health Adjustment					
Variable	*B*	β	*SE*	*p*	95% CI		*B*	β	*SE*	*p*	95% CI	
					LL	UL					LL	UL
Intercept	9.57	-	0.37	<.001	8.84	10.31	9.32	-	0.50	<.001	8.34	10.31
ADHD symptoms	-0.03	-0.06	0.03	0.38	-0.08	0.03	-0.01	-0.03	0.03	0.73	-0.07	0.05
Age							0.06	0.14	0.03	0.043	0.002	0.12
Female							0.16	0.02	0.45	0.72	-0.73	1.05
Depressive symptoms							0.003	0.008	0.03	0.91	-0.05	0.06
R^2		0.01						0.02				

Note. B=unstandardized parameter estimate. β=standardized parameter estimate.

Table 7. Linear Regression with Levels of Dispersion Regressed on Age

	Model 1: Unadjusted						Model 2: ADHD, Sociodemographic, and Mental Health Adjustment					
Variable	*B*	β	*SE*	*p*	95% CI		*B*	β	*SE*	*p*	95% CI	
					LL	UL					LL	UL
Intercept	9.30	-	0.21	<.001	8.89	9.72	9.29	-	0.36	<.001	8.59	10.00
Age	0.06	0.15	0.03	0.02	0.01	0.12	0.06	0.14	0.03	0.045	0.001	0.11
ADHD							-0.73	-0.07	0.80	0.34	-2.21	0.76
Female							0.13	0.02	0.46	0.73	-0.80	1.03
Depressive symptoms							0.008	0.02	0.03	0.80	-0.05	0.06
R^2		0.02						0.03				

Note. B=unstandardized parameter estimate. β=standardized parameter estimate.

Appendix B

Figures

Figure 1. Study Flow Chart

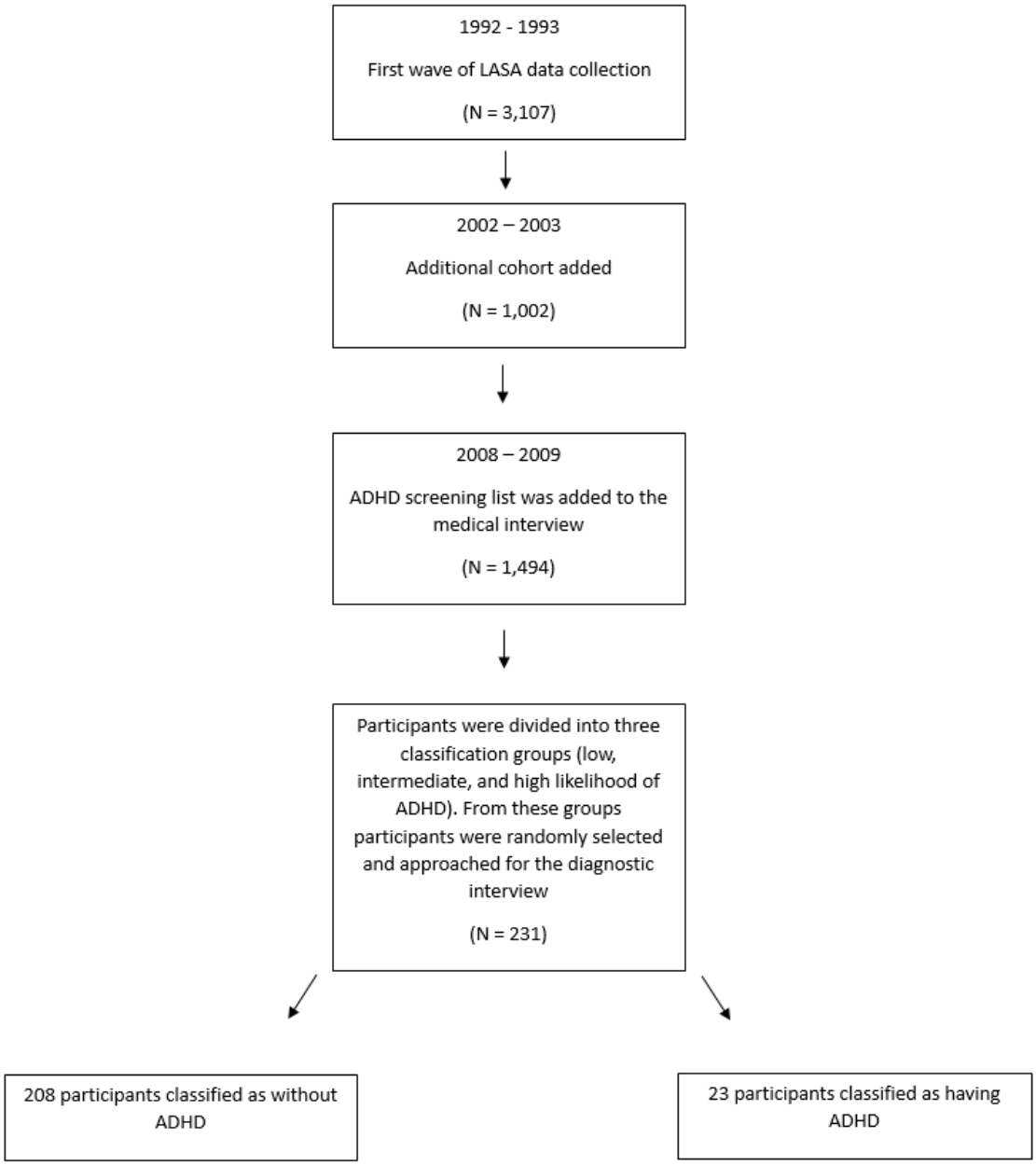
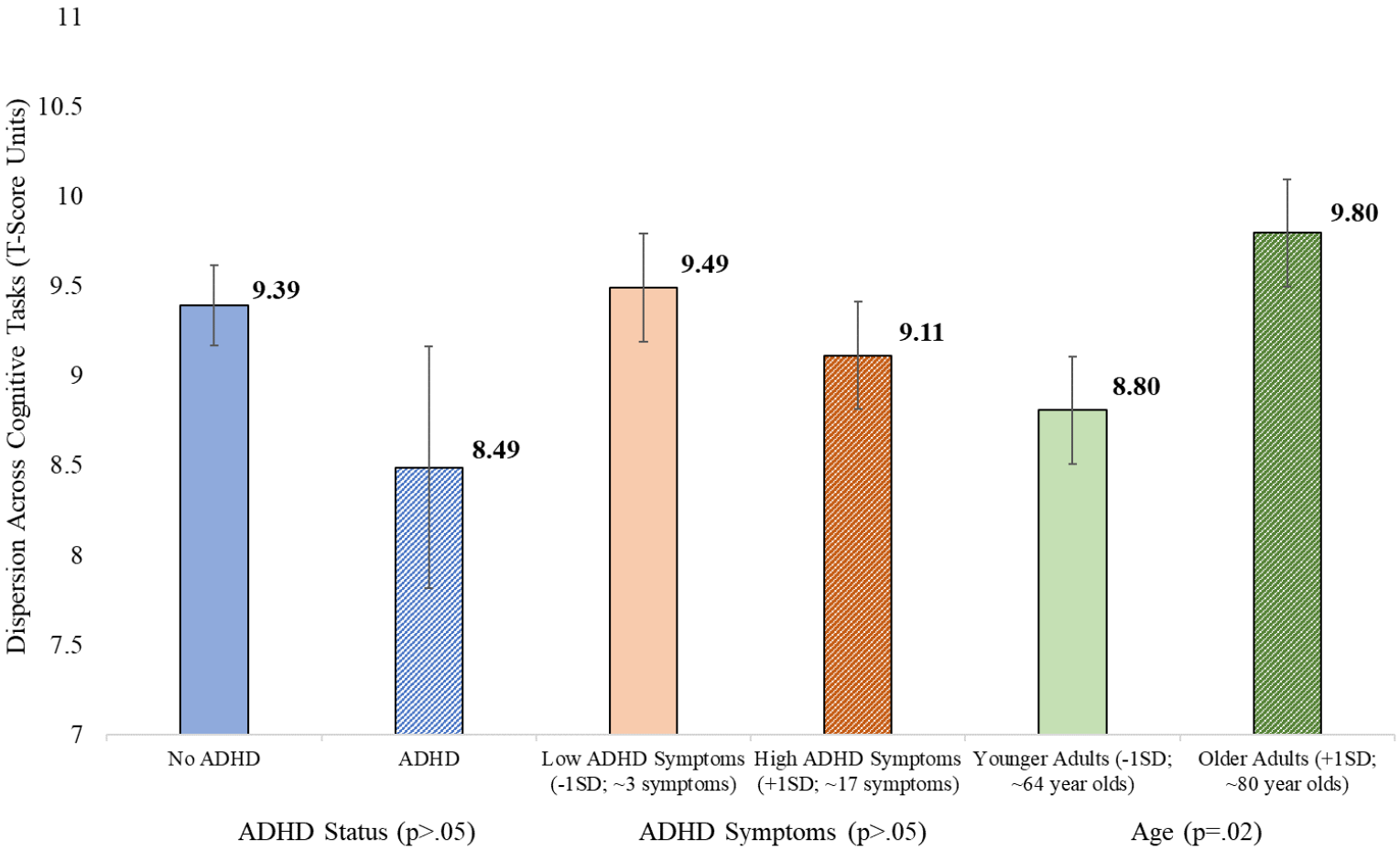


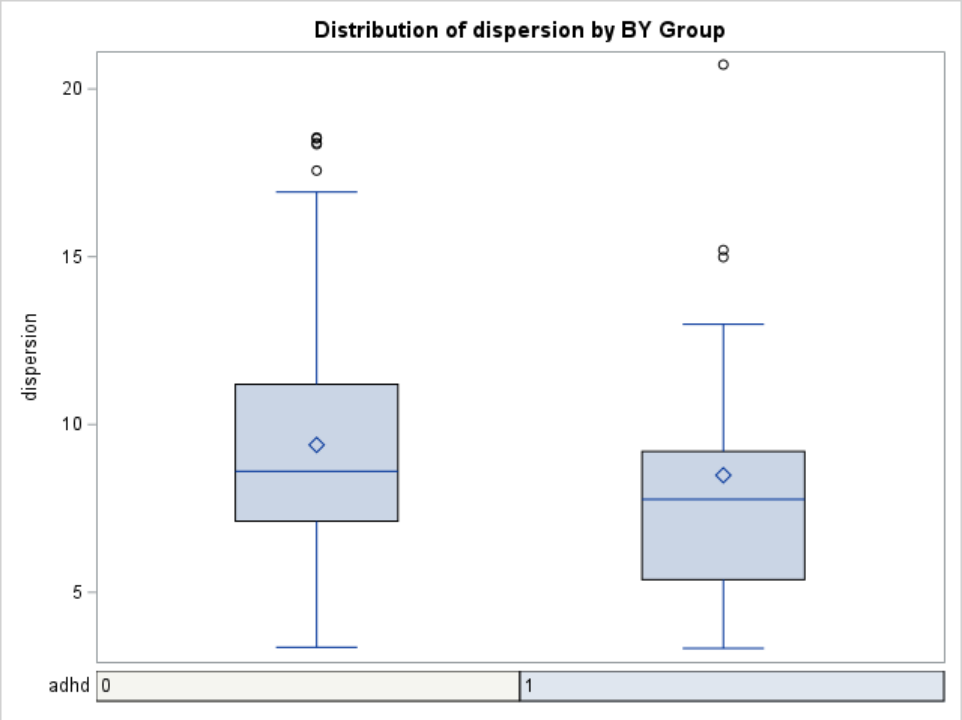
Figure 2.



Estimates of Dispersion at Different Levels of ADHD Status, ADHD Symptoms, and Age

Note. Estimates (with standard error bars) computed from regressions of dispersion on ADHD status (b=-0.90, SE=0.71, p>.05), ADHD total symptoms (b=-0.03, SE=0.03, p>.05), and age (b=0.06, SE=0.05, p=.02).

Figure 3. Research Question 1 Boxplot Showing Distribution of Dispersion for Participants without ADHD (ADHD = 0 below in leftmost boxplot) and Participants with ADHD (ADHD = 1 below in rightmost boxplot)



Note. The center horizontal line in each box corresponds to the sample median. The diamond symbol corresponds to the sample mean. Each box encompasses the interquartile range, with the bottom portion of each box specifying the 25th percentile (lower quartile) and top portion of the box specifying the 75th percentile (upper quartile). The whiskers indicate the most extreme points in the group that lie within the upper and lower fences of the plot. The upper fence is defined as the third quartile (upper edge of box) plus 1.5 times the interquartile range. The lower fence is defined as the first quartile (lower edge of box) minus 1.5 times the interquartile range. Observations outside the fences are specified as empty circle shapes.

Figure 4. Research Questions 2 and 3 Scatter Plots of Differences in Dispersion as a Function of ADHD Symptoms and Age

Figure 4A. ADHD Symptom Differences in Dispersion (Research Question 2)

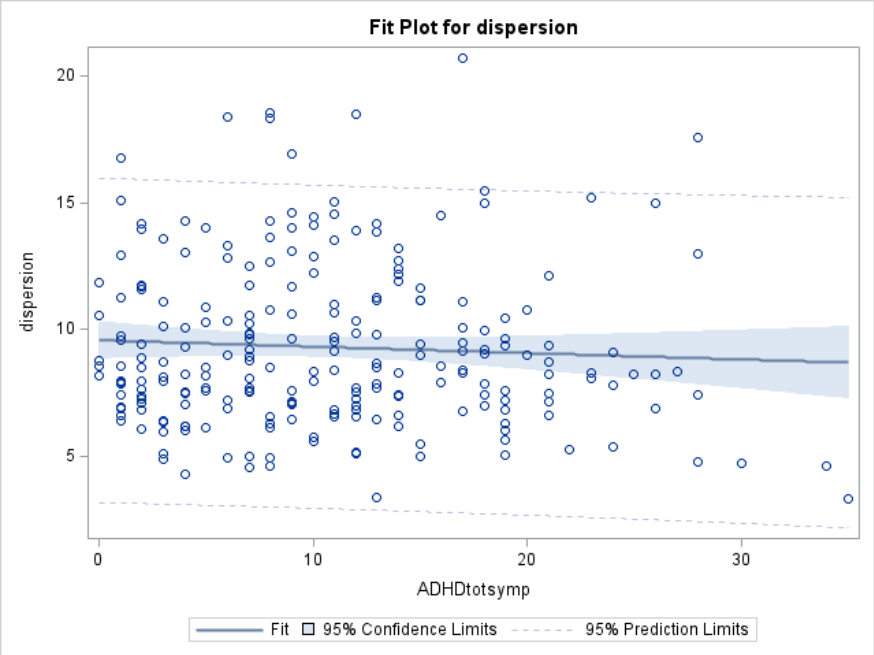


Figure 4B. Age Differences in Dispersion (Research Question 3)

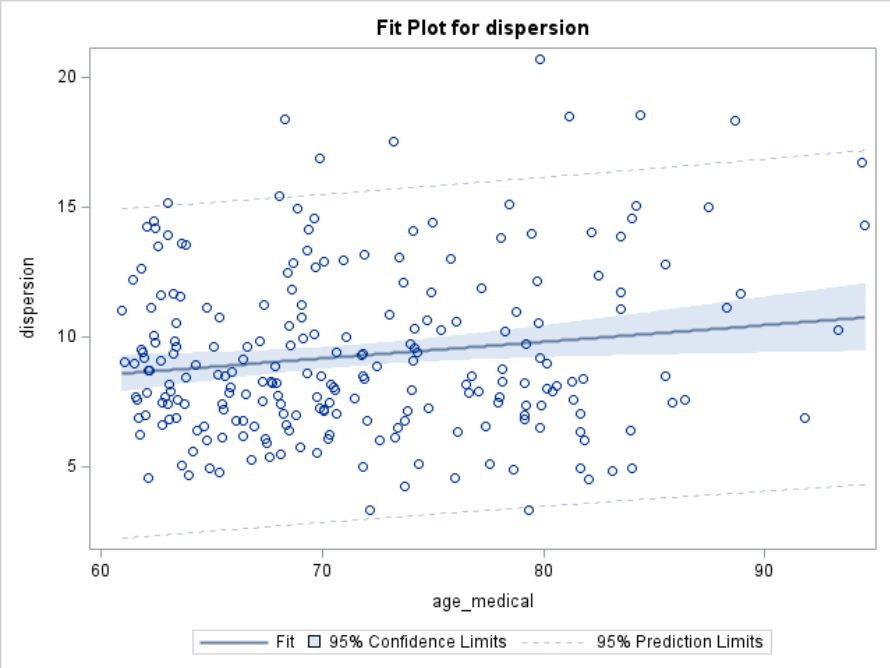


Figure 5. Boxplots Showing Distribution of Study Variables for Participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot).

Figure 5 Note. The center horizontal line in each box corresponds to the sample median. The diamond symbol corresponds to the sample mean. Each box encompasses the interquartile range, with the bottom portion of each box specifying the 25th percentile (lower quartile) and top portion of the box specifying the 75th percentile (upper quartile). The whiskers indicate the most extreme points in the group that lie within the upper and lower fences of the plot. The upper fence is defined as the third quartile (upper edge of box) plus 1.5 times the interquartile range. The lower fence is defined as the first quartile (lower edge of box) minus 1.5 times the interquartile range. Observations outside the fences are specified as empty circle shapes.

Figure 5A. ADHD Status Differences in ADHD Symptoms

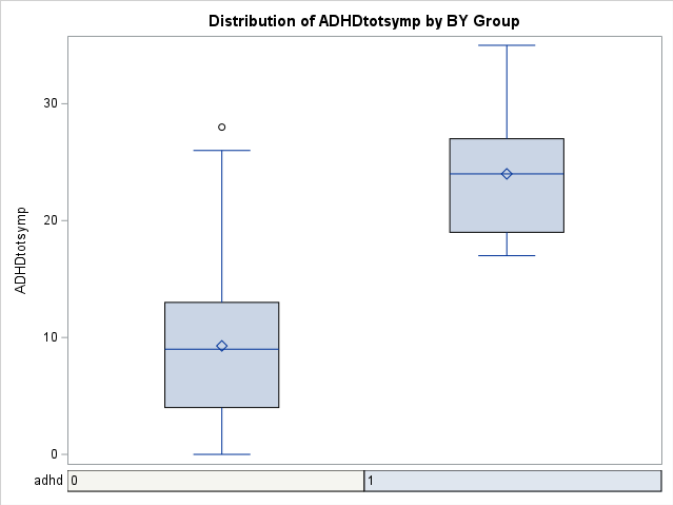


Figure 5B. ADHD Status Differences in Age

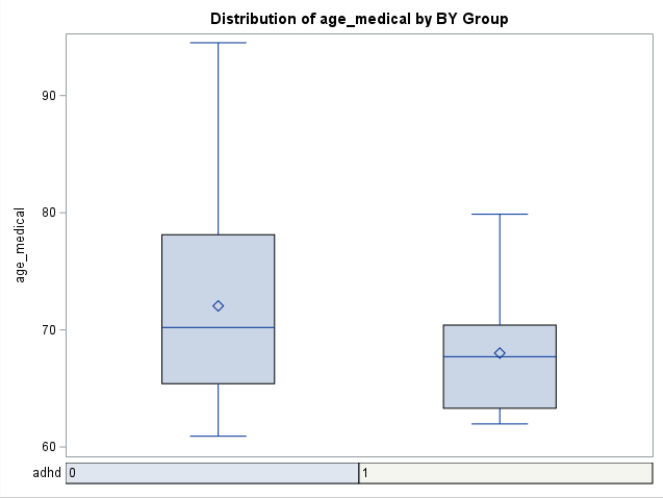


Figure 5C. ADHD Status Differences in Educational Attainment

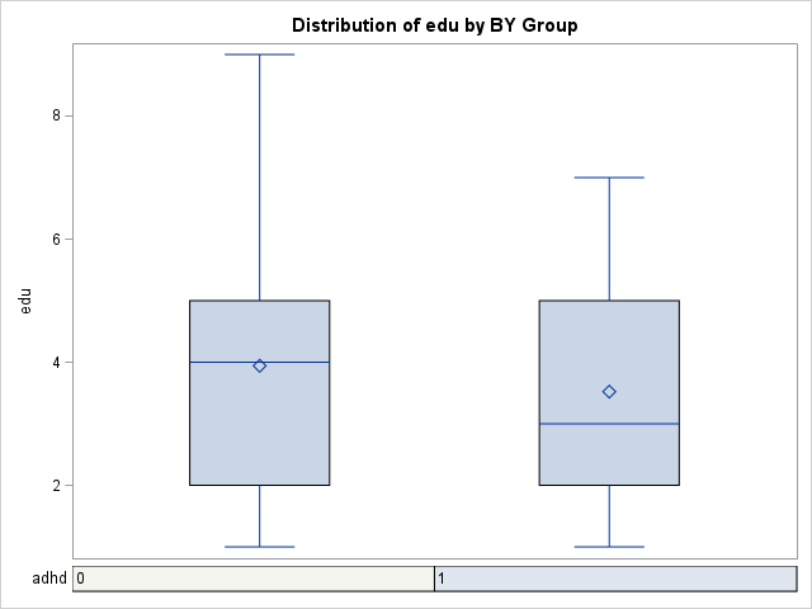


Figure 5D. ADHD Status Differences in Depressive Symptoms

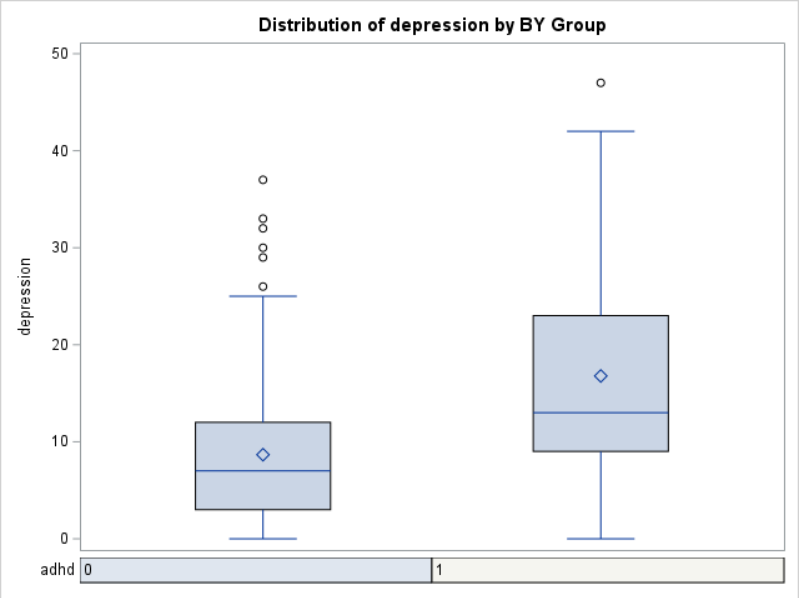


Figure 6. Boxplots Showing Distribution of Individual Cognitive Tasks and Mean Performance for Participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

Figure 6 Note. The center horizontal line in each box corresponds to the sample median. The diamond symbol corresponds to the sample mean. Each box encompasses the interquartile range, with the bottom portion of each box specifying the 25^{th} percentile (lower quartile) and top portion of the box specifying the 75^{th} percentile (upper quartile). The whiskers indicate the most extreme points in the group that lie within the upper and lower fences of the plot. The upper fence is defined as the third quartile (upper edge of box) plus 1.5 times the interquartile range. The lower fence is defined as the first quartile (lower edge of box) minus 1.5 times the interquartile range. Observations outside the fences are specified as empty circle shapes.

Figure 6A. ADHD status differences in Stroop Speed (before removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

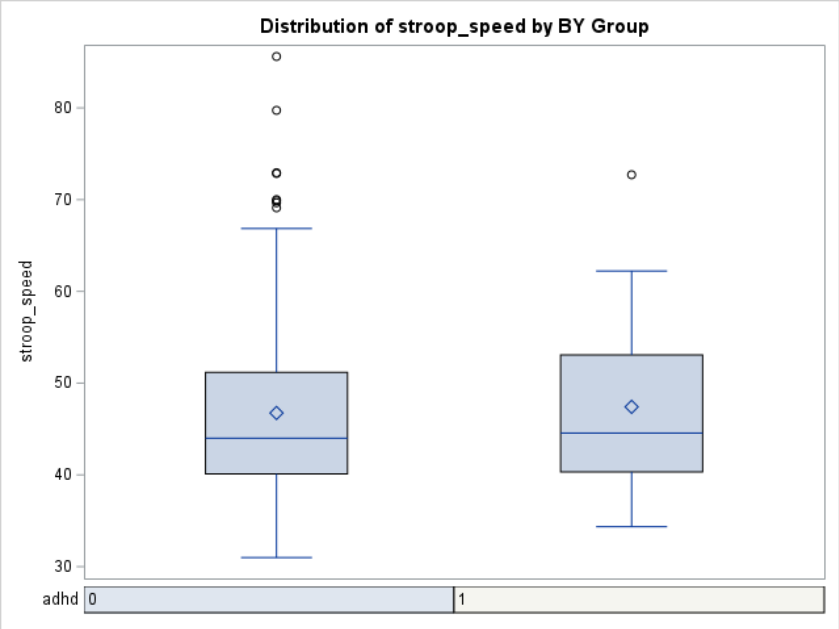


Figure 6B. ADHD status differences in Stroop Speed (after removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

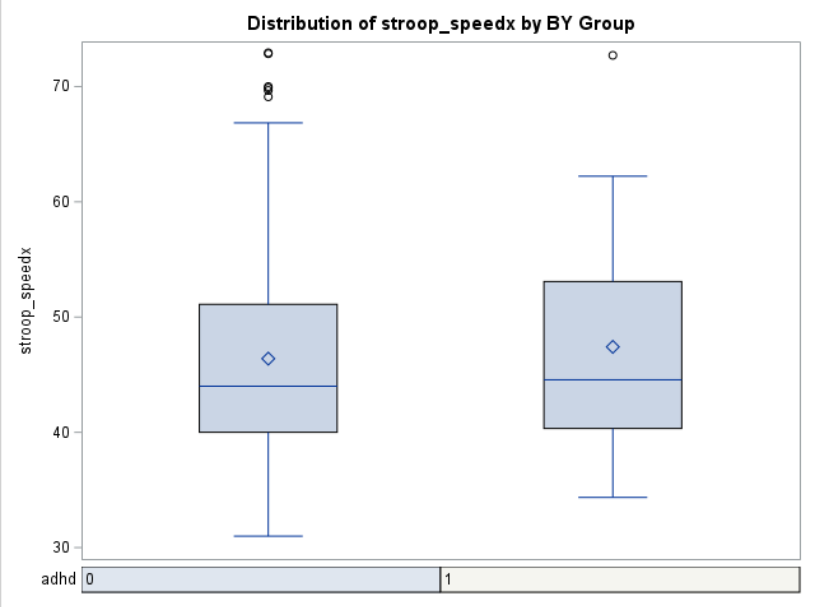


Figure 6C. ADHD status differences in Stroop Interference (before removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

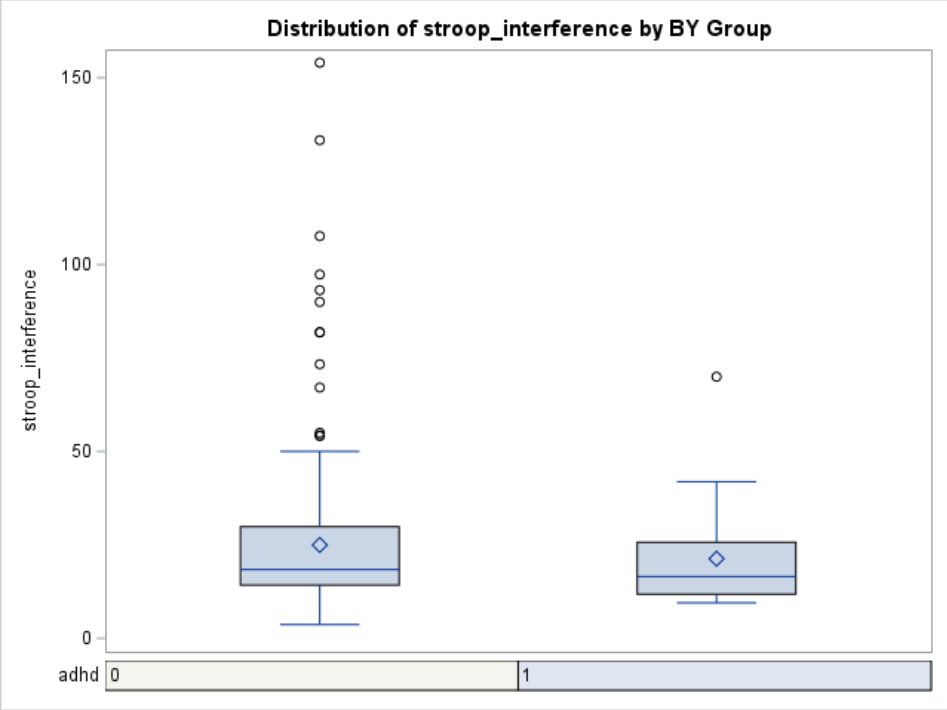


Figure 6D. ADHD status differences in Stroop Interference (after removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

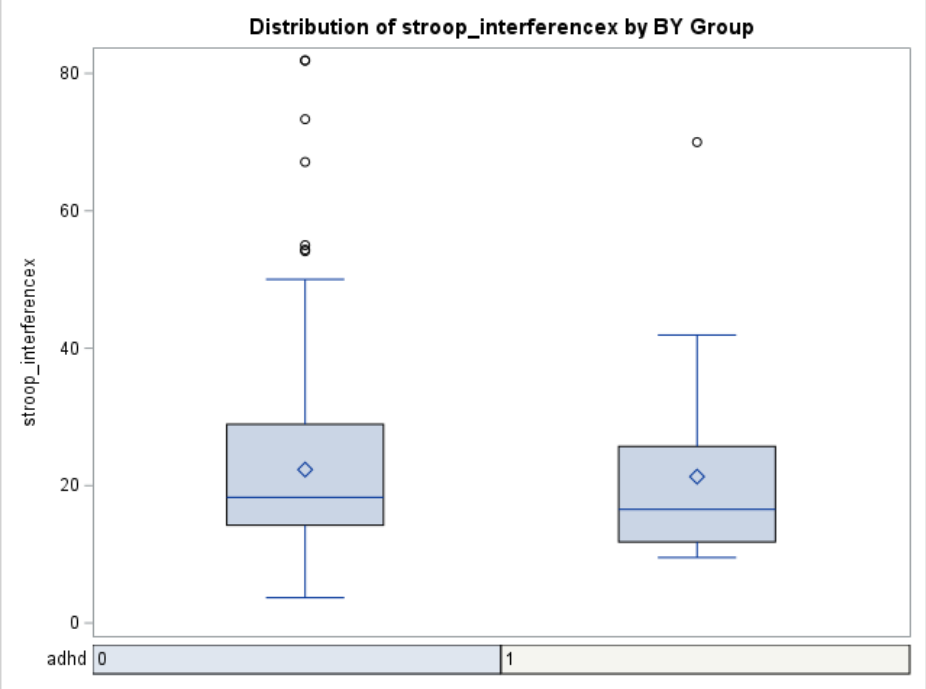


Figure 6E. ADHD status differences in Trail Making Task A (before removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

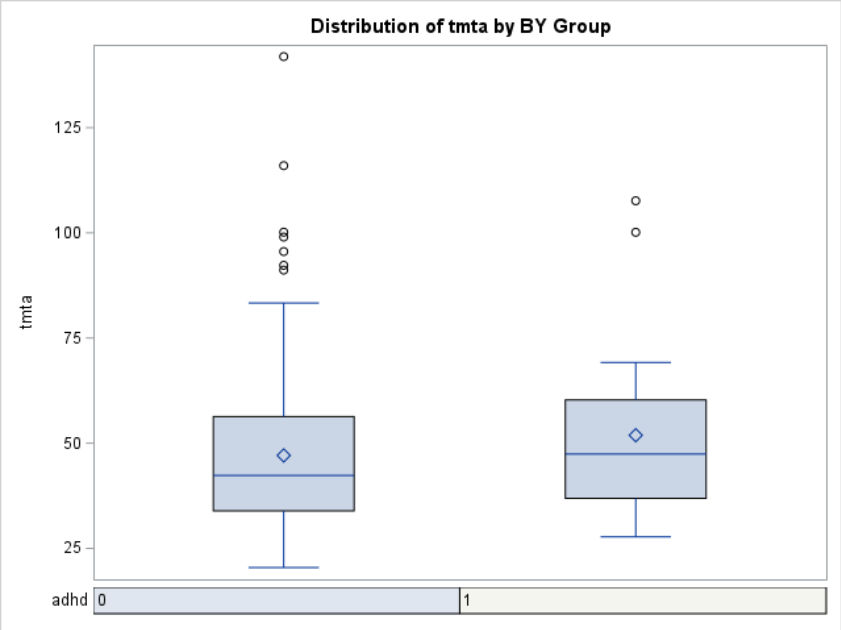


Figure 6F. ADHD status differences in Trail Making Task A (after removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

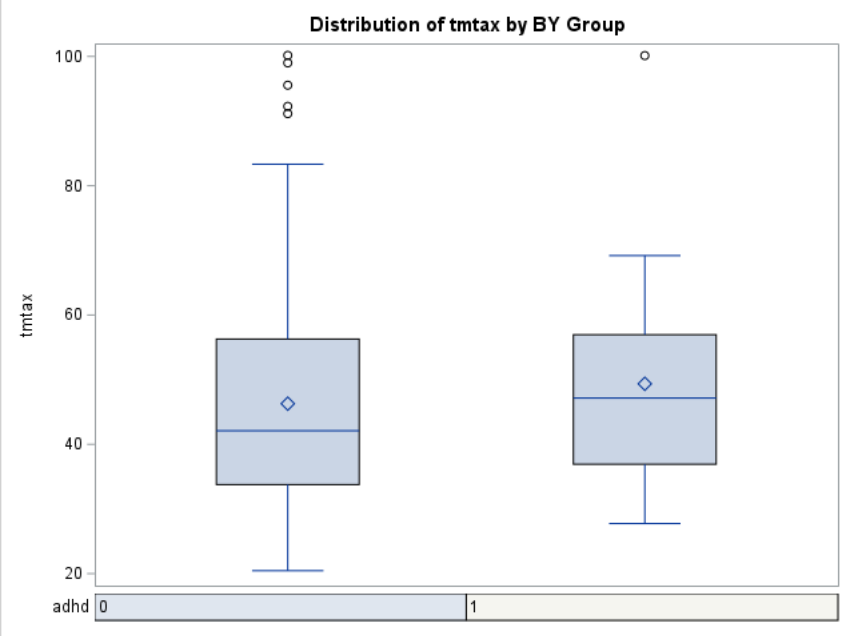


Figure 6G. ADHD status differences in Trail Making Task B (before removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

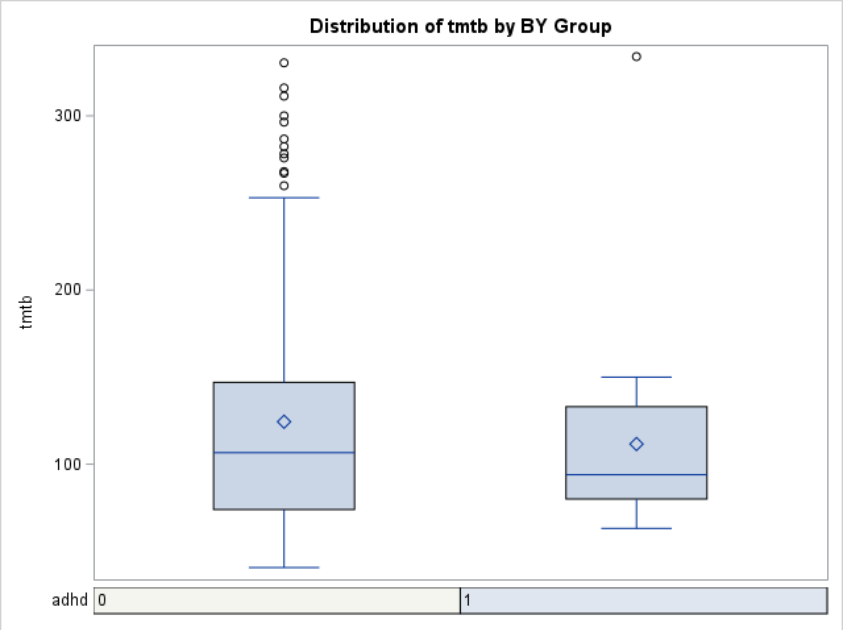


Figure 6H. ADHD status differences in Trail Making Task B (after removing outliers) for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

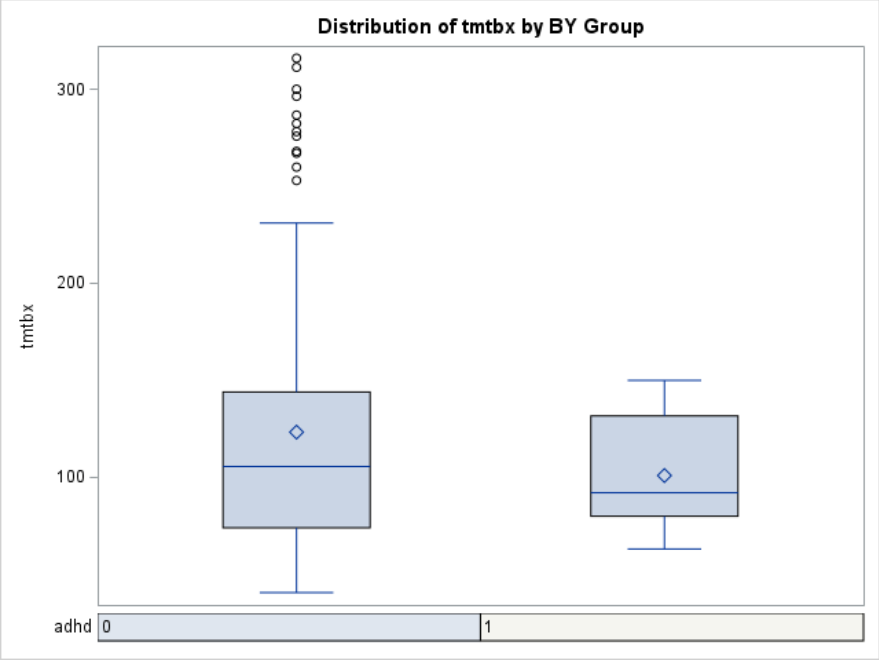


Figure 6I. ADHD status differences in Verbal Fluency Animal Naming for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

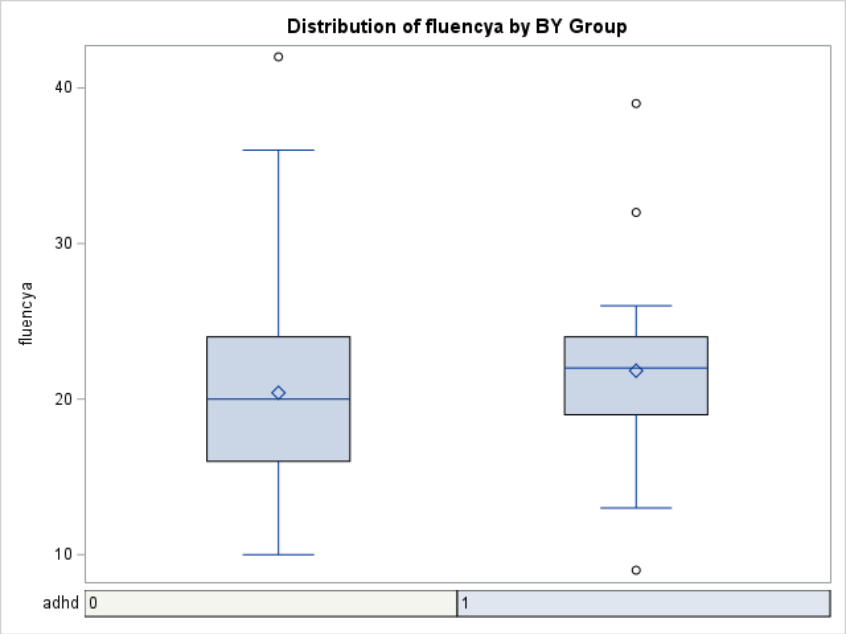


Figure 6J. ADHD status differences in Verbal Fluency Letter Naming for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

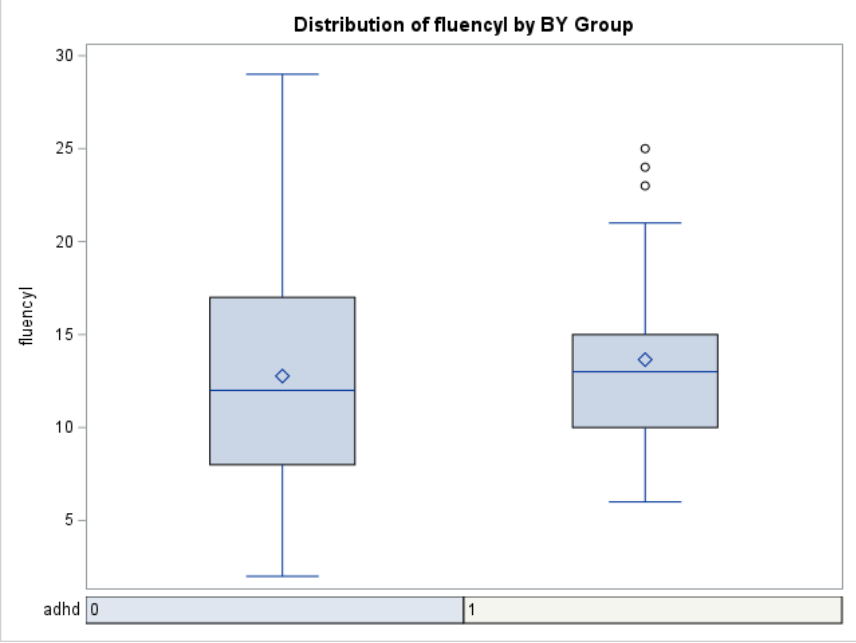


Figure 6K. ADHD status differences in Digit Span Forward for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

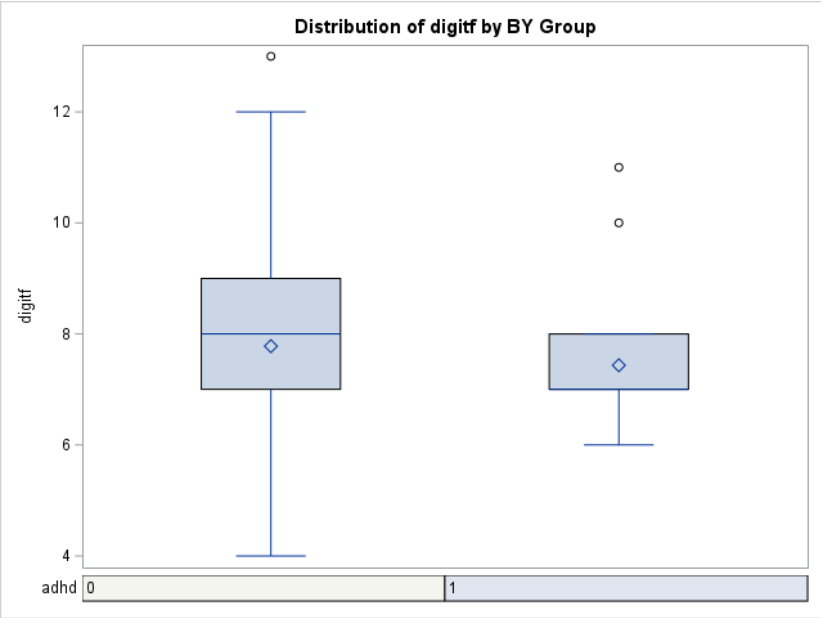


Figure 6L. ADHD status differences in Digit Span Backward for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

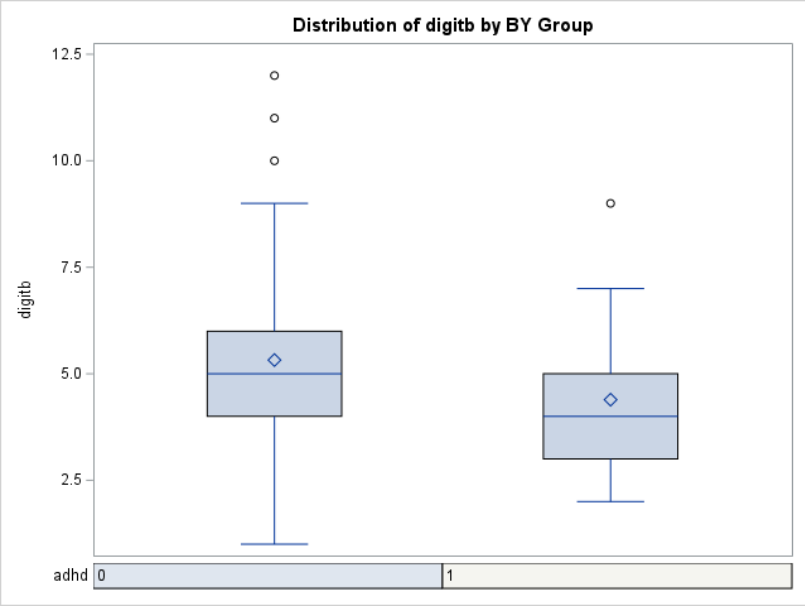


Figure 6L. ADHD status differences in Mini Mental State Examination for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

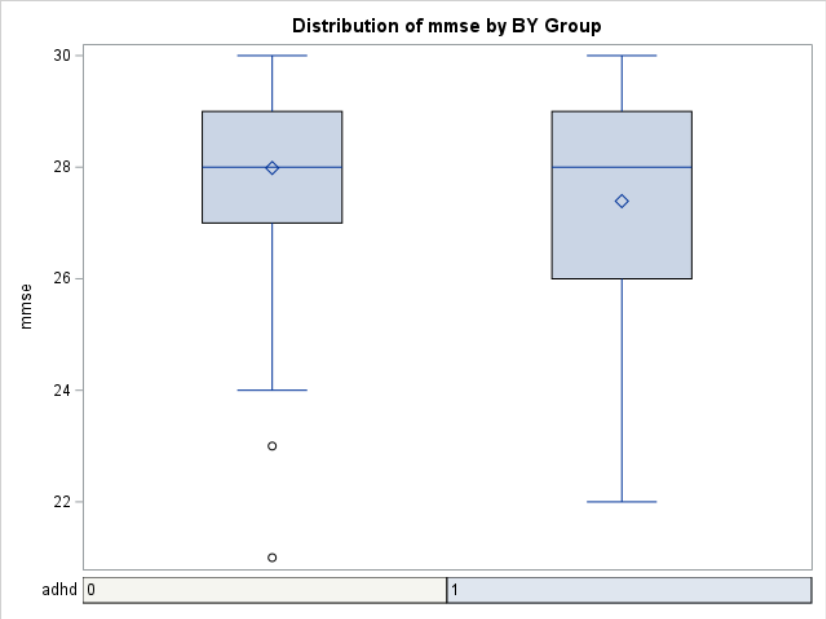


Figure 6M. ADHD status differences in Raven's Progressive Matrices for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

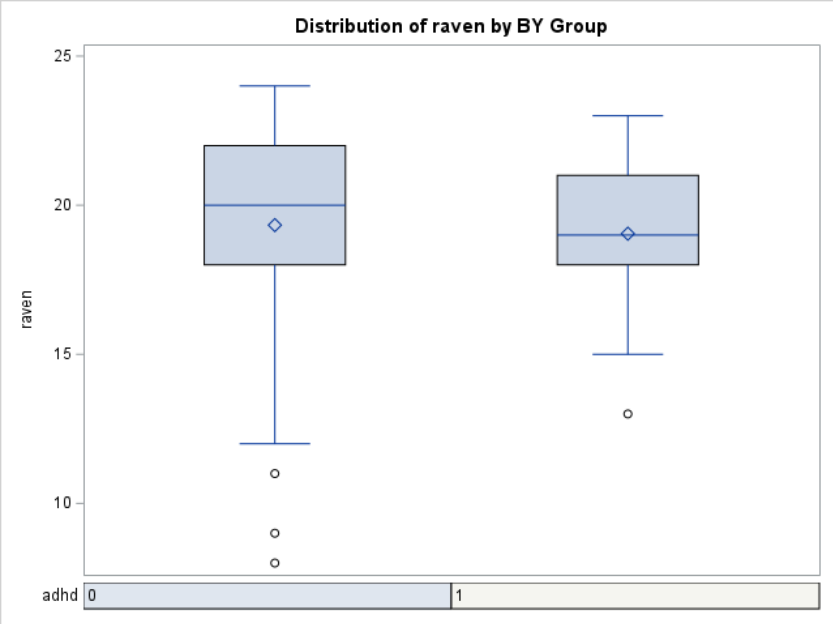


Figure 6N. ADHD status differences in Immediate Recall for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

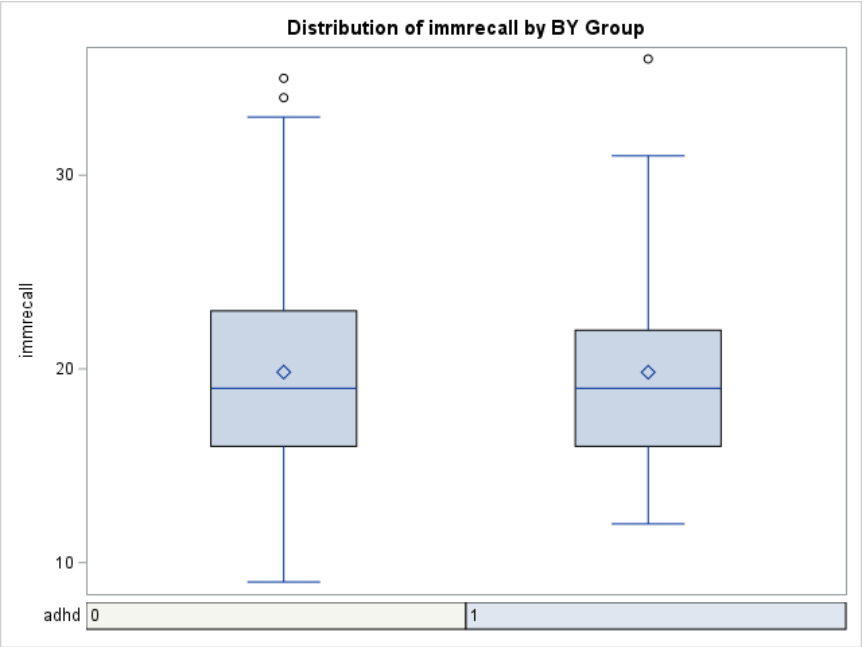


Figure 6O. ADHD status differences in Delayed Recall for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

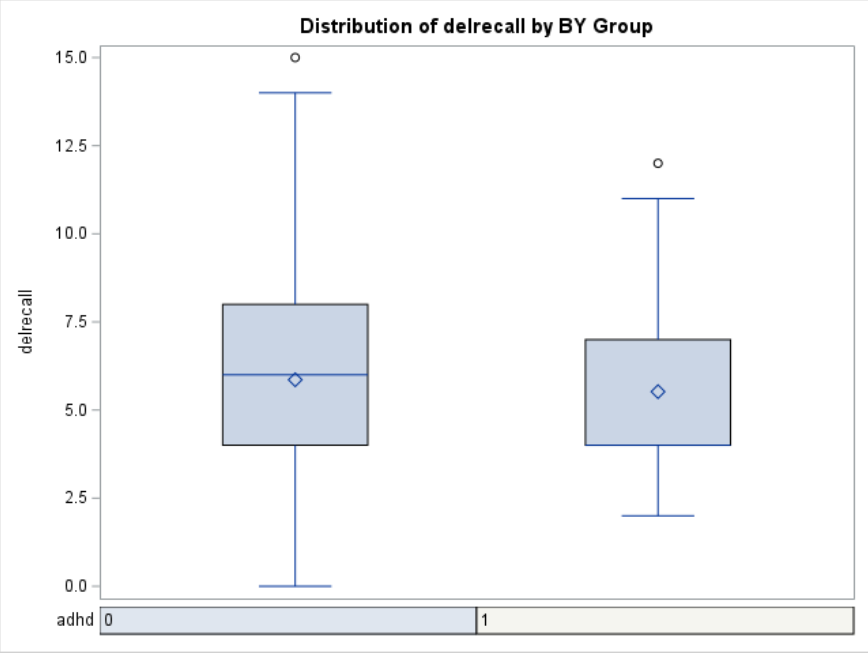


Figure 6P. ADHD status differences in Alphabet Coding Task for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)

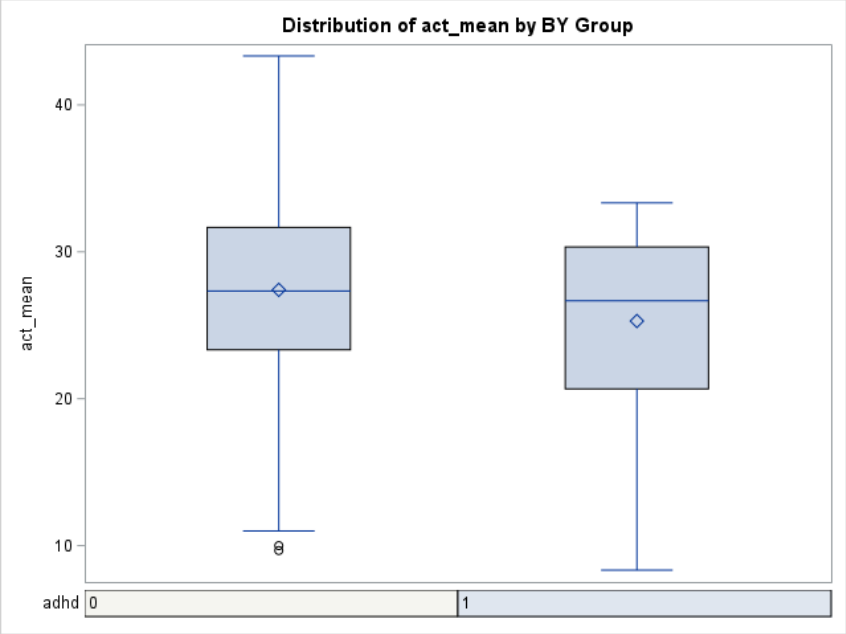
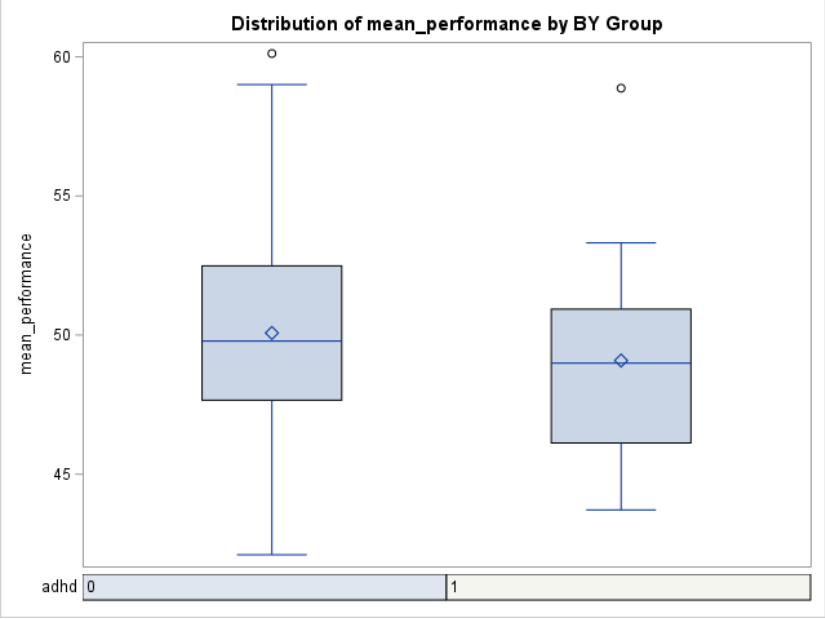


Figure 6Q. ADHD status differences in Mean Performance for participants without ADHD (ADHD = 0 below in leftmost boxplot) and participants with ADHD (ADHD = 1 below in rightmost boxplot)



Appendix C

Diagnostic Interview for ADHD in Adults 2.0

Part 1: Symptoms of attention-deficit (DSM-IV criterion A1)

Instructions: the symptoms in adulthood have to have been present for at least 6 months. The symptoms in childhood relate to the age of 5-12 years. For a symptom to be ascribed to ADHD it should have a chronic trait-like course and should not be episodic.

A1: Do you often fail to give close attention to detail, or do you make careless mistakes in your work or during other activities? And how was that during childhood?

A2: Do you often find it difficult to sustain your attention on tasks? And how was that during childhood?

A3: Does it often seem as though you are not listening when you are spoken to directly? And how was that during childhood?

A4: Do you often fail to follow through on instructions and do you often fail to finish jobs or fail to meet obligations at work? And how was that during childhood (when doing schoolwork as opposed to when at work)?

A5: Do you often find it difficult to organise tasks and activities? And how was that during childhood?

A6: Do you often avoid (or do you have an aversion to, or are you unwilling to do) tasks which require sustained mental effort? And how was that during childhood?

A7: Do you often lose things that are needed for tasks or activities? And how was that during childhood?

A8: Are you often easily distracted by external stimuli? And how was that during childhood?

A9: Are you often forgetful during daily activities? And how was that during childhood?

Part 2: Symptoms of hyperactivity-impulsivity (DSM-IV criterion A2)

Instructions: the symptoms in adulthood have to have been present for at least 6 months. The symptoms in childhood relate to the age of 5-12 years. For a symptom to be ascribed to ADHD it should have a chronic trait-like course and should not be episodic.

H/I 1: Do you often move your hands or feet in a restless manner, or do you often fidget in your chair? And how was that during childhood?

H/I 2: Do you often stand up in situations where the expectation is that you should remain in your seat? And how was that during childhood?

H/I 3: Do you often feel restless? And how was that during childhood?

H/I 4: Do you often find it difficult to engage in leisure activities quietly? And how was that during childhood?

H/I 5: Are you often on the go or do you often act as if "driven by a motor"? And how was that during childhood?

H/I 6: Do you often talk excessively? And how was that during childhood?

H/I 7: Do you often give the answer before questions have been completed? And how was that during childhood?

H/I 8: Do you often find it difficult to await your turn? And how was that during childhood?

H/I 9: Do you often interrupt the activities of others, or intrude on others? And how was that during childhood?

Part 3: Impairment on account of the symptoms (DSM-IV criteria B, C and D)

Criterion B

Q1: Have you always had these symptoms of attention deficit and/or hyperactivity/impulsivity?

Criterion C

Q1: In which areas do you have / have you had problems with these symptoms?

Adulthood

Work/education

- ❑ Did not complete education/training needed for work
- ❑ Work below level of education
- ❑ Tire quickly of a workplace
- ❑ Pattern of many short-lasting jobs
- ❑ Difficulty with administrative work/planning
- ❑ Not achieving promotions
- ❑ Under-performing at work
- ❑ Left work following arguments or dismissal
- ❑ Sickness benefits/disability benefit as a result of symptoms
- ❑ Limited impairment through compensation of high IQ
- ❑ Limited impairment through compensation of external structure
- ❑ Other

Relationship and/or family

- ❑ Tire quickly of relationships
- ❑ Impulsively commencing/ending relationships
- ❑ Unequal partner relationship owing to symptoms
- ❑ Relationship problems, lots of arguments, lack of intimacy
- ❑ Divorced owing to symptoms
- ❑ Problems with sexuality as a result of symptoms
- ❑ Problems with upbringing as a result of symptoms
- ❑ Difficulty with housekeeping and/or administration
- ❑ Financial problems or gambling
- ❑ Not daring to start a relationship
- ❑ Other:

Childhood and adolescence

Education

- ❑ Lower educational level than expected based on IQ
- ❑ Staying back (repeating classes) as a result of concentration problems
- ❑ Education not completed / rejected from school
- ❑ Took much longer to complete education than usual
- ❑ Achieved education suited to IQ with a lot of effort
- ❑ Difficulty doing homework
- ❑ Followed special education on account of symptoms
- ❑ Comments from teachers about behaviour or concentration
- ❑ Limited impairment through compensation of high IQ
- ❑ Limited impairment through compensation of external structure
- ❑ Other:

Family

- ❑ Frequent arguments with brothers or sisters
- ❑ Frequent punishment or hiding
- ❑ Little contact with family on account of conflicts
- ❑ Required structure from parents for a longer period than would normally be the case
- ❑ Other:

Adulthood *(continuance)*

Social contacts

- ❑ Tire quickly of social contacts
- ❑ Difficulty maintaining social contacts
- ❑ Conflicts as a result of communication problems
- ❑ Difficulty initiating social contacts
- ❑ Low self-assertiveness as a result of negative experiences
- ❑ Not being attentive (i.e. forget to send a card/ empathising/phoning, etc)
- ❑ Other:

Free time / hobby

- ❑ Unable to relax properly during free time
- ❑ Having to play lots of sports in order to relax
- ❑ Injuries as a result of excessive sport
- ❑ Unable to finish a book or watch a film all the way through
- ❑ Being continually busy and therefore becoming overtired
- ❑ Tire quickly of hobbies
- ❑ Accidents/loss of driving licence as a result of reckless driving behaviour
- ❑ Sensation seeking and/or taking too many risks
- ❑ Contact with the police/the courts
- ❑ Binge eating
- ❑ Other:

Self-confidence / self-image

- ❑ Uncertainty through negative comments of others
- ❑ Negative self-image due to experiences of failure
- ❑ Fear of failure in terms of starting new things
- ❑ Excessive intense reaction to criticism
- ❑ Perfectionism
- ❑ Distressed by the symptoms of ADHD
- ❑ Other:

Childhood and adolescence *(continuance)*

Social contacts

- ❑ Difficulty maintaining social contacts
- ❑ Conflicts as a result of communication problems
- ❑ Difficulty entering into social contacts
- ❑ Low self-assertiveness as a result of negative experiences
- ❑ Few friends
- ❑ Being teased
- ❑ Shut out by, or not being allowed, to do things with a group
- ❑ Being a bully
- ❑ Other:

Free time/hobby

- ❑ Unable to relax properly during free time
- ❑ Having to play lots of sport to be able to relax
- ❑ Injuries as a result of excessive sport
- ❑ Unable to finish a book or watch a film all the way through
- ❑ Being continually busy and therefore becoming overtired
- ❑ Tired quickly of hobbies
- ❑ Sensation seeking and/or taking too many risks
- ❑ Contact with the police/courts
- ❑ Increased number of accidents
- ❑ Other:

Self-confidence / self-image

- ❑ Uncertainty through negative comments of others
- ❑ Negative self-image due to experiences of failure
- ❑ Fear of failure in terms of starting new things
- ❑ Excessive intense reaction to criticism
- ❑ Perfectionism
- ❑ Other:

Summary of symptoms A and H/I

Indicate which criteria were scored in parts 1 and 2 and add up

Criterion DSM-IV TR	Symptom	Present during adulthood	Present during child-hood
A1a	**A1.** Often fails to pay close attention to details, or makes careless mistakes in schoolwork, work or during other activities		
A1b	**A2.** Often has difficulty sustaining attention in tasks or play		
A1c	**A3.** Often does not seem to listen when spoken to directly		
A1d	**A4.** Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace		
A1e	**A5.** Often has difficulty organizing tasks and activities		
A1f	**A6.** Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as school of homework)		
A1g	**A7.** Often loses things necessary for tasks or activities		
A1h	**A8.** Often easily distracted by extraneous stimuli		
A1i	**A9.** Often forgetful in daily activities		
	Total number of criteria Attention Deficit	☐ / 9	☐ / 9
A2a	**H/I 1.** Often fidgets with hands or feet or squirms in seat		
A2b	**H/I 2.** Often leaves seat in classroom or in other situations in which remaining seated is expected		
A2c	**H/I 3.** Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults this may be limited to subjective feelings of restlessness)		
A2d	**H/I 4.** Often has difficulty playing or engaging in leisure activities quietly		
A2e	**H/I 5.** Is often on the go or often acts as if 'driven by a motor'		
A2f	**H/I 6.** Often talks excessively		
A2g	**H/I 7.** Often blurts out answers before questions have been completed		
A2h	**H/I 8.** Often has difficulty awaiting turn		
A2i	**H/I 9.** Often interrupts or intrudes on others		
	Total number of criteria Hyperactivity/Impulsivity	☐ / 9	☐ / 9

Score form

DSM-IV criterion A	**Childhood** Is the number of A characteristics ≥ 6? Is the number of H/I characteristics ≥ 6? **Adulthood*** Is the number of A characteristics ≥ 6? Is the number of H/I characteristics ≥ 6?	 ❑ Yes / ❑ No ❑ Yes / ❑ No ❑ Yes / ❑ No ❑ Yes / ❑ No
DSM-IV criterion B	Are there signs of a lifelong pattern of symptoms and limitations?	❑ Yes / ❑ No
DSM-IV criterion C and D	The symptoms and the impairment are expressed in at least two domains of functioning Adulthood Childhood	 ❑ Yes / ❑ No ❑ Yes / ❑ No
DSM-IV criterion E	The symptoms cannot be (better) explained by the presence of another psychiatric disorder	❑ No Yes, by
	Is the diagnosis supported by collateral information? Parent(s)/brother/sister/other, i.e. ** Partner/good friend/other, i.e. ** School reports 0 = none/little support 1 = some support 2 = clear support	 ❑ N/A ❑ 0 ❑ 1 ❑ 2 ❑ N/A ❑ 0 ❑ 1 ❑ 2 ❑ N/A ❑ 0 ❑ 1 ❑ 2 Explanation:
	Diagnosis ADHD***	❑ No Yes, subtype ❑ 314.01 Combined type ❑ 314.00 Predominantly inattentive type ❑ 314.01 Predominantly hyperactive-impulsive type

Appendix D

SAS Syntax for Data Cleaning and Creation of Dispersion Index

```
*Cleaning data, creating dispersion index, and saving analytic dataset;

libname lasalib "Z:\"; *SAS 9.4;
options nofmterr;

data lasa;
set lasalib.lasadata;
run;

proc sort data=lasa;
by respnr;
run;

proc contents data=lasa;
run;

*Checking for outliers;

proc univariate data=lasa;
var stroop_speed;
histogram;
run;

proc print data=lasa;
where stroop_speed > 75.43;
var respnr adhd stroop_speed;
run;

proc univariate data=lasa;
var stroop_interference;
histogram;
run;

proc print data=lasa;
where stroop_interference > 84.13;
var respnr adhd stroop_interference;
run;

proc univariate data=lasa;
var tmta;
histogram;
run;

proc print data=lasa;
where tmta > 104.26;
var respnr adhd tmta;
run;

proc univariate data=lasa;
where adhd=1;
```

```
var tmtb;
histogram;
run;

proc print data=lasa;
where tmtb > 329.69;
var respnr adhd tmtb;
run;

proc univariate data=lasa;
var fluencya;
histogram;
run;

proc print data=lasa;
where fluencya > 38.16;
var respnr adhd fluencya;
run;

proc univariate data=lasa;
var fluencyl;
histogram;
run;

proc univariate data=lasa;
var digitf;
histogram;
run;

proc univariate data=lasa;
var digitb;
histogram;
run;

proc print data=lasa;
where digitb > 10.54;
var respnr adhd digitb;
run;

proc univariate data=lasa;
var mmse;
histogram;
run;

proc print data=lasa;
where mmse < 22.47;
var respnr adhd mmse;
run;

proc univariate data=lasa;
var raven;
histogram;
run;

proc print data=lasa;
where raven < 8.99;
var respnr adhd raven;
```

```
run;

proc univariate data=lasa;
var immrecall;
histogram;
run;

proc univariate data=lasa;
var delrecall;
histogram;
run;

proc print data=lasa;
where delrecall > 14.14;
var respnr adhd delrecall;
run;

proc univariate data=lasa;
var act_mean;
histogram;
run;


*removing outliers;

data lasax;
set lasa;
stroop_speedx = .;
stroop_interferencex = .;
tmtax = .;
tmtabx = .;
run;

proc sort data=lasax;
by respnr;
run;

proc contents data=lasax;
run;

proc print data=lasax(obs=50);
run;

data lasaxx;
set lasax;
if stroop_speed < 75.43 then stroop_speedx = stroop_speed;
if stroop_interference < 84.13 then stroop_interferencex =
stroop_interference;
if tmta < 104.26 then tmtax = tmta;
if tmtb < 329.69 then tmtbx = tmtb;
run;

proc sort data=lasaxx;
by respnr;
run;

proc print data=lasaxx;
```

```
var respnr stroop_speed stroop_speedx;
run;

*Now that cognitive tasks are cleaned we can create dispersion index;

*Put all 13 cogntiive tests on the same t-score metric;

proc standard data=lasaxx mean=50 std=10 out=tscoreraw;
var stroop_speedx stroop_interferencex tmtax tmtbx fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean;
run;

proc print data=tscoreraw;
run;

*Convert wide to long data to be able to create ISD;

data tscoreslongraw;
set tscoreraw;
cogtest=1; score=stroop_speedx; output;
cogtest=2; score=stroop_interferencex; output;
cogtest=3; score=tmtax; output;
cogtest=4; score=tmtbx; output;
cogtest=5; score=fluencya; output;
cogtest=6; score=fluencyl; output;
cogtest=7; score=digitf; output;
cogtest=8; score=digitb; output;
cogtest=9; score=mmse; output;
cogtest=10; score=raven; output;
cogtest=11; score=immrecall; output;
cogtest=12; score=delrecall; output;
cogtest=13; score=act_mean; output;
run;

proc print data=tscoreslongraw;
run;

*Create and Output Intraindividual Standard Deviation(ISD) that serves as our
dispersion index;

proc means data=tscoreslongraw  nway noprint;
by respnr;
var score;
output out=dispersiondataraw (drop=_type_ _freq_)
std(score)= dispersion mean(score)=mean_performance;
run;

proc sort data=dispersiondataraw;
by respnr;
run;

proc print data=dispersiondataraw;
run;

proc means data=dispersiondataraw;
run;
```

```
proc corr data=dispersiondataraw;
run;

*merging data;

data lasa2;
merge lasaxx dispersiondataraw;
by respnr;
run;

proc sort data=lasa2;
by respnr;
run;

proc print data=lasa2;
run;

*Saving permanent analytic dataset with dispersion index;

data lasalib.lasadataanalysis;
set lasa2;

run;
```

Appendix E

SAS Syntax evaluating Research Questions

```
libname lasalib "Z:\"; *SAS 9.4;
options nofmterr;

data lasa;
set lasalib.lasadataanalysis_15Apr2023;
run;

proc sort data=lasa;
by respnr;
run;

proc contents data=lasa;
run;

*Table 1;
*Descriptive statistics for primary study variables;

proc means data=lasa;
var dispersion adhdtotsymp age_medical female depression;
run;

proc ttest data=lasa;
class adhd;
var dispersion adhdtotsymp age_medical female depression;
run;

*Table 2;
*Descriptive Statistics for cognitive tasks including mean performance;

proc means data=lasa;
var stroop_speedx stroop_interferencex tmtax tmtbx fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean mean_performance;
run;

proc ttest data=lasa;
class adhd;
var stroop_speedx stroop_interferencex tmtax tmtbx fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean mean_performance;
run;

*Table 3;
*Bivariate correlation analysis;

proc corr data=lasa;
var adhd adhdtotsymp dispersion age_medical female depression;
run;

*Table 4;
*Independent samples t-test comparing ADHD status;

proc ttest data=lasa;
class adhd;
```

```
var dispersion;
run;


*Table 5: Linear Regression with Levels of Dispersion Regressed on ADHD
Status;

*Model 1: Unadjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = adhd/effectsize clparm;
run;

*Model 1: Unadjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion = adhd/stb;
run;

*Model 2: Adjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = adhd agem_medical female depression_gmc/effectsize clparm;
run;

*Model 2: Adjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion =  adhd agem_medical female depression_gmc/stb;
run;

*Table 6: Linear Regression with Levels of Dispersion Regressed on ADHD
symptoms;

*Model 1: Unadjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = adhdtotsymp/effectsize clparm;
run;

*Model 1: Unadjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion = adhdtotsymp/stb;
run;

*Model 2: Adjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = adhdtotsymp agem_medical female depression_gmc/effectsize
clparm;
run;


*Model 2: Adjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion =  adhdtotsymp agem_medical female depression_gmc/stb;
run;

*Table 7: Linear Regression with Levels of Dispersion Regressed on age;

*Model 1: Unadjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = agem_medical/effectsize clparm;
```

```
run;

*Model 1: Unadjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion = agem_medical/effectsize clparm;
run;

*Model 2: Adjusted PROC GLM to provide us with effect sizes;
proc glm data=lasa;
model dispersion = agem_medical adhd female depression_gmc/effectsize clparm;
run;

*Model 2: Adjusted PROC REG to provide us with standardized beta estimates;
proc reg data=lasa;
model dispersion = agem_medical adhd female depression_gmc/stb;
run;

*Supplementary analyses using categorical ADHD symptom variable;
proc anova data=lasa;
class adhdsympcat;
model dispersion= adhdsympcat;
means adhdsympcat/ tukey;
run;

proc glm data=lasa;
class adhdsympcat;
model dispersion= adhdsympcat/effectsize;
means adhdsympcat/ tukey;
run;


/* Templates for Calculated Cohen's D Effect Size:
https://www.spss-tutorials.com/cohens-d/
https://www.socscistatistics.com/effectsize/default3.aspx
*/


/*************************************************************************
**********************/
/*Obtaining Cronbach's Alpha Estimate of Internal Consistency Reliability for
CES-D Depression Scale*/
/*************************************************************************
**********************/
proc contents data=lasa;
run;

proc corr data=lasa nomiss alpha;
var gcesd01 gcesd02 gcesd03 gcesd04r gcesd05 gcesd06 gcesd07 gcesd08r gcesd09
gcesd10 gcesd11 gcesd12r gcesd13 gcesd14 gcesd15 gcesd16r gcesd17 gcesd18
gcesd19 gcesd20;
run;


*Figure 2;
/*************************************************************************
*********************************/
```

```
/*Writing Estimate Commands to Get Estimates of Dispersion at Different
Levels of Thesis Main Effects Variables*/
/*Useful for When You Are Interested in Plotting Differences in Values of
Outcome Based on Predictor Variables*/
/*************************************************************************
*********************************/

/*Estimate Commands for ADHD Status*/
proc glm data=lasa;
model dispersion = adhd/effectsize clparm;;
estimate 'Dispersion in People without ADHD' intercept 1 adhd 0;
estimate 'Dispersion in People with ADHD' intercept 1 adhd 1;
run;

/*Estimate Commands for Age*/
proc glm data=lasa;
model dispersion = agem_medical/effectsize clparm;
estimate 'Dispersion Among Younger Participants (-1SD; ~64 year olds)'
intercept 1 agem_medical - 7.6971757;
estimate 'Dispersion Among Average Age Participants (Mean; 72 year olds)'
intercept 1 agem_medical 1;
estimate 'Dispersion Among Older Participants (+1SD; ~80 year olds)'
intercept 1 agem_medical 7.6971757;
run;

/*Estimate Commands for ADHD Symptoms*/
proc glm data=lasa;
model dispersion = adhdtotsymp_gmc/effectsize clparm;
estimate 'Dispersion in People with Lower Number of ADHD Symptoms (-1SD; ~3
symptoms)' intercept 1 adhdtotsymp_gmc - 7.4686118;
estimate 'Dispersion in People with Average Number of ADHD Symptoms (Mean;
~10 symptoms)' intercept 1 adhdtotsymp_gmc 1;
estimate 'Dispersion in People with Higher Number of ADHD Symptoms (+1SD; ~17
symptoms)' intercept 1 adhdtotsymp_gmc 7.4686118;
run;


*Scatter Plots for Research Questions;

*Used proc glm to obtain the regression line overlaying data points in
scatter plot;
proc glm data=lasa;
model dispersion = adhd;
run;

proc corr data=lasa plots=matrix(histogram);
var dispersion adhd;
run;

proc glm data=lasa;
model dispersion = adhdtotsymp;
run;
proc corr data=lasa plots=matrix(histogram);
var dispersion adhdtotsymp;
run;

proc glm data=lasa;
```

```
model dispersion = age_medical;
run;
proc corr data=lasa plots=matrix(histogram);
var dispersion age_medical;
run;

*Boxplots by ADHD group;
proc sort data=lasa;
by adhd;
run;

proc univariate data=lasa plot;
by adhd;
var age_medical adhdtotsymp female depression dispersion mean_performance
stroop_speed stroop_speedx stroop_interference stroop_interferencex tmta
tmtax tmtb tmtbx
fluencya fluencyl digitf digitb mmse raven immrecall delrecall act_mean;
run;

*Sensitivity Analyses Using All Available Data;
*13 variables to use for dispersion index;
*stroop_speed stroop_interference tmta tmtb fluencya fluencyl digitf digitb
mmse raven immrecall delrecall act_mean;
libname lasalib "Z:\"; *SAS 9.4;
options nofmterr;

data lasa;
set lasalib.lasadataanalysis_15Apr2023;
run;

proc sort data=lasa;
by respnr;
run;

*Put all cognitive tests on the same t-score metric;
proc standard data=lasa mean=50 std=10 out=tscoreraw;
var stroop_speed stroop_interference tmta tmtb fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean;
run;

proc means data=tscoreraw;
var stroop_speed stroop_interference tmta tmtb fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean;
run;

proc means data=tscoreraw;
where adhd=0;
var stroop_speed stroop_interference tmta tmtb fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean;
run;

proc means data=tscoreraw;
where adhd=1;
var stroop_speed stroop_interference tmta tmtb fluencya fluencyl digitf
digitb mmse raven immrecall delrecall act_mean;
run;
```

```
*Convert wide to long data to be able to create ISD;
data tscoreslongraw;
set tscoreraw;
cogtest=1; score=stroop_speed; output;
cogtest=2; score=stroop_interference; output;
cogtest=3; score=tmta; output;
cogtest=4; score=tmtb; output;
cogtest=5; score=fluencya; output;
cogtest=6; score=fluencyl; output;
cogtest=7; score=digitf; output;
cogtest=8; score=digitb; output;
cogtest=9; score=mmse; output;
cogtest=10; score=raven; output;
cogtest=11; score=immrecall; output;
cogtest=12; score=delrecall; output;
cogtest=13; score=act_mean; output;
run;

proc print data=tscoreslongraw (obs=50);
run;

*Create and Output ISD;
proc means data=tscoreslongraw  nway noprint;
by respnr;
var score;
output out=dispersiondataraw (drop=_type_ _freq_)
std(score)= dispersion_alldata mean(score)=mean_performance_alldata;
run;

proc print data=dispersiondataraw;
run;
proc means data=dispersiondataraw;
run;
proc corr data=dispersiondataraw;
run;

proc sort data=dispersiondataraw;
by respnr;
run;

data lasa2;
merge lasa dispersiondataraw;
run;

proc sort data=lasa2;
by respnr;
run;

proc print data=lasa2;
var respnr dispersion_alldata mean_performance_alldata dispersion
mean_performance;
run;


*Sensitivity Analysis for Table 4;
proc ttest data=lasa2;
class adhd;
```

```
var dispersion_alldata;
run;


*Sensitivity Analysis for Table 5;
proc glm data=lasa2;
model dispersion_alldata = adhd/effectsize clparm;
run;

proc glm data=lasa2;
model dispersion_alldata = adhd agem_medical female depression_gmc/effectsize
clparm;
run;

proc reg data=lasa2;
model dispersion_alldata = adhd/stb;
run;

proc reg data=lasa2;
model dispersion_alldata = adhd agem_medical female depression_gmc/stb;
run;

*Sensitivity Analysis for Table 6;
proc glm data=lasa2;
model dispersion_alldata = adhdtotsymp/effectsize clparm;
run;

proc glm data=lasa2;
model dispersion_alldata = adhdtotsymp agem_medical female
depression_gmc/effectsize clparm;
run;

proc reg data=lasa2;
model dispersion_alldata = adhdtotsymp/stb;
run;

proc reg data=lasa2;
model dispersion_alldata =  adhdtotsymp agem_medical female
depression_gmc/stb;
run;

*Sensitivity Analysis for Table 7;
proc glm data=lasa2;
model dispersion_alldata = agem_medical/effectsize clparm;
run;

proc glm data=lasa2;
model dispersion_alldata = agem_medical adhd female depression_gmc/effectsize
clparm;
run;

proc reg data=lasa2;
model dispersion_alldata = agem_medical/effectsize clparm;
run;

proc reg data=lasa2;
model dispersion_alldata = agem_medical adhd female depression_gmc/stb;
```

```
run;

*Sensitivity Analysis for Supplementary analyses using categorical ADHD
symptom variable;
proc anova data=lasa2;
class adhdsympcat;
model dispersion_alldata= adhdsympcat;
means adhdsympcat/ tukey;
run;

proc glm data=lasa2;
class adhdsympcat;
model dispersion_alldata= adhdsympcat/effectsize;
means adhdsympcat/ tukey;

run;
```

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