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Abstract

Early-Adult Executive Functions in Girls with and without Childhood ADHD

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Executive functions (EFs) are neuropsychological constructs that help individuals perform complex, future-oriented actions that are necessary for successful performance of activities in academic and employment settings and in social interactions. Research on children with attention deficit hyperactivity disorder (ADHD) demonstrates that deficits in EFs are linked with a variety of functional impairments. However, there is limited research on the EF trajectories of girls with ADHD as they develop—and their associated impairments. Thus, my aim for this dissertation was to examine trajectories of EF deficits from childhood to emerging adulthood (i.e., ages 23-29) in an all-female ADHD sample, along with the impact these deficits may have on externalizing and internalizing behaviors and academic achievement by this period of emerging adulthood. For this investigation I utilized a sample of 140 girls diagnosed with ADHD and 88 comparison girls matched for age and ethnicity (Wave 1; mean age = 9.5 years). 209 (92%) participated in five-year follow-up assessments (Wave 2; mean age = 14.1); 216 (95%) participated in 10-year follow-up assessments (Wave 3; mean age = 19.6); and 211 (93%) participated in the 16-year follow-up assessments (Wave 4; mean age = 25.6). EF measurements assessing response inhibition, working memory, visual discrimination, and global EF were administered at all four waves. At Wave 4 the young women self-reported on their internalizing, externalizing, and depressive symptoms; parental reports on internalizing and externalizing symptoms and objective measures of reading and math were also utilized.

With the exception of Wave 1 response inhibition, the young women with ADHD performed worse on all EF measures at all waves, when compared to the matched subsample of women without ADHD histories. They also had poorer internalizing, externalizing, and depressive symptoms as well as poorer academic achievement. Growth curve modeling indicated that, even though all women experienced absolute increases in EF performance across waves, the women with histories of ADHD persistently lagged behind comparison women at all measurement points. In addition, young women with histories of ADHD had steeper rates of improvement than their comparison peers on the global executive functioning task, yet the opposite pattern was observed for the visual discrimination/inhibitory response task, on which comparison peers demonstrated steeper improvements. Importantly, the trajectory of working memory performance predicted self-reported externalizing behavior by emerging adulthood. In addition, improvements in the young women's performance in both working memory and visual discrimination/inhibition were associated with adult reading and math achievement. Moreover, higher levels of Wave 4 working memory were associated with higher concurrent levels of reading achievement in the ADHD than the comparison sample. Overall, these findings contribute to greater understanding of executive functioning in young women with ADHD and suggest that EF deficits should be considered when developing and implementing treatments for ADHD through emerging adulthood. Future research should be aimed at understanding the mechanisms behind the associations observed.

Early-adult executive functions in girls with and without childhood ADHD

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by persistent and developmentally extreme symptoms of inattention and/or hyperactivity and impulsivity, which can lead to major social, family, and academic impairments (American Psychiatric Association, 2013). Although evidence reveals that children with ADHD experience a decline in symptoms as they grow older (e.g., Kessler et al., 2006), researchers have also found that these children continue to experience impairments across multiple domains well into adolescence and adulthood, even when symptoms remit. In particular, when compared to their counterparts without childhood manifestations of ADHD, they experience lower educational attainment, have poorer work performance, and display more social problems (Barkley, 2015; Hinshaw & Scheffler, 2014). Furthermore, they have a greater risk of developing other psychiatric disorders—including antisocial, anxiety, mood, and substance use disorders—by early adulthood (Biederman et al., 2006; Charach, Yeung, Troy, & Lillie, 2011; Meinzer et al., 2013). Neuropsychological deficits—particularly executive functioning deficits—are thought to play a central role with regard to ADHD symptoms and related functional impairments (e.g. Aman, Roberts, & Pennington, 1998; Barkley, 1997).

There is a great need to understand the developmental course of ADHD in girls and women, because of the lack of relevant research on this population. In particular, although the neuropsychological functioning and life impairments of ADHD in predominantly or exclusively male samples are well documented, prospective longitudinal research on both females and neuropsychological functioning beyond adolescence lags far behind. Thus, the purpose of this dissertation is to examine the extent to which girls with ADHD continue to experience executive functioning deficits by emerging adulthood (i.e., ages 23-29), compared to their peers without childhood ADHD. I also investigate the impact these deficits may have on adult levels of externalizing and internalizing behaviors and academic achievement.

Executive Functions (EFs)

Although there has been some debate about what exactly constitutes executive functions (EFs), they are broadly defined as self-regulatory processes that incorporate a number of higher-order cognitive abilities, such as planning, inhibition, organization, set shifting, working memory, and problem solving (Seidman, 2006; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Pennington & Ozonoff, 1996). EFs help individuals attain future-oriented goals by allowing them to complete context-specific actions, in the face of other competing responses that are irrelevant or inappropriate (Pennington & Ozonoff, 1996). The frontal lobes of the brain—particularly the prefrontal cortex (PFC) and its intricate web of interconnections with other regions—are thought to play a central role in these processes. Specifically, the PFC promotes higher level functioning by prioritizing, integrating, and regulating other cognitive functions, which originate in other areas of the brain (Aman et al., 1998). The parietal lobes—specifically the right parietal lobe—also appear to play a role in executive functioning, as these areas specialize in integrating sensory input from the visual regions. In particular, these areas appear to be essential for visually guided motor activity and spatial perception and attention, processes necessary for higher-order executive functioning. (Aman et al., 1998).

Developmental Trajectory of Executive Functions

Typically developing individuals experience significant growth in EFs throughout childhood, which appear to correspond with maturation in the frontal lobes (Jurado & Rosselli, 2007). Although the basic foundations of the PFC are present at birth, this brain region remains underdeveloped throughout childhood (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001). In particular, whereas children experience peak increases in PFC gray matter (believed to be indicative of increased neural density and connection) by middle childhood, this peak is followed by significant reductions in early adolescence (Bunge & Wright, 2007). This adolescent reduction in gray matter, linked to pruning, is associated with improved efficiency of neuronal connections; concurrent improvements take place in basic information processing and logical reasoning during the adolescent years (Blakemore, 2012). At the same time, adolescents experience an increase in white matter (i.e., increased myelination and axon thickness), which is associated with increased speed and efficiency of neuronal impulses via enhanced connectivity in the PFC. Better EF performance ensues. Specifically, during adolescence, individuals experience improved response inhibition and better future planning (Blakemore, 2012).

Overall, by late adolescence, the brain has experienced extensive maturational changes that appear to be associated with improvements in EFs. Research suggests that the PFC continues to experience normative maturational changes (particularly increased myelination) up until the mid-20s, with the final refinement of the frontal lobes taking place mainly during the ages of 20-29. EF performance is believed to reach its peak levels during the ages of 20-29 as well (De Luca et al., 2003).

ADHD and Areas of the Brain Implicated in Executive Functioning

In recent years, there has been a shift from focusing on the attentional deficits in ADHD to the primacy of EFs deficits, which appear to be strongly implicated in the disorder (Seidman, 2006). Children with ADHD present with a number of structural and functional disparities in the frontal areas of the brain, which support the association between executive functioning and ADHD (see Faraone & Biederman, 1998, for an early review). For example, unlike typically developing children, children with ADHD show symmetry in the anterior regions of the frontal lobes (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990). In addition, the areas of the corpus callosum that connect the frontal and the parietal lobes are also smaller in children with ADHD (Hynd , Semrud-Clikeman, Lorys, Novey, Eliopulos, & Lyytinen, 1991). These children also present with reduced cerebral blood flow in the prefrontal areas, while adults with ADHD and no stimulant medication history show reduced cerebral glucose metabolism in both the prefrontal and parietal regions (Lou, Henriksen, Bruhn, Borner, & Nielson, 1989; Zamertrin, et al., 1990). The areas of the brain that appear to be affected in ADHD are rich in catecholamines (i.e., dopamine and norepinephrine). Thus, the clinical efficacy of stimulant drugs—which block the reuptake of dopamine and norepinephrine—further supports the theory that frontal lobe deficits and associated brain areas are implicated in ADHD (Faraone & Biederman, 1998)—although the exact mechanisms behind these deficits and the development of ADHD is still unknown (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005).

More recent research on ADHD and frontal problems, using structural and functional imaging, implicates both frontal-striatal and frontal-cerebellar inefficiency in the neural processing of individuals with ADHD (for reviews, see Ahmad & Hinshaw, 2015; Rubia, 2010). Although the frontal regions and executive functions are still viewed as central to relevant psychopathology and impairment, recent research also implicates additional brain regions in ADHD—for example, reduced volume and cortical thickness in the parietotemporal areas, the basal ganglia, and the cerebellum (Krain & Castellanos, 2006; Shaw et al, 2006; Castellanos et. al, 2002). Furthermore, individuals with ADHD display complex patterns of limited brain-wide connectivity (e.g., Castellanos & Proal, 2012; Castellanos et al., 2006).

There is also some debate on whether the structural and functional brain disparities observed between individuals with ADHD and those without ADHD represent a divergence from normal development or a maturational lag, in which the brain development of individuals with ADHD may eventually catch up to that of their peers (see El Sayed, Larsson, Persson, Santosh, & Rydelius, 2003). For example, studies of EEG patterns revealed that boys with ADHD exhibited EEG abnormalities that were significantly divergent from normal development and not indicative of a maturational lag (Hobbs, Clarke, Barry, McCarthy, & Selikowitz, 2006). In contrast, other researchers have found patterns of brain-wide connectivity and structural changes suggesting that ADHD involves a maturational lag in brain development (e.g. Sripada, Kessler, & Angstadt, 2014). In particular, one study found that early differences in cortical thickness between individuals diagnosed with ADHD in childhood and comparisons diminished by early adulthood, with no differences being found by this developmental period between those who had remitted ADHD and those with no history of ADHD (Shaw, Malek, Watson, Greenstein, de Rossi, & Sharp, 2013). Thus, it is possible that at least some individuals with ADHD experience a maturational lag regarding specific aspects of brain development. However, because of limited research, it is unclear whether this same lag would be observed in EF performance, given the close association observed between the above-mentioned abnormalities in brain and executive functioning (El Sayed et al., 2003).

ADHD and Associated Executive Functioning Deficits

A number of EF deficits appear in ADHD samples. For example, an early but seminal meta-analysis of studies comparing participants with ADHD with controls, revealed that 15 of 18 investigations showed significant differences between the groups on one or more EF measures (Pennington & Ozonoff, 1996). Across these studies, participants with ADHD performed worse than controls on 40 of the 60 tasks assessed and did not perform better than controls on any of the tasks. EF tasks that appeared to be the most affected by ADHD status included the Tower of Hanoi (which assesses planning ability), Stroop (which is indicative of cognitive speed and interference control), Matching Familiar Figures Test (a measure of cognitive tempo), Trail-making (a measure of visual attention and task switching), tasks of working memory, and several measures of motor inhibition (e.g., Go No-Go task). Notably, significant differences in non-EF tasks (e.g., verbal tasks) were less common (Pennington & Ozonoff, 1996).

Another meta-analysis revealed significant differences between ADHD participants and controls across 13 distinct EF tasks. In particular, ADHD participants displayed consistent weaknesses in those tasks that indexed response inhibition, vigilance, working memory, and planning, even when adjusting for IQ, academic achievement, and symptoms of other disorders (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2006). Moreover, Aman et al. (1998) found that, in addition to demonstrating impairments with inhibitory control and planning ability, boys with ADHD demonstrated impairments with tasks that are thought to be associated with parietal lobe functioning—i.e., mental rotation and visual processing. Recent meta-analyses with expanded databases of more current empirical research have shown clear evidence for accentuated impulsivity and deficient response inhibition in individuals with ADHD, as well (Meza et al., 2017; Patros et al., 2015).

Despite the large number of studies supporting the link between ADHD and EF deficits, it is important to note that findings are not always consistent. For individual EF tasks, differences between ADHD and comparison participants have been found in some studies but not others (Pennington & Ozonoff, 1996). Furthermore, not every individual with ADHD displays EF deficits (Wilcutt et al., 2006). One proposed explanation is that there is heterogeneity in the mechanisms underlying ADHD. That is, for some individuals, ADHD may be a condition of underlying executive dysfunction, but for others it presents as a motivational style that is characterized by an aversion to time delays (e.g. Sonuga-Barke, 2002). However, even when these inconsistencies are taken into account, the vast majority of research suggests that most individuals with ADHD experience some type of EF deficit.

Furthermore, although some age-related improvement in their EF performance occurs as they age, research reveals that individuals with ADHD continue to experience EF deficits throughout the lifespan. For example, a cross-sectional study of younger (ages 9-15) and older (ages 15-22) males with and without ADHD revealed that the older boys performed better than younger boys on EF tasks regardless of their ADHD status. However, boys with ADHD continued to demonstrate poor EF performance—particularly for the Stroop test, the Wisconsin Card Sort Task (WCST), and the Rey-Osterrieth Complex Figure (ROCF; a global measure of EF)—when compared to their respective peers in both age groups (Siedman, Biederman, Faraone, Weber, & Ouellette, 1997). Similar results were found in both cross-sectional (ages 9-17: Siedman, Biederman, Monuteaux, Valera, Doyle, & Faraone, 2005) and longitudinal studies of girls with ADHD (Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Biederman et al., 2007). EF deficits also continue into early adulthood. For example, Biederman et al. (2006) found that males with ADHD who demonstrated EF deficits in early childhood and adolescence (9-22 years) continued to experience these deficits in the early adult years (16-30 years). Similarly, a sample of girls with ADHD, followed longitudinally, continued to display deficits in EFs in the earliest years of adulthood (mean age 19.6)—particularly on tasks of global EF, response inhibition, and working memory (Miller, Ho, & Hinshaw, 2012).

In addition, the EF deficits observed in ADHD have been linked to a wide range of poor outcomes. For example, in children and adolescents with ADHD, EF deficits are associated with a higher risk of grade retention and poor academic performance (Biederman et al., 2004). These deficits are also associated with poor social skills (Diamantopoulou, Rydell, Thorell, & Bohlin, 2007). Longitudinally, childhood EF deficits in females with ADHD have also been linked with poor social functioning and comorbid internalizing/externalizing disorders in adolescence (Rinsky & Hinshaw, 2011) and with poorer academic and occupational functioning in early adulthood (Miller, Nevado-Montenegro, & Hinshaw, 2012). The strong suggestion is that treatments aimed at individuals with ADHD should consider the role of EF deficits underlying the disorder and its related impairments (Brown, 2013).

However, there has been limited research exploring EF deficits in ADHD beyond the earliest years of adulthood—particularly in females. Such research is important because gender differences appear to exist in brain maturation. For example, the frontal lobe volume of girls tends to peak earlier than in boys, girls demonstrate different patterns of cerebral organization, and girls experience increases in dopamine receptors due to pubertal increases in estrogen (which has been associated with increased ADHD symptoms in adolescence; see Mahone & Wodka, 2008, for a review). In addition, gender differences in executive functioning performance have been found in both normative samples and children with ADHD (Mahone & Wodka). Thus, it is important to clarify whether the EF deficits observed in research utilizing mostly male samples are present in female samples as well.

As noted above, EF performance appears to reach its optimal peak between the ages of 20 to 29. Thus, in the present dissertation, I expand on previous research by exploring whether girls with ADHD continue to demonstrate EF impairments by their mid-twenties—a period of time known as emerging adulthood (Arnett, 2000). In particular, I explore their trajectories of global EF, working memory, sustained attention, and response inhibition from the period of childhood (ages 6-12 years), adolescence (12-17 years), the earliest years of adulthood (17-23 years), and emerging adulthood (23-29 years). Furthermore, I explore whether the EF performance of young women with ADHD histories continues to be divergent from that of their peers by adulthood, despite these potential improvements in overall raw scores. Finally, I investigate the linkages between (a) both EF trajectories and adult levels of EF performance and (b) core behavioral and academic impairments (i.e., externalizing, internalizing, and depressive symptoms as well as math and reading achievement) during emerging adulthood.

Hypotheses

1. Despite at least some evidence that certain disparities in the brain development of individuals with ADHD may be related to a maturational lag, additional evidence exists that individuals with ADHD continue to experience EF deficits into late adolescence and the early years of adulthood. Thus, I predict that, in the present sample, young women with histories of ADHD will continue to demonstrate deficits in global EF by emerging adulthood. I also predict that these young women will display more behavioral and academic impairments (e.g., higher internalizing, externalizing, and depressive symptoms and lower math and reading academic achievement) than their typically developing peers.
2. Given, however, that EFs tend to improve with age, in boys with ADHD, I hypothesize that this trend will continue in girls with histories of ADHD. That is, using growth curve modeling (GCM), I expect to find that young women with ADHD will demonstrate a trajectory of improvement in EFs—as will their counterparts without ADHD.
3. Past research exploring changes in EFs over childhood, adolescence, and the early twenties for females with ADHD suggests that this trajectory of EF improvement may be different for young women with ADHD vs. those without ADHD. Thus, I predict that the ADHD sample will demonstrate higher rates of improvement than those without ADHD

(see Miller, Loya, & Hinshaw, 2013). Even so, their performance will remain lower than that of the comparison sample.

4. Given that EFs are essential for future-oriented goals and context-specific actions, I expect that the poor EF performance in the ADHD sample will be correlated with poor behavioral and academic outcomes. For this hypothesis, I examine both (a) growth parameters of EF (e.g., linear slope) performance over the age span of childhood through emerging adulthood and (b) emerging-adult levels of EF performance, as predictors of behavioral and academic impairments in emerging adulthood.

Method

Overview

Data were drawn from a longitudinal study of elementary-school-aged girls with and without ADHD, known as the Berkeley Girls with ADHD Longitudinal Study (BGALS). Initial data were collected during three summer enrichment programs that took place from 1997 to 1999. Each summer, a new cohort of girls with ADHD participated in a five-week program that offered a combination of classroom, art, drama, and playground activities, along with a group-matched comparison sample of girls without ADHD. Specifically, comparison girls were recruited to be similar, in terms of age and ethnicity, to the ADHD sample. Parents and teachers completed questionnaires as part of the screening process; girls and their families then went through a thorough assessment battery pertaining to ADHD status as well as comorbidities, impairments, and academic, social, and cognitive functioning. All evaluations were conducted during a period in which girls with prior medication histories were not receiving stimulant medication. Well-trained graduate students and bachelor's level research assistants, who were closely supervised by a licensed clinical psychologist, administered the evaluations. The assessors were unaware of the diagnostic status of the participants. Initial and follow-up assessments received full approval from UC Berkeley's Committee for the Protection of Human Subjects.

The families were invited to participate in 5-year, 10-year and 15-year follow-up studies after their initial participation. Because of funding delays, the last follow-up was actually a 16-year evaluation, on average. Participants completed a thorough evaluation, spanning two half-days at our lab/clinic (occasionally, telephone interviews or home visits were performed). Data were gathered from participants and from informants (particularly parents, even if the young adults were no longer living at home).

Participants

The original sample consisted of 140 girls rigorously diagnosed with childhood ADHD (mean age = 9.6) and 88 comparison girls (mean age = 9.4) at baseline (Wave 1). The sample was ethnically diverse (53% White, 27% African American, 11% Latina, and 9% Asian American). The ADHD sample was recruited through medical settings (e.g., health maintenance organizations), mental health centers, pediatric practices, and local school districts. Advertisements were also placed in local newspapers and parenting newsletters. Comparison girls were recruited through school districts and local community centers and through advertisements in the local newspapers and parenting newsletters. Eligible families were sent packets about the program and then screened for ADHD status. ADHD diagnosis was made on the basis of initial ratings from parents and teachers using the Swanson, Nolan, and Pelham (SNAP) Parent and Teacher Inattention and Hyperactivity/Impulsivity Scales (Swanson, 1992), in addition to the Attention Problems scores on the Child Behavior Checklist (CBCL) and Teacher Report Form (Achenbach, 1991). Initial screening criteria were set somewhat low to avoid prematurely excluding potentially eligible girls. Yet for final eligibility in the ADHD group, the girl had to meet full criteria for ADHD (either Combined or Inattentive type) with respect to diagnostic interview criteria (i.e., at least six impairing symptoms of inattention for ADHD-Inattentive or at least six inattention and six hyperactivity-impulsivity symptoms for ADHD-Combined) on the Diagnostic Interview Schedule for Children, 4th edition (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). For eligibility in the comparison group, a diagnosis of ADHD needed to be absent based on both the SNAP and DISC-IV. The age range for both the ADHD and comparison group was 6-12 years. Exclusion criteria included an IQ lower than 70, overt neurological damage, psychosis, or pervasive developmental disorder, and medical conditions that permitted participation in the summer camp. Full data from the childhood phase of this investigation can be found in Hinshaw (2002) plus companion investigations (e.g., Hinshaw et al., 2002, for neuropsychological data at baseline).

Of the original 228 families, 209 (92%) participated in the five-year follow-up (Wave 2; mean age = 14.1); see Hinshaw et al. (2006) and Hinshaw et al. (2007) for neuropsychological data. Next, 216 (95%) participated in the 10-year follow-up study (designated as Wave 3: mean age = 19.6); see Hinshaw et al. (2012) for an overview and Miller et al. (2012) for information neuropsychological performance at this age. Finally, 211 (93%) participated in the 16-year follow-up (designated as Wave 4; mean age = 25.6). Extensive measures were taken to maximize participant retention at all waves.

Measures

Executive Functioning

EF measures were selected for (a) their establishment as well-validated measures at the study's baseline, in the 1990s, (b) past research evidence differentiating ADHD from comparison individuals, and (c) their repeated administration in the present sample. Except for the Cancel-Underlining task, these measures were administered at all four waves.

Rey-Osterrieth Complex Figure Test (ROCF; Osterrieth, 1944). The ROCF was used to assess global EF, as it taps multiple domains of EF, including planning, response inhibition, attention, and organization. This task was administered at Wave 1, Wave 3, and Wave 4 (see below for the parallel measure administered at Wave 2); thus, it was considered to be time varying in the growth curve modeling analyses. In this task, the individual was asked to copy and recall a complex figure that was comprised of 64 segments. Only the copy condition was used in the analyses, because it was the only condition that differentiated ADHD from comparison status at baseline (Sami, Carte, Hinshaw, & Zupan, 2003). The error proportion score (EPS)—which is derived by dividing the number of errors by the total number of segments drawn—was used to index the participants' efficiency in drawing the figure (Sami et al., 2003). This index has shown excellent psychometric properties in subsequent research (e.g., Hinshaw et al., 2002; Hinshw et al., 2007; Miller et al., 2012). Higher scores indicate greater EF impairment. In terms of inter-scorer agreement, intraclass correlations between the pairs of the three primary scorers for the EPS at Wave 1 ranged from .91 to .94 (based on 84-195 drawings completed across rater pairs). The intraclass correlation at Wave 3 between pairs of the two primary scorers was .91 on a sample of 70 drawings and; at Wave 4, it was .94 between similar pairs for 79 drawings.

Taylor Complex Figure Test (TCFT; Taylor, 1969). The TCFT was administered at Wave 2 to serve as a parallel test for the ROCF. It measures the same constructs as the ROCF. The TCFT is considered the only major alternative to the ROCF in test-retest situations (Helmes, 2000). It was used to address possible practice effects for the recall condition. However, as noted above, later analyses found that this condition did not differentiated the ADHD participants from the comparisons. Thus, since the recall condition was not subsequently used in Wave 3 and 4, use of the TCFT was limited to Wave 2. As with the ROCF, only the copy condition of the TCFT was used in the current analyses and a parallel error proportion score was used to index the participants' efficiency in drawing the figure. The intraclass correlation between pairs of three scorers ranged from .77 to .94 (mean = .84) in a subsample of 60 drawings.

Conner's Continuous Performance Task (CPT; Conners, 1995). The CPT was used to assess attentional processing and response inhibition. The task requires participants to press the spacebar when all target letters (except the letter "X") appear on the screen. Simultaneously, participants are instructed to refrain from pressing the spacebar when the "X" appears on the screen. The 14-minute task consists of trials presented in six blocks, during which the stimulus is presented for 250 ms (with interstimulus intervals of 1, 2, and 4 secs). Only the percentages of commission errors (indicative of response inhibition) were used in the analyses. There was not enough variance in the percentages of omission errors (indicative of sustained attention) to compute estimates (for example, at Wave 4, 40% of the participants made no errors and 75% of participants made fewer than 1% omission errors). Higher percentages of commission errors were indicative of poorer inhibitory executive functioning; this variable was utilized as a time-varying variable in the growth curve modeling analyses.

WISC-III Digit Span (Wechsler, 1991). The Digit Span was used to assess working memory. During this task, participants are asked to recall digit sequence of increasing length in original (Digits Forward) and reverse order (Digits Backwards). The raw scores of these tasks (i.e., Digits Forward and Backwards) were combined, so that lower scores indicated greater working memory impairment, and were utilized as a time varying variable. The WISC-III is a well-established, psychometrically sound measure, with the Digit Span subtest having an internal consistency of .85 and re-test reliability of .75 (Wechsler, 1991).

Cancel Underlining (CUL). A modification of the Underlining Test (Rourke & Orr, 1977) was used to measure rapid, accurate visual discrimination, as well as inhibition. Previous research has found that the errors in discriminating consonant sequences optimally distinguished ADHD participants from typically developing comparison participants (e.g. Nigg, Hinshaw, Carte, & Treuting, 1998). The girls were instructed to underline targets (i.e. a sequence consonants and shapes) and to cancel out non-targets (ratio 1:5). The correct minus incorrect responses were analyzed. The CUL task was administered at Wave 1, 3, and 4. Thus, three waves of data for the CUL were used and analyzed as a time varying variable.

Measures of Adult Outcome

A variety of measures from various sources (parent- and self-report; objective testing) assessed the young women's emerging-adult behavioral and academic outcomes (Wave 4).

Self-reported externalizing and internalizing behaviors. The young women self-reported on their externalizing and internalizing behavior using the Adult Self-Report (ASR), a frequently utilized 126-item measure that has well-established internal consistency, test-retest reliability, and validity (Achenbach & Rescorla, 2003). For adults, Cronbach's alphas for the broad-band internalizing and the externalizing scales = .93 and .89, respectively, (one week test-retest reliabilities = .89 and .91). The young women rated items assessing their own behavior using a 3-point scale (0 = *not true*; 2 = *very true or often true*). The raw scores were converted to T-scores using age and gender norms.

Parent-reported externalizing and internalizing behaviors. Parent reported externalizing and internalizing behaviors were measured using the Adult Behavior Checklist (ABCL). For a majority of cases (86.8%), the young women's mother completed this measure. The ABCL is a 126-item measure that parallels the ASR and, likewise, has well-established internal consistency, test-retest reliability, and validity (Achenbach & Rescorla, 2003). Cronbach's alphas for the broad-band internalizing and the externalizing scales = .92 and .93, respectively, for the young women's behavior as reported by the parent (one week test-retest reliabilities = .80 and .92). Their parents rated items assessing their child's behavior using the same 3-point scale (0 = *not true*; 2 = *very true or often true*) as the ASR. The raw scores were also converted to T-scores using age and gender norms. The cross informant correlation between the ASR and ABCL was .43 for the internalizing scales and .44 for the externalizing scales.

Self-reported depression. The young women's self-reported depressive symptoms were measured via the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). This measure comprises 21 items and is used to assess the presence of depressive symptoms including negative mood, interpersonal problems, and negative self-esteem. Participants are asked to rate the presence of each symptom during the past two weeks on a 4-point, 0-3 scale("0" indicates an absence of a symptom; "3" indicates the presence of an extreme form of a symptom). Both the test-retest reliability and internal consistency of the CDI have been well established: internal consistency = .92 in outpatient populations and .93 in college age students; test–retest reliability averages .93 (Beck et al., 1996).

Wechsler Individual Achievement Test, 2nd Edition (WIAT-II; Weschler 1992). On this individually administered test, the Word Reading subtest measures sight-reading ability of known words, and the Math Reasoning subtest indexes understanding of numbers, consumer math concepts, geometric measurement, and basic graphs in order to solve multi-step word problems. The WIAT-II is considered a psychometrically sound assessment of academic achievement, with both internal consistency and test-retest reliability estimates above .85 for most subtests (Wechsler, 1992). Standard scores (which were normed based on the participants' age and grade) from both the Word Reading subtest and Math Reasoning subtest were used as a measure of academic functioning at Wave 4, with higher scores indicating higher achievement.

Covariates. The young women's diagnostic status, which was determined by the SNAP and DISC-IV as described above, at baseline (i.e., Wave 1) was dummy coded: those with childhood ADHD were coded as 1 and the comparison group was coded as 0. The young women's age in years was also collected at each assessment. Baseline (i.e., Wave 1) family annual income as reported by the primary parent (which could range from 1 to 9; with 1 indicating "<$10,000", 5 indicating "$40,001 to $50,000", and 9 indicating ">$75,000") and maternal education (which could range from 1 to 6; with 1 indicating "less than 8th grade", 3 indicating "high school graduate" and 5 indicating "advanced or professional degree") were included as covariates in the final prediction analyses.

Statistical Analyses

All statistical analyses were performed using STATA version 14. After examining descriptive statistics and correlations among the variables, I conducted t-tests to assess diagnostic group differences with respect to both the EF measures at all waves and emerging-adult outcomes at Wave 4.

The repeated, prospective design resulted in a two-level hierarchical structure, with each wave of data collection nested within participants. Using hierarchical linear modeling, growth curves were used to model the average change of EF over time, each participant's change in executive functioning, and predictors (i.e., baseline ADHD status) that may account for individual differences in change over time. All data available were utilized and maximum likelihood estimation (MLE), which is considered a common and well-established estimation method (Garson, 2013), was used to derive the estimates of the growth curve models. These analyses involved four steps: First, I investigated individual and group-level EF performance as a function of time, without predictors (i.e., an unconditional growth model). Both the intercepts and slopes were allowed to vary, as individual variability was expected to be high.

Second, given the expected nature of EFs (i.e., EF performance generally improves throughout childhood and adolescence before peaking and plateauing in early adulthood), I considered a non-linear trajectory of EF and investigated adding polynomial terms to the model. Third, I added predictors (i.e., ADHD diagnostic status) of variance in the growth curve slope and intercept of each EF measure. Finally, I examined cross-level interaction terms between the participants' age and childhood diagnostic status to investigate possible differences in EF trajectories between those young women with childhood ADHD and those without. These analyses were repeated separately for each of the four measures of EF: ROCF (a global measure of EF), CPT commission errors (indicative of response inhibition), Digit Span (working memory), and CUL (visual discrimination and inhibition).

To test the effects of EF performance over time on the young women's behavioral and academic impairments in emerging adulthood, I used sample-level estimates from the final growth curve models to estimate an individual slope and intercept for each woman's EF trajectory. Hierarchical multiple regression was then used to explore the relation between a young woman's slope and Wave 4 behavioral and academic impairment measures. That is, the steepness of a young woman slope was explored as a predictor of her emerging adult impairment. At Step 1, the women's childhood diagnostic status (dummy coded) was entered, along with maternal education and family income as covariates. The individual-level slopes from the previous growth curve model were entered as a predictor at Step 2. At Step 3, two-way interactions between these slopes and childhood ADHD status were entered to examine possible diagnostic group differences in the association between EF trajectories and emerging adult impairments. These analyses were repeated for each of the emerging-adult impairment measures, yielding six separate hierarchical regression models.

A final set of hierarchical multiple regressions was used to explore whether a young woman's EF during emerging adulthood (i.e., at Wave 4) predicted her concurrent behavioral and academic impairment. At Step 1, I entered childhood diagnostic status, along with maternal education, income, and the young women's age at testing (because raw scores for EF measures are utilized in these analyses). At Step 2, I entered EF scores (with separate models entered for each of the four EF measures); two-way interactions between EF scores and diagnostic status were entered at Step 3. These analyses were repeated for each of the emerging-adult impairment measures, yielding six separate hierarchical regression models for each EF measure.

Because this statistical analysis involved performing multiple hypothesis tests simultaneously, the Benjamini-Hochberg (BH) procedure was used as a multiple testing correction. The BH procedure protects against Type I error by controlling for a false discovery rate (see What Works Clearinghouse, 2008). An overall BH procedure was performed on the t-tests to assess diagnostic differences. Subsequently, BH procedures for each Wave 4 outcome variable were performed after both sets of hierarchical multiple regressions. That is, for each Wave 4 outcome variable, a single BH correction was made for all significant p-values found when examining the individual-level slopes and concurrent EF performance scores as predictors.

Results

Descriptive Statistics

Table 1 presents scores for the ADHD and comparison samples with respect to demographic variables. The groups were statistically indistinguishable with respect to age, family income, and maternal education. However, a chi-square test did reveal a significant difference in the ethnic composition of the groups, $\chi2(4)$, $N = 201) = 9.298$, $p <.05$, with a higher percentage of Asian American girls in the comparison group. Tables 2, 3, and 4 present the intercorrelations between the EF measures from each wave and outcome variables from emerging adulthood (i.e., Wave 4). As shown in Table 5, the young women with ADHD, when compared to their peers without ADHD, had far worse scores on all EF measures across all waves with the exception of Wave 1 CPT scores. They also had worse parent-reported internalizing symptoms, self- and parent-reported externalizing symptoms, self-reported depressive symptoms, and academic achievement (i.e., math and reading) outcomes than their counterparts at Wave 4. However, no significant differences were found for self-reported internalizing symptoms (see Table 6). The results for these t-tests remained significant when the BH correction was applied.

Growth Curve Modeling

Developmental Trajectory of ROCF/TCFT. Table 7 presents the estimates for the hierarchical linear model exploring the developmental change in ROCF error proportion scores, representing global EF. In the unconditional growth model, the estimated slope for the growth curve across all participants indicated that error proportion scores decreased over time, from childhood to emerging adulthood, ($B = -.01$, $p < .001$). Given the pattern based on a plot of the observed means of the error proportion scores for the ROCF/TCFT across the 4 waves (see Figure 1a), the appropriateness of a quadratic model was considered. In Model 2, with the quadratic term added, both the linear slope ($B = -.05$, $p < .001$), and quadratic term ($B = .001$, $p < .001$) remained significant, suggesting that the young women's error proportion scores decreased over time, with rapid improvements occurring in early childhood and adolescence before plateauing (and even reversing) in early/emerging adulthood. A significant likelihood-ratio test (comparing Model 2 against Model 1) provided strong evidence that the quadratic term should be retained: $\Delta\chi^2_{(1)}=127.70$, $p < .001$. In Model 3, childhood (Wave 1) diagnostic status was entered as a predictor of the variance around the slope and intercept. Results indicated that the participants with childhood ADHD had error proportion scores that were on average .08 greater (i.e., worse) than those of the young women without childhood ADHD. A significant likelihood-ratio test comparing this model to the previous provided strong evidence that this predictor should be retained: $\Delta\chi^2_{(1)}=40.41$, $p < .001$. In the final model (Model 4), a significant two-way interaction between diagnostic status and time was found ($B = -.003$, $p < .05$), with a significant likelihood-ratio test indicating that the interaction term should be retained: $\Delta\chi^2_{(1)}=4.44$, $p < .05$. Post-hoc analyses suggested that the young women with ADHD had steeper decreases in errors over time ($B = -.06$, $p < .001$) than did their typically developing counterparts ($B = -.03$, $p < .001$), although both groups experienced declines over time (see Figure 2).

Developmental Trajectory of CPT. Table 8 presents the estimates for the hierarchical linear model exploring the developmental change in CPT commission errors, representing response inhibition. In the unconditional growth model, the estimated slope for the growth curve across all participants for the CPT indicated that commission error scores decreased over time, from childhood to emerging adulthood, ($B = -1.21$, $p < .001$). Given the pattern based on a plot of the observed means (see Figure 1b), a quadratic term was considered. In Model 2, with the quadratic term added, both the linear slope ($B = -5.48$, $p < .001$), and quadratic term ($B = .12$, $p < .001$) were significant. In conjunction with a plot of the means, this pattern suggests that young women's commission errors followed a similar trajectory as the ROFT/TCFT error proportion scores: decreasing over time, with rapid improvements occurring in early childhood and adolescence before beginning plateauing (and even reversing) in early/emerging adulthood. A significant likelihood-ratio test comparing this model against the previous provided strong evidence that the quadratic term should be retained: $\Delta\chi^2_{(1)}=46.65$, $p < .001$. When childhood diagnostic status was entered as a predictor in Model 3, results indicated that the young women with childhood ADHD had a commission error percentage that was on average 8.49 points greater than those young women without childhood ADHD. A significant likelihood-ratio test provided strong evidence that this predictor should be retained: $\Delta\chi^2_{(1)}=16.42$, $p < .001$. In Model 4, a significant two-way interaction between diagnostic status and time was not found ($B = .31$, *n.s.*), with a significant likelihood-ratio test indicating that the interaction term should not be retained: $\Delta\chi^2_{(1)}=2.35$, *n.s.*

Developmental Trajectory of Digit Span. Table 9 presents the estimates for the hierarchical linear model exploring the developmental change in the young women's digit span raw scores, representing working memory. In the unconditional growth model, the estimated slope for the growth curve across all participants indicated that digit span scores increased over time, from childhood to emerging adulthood (B = .24, $p < .001$). Given the pattern based on a plot of the observed means (see Figure 1c), a quadratic term was considered. In Model 2, with the quadratic term added, both the linear slope (B = 1.01, $p < .001$), and quadratic term (B = -.02, $p < .001$) were significant. In conjunction with a plot of the means, a parallel trend to those for ROCF and CPT Commission errors was found, with rapid improvements occurring in early childhood and adolescence before plateauing in early/emerging adulthood. A significant subsequent likelihood-ratio test provided strong evidence that the quadratic term should be retained: $\Delta\chi^2_{(1)}=104.78$, $p < .001$. When childhood diagnostic status was entered as a predictor in Model 3, results indicated that the digit span scores for young women with childhood ADHD were significantly lower (i.e., 2.26 points lower) than those young women without childhood ADHD. A significant likelihood-ratio test provided strong evidence that this predictor should be retained: $\Delta\chi^2_{(1)}=38.97$, $p < .001$. In Model 4, a significant two-way interaction was not found between diagnostic status and time (B = -.009, *n.s.*), with a significant likelihood-ratio test indicating that the interaction term should not be retained: $\Delta\chi^2_{(1)}=.11$, *n.s.*

Developmental Trajectory of CUL. Table 10 presents the estimates for the hierarchical linear model exploring the developmental change in the young women's CUL raw scores, representing visual discrimination/response inhibition. In the unconditional growth model, the estimated slope for the growth curve across all participants indicated that CUL scores increased over time, from childhood to emerging adulthood, (B = .79, $p < .001$). Because only three time points were used for this model, a quadratic term was not considered, as four or more points are required to investigate a quadratic term (Anderson, 2012). When childhood diagnostic status was entered as a predictor in Model 2, results indicated that the CUL scores for young women with childhood ADHD were significantly lower (i.e., 2.95 points lower) than those of young women without childhood ADHD. A significant likelihood-ratio test provided strong evidence that this predictor should be retained: $\Delta\chi^2_{(1)}=16.86$, $p < .001$. In Model 3, a significant two-way interaction was found between diagnostic status and time (B = -.24, $p < .001$), with a significant likelihood-ratio test indicating that the interaction term should be retained: $\Delta\chi^2_{(1)}= 14.29$, $p < .001$. Post hoc analyses suggest that the young women without ADHD had steeper increases in CUL performance (i.e., improvement) over time (B = .92, $p < .001$) than their counterparts with ADHD (B = .68, $p < .001$), though both groups showed increases over time (see Figure 3).

Emerging Adult Outcomes Predicted by EF Trajectories

See Table 11.

Covariates. Childhood ADHD status was predictive of all Wave 4 outcome variables, with the exception of self-reported externalizing symptoms and self-reported depressive symptoms. Specifically, ADHD diagnostic status was associated with more self-reported externalizing ($\beta = .19$, $p < .01$), parent-reported internalizing ($\beta = .45$, $p < .001$), and parent-reported externalizing ($\beta = .56$, $p < .001$) problems, as well as lower reading ($\beta = -.40$, $p < .001$) and math achievement scores ($\beta = -.42$, $p < .001$). These remained significant with the BH correction. After the BH correction, higher annual family income at baseline (i.e. Wave 1) was associated with lower self-reported externalizing ($\beta = -.18$, $p < .05$) and self-reported depressive symptoms ($\beta = -.21$, $p < .01$) as well as higher reading achievement ($\beta = .27$, $p < .001$). Maternal education was not predictive of any of the Wave 4 adult outcomes.

Trajectory of ROCF/TCFT. The trajectory (i.e., slope) of ROCF/TCFT performance was not predictive of any of Wave 4 adult outcomes. No two-way interactions between the trajectory and diagnostic status were found.

Trajectory of CPT. Likewise, the trajectory of CPT performance was not predictive of any of the Wave 4 outcomes. No two-way interactions between the trajectory and diagnostic status were found.

Trajectory of Digit Span. The trajectory of digit span performance was predictive of self-reported externalizing symptoms ($\beta = .17$, $p < .05$) and explained a significant proportion of the variance in these symptoms, $R^2 = .11$, $F(4, 200) = 6.20$, $p < .001$. That is, increases in digit span performance were associated with higher levels of externalizing symptoms. This relation continued to be significant after the BH correction. The trajectory of digit span performance was not predictive of any other Wave 4 outcomes. No two-way interactions between the trajectory and diagnostic groups were found.

Trajectory of CUL. The trajectory of CUL performance was initially predictive of the young women's self-reported externalizing symptoms ($\beta = .15$, $p < .05$) and reading achievement ($\beta = .15$, $p < .05$). However, these predictions did not remain significant with the BH correction. No two-way interactions between the trajectory and diagnostic groups were found.

Emerging Adult Outcomes Predicted by Concurrent EF Performance

Note that for these analyses, the prediction is from the Wave 4 level of EF performance, rather than the Wave 1-Wave 4 trajectory of EF performance. See Table 12.

Covariates. After the BH correction, childhood ADHD status continued to predict all Wave 4 outcome variables with the exception of self-reported externalizing symptoms and self-reported depressive symptoms. That is, having ADHD in childhood was associated with more self-reported externalizing ($\beta = .20$, $p < .01$), parent-reported internalizing ($\beta = .45$, $p < .001$), and parent-reported externalizing ($\beta = .56$, $p < .001$) problems, as well as lower reading ($\beta = -.40$, $p < .001$) and math achievement scores ($\beta = .43$, $p < .001$). After the BH correction, higher annual family income at baseline also continued to be associated with self-reported externalizing ($\beta = -.17$, $p < .05$) and self-reported depressive symptoms ($\beta = -.21$, $p < .01$) as well as higher reading achievement ($\beta = .27$, $p < .001$). Age at Wave 4 was negatively associated with self-reported externalizing symptoms ($\beta = -.19$, $p < .001$) after the BH correction, such that older women reported fewer externalizing behaviors. Maternal education was not predictive of any of the emerging adult outcomes.

Emerging Adult ROCF/TCFT Performance. Wave 4 ROFC/TCFT performance was predictive of the young women's WIAT math scores ($\beta = -.22$, $p < .01$) and explained a significant proportion of the variance, $R^2 = .26$, $F(6, 184) = 10.66$, $p < .001$. That is, the higher a young women's error proportion score in emerging adulthood, the lower her concurrent performance in math. This association remained significant after the BH correction was applied. ROCF/TCFT performance was not predictive of any of the other emerging adult outcomes. No two-way interactions between ROCT/TCFT performance and diagnostic group were found.

Emerging Adult CPT Performance. Wave 4 adult CPT performance was not predictive of any of the emerging adult outcomes. No two-way interactions between CPT performance and diagnostic group were found.

Emerging Adult Digit Span Performance. Wave 4 digit span performance was predictive of the young women's WIAT reading scores ($\beta = .33$, $p < .001$) and explained a significant proportion of the variance in these scores, $R^2 = .40$, $F(6, 198) = 21.98$, $p < .001$. That is, the higher a young women's digit span score in emerging adulthood, the higher her concurrent WIAT reading scores. A two-way interaction between digit span performance and diagnostic group was also found ($\beta = .78$, $p < .01$), such that higher digit span scores were associated with higher reading achievement scores in the young women with ADHD ($\beta = .46$, $p < .001$), but not in their counterparts without ADHD ($\beta = .19$, $p >.05$; see Figure 4.) These relations remained significant after the BH correction was applied.

Wave 4 digit span performance was also predictive of the young women's WIAT math achievement scores ($\beta = .41$, $p < .001$), such that the higher a young women's digit span score during emerging adulthood, the higher her concurrent WIAT math scores. This association explained a significant proportion of the variance in these scores, $R^2 = .37$, $F(6, 196) = 18.86$, $p < .001$, and remained significant after the BH correction No two-way interaction between digit span performance and diagnostic group was found for this association. Digit span performance was not predictive of any of the other emerging adult outcomes, and no other significant two-way interactions were found.

Emerging Adult CUL Performance. Wave 4 CUL performance was predictive of WIAT reading scores ($\beta = .32$, $p < .001$) and explained a significant proportion of the variance in these scores, $R^2 = .36$, $F(5,193) = 21.84$, $p < .001$. The higher a young women's CUL score in emerging adulthood, the higher her concurrent WIAT reading scores. This association remained after the BH correction. A two-way interaction between CUL performance and diagnostic group was not found ($\beta = .37$, $p > .05$).

Emerging adult CUL performance was also predictive of WIAT math scores ($\beta = .34$, $p < .001$), such that the higher a young women's CUL score during emerging adulthood, the higher her concurrent WIAT math scores. This association explained a significant proportion of the variance in these scores, $R^2 = .31$, $F(5, 191) = 17.34$, $p < .001$, and remained significant after the BH correction. No two-way interaction between CUL and diagnostic group for this association was found. CUL performance was not predictive of any of the other emerging adult outcomes, and no other two-way interactions were found.

Discussion

As predicted, girls diagnosed with ADHD in childhood continued to exhibit poor performance on all aspects of EF—global executive functioning, inhibitory control, working memory, and visual discrimination—than their typically developing counterparts, in the developmental period of emerging adulthood. This core finding is congruent with previous research in younger and predominately male samples, which has revealed that individuals with ADHD consistently experience more EF deficits than their peers without ADHD (e.g., Pennington & Ozonoff, 1996). Furthermore, the overall trajectory on these aspects of EF (with the exception of visual discrimination, which had too few data points to estimate non-linear terms), followed a quadratic trend, with rapid improvements observed from childhood to adolescence before plateauing in the initial years of adulthood and emerging adulthood. Although both diagnostic groups exhibited improvements in all four aspects of EF across this developmental span, the young women with ADHD consistently lagged behind their counterparts. Because the young women with ADHD did not catch up to their peers on any aspect of EF tasks at any point of the developmental trajectories observed, the current findings do not support the idea that individuals with ADHD are experiencing merely a maturational lag with regard to EF.

Minor diagnostic group differences emerged in the rates of these trajectories, particularly for global executive functioning (as measure by the ROCF task) and visual discrimination and inhibitory response (as measured by the CUL task). The young women with ADHD demonstrated steeper improvements than their peers in global executive functioning. A plot of this trajectory (Figure 2) revealed that the young women with childhood ADHD experienced steeper improvements in global EF than their peers from childhood to late adolescence before rapidly leveling off, whereas their peers experienced a more gradual increase in global EF performance. Thus, young women with ADHD experience early rapid changes in global EF that bring them closer to (but not at the same level as) their peers' EF performance. Mechanisms underlying these sudden rapid improvements should be further investigated. It is still possible that a delayed maturational process related to certain brain structures or connectivity networks fosters this rapid change.

The opposite trend was observed for the CUL task, which indexes visual discrimination and inhibitory control. Here, the comparison women had steeper improvements. This finding suggests that young women with ADHD displaying difficulties with visual discrimination and inhibitory control not only have difficulties performing at a level comparable with their peers but also show slower rates of improvement. Further research is needed to fully understand the underlying mechanisms. In addition, early interventions targeting these aspects of EF may be beneficial for girls with ADHD.

I also found that the trajectory for working memory was predictive of the young women's behavioral functioning in emerging adulthood. In particular, and surprisingly, steeper improvements in the rate of working memory were associated with *more* self-reported externalizing symptoms. This pattern may reflect a compensatory process by which young women who started with lower working memory scores—predictive of externalizing problems—had more room for improvement. Alternatively, given that this association was only found for self-reported externalizing problems, the rapid improvement in working memory capacity may have led the girls to become more aware of their externalizing problems and subsequently led to them reporting more. Further investigation is needed to fully understand this association.

Finally, I found that (a) higher levels of global EF in emerging adulthood were associated with higher concurrent adult reading scores, and (b) higher levels of working memory and visual discrimination/inhibitory response performance in emerging adulthood were associated with concurrent reading and math achievement. These findings are consistent with research on EF in males and in girls with ADHD, which reveals that EF deficits are associated with poorer academic performance (e.g., Biederman et al., 2004; Miller et al., 2012). Furthermore, the association between working memory and reading was stronger for young women with ADHD than for the comparison sample, suggesting that working memory deficits in young women with ADHD may be particularly salient.

However, contrary to expectations and previous research in younger samples of females with ADHD (e.g., Rinsky & Hinshaw, 2011), we did not find evidence that EF deficits in emerging adulthood predicted concurrent internalizing, externalizing, or depression symptoms. This negative finding suggests that earlier EF performance (i.e., childhood and adolescence) may play a bigger role in behavioral impairments than later EF performance in relation to problematic behavior. In addition, given the variation in occupational/educational status that often characterizes young adulthood, the association between EF deficits and these impairments during this developmental period may vary depending on the extent to which a young adult's current occupational/educational status requires the use of EF skills (e.g. some may be employed in jobs where the demand on their EF performance is high, while others may be employed in jobs where the demand is low). Further research should be done to explore such group differences.

Limitations

Several limitations should be taken into consideration when interpreting these results. First, because research examining executive functioning in young women with ADHD has been quite limited, we focused on females with ADHD. Given the lack of a male sample, however, sex differences could not be directly investigated. To date, a dearth of studies investigates sex differences in the trajectory of EF. As highlighted in the Introduction, a number of known sex differences in brain development, structure, and function exist (see Mahone &Wodka, 2008), suggesting that differences in EF may be observed between men and women. In addition, the associations between these EF trajectories (and concurrent EFs) with emerging-adult academic and behavioral performance may differ for boys with ADHD, especially because the disorder tends to manifest and influence impairments in boys and girls in different ways (Taylor & Keltner, 2002). Future studies should examine these differences.

Furthermore, I did not examine the influence of diagnostic subtypes on EFs (i.e. predominately inattentive, hyperactive-impulsive, or combined type) and associated impairments. It may be the case that the observed trajectories on EF and subsequent impairments may be different for those with one type versus another, although the BGALS sample has revealed almost no significant subtype differences for any neuropsychological variable (see Hinshaw et al., 2002, 2007). In addition, my investigation did not distinguish between young women whose ADHD had remitted by adulthood (i.e., had < 5 symptoms) and those who had persisting symptoms. It was beyond the scope of this dissertation to analyze this potentially important variable. Previous research has found that remitters, by adulthood, tend to have brain connectivity that resembles that of their peers, whereas those with persistent ADHD symptoms continue to demonstrate brain connectivity that is divergent from both remitters and comparison participants (Shaw et al., 2013). Furthermore, not all individuals with ADHD demonstrate EF deficits (Wilcutt et al., 2005). The present investigation did not examine possible idiosyncratic differences in EF deficit among those with ADHD, even though understanding the heterogeneity of underlying mechanisms is crucial.

It is also important to note that this investigation was limited to four aspects of EF (i.e., global EF, inhibitory control, working memory, and visual discrimination). Other aspects of EF, such as planning and set shifting, should also be investigated to examine whether similar trajectories and associated impairments are found. Furthermore, other important emerging- adult outcomes (such as educational and occupational attainment) should be examined.

Finally, regarding methodology, only three time points were available for the CUL task, which limited the ability to consider a quadratic trajectory. Also, although the majority of parent reporters for internalizing and externalizing behaviors were the young women's mothers, our analyses included reports from alternative caregivers (i.e. fathers, step-parents, grandparents, aunts, uncles, etc.), which may have affected the comparability of these measures across different participants. Future investigations could be improved by including more objective measures of externalizing and internalizing symptoms.

Overall, despite these limitations, this study features several key strengths—in particular, multi-wave data, a low participant attrition rate, and an ethnically diverse sample—which contribute to the overall validity and generalizability of the findings.

Implications

The present findings provide valuable insight into how executive functioning develops over time, from childhood to emerging adulthood, for females with ADHD. Young women with ADHD not only continue to experience EF deficits in emerging adulthood, but these deficits continue to be associated with significant reading and math achievement impairments, with strong implications for occupational attainment. It is conceivable that interventions (including academic accommodations) aimed toward enhancing EF, particularly working memory, could alter the negative developmental trajectories so often associated with ADHD through adolescence and adulthood. Although research on working memory training has been mixed (Randall & Tyldesley, 2016), there is some evidence for other ways to improve working memory. For example, more interactive forms of working memory training have demonstrated promising results (Alloway, 2012), and physical exercise has been shown to improve working memory as well as other aspects of EF (Grassman, Alves, Santos-Galduroz, and Galduroz, 2017; Zieris & Jansen, 2015).

The development of early interventions targeting EF deficits more broadly should also be considered, in order to help lessen the pervasive EF performance gap observed between ADHD and comparison samples throughout childhood, adolescence, and early adulthood. In addition, it is important to consider how the persistent presence of EF deficits in young women with ADHD—particularly through emerging adulthood—may influence choice of treatments. Indeed, a popular treatment for adult ADHD—cognitive behavioral therapy (CBT)—targets key cognitive processes. In particular, a study of a CBT treatment for adults with ADHD specifically targeting EF deficits (i.e., poor time management, planning, and organizational skills) has shown promise in both reducing participants' symptoms of inattention and EF difficulties (Solanto, Marks, Mitchell, Wasserstien, & Kofman, 2008). Research examining the effects of EF deficits on other types of treatments in other clinical populations (e.g., depression) suggests that EF deficits can hamper patients' response to psychotherapy more broadly (Julian &Mohr, 2006). However, more research is required, to obtain a more comprehensive picture of how EF deficits may affect treatment responses to psychotherapy in general and for individuals with ADHD in particular.

Summary: Conclusions and Future Directions

In summary, I demonstrated that, despite overall executive functioning improvements over time, young women diagnosed with childhood ADHD consistently experience EF problems from childhood through emerging adulthood. These deficits are associated with a number of behavioral and academic impairments in emerging adulthood, including poorer math and reading achievement and increased externalizing symptoms. These findings yield key implications for the need to provide remediation for EF deficits in girls and women with ADHD. Further research should aim to understand individual difference in processes and mechanisms, with particular focus on girls and women experiencing clinically significant symptoms.

References

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist 4/18 and Profile 1991*. Burlington, VT: University Associates in Psychiatry.

Achenbach, T. M., & Rescorla, L. A. (2003). *Manual for the ASEBA adult forms and profiles.* Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Ahmad, S. I., & Hinshaw, S. P. (2015). Attention-deficit hyperactivity disorder: Similarities to and differences from other externalizing disorders. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Oxford handbook of externalizing spectrum disorders.* New York: Oxford University Press.

Aman, C. J., Roberts, R. J., Jr., & Pennington, B. F. (1998). A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder: Frontal lobe versus right parietal lobe theories. *Developmental Psychology, 34*(5), 956-969. doi:http://dx.doi.org/10.1037/0012-1649.34.5.956

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). *Arlington, VA: American Psychiatric Publishing.*

Anderson, D. (2012). Hierarchical Linear Modeling (HLM): An introduction to key concepts within cross-sectional and growth modeling frameworks (Report No. 1308). Eugene, OR: Behavioral Research and Teaching, University of Oregon.

Anderson, V A, Anderson, P, Northam, E, Jacobs, R & Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology, 20 (1),* 385-406.

Arnett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist, 55,* 469-480.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94. doi:http://dx.doi.org/10.1037/0033-2909.121.1.65

Barkley, R. A. (Ed.). (2015). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment (4th ed.).* New York: Guilford Press.

Beck A. T., Steer R. A., & Brown G. K. (1996). *Manual for the Beck Depression Inventory-II.* San Antonio (TX): The Psychological Corporation.

Blakemore, S. (2012). Imaging brain development: The adolescent brain. *NeuroImage, 61*(2), 397-406. doi:http://dx.doi.org/10.1016/j.neuroimage.2011.11.080

Biederman, J., Petty, C. R., Doyle, A. E., Spencer, T., Henderson, C. S., Marion, B., . . . Faraone, S. V. (2007). Stability of executive function deficits in girls with ADHD: A prospective longitudinal follow-up study into adolescence. *Developmental Neuropsychology, 33*, 44-61. doi:10.1080/8756564070172975510.1080/875656407017297552008-01304-003

Biederman, J., Monuteaux, M. C., Mick, E., Spencer, T., Wilens, T. E., Silva, J. M., . . . Faraone, S. V. (2006). Young adult outcome of attention deficit hyperactivity disorder: A controlled 10-year follow-up study. *Psychological Medicine, 36*(2), 167-179. doi:http://dx.doi.org/10.1017/S003329170500641

Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., . . . Faraone, S. V. (2004). Impact of executive function deficits and attention-deficit/hyperactivity disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology, 72*(5), 757-766. doi:http://dx.doi.org/10.1037/0022-006X.72.5.757

Bird, H. R., Shaffer, D., Fisher, P., & Gould, M. S. (1993). The Columbia Impairment Scale (CIS): Pilot findings on a measure of global impairment for children and adolescents. *International Journal of Methods in Psychiatric Research, 3*, 167–176.

Brown, T. E. (2013). *A new understanding of ADHD in children and adults: Executive function impairments.* New York: Routledge.

Bunge, S.A. & Wright, S.B. (2007). Neurodevelopmental changes in working memory and cognitive control. *Current Opinion in Neurobiology*, 17(2), 243-50.

Castellanos, F., Lee, P.P., Sharp, W., Jeffries, N., Greenstein, D. K., Clasen, L. S., ….Rapoport, J. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *Journal of the American Medical Association, 288(14)*, 1740-1748. doi:10.1001/jama.288.14.1740.

Castellanos, F. X., & Proal, E. (2012). Large-scale brain systems in ADHD: Beyond the prefrontal–striatal model. *Trends in Cognitive Sciences, 16*, 17-26.

Castellanos, F. X., Sonuga-Barke, E., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences, 10*(3), 117-123. doi:http://dx.doi.org/10.1016/j.tics.2006.01.011

Charach, A., Yeung, E., Climans, T., & Lillie, E. (2011). Childhood attention-deficit/hyperactivity disorder and future substance use disorders: Comparative meta-analyses. *Journal of the American Academy of Child & Adolescent Psychiatry, 50*(1), 9-21. doi:http://dx.doi.org/10.1016/j.jaac.2010.09.019

Claes, L., & Vandereycken, W. (2007). The Self-Injury Questionnaire-Treatment Related (SIQ-TR): Construction, reliability, and validity in a sample of female eating disorder patients. In P.M. Goldfarb (Ed.), *Psychological tests and testing research* trends (pp. 111-139). New York: Nova Science Publishers.

Conners, C. K. (1995). *Conners' Continuous Performance Test computer program: User's manual.* Toronto, Canada: Multi-Health Systems.

De Luca, C. R., Wood, S. J., Anderson, V., Buchanan, J., Proffitt, T. M., Mahony, K., & Pantelis, C. (2003). Normative data from the CANTAB. I: Development of executive function over the lifespan. *Journal of Clinical and Experimental Neuropsychology, 25*(2), 242-254. doi:http://dx.doi.org/10.1076/jcen.25.2.242.13639

Diamantopoulou, S., Rydell, A., Thorell, L. B., & Bohlin, G. (2007). Impact of executive functioning and symptoms of attention deficit hyperactivity disorder on children's peer relations and school performance. *Developmental Neuropsychology, 32*(1), 521-542. doi:http://dx.doi.org/10.1080/87565640701360981

El-Sayed, E., Larsson, J.-O., Persson, H., Santosh, P. and Rydelius, P.-A. (2003), “Maturational lag” hypothesis of attention deficit hyperactivity disorder: An update. *Acta Pædiatrica, 92,* 776–784. doi:10.1111/j.1651-2227.2003.tb02531.x

Faraone, S. V., & Biederman, J. (2004). Neurobiology of attention deficit/hyperactivity disorder. In D. S. Charney & E. J. Nestler (Eds.), .*Neurobiology of mental illness (2nd ed.)* New York: Oxford University Press.

Garson, D. (2013). *Hierarchical linear modeling guide and applications*. California: Thousand Oaks, CA: Sage.

Grassmann, V., Alves, M. V., Santos-Galduróz, R. F., & Galduróz, J.,Carlos Fernandes. (2017). Possible cognitive benefits of acute physical exercise in children with ADHD: A systematic review. *Journal of Attention Disorders, 21*(5), 367-371. doi:http://dx.doi.org/10.1177/1087054714526041

Helmes, E. (2000). Learning and memory. In G. Groth-Marnat (Ed.), *Neuropsychological assessment in clinical practice: A guide to test interpretation and integration* (pp. 293–334). Hoboken, NJ: Wiley

Hinshaw, S. P. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *Journal of Consulting and Clinical Psychology, 70,* 1086-1098.

Hinshaw, S. P., Carte, E. T., Fan, C., Jassy, J. S., & Owens, E. B. (2007). Neuropsychological functioning of girls with attention-deficit/hyperactivity disorder followed prospectively into adolescence: Evidence for continuing deficits? *Neuropsychology, 21*(2), 263-273. doi:http://dx.doi.org/10.1037/0894-4105.21.2.263

Hinshaw, S. P., Carte, E. T., Sami, N., Treuting, J. J., & Zupan, B. A. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification. *Journal of Consulting and Clinical Psychology, 70*, 1099-1111.

Hinshaw, S. P., Owens, E. B., Sami, N., & Fargeon, S. (2006). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence for continuing cross-domain impairment. *Journal of Consulting and Clinical Psychology, 74,* 489-499.

Hinshaw, S. P., Owens, E. B., Zalecki, C., Huggins, S. P., Montenegro-Nevado, A., Schrodek, E., & Swanson, E. N. (2012). Prospective follow-up of girls with attention-deficit hyperactivity disorder into young adulthood: Continuing impairment includes elevate risk for suicide attempts and self-injury. *Journal of Consulting and Clinical Psychology 80,* 1041-1051.

Hinshaw, S. P., & Scheffler, R. M. (2014). *The ADHD explosion: Myths, medication, money, and today's push for performance*. New York: Oxford University Press.

Hobbs, M. J., Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2007). EEG abnormalities in adolescent males with AD/HD. *Clinical Neurophysiology, 118*(2), 363-371. doi:http://dx.doi.org/10.1016/j.clinph.2006.10.013

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E. S., & Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. *Archives of Neurology, 47*(8), 919-926.

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E. S., Eliopulos, D., & Lyytinen, H. (1991). Corpus callosum morphology in attention deficit-hyperactivity disorder: Morphometric analysis of MRI. *Journal of Learning Disabilities, 24*(3), 141-146

Julian, L. J., & Mohr, D. C. (2006). Cognitive predictors of response to treatment for depression in multiple sclerosis. *The Journal of Neuropsychiatry and Clinical Neurosciences, 18*(3), 356-363. doi:http://dx.doi.org/10.1176/appi.neuropsych.18.3.356

Jurado, M. B., & Rosselli, M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychology Review, 17*(3), 213-233. doi:http://dx.doi.org/10.1007/s11065-007-9040-z

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O., . . . Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the united states: Results from the national comorbidity survey replication. *American Journal of Psychiatry, 163(4),* 716-723. doi:http://dx.doi.org/10.1176/appi.ajp.163.4.716

Krain, A. L., & Castellanos, F. X. (2006). Brain development and ADHD. *Clinical Psychology Review, 26(4),* 433-444. doi:http://dx.doi.org/10.1016/j.cpr.2006.01.005

Lou, H. C., Henriksen, L., Bruhn, P., Børner, H., & Nielsen, J. B. (1989). Striatal dysfunction in attention deficit and hyperkinetic disorder. *Archives of Neurology, 46*(1), 48-52.

Mahone, E. M., & Wodka, E. L. (2008). The neurobiological profile of girls with ADHD. *Developmental Disabilities Research Reviews, 14*(4), 276-284. doi:http://dx.doi.org/10.1002/ddrr.41

Meinzer, M. C., Lewinsohn, P. M., Pettit, J. W., Seeley, J. R., Gau, J. M., Chronis-Tuscano, A., & Waxmonsky, J. G. (2013). Attention–deficit/hyperactivity disorder in adolescence predicts onset of major depressive disorder through early adulthood. *Depression and Anxiety, 30*(6), 546-553. doi:http://dx.doi.org/10.1002/da.22082

Meza, J. I., Belendiuk, K., Loera, A., Teh, S. R., Valentine, R., & Hinshaw, S. P. (2017). A meta-analytic review of attention-deficit/hyperactivity disorder and response inhibition deficits: Implications for clinical assessment. Submitted to *Clinical Psychology Review*.

Miller, M., Ho, J., & Hinshaw, S. P. (2012). Executive functions in girls with ADHD followed prospectively into young adulthood. *Neuropsychology, 26*(3), 278-287. doi:http://dx.doi.org/10.1037/a0027792

Miller, M., Loya, F., & Hinshaw, S. P. (2013). Executive functions in girls with and without childhood ADHD: Developmental trajectories and associations with symptom change. *Journal of Child Psychology and Psychiatry, 54,* 1005-1015.

Miller, M., Nevado-Montenegro, A., & Hinshaw, S. P. (2012). Childhood executive function continues to predict outcomes in young adult females with and without childhood-diagnosed ADHD. *Journal of Abnormal Child Psychology, 40*(5), 657-668. doi:http://dx.doi.org/10.1007/s10802-011-9599-y

Nigg, J. T., Hinshaw, S. P., Carte, E. T., & Treuting, J. J. (1998). Neuropsychological correlates of childhood attention-deficit/hyperactivity disorder: Explainable by comorbid disruptive behavior or reading problems? *Journal of Abnormal Psychology*,107, 468–480.

Nigg, J. T., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. (2005). Causal heterogeneity in attention-deficit/ hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry, 57*(11), 1224-1230. doi:http://dx.doi.org/10.1016/j.biopsych.2004.08.025

Nock, M. (2012). Future directions for the study of suicide and self-injury. *Journal of Clinical Child and Adolescent Psychology, 41,* 255-259.

Osterrieth, P. A. (1944). Le test de copie d'une figure complex [A test of copying a complex figure]. *Archives de Psychologie, 30,* 206-356.

Patros, C. H., Alderson, R. M., Kasper, L. J., Tarle, S. J., Lea, S. E., & Hudec, K. L. (2015). Choice-impulsivity in children and adolescents with attention-deficit/hyperactivity disorder (ADHD): a meta-analytic review. *Clinical Psychology Review, 43,* 162-74

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology & Psychiatry, 37*(1), 51-87.

Randall, L., & Tyldesley, K. (2016). Evaluating the impact of working memory training programmes on children—A systematic review. *Educational and Child Psychology, 33*(1), 34-50.

Rinsky, J. R., & Hinshaw, S. P. (2011). Linkages between childhood executive functioning and adolescent social functioning and psychopathology in girls with ADHD. *Child Neuropsychology, 17*(4), 368-390. doi:http://dx.doi.org/10.1080/09297049.2010.544649

Rourke, B. P., & Orr, R. R. (1977). Prediction of the reading and spelling performances of normal and retarded readers: A four-year follow-up. *Journal of Abnormal Child Psychology, 5*(1), 9.

Rubia, K. (2010). "Cool" inferior frontostriatal dysfunction in attention-deficit/hyperactivity disorder versus "hot" ventromedial orbitofrontal-limbic dysfunction in conduct disorder: A review. *Biological Psychiatry, 69,* e69-e87.

Sami, N., Carte, E. T., Hinshaw, S. P., & Zupan, B. A. (2004). Performance of girls with ADHD and comparison girls on the Rey-Osterrieth Complex Figure: Evidence for executive processing deficits. *Child Neuropsychology, 9*, 237-254.

Seidman, L. J. (2006). Neuropsychological functioning in people with ADHD across the lifespan. *Clinical Psychology Review, 26*(4), 466-485. doi:http://dx.doi.org/10.1016/j.cpr.2006.01.004

Seidman, L. J., Biederman, J., Monuteaux, M. C., Valera, E., Doyle, A. E., & Faraone, S. V. (2005). Impact of gender and age on executive functioning: Do girls and boys with and without attention deficit hyperactivity disorder differ neuropsychologically in preteen and teenage years? *Developmental Neuropsychology, 27*(1), 79-105. doi:http://dx.doi.org/10.1207/s15326942dn2701_4

Seidman, L. J., Biederman, J., Weber, W., Hatch, M., & Faraone, S. V. (1998). Neuropsychological function in adults with attention-deficit hyperactivity disorder. *Biological Psychiatry, 44*(4), 260-268.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. (2000). NIMH diagnostic interview schedule for children version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(1), 28-38. doi:http://dx.doi.org/10.1097/00004583-200001000-00014

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., . . . Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-Deficit/Hyperactivity disorder. Archives of General Psychiatry, 63(5), 540-549. doi:http://dx.doi.org/10.1001/archpsyc.63.5.540

Shaw, P., Malek, M., Watson, B., Greenstein, D., de Rossi, P., & Sharp, W. (2013). Trajectories of cerebral cortical development in childhood and adolescence and adult attention-deficit/hyperactivity disorder. *Biological Psychiatry, 74*(8), 599-606. doi:http://dx.doi.org/10.1016/j.biopsych.2013.04.007

Skegg, K. (2005). Self-harm. *The Lancet*, *366*, 1471–1483.

Solanto, M. V., Marks, D. J., Mitchell, K. J., Wasserstein, J., & Kofman, M. D. (2008). Development of a new psychosocial treatment for adult ADHD. *Journal of Attention Disorders, 11*(6), 728-736. doi:http://dx.doi.org/10.1177/1087054707305100

Sonuga-Barke, E. (2002). Psychological heterogeneity in AD/HD--A dual pathway model of behaviour and cognition. *Behavioural & Brain Research, 130*(1-2), 29-36. doi:http://dx.doi.org/10.1016/S0166-4328(01)00432-6

Sripada, C. S., Kessler, D., & Angstadt, M. (2014). Lag in maturation of the brain's intrinsic functional architecture in attention-deficit/hyperactivity disorder. *Proceedings of the National Academy of Sciences,111*(39), 14259-14264. doi:http://dx.doi.org/10.1073/pnas.1407787111

Swanson, J. M. (1992). *Assessment and treatment of ADD students*. Irvine, CA: K.C. Press.

Taylor, E. W., & Keltner, N. L. (2002). Messy purse girls: Adult females and ADHD. *Perspectives in Psychiatric Care, 38*(2), 69-72. doi:http://dx.doi.org/10.1111/j.1744-6163.2002.tb00659.x

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children (3rd. ed.).* San Antonio, TX: Psychological Corporation.

Wechsler, D. (1992). *Wechsler Individual Achievement Test*. San Antonio, TX: Psychological Corporation.

What Works Clearinghouse. (2008). *Procedures and standards handbook (version 2.1*). Retrieved from http://ies.ed.gov/ncee/wwc/pdf/reference_resources/wwc_procedures_v2_1_standards_handbook.pdf

Wilcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-Deficit/Hyperactivity disorder: A meta-analytic review. *Biological Psychiatry, 57*(11), 1336-1346. doi:http://dx.doi.org/10.1016/j.biopsych.2005.02.006

Zametkin, A. J., Nordahl, T. E., Gross, M., King, A. C, Semple, W. E., … Cohen R. M. (1990) Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *New England Journal of Medicine, 323(20),* 1361-1366.

Ziereis, S., & Jansen, P. (2015). Effects of physical activity on executive function and motor performance in children with ADHD. *Research in Developmental Disabilities, 38,* 181-191. doi:http://dx.doi.org/10.1016/j.ridd.2014.12.00

Table 1

Demographic Variables

	ADHD[a]		**Comparison**[b]		**T-test**	**Cohen's d**
	M	*SD*	*M*	*SD*	*t* (*df*)	
Demographic Variables						
Age (in years)						
Wave 1	9.64	.14	9.43	.18	-0.89 (226)	-.11
Wave 2	14.26	.15	13.84	.18	-1.79 (207)	-.25
Wave 3	19.62	.15	19.45	.19	-0.74 (215)	-.10
Wave 4	25.64	.16	25.42	.20	-0.85 (209)	-.11
Ethnicity (%)						
European American	60.8		46.9			
African American	22.5		27.2			
Hispanic American	11.7		11.1			
Asian American[c]	4.2		14.8			
Total Annual Family Income[d]	6.47	2.59	6.81	2.37	-.94 (197)	-.14
<$10,000 (%)	2.5		5.8			
$10,001 to $20,000	3.7		2.5			
$20,001 to $30,000	6.2		7.6			
$30,001 to $40,000	7.4		6.8			
$40,001 to $50,000	7.4		14.4			
$50,001 to $60,000	12.3		7.6			
$60,001 to $70,000	12.3		11			
$70,001 to $75,000	7.4		6.8			
>$75,000	40.7		36.4			
Maternal Education[e]	4.76	.88	4.98	.95	-1.66 (199)	-.24
Less than 8th grade (%)	0.0		0.0			
Some high school	1.2		0.0			
High school graduate	2.5		4.2			
Some College	30.9		40.8			
College graduate	28.4		30.0			
Advanced or prof. degree	37.0		25.0			

[a] For Wave 1, n=140. For Wave 2, n=127. For Wave 3, n = 131. For Wave 4, n= 126

[b] For Wave 1, n=88. For Wave 2, n=82. For Wave 3, n = 86. For Wave 4, n= 85

[c] There was a significantly higher percentage of Asian American girls in the comparison group: $\chi^2(4)$, $N = 201) = 9.298$, $p < .05$

[d] For total annual family income, 1 <$10,000; 9 >$75,000.

[e] For maternal education, 1 = less than 8th grade; 6 = advanced or professional degree

$*p < .05$. $**p < .01$. $***p < .001$.

Table 2

Summary of Correlations for EF Measures and Emerging Adult Outcomes

Measures	1	2	3	4	5	6	7
1. W1 ROCF	--						
2. W1 CPT	.15*	--					
3. W1 Digit Span	-.44**	-.08	--				
4. W1 CUL	-.41**	-.13	.44**	--			
5. W2 ROCF	.48**	-.00	-.20**	-.32**	--		
6. W2 CPT	.27**	.35**	-.16*	-.21**	.25**	--	
7. W2 Digit Span	-.28**	-.04	.66**	.27**	-.19**	-.14	--
8. W3 ROCF	.40**	.07	-.18*	-.15*	.46**	.12	-.18*
9. W3 CPT	.30**	.24**	-.14	-.13	.16	.51**	-.09
10. W3 Digit Span	-.27**	-.09	.55**	.24**	-.22**	-.15	.66**
11. W3 CUL	-.32**	.04	.23**	.45**	-.34**	-.16*	.28**
12. W4 ROCF	.29**	-.04	-.12	-.03	.21**	.18*	-.15*
13. W4 CPT	.20**	.31**	-.11	-.15*	.15*	.49**	-.22**
14. W4 Digit Span	-.27**	-.04	.54**	.27**	-.32**	-.18*	.67**
15. W4 CUL	-.33**	-.01	.20**	.45**	-.21**	-.15*	.33**
16. Self-Reported ASR Ext	.25**	.21**	-.12	-.18**	.24	.08	-.13
17. Self-Reported ASR Int	.15*	.09	-.04	-.21**	.10	.04	-.07
18. Self-reported BDI	-.13	.06	-.02	-.15*	.12	.06	-.07
19. Parent Reported ABCL Ext	.24**	17*	-.16*	-.14	.23**	.16*	-.28**
20. Parent Reported ABCL Int	.25**	.14*	-.12	-.22**	.19*	.14	-.23**
21. WIAT Reading	-.23**	-.03	.37**	.25**	-.22**	-.01	.43**
22. WIAT Math	-.40**	-.02	.42**	.30**	-.40**	-.13	.54**

*p < .05, **p < .01.

Table 3

Summary of Correlations for EF Measures and Emerging Adult Outcomes (Continued)

Measures	8	9	10	11	12	13	14
1. W1 ROCF							
2. W1 CPT							
3. W1 Digit Span							
4. W1 CUL							
5. W2 ROCF							
6. W2 CPT							
7. W2 Digit Span							
8. W3 ROCF	--						
9. W3 CPT	-.11	--					
10. W3 Digit Span	-.23**	-.14	--				
11. W3 CUL	-.31**	-.29**	.28**	--			
12. W4 ROCF	.52**	.23**	-.18*	-.32**	--		
13. W4 CPT	-12	.50**	-.22**	-.20**	.14	--	
14. W4 Digit Span	-.27**	-.07	.73**	.38**	-.21**	-.15*	--
15. W4 CUL	-.40**	-.21*	.29**	.76**	-.39**	-.20**	.38**
16. ASR Ext	.21**	.16	-.07	-.07	-.11	-.17*	-.04
17. ASR Int	.10	.12	-.14*	-.14*	-.01	.10	-.02
18. Self-reported BDI	.10	.19*	-.11	-.11	.08	.10	-.05
19. ABCL Ext	.22**	.21*	-.26**	-.22**	.24**	.17	-.24**
20. ABCL Int	.16*	.24**	-.31**	-.20**	-.19*	.11	-.23**
21. WIAT Reading	-.28**	-.18*	.48**	.45**	-.17*	-.18*	.46**
22. WIAT Math	.34**	-.18*	.49**	.43**	-.34**	-.23**	.52**

__*p < .05, **p < .01.__

Table 4

Summary of Correlations for EF Measures and Emerging Adult Outcomes (Continued)

Measures	15	16	17	18	19	20	21
1. W1 ROCF							
2. W1 CPT							
3. W1 Digit Span							
4. W1 CUL							
5. W2 ROCF							
6. W2 CPT							
7. W2 Digit Span							
8. W3 ROCF							
9. W3 CPT							
10. W3 Digit Span							
11. W3 CUL							
12. W4 ROCF							
13. W4 CPT							
14. W4 Digit Span							
15. W4 CUL	--						
16. ASR Ext	-.08	--					
17. ASR Int	-.06	.67**	--				
18. Self-reported BDI	-.10	.58**	.70**	--			
19. ABCL Ext	-.28**	.52**	.36**	.45**	--		
20. ABCL Int	-.24**	.37**	.46**	.45**	.77**	--	
21. WIAT Reading	.44**	-.11	-.10	-.23**	-.32**	-.33**	--
22. WIAT Math	.46**	-.18	-.08	-.18*	-.41**	-.34**	.55**

***p < .05, **p < .01.**

Table 5

Group Comparisons of Executive Functioning Across All Waves

	ADHD (n= 140)		**Comparison (n= 88)**		**T-test**		**Cohen's D**
	M	*SD*	*M*	*SD*	*t* (*df*)	*p*	
EF Measures							
W1 CPT Commission Errors (%)	56.41	20.99	53.33	20.76	-1.06 (217)	*ns*	-.15
W1 ROCF Error Proportion	.35	.19	.22	.15	-5.46 (222)	<.001*	-0.76
W1 WISC Digit Span (Raw Score)	11.83	2.99	13.58	3.02	4.04 (195)	. <.001*	.58
W1 CUL	17.18	6.32	18.87	5.35	2.06 (222)	.04*	.28
W2 CPT Commission Errors (%)	44.39	23.95	35.97	18.69	-2.59 (184)	.01*	-0.36
W2 ROCF Error Proportion	.23	.12	.17	.09	-4.04 (194)	<.001*	-.56
W2 WISC Digit Span (Raw Score)	13.68	3.53	16.14	3.36	4.93 (198)	<.001*	.71
W3 CPT Commission Errors (%)	37.23	17.90	28.17	19.09	-3.03 (151)	.002*	-.49
W3 ROCF Error Proportion	.21	.10	.15	.08	-4.65 (206)	<.001*	-.66
W3 WISC Digit Span (Raw Score)	15.22	3.57	17.82	3.24	5.35 (205)	<.001*	.76
W3 CUL	25.68	7.90	30.35	5.98	4.54 (201)	<.001*	
W4 CPT Commission Errors (%)	40.13	20.24	29.85	18.14	-3.68 (198)	. <.001*	-.53
W4 ROCF Error Proportion	.24	.13	.16	.09	-4.52 (195)	<.001*	-.71
W4 WISC Digit Span (Raw Score)	15.35	3.41	17.71	3.45	4.87 (206)	<.001*	.69
W4 CUL	27.52	7.60	33.30	6.47	5.61 (200)	<.001*	.79

*Significant at $p<.05$ after Benjamini-Hochberg false discovery rate correction (taking into account all t-tests performed).

Table 6

Group Comparisons of Behavioral and Academic Achievement in Emerging Adulthood

	ADHD (n= 140)		**Comparison (n= 88)**		**T-test**		**Cohen's D**
	M	*SD*	*M*	*SD*	*t* *(df)*	*p*	
Emerging Adult Outcomes							
Mother-reported Internalizing	58.40	13.13	45.67	10.78	-7.08 (187)	<.001*	-1.06
Self-reported Internalizing	21.23	12.84	17.99	13.46	-1.75 (205)	.08	-.24
Mother-reported Externalizing	58.81	10.07	46.20	7.94	-3.24 (187)	<.001*	-1.39
Self-reported Externalizing	16.23	10.53	11.61	9.42	-9.28 (205)	.001*	-.46
Self-Reported Depression	9.51	.75	6.33	.87	-5.47 (206)	.01*	-0.76
WIAT Reading Comp	92.90	15.12	105.32	8.37	6.88 (205)	<.001*	1.02
WIAT Math Fluency	85.20	17.25	101.93	15.75	7.09 (203)	<.001*	1.01

*Significant at *p*<.05 after Benjamini-Hochberg false discovery rate correction (taking into account all t-tests performed).

Table 7

Growth Curve Models of ROCF/TCFT Error Proportion

	Model 1		**Model 2**		**Model 3**		**Model 4**	
	Est	(SE)	Est	(SE)	Est	(SE)	Est	(SE)
Fixed Part								
Intercept	.32	(.02) ***	.64	(.03)***	.59	(.03)***	.56	(.04)***
Age	-.01	(.001)***	-.05	(.003)***	-.05	(.003)***	-.05	(.003)***
Age^2			.001	(.001)***	.001	(.001)***	.001	(.001)***
ADHD					.08	(.01)***	.14	(.03)***
ADHD x Age							-.003	(.001)*
Random Part								
Between level								
$\sqrt{\boldsymbol{\psi 11}}$	.18		.17		.16		.16	
$\sqrt{\boldsymbol{\psi 22}}$ (Age)	.01		.01		.01		.01	
ρ_{12}	-.93		-.90		-.91		-.91	
Within level								
$\sqrt{\boldsymbol{\theta}}$	.10		.09		.09		.09	
Log likelihood	526.96		590.81		611.01		613.23	

*p < .05. **p < .01. ***p<.001.

Table 8

Growth Curve Models of CPT Commission Error %

	Model 1		**Model 2**		**Model 3**[a]		**Model 4**	
	Est	(SE)	Est	(SE)	Est	(SE)	Est	(SE)
Fixed Part								
Intercept	63.00	(1.92)***	95.5	(4.97)***	90.18	(5.14)***	92.88	(5.44)***
Age	-1.21	(.10)***	-5.48	(.61)***	-5.45	(.61)***	-5.59	(.62)***
Age^2			.12	(.02)***	.12	(.02)***	.12	(.02)***
ADHD					8.49	(2.05)***	3.35	(3.92)
ADHD x Age							.31	(.20)
Random Part								
Between level								
$\sqrt{\boldsymbol{\psi 11}}$	11.52		14.97		15.29		15.06	
$\sqrt{\boldsymbol{\psi 22}}$ (Age)	.07		.54		.56		.54	
ρ_{12}	.99		-.52		-.60		-.58	
Within level								
$\sqrt{\boldsymbol{\theta}}$	16.77		15.64		15.60		15.61	
Log likelihood	-3331.73		-3308.41		-3300.20		-3299.02	

***p < .05. **p < .01. ***p<.001.**

[a] Final model retained for follow-up analyses

Table 9

Growth Curve Models of Digit Span Raw Scores

	Model 1		**Model 2**		**Model 3[a]**		**Model 4**	
	Est	(SE)	Est	(SE)	Est	(SE)	Est	(SE)
Fixed Part								
Intercept	10.81	(.28)***	4.83	(.61)***	6.08	(.64)***	6.02	(.66)***
Age	.24	(.01)***	1.01	(.07)***	1.03	(.07)***	1.03	(.07)***
Age^2			-.02	(.002)***	-.02	(.002)***	-.02	(.002)***
ADHD					-2.26	(.36)***	-2.14	(.50)***
ADHD x Age							-.008	(.03)
Random Part								
Between level								
$\sqrt{\boldsymbol{\psi 11}}$	2.20		2.16		1.83		1.83	
$\sqrt{\boldsymbol{\psi 22}}$ (Age)	.07		.07		.07		.07	
ρ_{12}	.21		.25		.31		.31	
Within level								
$\sqrt{\boldsymbol{\theta}}$	2.15		1.97		1.97		1.97	
Log likelihood	-1999.66		-1947.27		-1929.29		-1929.23	

***p < .05. **p < .01. ***p<.001.**

[a] Final model retained for follow-up analyses

Table 10

Growth Curve Models of CUL Raw Scores

	Model 1		**Model 2**[a]		**Model 3**	
	Est	(SE)	Est	(SE)	Est	(SE)
Fixed Part						
Intercept	10.73	(.23)***	12.53	(.68)***	10.62	(.84)***
Age	.79	(.03)***	.79	(.03)***	.93	(.65)***
ADHD			-2.95	(.68)***	.23	(1.08)
ADHD x Age					-.24	(.06)***
Random Part						
Between level						
$\sqrt{\boldsymbol{\psi 11}}$	2.69		2.96		2.65	
$\sqrt{\boldsymbol{\psi 22}}$ (Age)	.29		.28		.26	
ρ_{12}	-.22		-.35		-.19	
Within level						
$\sqrt{\boldsymbol{\theta}}$	4.34		4.36		4.35	
Log likelihood	-2018.90		-2010.47		-2003.323	

***p < .05. **p < .01. ***p<.001.**

[a] A quadratic model was not calculated for this variable

Table 11

EF Trajectory Predicting Emerging Adult Outcome Variables

	Self-Reported Internalizing Symptoms		**Self-Reported Externalizing Symptoms**		**Parent Reported Internalizing Symptoms**		**Parent Reported Externalizing Symptoms**		**Self-Reported Depressive Symptoms**		**WIAT Reading**		**WIAT Math**	
	β	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*
Step 1														
ADHD Status	.09	*ns*	.19	.001*	.45	<.001*	.56	<.001*	.16	.02	-.40	<.001*	-.42	<.001*
Family Income	-.17	.02	-.18	.01*	-.15	.03	-.13	.05	-.21	.003*	.27	<.001*	.12	*ns*
Maternal Educ	-.03	*ns*	-.03	*ns*	-.01	*ns*	.06	*ns*	-.05	*ns*	.05	*ns*	.03	*ns*
Step 2[a]														
ROCF Slope	-.10	*ns*	-.13	.06	-.04	*ns*	-.02	*ns*	-.02	*ns*	-.01	*ns*	.08	*ns*
CPT Slope	-.01	*ns*	-.06	*ns*	-.05	*ns*	-.07	*ns*	.004	*ns*	-.06	*ns*	-.11	*ns*
Digit Span Slope	.06	*ns*	.17	.01*	-.002	*ns*	.03	*ns*	.05	*ns*	.02	*ns*	.05	*ns*
CUL Slope	.14	*ns*	.15	.04	.05	*ns*	-.01	*ns*	.08	*ns*	.15	.02	.12	*ns*
Step 3[a]														
ROCF Slope X ADHD	-.08	*ns*	-.12	*ns*	-.10	*ns*	-.03	*ns*	-.12	*ns*	-.02	*ns*	-.03	*ns*
CPT Slope X ADHD	.02	*ns*	.14	*ns*	-.03	*ns*	.03	*ns*	.05	*ns*	-.10	*ns*	.07	*ns*
Digit Span Slope X ADHD	.07	*ns*	.15	*ns*	.16	*ns*	-.05	*ns*	.15	*ns*	-.08	*ns*	-.10	*ns*
CUL Slope X ADHD	.18	*ns*	.05	*ns*	.06	*ns*	-.13	*ns*	.23	*ns*	.11	*ns*	.14	*ns*

*Significant at $p<.05$ after Benjamini-Hochberg false discovery rate correction for each outcome variable.

[a] Each predictor at Step 2 and their representative two-way interactions at Step 3 were entered as separate models.

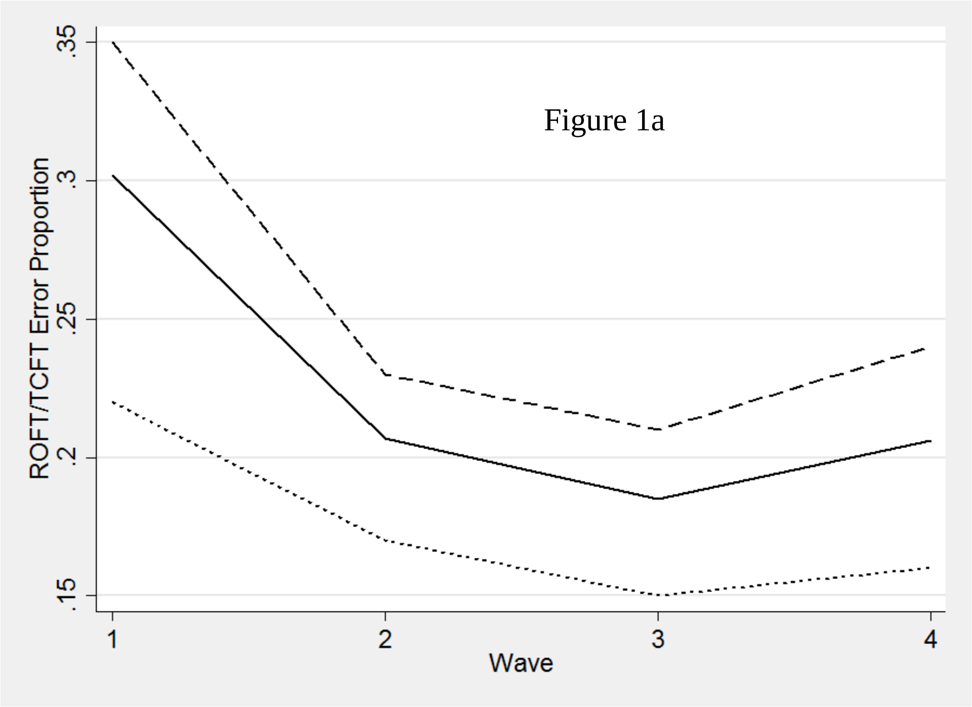
Table 12

Concurrent EFs Predicting Emerging Adult Outcome Variables

	Self-Reported Internalizing Symptoms		**Self-Reported Externalizing Symptoms**		**Parent Reported Internalizing Symptoms**		**Parent Reported Externalizing Symptoms**		**Self-Reported Depressive Symptoms**		**WIAT Reading**		**WIAT Math**	
	β	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*
Step 1														
ADHD Status	.10	*ns*	.20	.003*	.45	<.001*	.56	<.001*	.17	.02	-.40	<.001*	-.43	<.001*
Family Income	-.17	.02	-.17	.01*	-.15	.03	-.13	*ns*	-.21	.003*	.27	<.001*	.12	*ns*
Maternal Educ	-.04	*ns*	-.04	*ns*	-.01	*ns*	.06	*ns*	-.06	*ns*	.05	*ns*	.03	*ns*
Wave 4 Age	-.16	.02	-.19	.004*	-.09	*ns*	-.03	*ns*	-.16	.02	.03	*ns*	.02	*ns*
Step 2^a														
ROCF	-.05	*ns*	.05	*ns*	.05	*ns*	.08	*ns*	.03	*ns*	-.03	*ns*	-.22	.001*
CPT	.08	*ns*	.12	*ns*	-.01	*ns*	.03	*ns*	.06	*ns*	-.07	*ns*	-.13	*ns*
Digit Span	.06	*ns*	.07	*ns*	-.04	*ns*	-.04	*ns*	.04	*ns*	.33	<.001*	.41	<.001*
CUL	-.01	*ns*	.01	*ns*	-.07	*ns*	-.07	*ns*	-.02	*ns*	.32	<.001*	.34	<.001*
Step 3^a														
ROCF X ADHD	-.11	*ns*	-.11	*ns*	.30	*ns*	.17	*ns*	-.10	*ns*	.05	*ns*	.02	*ns*
CPT X ADHD	.29	*ns*	.08	*ns*	-.15	*ns*	-.09	*ns*	.04	*ns*	-.32	*ns*	.21	*ns*
Digit Span X ADHD	.08	*ns*	.09	*ns*	.12	*ns*	-.25	*ns*	-.02	*ns*	.78	.003*	.28	*ns*
CUL X ADHD	.24	*ns*	.08	*ns*	.02	*ns*	-.04	*ns*	.36	*ns*	.36	*ns*	.38	*ns*

*Significant at $p<.05$ after Benjamini-Hochberg false discovery rate correction for each outcome variable.

[a] Each predictor at Step 2 and their representative two-way interactions at Step 3 were entered as separate models.



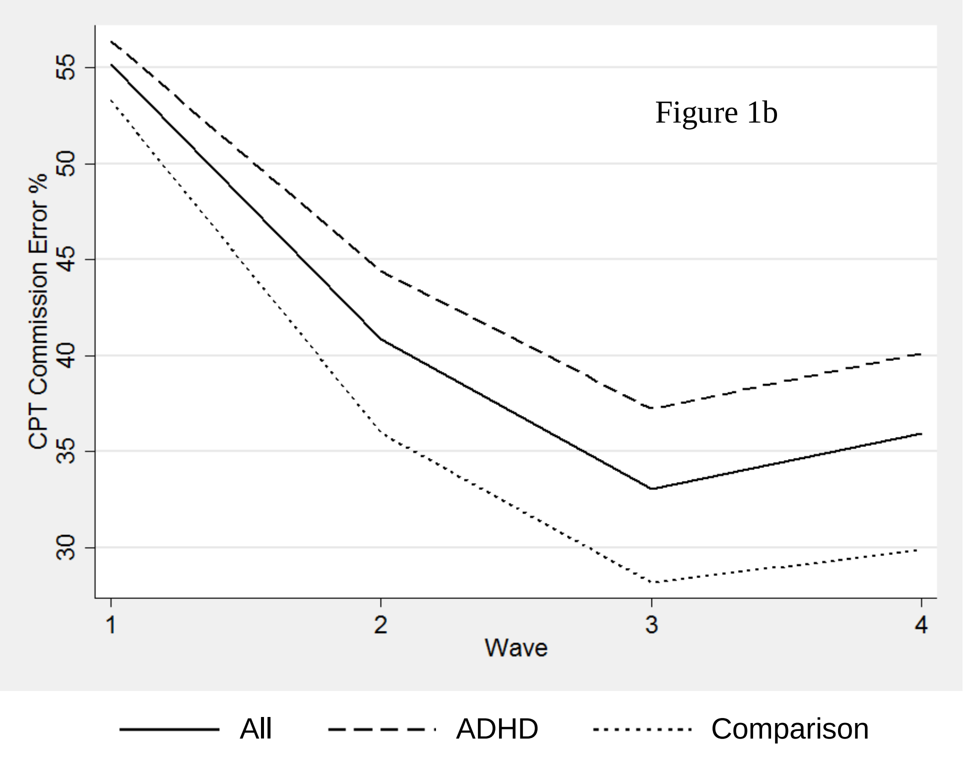
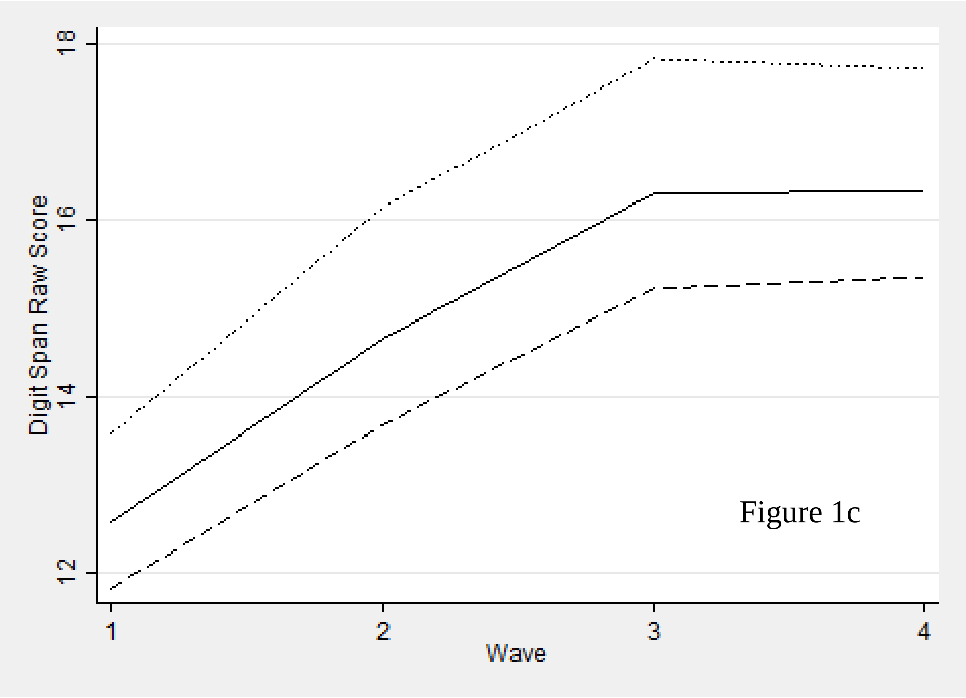


Figure 1a. Change in observed means of ROCF/TCFT error proportion across waves

Figure 1b. Change in observed means of CPT % error across waves



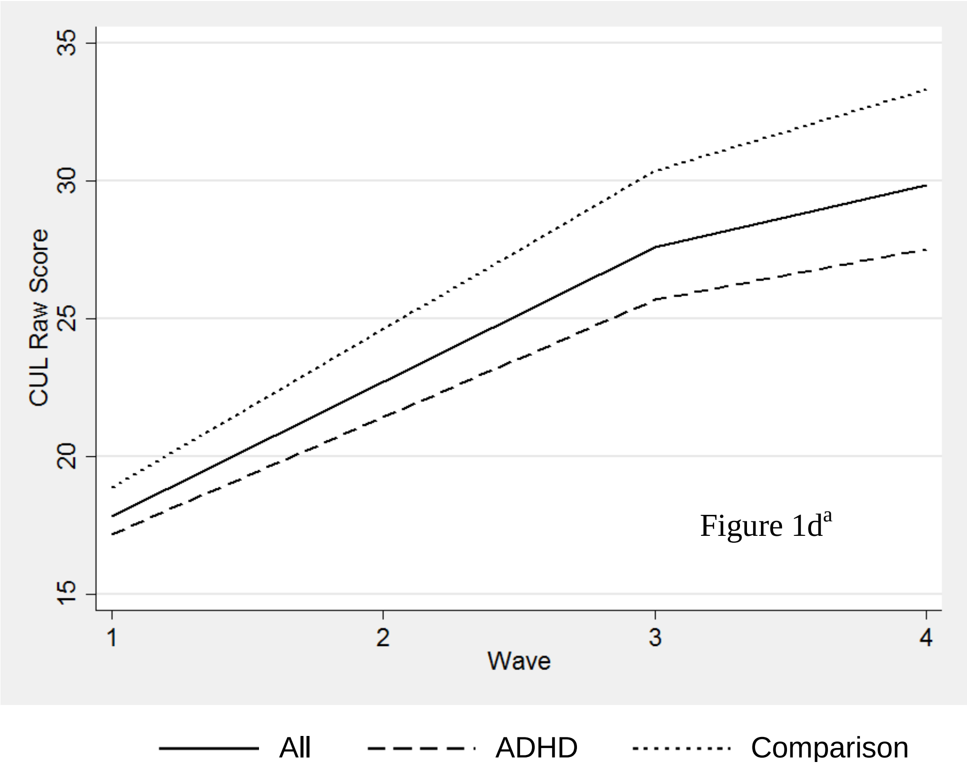


Figure 1c. Change in observed means of raw digit span scores across waves

Figure 1d. Change in observed means of raw CUL scores across waves (Note: Wave 2 data were not included for this variable)

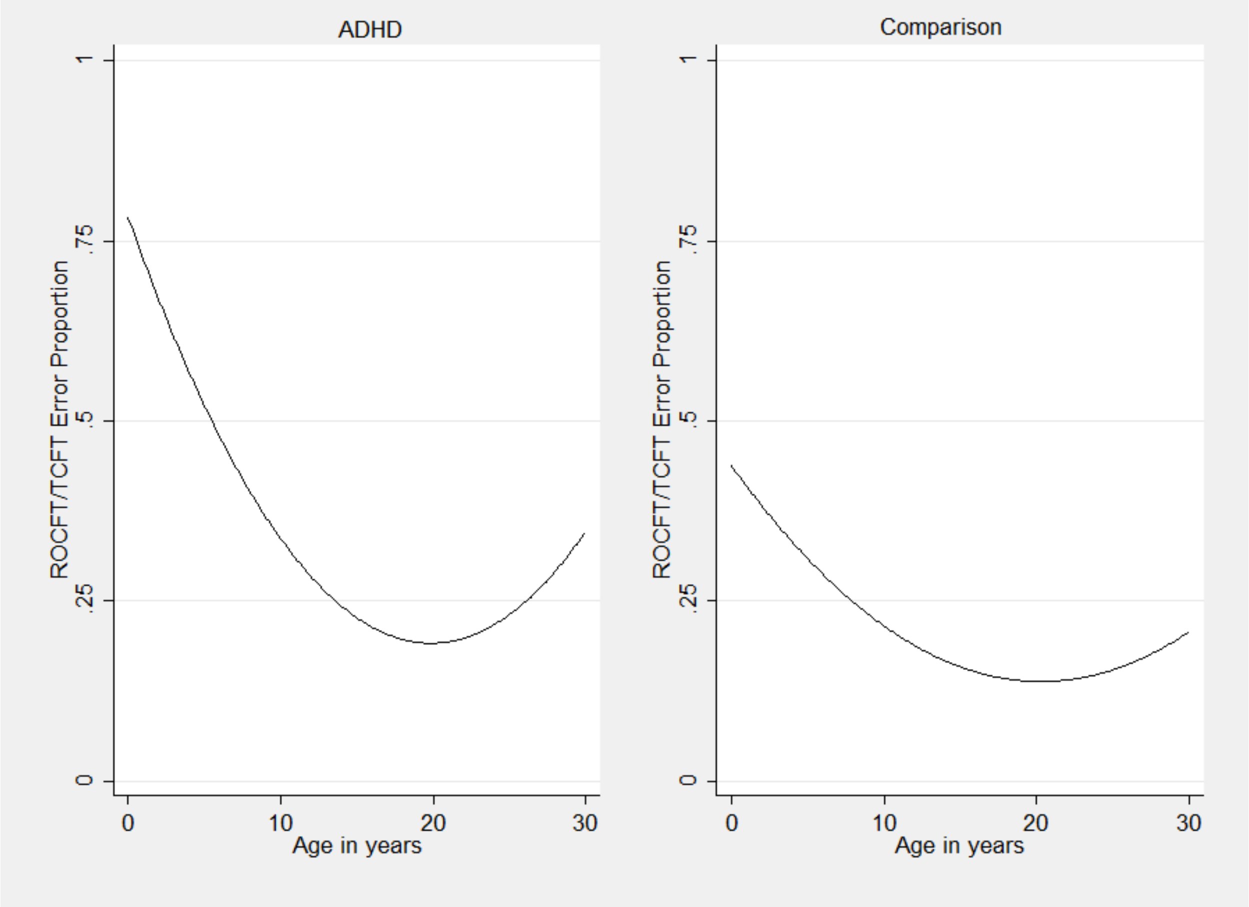


Figure 2. Predicted Trajectory of ROCF/TCFT error proportion scores across age for ADHD and Comparison Women

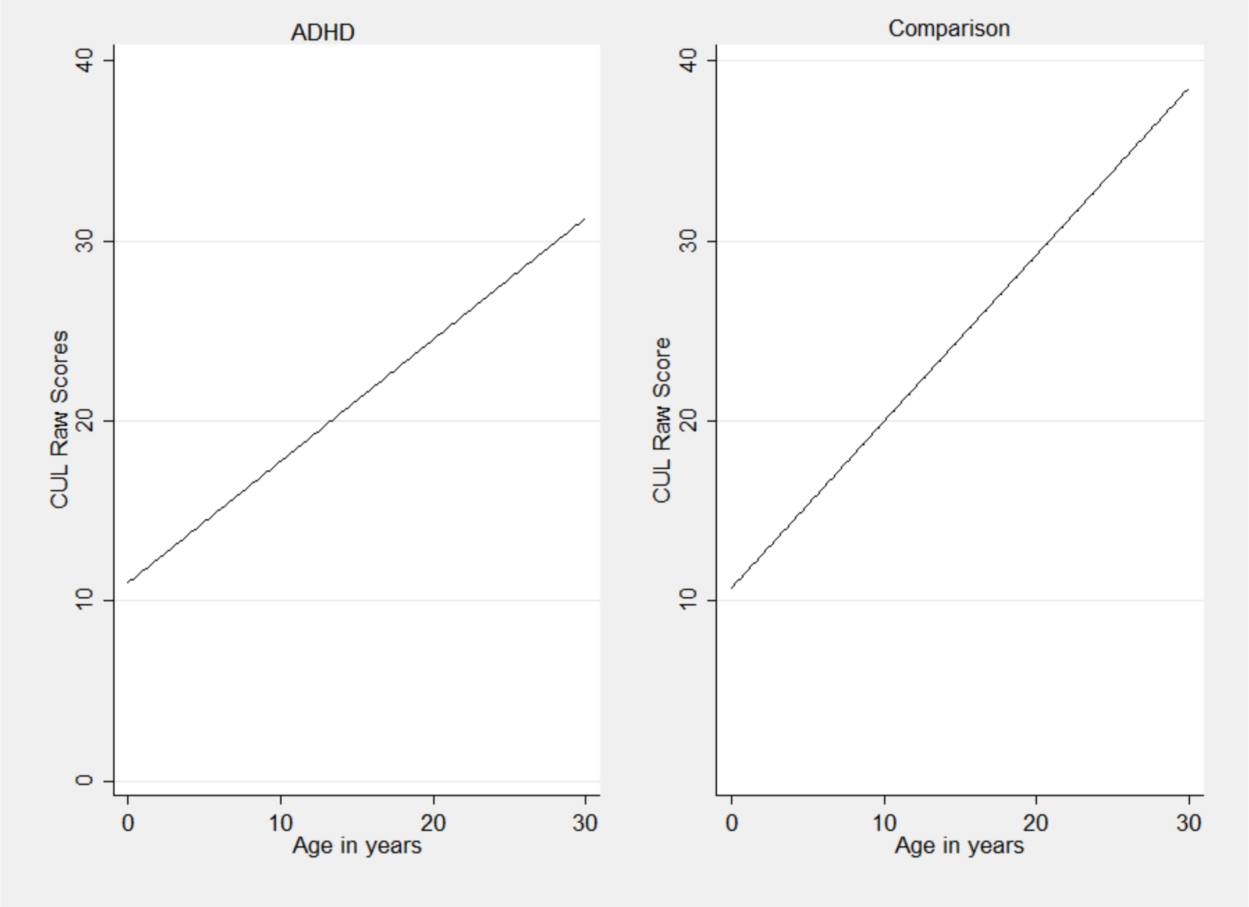


Figure 3. *Predicted Trajectory of CUL scores across age for ADHD and Comparison Women*

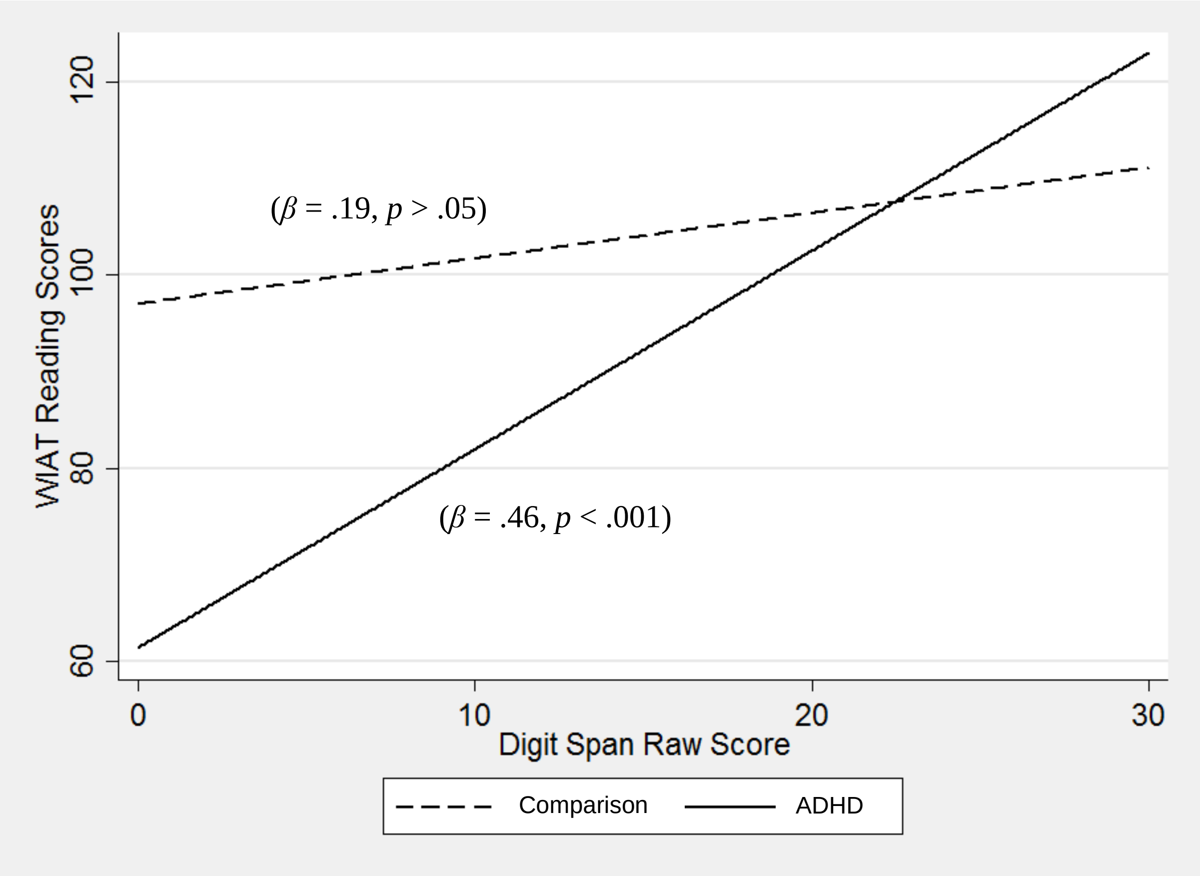


Figure 4. Estimated effect of diagnostic status on the association between digit span performance and emerging adult reading scores.