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Examining Relationships Between Executive Functioning and Delay Aversion in Attention Deficit Hyperactivity Disorder

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Abstract

Although motivation and cognition are often examined separately, recent theory suggests that a delay-averse motivational style may negatively impact development of executive functions (EFs), such as working memory (WM) and response inhibition (RI) for children with Attention Deficit Hyperactivity Disorder (ADHD; Sonuga-Barke, 2002). This model predicts that performance on delay aversion and EF tasks should be correlated for school-age children with ADHD. However, tests of these relationships remain sparse. Forty-five children ages 8 to 12 with ADHD and 46 non-ADHD controls completed tasks measuring EFs and delay aversion. Children with ADHD had poorer WM and RI than non-ADHD controls, as well as nonsignificantly worse delay aversion. Consistent with previous research, RI was not related to delay aversion. However, delay aversion did predict WM scores for children with and without ADHD. Implications for the dual-pathway hypothesis and future research on cognitive and motivational processing in ADHD are discussed.

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common disorders of childhood, with prevalence rates in large-scale epidemiological studies ranging from 2% to 7% (American Psychiatric Association, 2000). The core behavioral symptoms of the disorder include difficulties with sustained attention, hyperactivity, and impulsivity, and many children with ADHD also experience impairment in academic performance, peer relationships, and parent–child/teacher–child relationships (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Biederman et al., 2004; Dumas, 1998; Erhardt & Hinshaw, 1994; Greene, Beszterczey, Katenstein, Park, & Goring, 2002; Schachar, Taylor, Wieselberg, Thorley, & Rutter, 1987).

A variety of possible underlying mechanisms have been put forward to explain both the behavioral symptoms and functional impairments associated with ADHD. In particular, many children with ADHD demonstrate deficits in executive functioning (EF), including in working memory or response inhibition, as compared to their typically -developing peers (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Pennington & Ozonoff, 1996; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). These findings have led to suggestions that EF deficits are at the core of the etiology for the disorder. However, at least half of children with ADHD do not show EF

impairments as compared to typically developing children, so these deficits may not be causal for all children with the disorder (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005).

Other etiological models of ADHD have focused on the role of reward sensitivity in leading to the behavioral symptoms of the disorder (for review, see Luman, Oosterlaan, & Sergeant, 2005). Most recently, research has focused on the possibility that children with ADHD may be less sensitive to *delayed* rewards than children without ADHD (Aase & Sagvolden, 2006; Sagvolden, Aase, Johansen, & Russell, 2005; Sagvolden, Aase, Zeiner, & Berger, 1998; Sagvolden & Sergeant, 1998; Sonuga-Barke, Taylor, Sembi, & Smith, 1992). Sonuga-Barke (1992, 2003) has additionally suggested that children with ADHD who are less sensitive to delayed rewards also learn to dislike and avoid situations associated with delay (i.e., become “delay-averse” or develop a “delay-averse motivational style”). This occurs because delayed rewards do not adequately reinforce behavior that occurred before the delay. Insensitivity to delayed rewards is hypothesized to be related to a dopaminergic deficit present from birth (Sagvolden et al., 2005; van den Bergh, Bloemarts, Groenink, Olivier, & Oosting, 2006), suggesting that conditioning of delay aversion begins in infancy. Typical delay aversion tasks measure a child’s preference for immediate versus delayed rewards by asking them to choose between small, immediate rewards and large, delayed rewards. Children are not required to sustain their choice after the initial selection (i.e., they cannot change their mind and opt for an immediate reward after making a choice for delayed reward), and so these tasks measure children’s learned preference for immediate versus delayed rewards rather than their abilities to actively delay gratification.

Although many etiological models have focused on finding a primary causal pathway leading to disorder, it is also recognized that multiple deficits or developmental pathways can lead to the same behavioral or cognitive outcomes (Garber, 1984; Sroufe & Rutter, 1984). Partly for this reason, recent research in ADHD has proposed using cognitive markers not only to bring the field closer to understanding genetic etiology (i.e., as endophenotypes) but also to help define etiologically homogeneous subgroups within the broader ADHD diagnosis (Doyle et al., 2005; Waldman et al., 2006; Willcutt et al., 2005). Consistent with the possibility of heterogeneous etiologies that manifest in distinct cognitive and motivational profiles, Sonuga-Barke (2002, 2003, 2005) has proposed a dual-pathway hypothesis highlighting how EF deficits and motivational style, specifically delay aversion, may be two distinct causal pathways for ADHD.

Although primary hypotheses of the dual-pathway model are that delay aversion and EF are two core deficits that should be at least partially dissociable, secondary hypotheses suggest that, over time, deficits in either delay aversion or EF could negatively impact the other area (Sonuga-Barke, 2002). In particular, children make some of the largest age-related improvements in response inhibition and working memory between early and late childhood (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Williams, Ponesse, Schachar, Logan, & Tannock, 1999), after a delay-averse motivational style has been well established. If children with hyperactivity and inattention have learned to avoid delay by preschool, this motivational style may have a significant negative impact on their development of EF skills,

such that by school age, children would be expected to be both delay averse and have weaker EF skills.

In the case of response inhibition, this prediction has not been supported in past research. Instead, several studies have found that a delay-averse motivational style does not predict inhibitory deficits in school-aged children with ADHD and that deficits in these separate processes effectively identify two distinct subgroups of children with ADHD (Lambek et al., 2010; Solanto et al., 2001; van den Bergh, Spronk, et al., 2006; Wahlstedt, 2009). These findings are consistent with the suggestion that response inhibition and delay aversion may be two distinct endophenotypes. However, despite being used as an umbrella term, EF processes are not unitary (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000; Tsujimoto, Kuwajima, & Sawaguchi, 2007). It may be that some EFs would be more likely than others to be affected by a delay-averse motivational style. In particular, EFs that rely on engagement across periods of delay may be more likely to be affected by a delay-averse motivational style than are EFs that do not rely on engagement across delay. Response inhibition, as most commonly defined in the ADHD literature (Barkley, 1997), is the ability to stop a prepotent or ongoing response. The stop task used in previous studies of relationships between delay aversion and response inhibition measures a child's ability to alter an immediate motor response. Given that response inhibition does not rely on engagement over periods of delay, it is unlikely to be impacted by a delay-averse motivational style.

In contrast, working memory is emblematic of EFs requiring engagement over delay. Although a variety of EFs require this ability, engagement over periods of delay is incorporated into the most commonly accepted definition of working memory (Baddeley & Logie, 1999; Logie, Della Sala, Laiacina, Chalmers, & Wynn, 1996). Thus, children who are delay averse are more likely to avoid and miss out on culturally common situations that require working memory than on situations requiring response inhibition. Further, working memory may be more likely than response inhibition to be impaired in children with ADHD who are delay averse. However, the few studies that have examined relationships between working memory and delay aversion have offered conflicting results. Wahlstedt, Thorell, and Bohlin (2009) found no correlation between working memory and delay aversion. However, the average age of their sample was only 8 years old. Because relationships between delay aversion and working memory are theorized to be age and experience dependent, it is possible that the inclusion of relatively younger children accounts for the lack of relationships. In an older school-age sample (M age = 12.12 years), Sonuga-Barke, Bitsakou, and Thompson (2010) completed a factor analysis including measures delay aversion, response inhibition, working memory, and time perception. They reported that working memory performance loaded on a factor with some measures of delay aversion but not others.

Given contradicting results and the burgeoning interest in assessing EFs and motivational style simultaneously to identifying more homogeneous subgroups of children within the ADHD diagnosis, it is important to determine relationships between measures of EF and delay aversion in ADHD. In particular, if delay aversion and working memory are closely related in school-age children, then they cannot be used in that age group to effectively

differentiate subgroups of children. Currently, the relationships between working memory, response inhibition, and delay aversion are not well defined. Further, despite EF and delay aversion being conceptualized as continuous constructs, these relationships have not been evaluated in typically developing populations.

CURRENT STUDY

Given the implications of relationships between EFs and motivational style for their use in identifying etiologically distinct subgroups of children with ADHD, the current study focused on testing the degree to which delay aversion is associated with two EFs that place either low or high demands on engagement over periods of delay: response inhibition and working memory, respectively. We predicted that a delay-averse motivational style would not be associated with a child's ability to inhibit a motor response (consistent with previous research). However, due to the theoretically stronger developmental interdependence, we predicted that delay aversion would be associated with a child's working memory capacity. If the degree of delay aversion significantly predicted working memory, our second goal was to determine whether that relationship was specific to children with ADHD or whether it would also be seen in typically developing children.

Several theories suggest that the two primary subtypes of ADHD are distinct disorders with distinct cognitive mechanisms (Diamond, 2005; Milich, Balentine, & Lynam, 2001). However, reliable subtype differences on neuropsychological tasks have proven difficult to find (Baeyens, Roeyers, & Walle, 2006; Chhabildas, Pennington, & Willcutt, 2001; Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2005; Huang-Pollock & Nigg, 2003). Further, the delay aversion theory has been proposed to apply to both ADHD-Combined (ADHD-C) and ADHD-Inattentive (ADHD-I) subtypes. For these reason, subtypes were combined for all analyses in the current study.

METHOD

Participants

Ninety-one children between the ages of 8 and 12 were recruited through fliers and advertisements distributed in the Centre County and York County areas of Pennsylvania ($N_{\text{Control}} = 46$; $N_{\text{ADHD-I}} = 27$; $N_{\text{ADHD-C}} = 18$). All data were collected in compliance with American Psychiatric Association ethical guidelines and with human subjects' approval from the Pennsylvania State University Institutional Review Board (IRB# 32126). A parent or legal guardian provided written consent, and the child provided verbal assent prior to participation. Eligibility for participation was a multitiered process. After expressing interest in participating, all participants completed a phone screen to determine initial eligibility. Children with significant visual or hearing impairments; who did not speak English as a first language; or for whom parents reported the presence of mental retardation, autism, or psychosis were excluded. Children who were prescribed nonstimulant psychotropic medications, which cannot be abruptly discontinued, were also screened out due to possible contaminating effects of medication on performance. Children who were prescribed a stimulant medication were asked to discontinue medications 24 [short-acting] to 48 [long-acting] hr prior to testing.

Procedure

A parent or legal guardian provided written consent for each child (The Pennsylvania State University IRB 32126), and the child's written assent was also obtained in all cases. Families still eligible after the phone screen received two packets of questionnaires, each of which included the Behavioral Assessment System for Children, 2nd edition (BASC-2; Reynolds & Kamphaus, 2004); the Conners' Rating Scale-Revised (CRS-R; Conners, 2003); and ADHD Rating Scale-IV (ADHD-RS-IV; DuPaul, Power, Anastopoulos, & Reid, 1998). The child's primary caretaker completed one of the sets of questionnaires. The child's classroom teacher completed the other set of questionnaires.

To be screened in as *potentially ADHD*, the child's parent *and* primary teacher were required to rate the child as having significant age-inappropriate levels of hyperactivity, inattention, or oppositional behavior on either the BASC-2 or CRS-R (*T* scores >61, 85th percentile). To be *diagnosed as ADHD*, children were required to meet *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV-TR]; American Psychiatric Association, 2000) criteria for ADHD during an in-person interview with the primary caregiver using the Diagnostic Interview Schedule for Children, 4th edition (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). On the DISC-IV, each reported symptom is only counted as present if the parent also endorses that the behavior leads to significant impairment in academic, family, or social functioning. Age of onset, chronicity, and cross-situational severity criteria are also required. If all ADHD criteria were met, then following DSM-IV field trials (Frick et al., 1994) the appropriate ADHD subtype was determined using an "or" algorithm: If the parent (on the DISC) or teacher (on the ADHD-RS) endorsed a symptom as present, then the symptom was counted as present. Requiring that the child meet impairment criteria prior to determining a final diagnosis, as well as requiring that they also show age-inappropriate impairment on parent and teacher BASC-2 or CRS-R measures protects against inclusion of children who might be falsely classified as ADHD when diagnosis is based solely on parent and teacher symptom counts (Gathje, Lewandowski, & Gordon, 2008; Gomez, Harvey, Quick, Scharer, & Harris, 1999).

To be screened in as *potentially non-ADHD control*, children must have been rated as having age-normative (*T* score <58) level of inattention, hyperactivity, and oppositional behaviors by both parent and teacher report. To be *diagnosed as a non-ADHD control*, using the "or" algorithm just described, children needed to have three or fewer attention symptoms, three or fewer hyperactivity symptoms, and fewer than four *total* symptoms.

Children completed a two-subtest short form of the Wechsler Intelligence Scale for Children, 4th edition (WISC-IV; Wechsler, 2003), including Vocabulary and Matrix Reasoning (predictive validity with the Full Scale Intelligence Quotient $r = .873$; Sattler, 2008). Children with estimated IQs below 80 were excluded.

No exclusions were made for comorbid diagnoses or behavioral problems because this would have drastically reduce both sample size and the generalizability of results given high rates of comorbidity among children with ADHD (60% or more of children with ADHD have another comorbid psychiatric disorder; Gillberg et al., 2004). Instead, oppositional/conduct problems or anxiety were used as covariates in analyses. To justify the creation of a

composite oppositional and conduct problems score to be used as a covariate, the Parent and Teacher BASC–2 Conduct Problems and Conners' Oppositional scores were analyzed using exploratory factor analysis. Parent and teacher ratings of behavior problems loaded onto a single factor that accounted for 73.97% of the variance (eigenvalue = 2.96 factor loading range = .792–.905). Given these results, a composite externalizing behavior problem score to be used as a covariate in analyses was created by averaging the parent and teacher ratings on these indices. Parent and teacher anxiety ratings (BASC–2 Anxiety) were significantly correlated ($r = .29$, $p = .006$), and so these two ratings were also averaged to create an anxiety composite used as a covariate in analyses.

Measures

Working memory—All children completed the working memory subtests from the WISC–IV (Digit Span Forward and Backwards, Letter-Number Sequencing, and Arithmetic), as well as the Verbal Working Memory, Symbolic Working Memory, and Finger Windows subtests from the WRAML–2 (Sheslow & Adams, 2003). The working memory measures from the WISC–IV and WRAML–2 were submitted to factor analytic procedures. Exploratory factor analysis in which factors with eigenvalues less than 1 were not considered in the final solution indicated that a one-factor solution provided the best fit for the data. The single factor accounted for 56.83% of the variance (eigenvalue = 3.41, factor loadings range = .639 [Finger Windows Forward] to .830 [Letter-Number Sequencing]), justifying the creation of a working memory composite score that was the average of scaled scores on each of the working memory tests.

Stop task (logan, 1994)—Children were administered a tracking version of the Logan Stopping Task. For each trial, a central fixation point appeared for 200 ms. An “X” or an “O” then appeared for 1,000 ms. On 75% of trials (“go” trials), children were asked to indicate with a key press whether an “X” or an “O” (the “.” and “,” keys, respectively) appeared in the center of the screen. Stickers were placed over the appropriate symbols. Children were given 2,300 ms to respond, after which the next trial automatically commenced. On 25% of trials (“stop” trials), an auditory tone was presented to indicate that they should not respond.

An initial mean reaction time (MRT) was determined based on 20 practice trials, and the auditory stop tone was initially set to occur 250 ms before the MRT. MRT was then recalculated following every successful go trial. The delay at which the stop tone was presented was adjusted dynamically in 50 ms increments. If the child successfully inhibited on a given stop trial, on the following stop trial, the tone was presented 50 ms closer to the MRT to make it more difficult to stop. If the child was unsuccessful, the tone was presented 50 ms farther from the MRT. Stop Signal Reaction Time (SSRT), the amount of time a child needs in order to successfully inhibit a response 50% of the time, was calculated by subtracting the mean delay from the child's MRT. Children were given 20 practice trials, after which followed five blocks of 40 experimental trials with optional rest periods between blocks. Stop trials were semirandomized within each block with the exception that each stop trial was followed by a go trial, except once in each block where two stop signal trials occurred back to back to prevent children from anticipating a go trial following a stop trial.

Children who had fewer than 80% correct hits were excluded from the final analyses. Six non-ADHD controls and 14 children with ADHD were excluded based on this criterion. Excluded children did not differ from those included in analysis in age, socioeconomic status, or estimated IQ. Reliability (α) of SSRT between the five blocks of the task was 0.853. Although no normative data are available for the stop task, mean SSRTs for control and ADHD children were similar to values found in previous studies (Huang-Pollock, Mikami, Pfiffner, & McBurnett, 2007; Solanto et al., 2001; Williams et al., 1999).

Choice-delay task (Sonuga-Barke et al., 1992)—In this task, children chose between two rewards each requiring a different waiting period: (a) a 1-point reward available after 2 s, or (b) a 2-point reward available after 30 s. Each trial began immediately after the reward was received from the preceding trial. Children had 20 trials in which they were instructed to earn as many points as possible. Prior to beginning the 20 trials, the experimenter provided 5 practice trials during which she or he ensured that the child understood how to make reward choices. In addition, in each of the 5 practice trials, the experimenter instructed the child to alternate between choosing the large and small rewards and then drew the child's attention to the point value, as well as to the difference in waiting time associated with each. Children were told that they would receive a prize at the end of the game based on the number of points that they earned. (In reality, all children received the prize, regardless of points earned.) The variable used in analyses is the percentage of choices for the 2-point, delayed reward. Reliability (α) of reward choices across trials was 0.90. Again, although no normative data are available for this task, the percentage of choice for the delayed reward was consistent with results in previous studies of children the same age (Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Solanto et al., 2001).

Data Analyses

Between-group comparisons—For demographics and sample characteristics, all between-group comparisons were conducted using one-way analysis of variance (ANOVA) tests. For experimental variables, including SSRT, Choice-Delay Task (C-DT), and working memory scores, a multivariate analysis of variance (MANOVA) test was first performed followed by one-way ANOVA tests.

Moderation analyses—Separate multiple regression analysis were conducted to determine if performance on the C-DT predicted either children's response inhibition or working memory abilities, as well as whether ADHD status moderated this relationship. To test relationships between C-DT performance and EF task performance, ADHD status (control = 0, ADHD = 1) was entered in Step 1 and C-DT scores were entered in Step 2 of the analysis. For moderation analyses, centered C-DT scores and ADHD status were entered in Step 1, and a moderator variable created by multiplying ADHD status with C-DT scores, which was also centered, was entered in Step 2 (Holmbeck, 1997). Either SSRT or the working memory composite score was used as the dependent variable.

Covariates—Current theory suggests that the etiology of ADHD may overlap with that of other externalizing disorders and that children with other externalizing disorders may also have dysfunctional reward processing (Burt, Krueger, McGue, & Iacono, 2001; Krueger,

Caspi, Moffitt, & Silva, 1998). In addition, working memory abilities are often highly correlated with overall IQ, making group differences in IQ a potential confound in interpreting results of analyses related to working memory. For these reasons, all between-group analyses and regression analyses were also conducted with the oppositional and conduct problems composite and estimated IQ as covariates. Patterns of significance did not change for any of the analyses, and so results reported next are from tests without covariates included.

Power analyses—Power analyses with G*Power3 (Erdfeider, Faul, & Buchner, 1996) indicated that the total sample size ($N=91$) provides adequate power (power $>.80$) for detecting effect sizes that are in the medium range ($d>0.64$; Cohen, 1988), which is consistent with previous effect sizes for between group differences in measures of EF and delay aversion (Lijffijt et al., 2005; Martinussen et al., 2005; Solanto et al., 2001; Willcutt et al., 2005). Power for omnibus (R^2 different from zero) and R^2 change tests in the moderation analyses achieved adequate power (power for all tests $>.80$) for detecting relationships with medium effect sizes. Where direct comparison was made between correlation coefficients, power was adequate to detect differences in r of a magnitude of .30.

RESULTS

Sample Characteristics

As rated by both parents and teachers, children with ADHD had significantly more total symptoms of inattention and hyperactivity than controls (all $\eta^2=0.92$ and 0.48 , respectively; see Table 1), as well as significantly more oppositional behaviors (all $\eta^2=0.42$; see Table 1) and higher levels of anxiety (all $\eta^2=0.16$; see Table 1) than control children. There were no significant group differences between children with and without ADHD in either age ($\eta^2=0.10$) or IQ ($\eta^2=0.01$). Median household income for families of children with and without ADHD did not differ and was equal to \$51,000 to \$60,000 a year. Parents of the majority of children (96% of children in the control group and 91% of children in the ADHD group) identified their child's ethnicity as non-Hispanic White.

Group Differences in Working Memory, Response Inhibition, and Delay Aversion

A MANOVA that included the working memory composite, SSRT, and C-DT scores indicated that children with ADHD performed significantly more poorly than non-ADHD controls, $F(3, 87)=12.84$, $p<.001$. Follow-up ANOVA tests indicated that children with ADHD had worse working memory than controls, $F(1, 90)=24.76$, $p<.001$, $\eta^2=0.22$. Children with ADHD also had slower SSRTs than did controls: SSRT, $F(1, 90)=17.16$, $p<.001$, $\eta^2=0.16$, but were not more delay averse than non-ADHD controls, $F(1, 90)=2.06$, $p=.154$, $\eta^2=0.02$ (see Table 2).

Relationships Between Delay Aversion and Executive Function

Simple correlations between all tasks were significant (all $p<.05$; $r_{WM \& SSRT}=-.24$, $r_{WM \& C-DT}=.316$, $r_{SSRT \& C-DT}=-.224$). A multiple regression analysis was conducted to determine if delay aversion was related to SSRT beyond ADHD diagnostic status. In Step 1 of the analysis, ADHD status predicted SSRT ($R^2=.163$, $p<.001$). However, C-DT scores

entered in Step 2 did not add to the prediction of SSRT scores ($R^2 = .027, p = .100$; see Table 3). Further, in a separate multiple regression analysis with ADHD status and C-DT scores entered in Step 1 and the interaction of ADHD status and C-DT entered on Step 2, the moderation effect based on the presence or absence of an ADHD diagnosis was not significant ($R^2 = .003, p = .588$).

Similarly, multiple regression analyses were used to test the relationship between working memory and C-DT performance. Again, in Step 1 of the analysis ADHD status predicted working memory ($R^2 = .220, p < .001$). In this case, however, C-DT was related to working memory in Step 2 of the regression ($R^2 = .061, p = .008$; see Table 3). Further, a follow-up t test indicated that the magnitude of the correlation between working memory and C-DT ($r = .25, p < .01$) was significantly larger than the correlation between SSRT and C-DT ($r = -.16, p > .05$), $t(76) = 3.19, p < .01$. The relationship between working memory and C-DT performance was not moderated based on the presence or absence of an ADHD diagnosis (i.e., the relationship between delay aversion and working memory did not differ between children with and without ADHD; $R^2 = .003, p = .543$).

Logistic Regression

Forward stepwise logistic regression was used to test whether working memory, response inhibition, and delay aversion contribute separately to the prediction of ADHD diagnostic status. The Hosmer and Lemeshow test indicated that the model adequately fit the data, $\chi^2(8) = 3.73, p = .881$. Both working memory and SSRT contributed significantly to the final model ($\beta = -.499, p < .001$ and $\beta = .007, p = .003$, respectively), which correctly predicted 72.2% of cases (33 of 45 control and 32 of 46 ADHD). Performance on the C-DT did not add to the prediction of diagnostic status over and above SSRT and working memory ($p = .151$). In addition, when impairment for each measure was defined as 1.5 standard deviations below the control mean, 16 children with ADHD (36%) were unimpaired on all measures and 20 children (44%) had *only* EF impairment (either SSRT or working memory). None of the children with ADHD were impaired on only the delay aversion measure. However, 9 children (20%) had *both* delay aversion and EF impairment.

DISCUSSION

In the present study, children with ADHD performed more poorly than non-ADHD controls on tests of working memory and response inhibition, which is consistent with previous research (Lijffijt et al., 2005; Martinussen et al., 2005; Willcutt et al., 2005). In contrast, as a group, children with ADHD were not more delay averse than their typically developing counterparts. Although this contradicts some previous research (Kuntsi, Oosterlaan, & Stevenson, 2001; Solanto et al., 2001; Sonuga-Barke, Auerbach, Campbell, Daley, & Thompson, 2005; Sonuga-Barke et al., 1992), it is consistent with other large-scale studies of children with ADHD (Bidwell, Willcutt, DeFries, & Pennington, 2007; Scheres, et al., 2006; Solanto et al., 2007). A failure to find group differences in delay aversion is contradictory to the delay aversion hypothesis. However, even under this hypothesis, not all children are expected to have deficits in delay aversion. This heterogeneity may contribute to a failure to find significant differences at the group level. The age of our sample may also

have prevented finding group differences. Several studies have demonstrated that effect sizes for delay aversion are largest in young children and become smaller in older children and teenagers (Bitsakou et al., 2009; Marco et al., 2009). This is consistent with research indicating that young children often lack strategies for effectively delaying gratification, but learn these strategies in middle and late childhood, and suggests that delay aversion may have its largest effects on behavior and development early in childhood.

In addition to group differences in EF and delay aversion, the current study investigated the relationships among these measures. In the current sample, delay aversion did not independently predict ADHD diagnosis, and *no* children experienced impairment in delay aversion in the absence of EF impairment, suggesting that in this age group motivational and EF impairments co-occur. Although results are consistent with secondary hypotheses of the dual-pathway model, which suggest that children with a delay-averse motivational style will develop EF deficits, they contradict the primary hypotheses and most common interpretation of the dual-pathway model, which suggests that motivational and EF deficits are etiologically distinct and should identify different subgroups of children with ADHD. Although the current study does not speak to etiology of either delay aversion or EF deficits, the significant overlap in these cognitive symptoms in school-age children suggests that combining assessment of these domains cannot improve diagnostic sensitivity or differentiate distinct subgroups in this age range.

Current findings contradict some previous research suggesting that EF deficits and delay aversion do characterize two distinct groups of children (Lambek et al., 2010; Solanto et al., 2001; van den Bergh, Spronk, et al., 2006). Previous research, however, has focused primarily on motor inhibitory control as indexed by the SSRT as the measure of EF. The current study replicated the lack of relationship between SSRT and delay aversion performance, and further demonstrated that this relationship did not differ between children with and without ADHD. However, consistent with the hypotheses of the current study that working memory would be more likely than response inhibition to be impaired in children with a delay-averse motivational style, there was a significant predictive relationship between working memory and delay aversion performance over and above ADHD status. Further, the relationship between working memory and delay aversion did not differ in ADHD and typically developing populations, indicating that a child's ability to tolerate delay is related to working memory development whether or not they are delay averse.

One possibility for the dissociation between EFs may be that tests of delay aversion require working memory to complete (e.g., remembering the instructions for the task and the wait time associated with each reward). Although this cannot be ruled out definitively, the working memory demands of a delay aversion task are minimal compared to standard tests of working memory (e.g., WISC-IV Digit Span task, n-back tasks), and are reduced further by the practice trials given to children at the beginning of the task. Furthermore, studies in preschool-age children have failed to find correlations between delay aversion and working memory tasks. If these two tasks measured overlapping constructs, they would be likely to be correlated at all ages, rather than only in school-age children. Last, tests of working memory and delay aversion have been found to predict different symptom domains and outcomes in children with ADHD (Thorell, 2007; Wåhlstedt et al., 2009), suggesting that the

tasks are measuring two independent constructs. Given this, it is unlikely that shared working memory demands explain the association between the delay and working memory tasks; however, future studies manipulating working memory load will be important in fully assessing this possibility.

Limitations

The current study examined relationships in school-age children between a delay-averse motivational style and EF. If the hypothesis that a delay-averse motivational style contributes to deficits in working memory via poor task engagement (Sonuga-Barke, 2005) is to be supported, three things must be true: (a) At an early age, deficits in motivation and working memory should be uncorrelated (Sonuga-Barke et al., 2005); (b) children with ADHD should show poorer task engagement across early childhood than typically developing children (Lauth, Heubeck, & Mackowiak, 2006); and (c) by the time children are school-age, delay aversion and working memory should be correlated. Individually, each of these components has been supported in cross-sectional samples. However, to demonstrate a direct causal relationship between delay aversion and working memory performance, longitudinal research simultaneously examining motivational style, task engagement, and working memory is necessary.

In addition, to understand the relationships between delay aversion and EFs, future research will need to fully explore alternative hypotheses for the results. For example, rather than being developmentally related, working memory and delay aversion may be correlated because they each rely on similar structural and functional networks. If this is the case, it suggests that these relationships should be present even in early childhood, rather than emerging over the course of development. Alternatively, the cognitive-energetic model (Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003) has implicated factors such as arousal and effort as potential contributors to cognitive deficits in ADHD, and these factors may also contribute to a desire for immediate rewards. Future research to determine whether state manipulations impact performance on either delay aversion or working memory tests may clarify whether relationships reflect established traits or instead reflect variations with state factors.

In addition, it is unclear how environmental and contextual factors may influence either delay aversion or its impact on EF. Sonuga-Barke (2002, 2003, 2005) has specifically suggested that a delay-averse motivational style develops over time in response to negative affect experienced during periods of delay and negative feedback from others during tasks requiring delay (e.g., consistently getting in trouble because of difficulty waiting turns). This suggests that parent and teacher efforts to provide more positive reinforcement during delay periods (e.g., providing praise several times while the child is waiting his or her turn) may reduce the impact of delay intolerance on a child's cognitive development. Alternatively, stimulant medications are used to reduce behavioral symptoms of inattention and impulsivity, but their effects on a delay-averse motivational style are not understood. If stimulant medications effectively reduce delay aversion for children with ADHD, it may be possible that early identification and treatment could also reduce the impact of delay aversion on cognitive development. In the current study, only 10 children were prescribed

medications for treatment of ADHD, and so differences between children with and without medication treatment were not examined. However, this will be an important area for future studies to address.

Finally, the current study only examined relationships between delay aversion and two EFs: response inhibition and working memory. Hypotheses and results of the current study suggest that any EF that develops significantly during early and middle childhood and requires engagement over periods of delay may be impacted by a delay-averse motivational style. By this logic, it may be that planning (which requires waiting while a plan is developed) is more negatively impacted by a delay-averse motivational style than is flexibility (which does not require waiting). However, more research is needed to clearly delineate the impact of motivational style on the full range of EFs.

Implications for Research, Policy, and Practice

Although the current study has a small sample size, and so results require replication, the findings highlight the importance of considering EFs as a set of heterogeneous cognitive processes that differ with respect to the interdependence of their developmental trajectories. Despite empirical evidence that EFs are at least partially dissociable processes (Miyake et al., 2000; Tsujimoto et al., 2007), current theories of ADHD often implicate “executive functioning” as a unitary construct and contrast impairment in EFs to motivational or arousal deficits (Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008; Wählstedt et al., 2009). The current study suggests that relationships between motivation and EFs vary based on the particular EF process measured. Results imply that success in identifying etiologically distinct subgroups of children with ADHD based on intermediate cognitive and motivational profiles cannot rely on comparison of groups with working memory and delay aversion deficits. The utility of response inhibition and delay aversion in identifying distinct subgroups of children with ADHD is less clear because the relationship between these two constructs was weaker, implying a greater degree of dissociability.

Clinically, results of a logistic regression predicting diagnosis indicate that using a combination of EF and delay aversion tasks does not improve identification of children with ADHD over EF tests alone. Delay aversion demonstrated little sensitivity or specificity for predicting diagnosis in school-age children as compared to response inhibition and working memory. This suggests not only that tests of delay aversion should *not* be used diagnostically (which is also true of EF tests) but also that even the combination of these tests cannot be used to rule in or rule out a diagnosis of ADHD. That said, assessment of children who may have attention disorders is concerned not only with establishing a diagnosis but also with understanding factors that contribute to functional impairments, and tests of EF and delay aversion may add to the assessment of functional impairment and aid treatment planning.

Finally, it is important to highlight that the relationship between motivational style and working memory did not differ between children with and without ADHD. Although symptom domains associated with ADHD are largely agreed to be dimensional, much of the research focused on identifying cognitive and motivational deficits emphasizes qualitative differences between those with the disorder and those without. The current results highlight

that nondiagnostic associated features, such as EF impairment and motivational style, may also exist on a continuum and that degree of impairment on these measures may also represent an extreme of normative relationships between cognitive processes.

References

- Aase H, Sagvolden T. Infrequent, but not frequent, reinforcers produce more variable responding and deficient sustained attention in young children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry*. 2006; 47:457–471. [PubMed: 16671929]
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2000. text rev
- Anastopoulos AD, Guevremont DC, Shelton TL, DuPaul GJ. Parenting stress among families of children with attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 1992; 20:503–520. [PubMed: 1487593]
- Anderson VA, Anderson P, Northam E, Jacobs R, Catroppa C. Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology*. 2001; 20(1):385–406. [PubMed: 11827095]
- Baddeley, A.; Logie, R. Working memory: The multi-component model. In: Miyake, A.; Shah, P., editors. *Models of working memory: Mechanisms of active maintenance and executive control*. Cambridge, UK: Cambridge University Press; 1999. p. 28-61.
- Baeyens D, Roeyers H, Walle JV. Subtypes of Attention-Deficit/Hyperactivity Disorder (ADHD): Distinct or related disorders across measurement levels? *Child Psychiatry and Human Development*. 2006; 36:403–417. [PubMed: 16755403]
- Barkley RA. Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*. 1997; 121(1):65–94. [PubMed: 9000892]
- Bidwell LC, Willcutt EG, DeFries JC, Pennington BF. Testing for neuropsychological endophenotypes in siblings discordant for attention-deficit/hyperactivity disorder. *Biological Psychiatry*. 2007; 62:991–998. [PubMed: 17585884]
- Biederman J, Monuteaux MC, Doyle AE, Seidman LJ, Wilens TE, Ferrero F, et al. Impact of executive function deficits and Attention-Deficit/Hyperactivity Disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology*. 2004; 72:757–766. [PubMed: 15482034]
- Bitsakou P, Psychogiou L, Thompson M, Sonuga-Barke E. Delay aversion in Attention Deficit/Hyperactivity Disorder: An empirical investigation of the broader phenotype. *Neuropsychologia*. 2009; 47:446–456. [PubMed: 18929587]
- Burt SA, Krueger RF, McGue M, Iacono WG. Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of shared environment. *Journal of Abnormal Psychology*. 2001; 110:516–525. [PubMed: 11727941]
- Castellanos FX, Sonuga-Barke E, Milham MP, Tannock R. Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences*. 2006; 10:117–123. [PubMed: 16460990]
- Chhabildas N, Pennington BF, Willcutt EG. A comparison of the neuropsychological profiles of the DSV-IV subtypes of ADHD. *Journal of Abnormal Child Psychology*. 2001; 29:529–540. [PubMed: 11761286]
- Cohen, J. *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum; 1988.
- Conners, CK. *Conners' Rating Scales: Revised technical manual*. New York: Multi-Health Systems; 2003.
- Diamond A. Attention-deficit disorder (attention-deficit/ hyperactivity disorder without hyperactivity): A neurobiologically and behaviorally distinct disorder from attention-deficit/ hyperactivity disorder (with hyperactivity). *Development and Psychopathology*. 2005; 17:807–825. [PubMed: 16262993]

- Doyle AE, Faraone SV, Seidman LJ, Willcutt EG, Nigg JT, Waldman ID, et al. Are endophenotypes based on measures of executive functions useful for molecular genetic studies of ADHD? *Journal of Child Psychology and Psychiatry*. 2005; 46(7):778–803.
- Dumas MC. The risk of social interaction problems among adolescents with ADHD. *Education & Treatment of Children*. 1998; 21:447–460.
- DuPaul, G.; Power, T.; Anastopoulos, A.; Reid, R. *ADHD Rating Scale–IV: Checklists, norms, and clinical interpretation*. New York: Guilford; 1998.
- Erdfelder E, Faul F, Buchner A. GPOWER: A general power analysis program. *Behavior Research Methods, Instruments & Computers*. 1996; 28(1):1–11.
- Erhardt D, Hinshaw SP. Initial sociometric impressions of attention-deficit hyperactivity disorder and comparison boys: Predictions from social behaviors and from nonbehavioral variables. *Journal of Consulting and Clinical Psychology*. 1994; 62:833–842. [PubMed: 7962888]
- Frick PJ, Lahey BB, Applegate B, Kerdyck L, Ollendick T, Hynd GW, et al. *DSM–IV* field trials for the disruptive behavior disorders: Symptom utility estimates. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1994; 33:529–539. [PubMed: 8005906]
- Garber J. Classification of childhood psychopathology: A developmental perspective. *Child Development*. 1984; 55(1):30–48. [PubMed: 6705631]
- Gathje RA, Lewandowski LJ, Gordon M. The role of impairment in the diagnosis of ADHD. *Journal of Attention Disorders*. 2008; 11:529–537. [PubMed: 18259000]
- Geurts H, Verté S, Oosterlaan J, Roeyers H, Sergeant JA. ADHD subtypes: Do they differ in their executive functioning profile? *Archives of Clinical Neuropsychology*. 2005; 20:457–477. [PubMed: 15896560]
- Gillberg C, Gillberg IC, Rasmussen P, Kadesjö BR, Söderström H, Råstam M, et al. Co-existing disorders in ADHD—Implications for diagnosis and intervention. *European Journal of Child & Adolescent Psychiatry*. 2004; 13:80–92.
- Gomez R, Harvey J, Quick C, Scharer I, Harris G. *DSM–IV* AD/HD: Confirmatory factor models, prevalence, and gender and age differences based on parent and teacher ratings of Australian primary school children. *Journal of Child Psychology and Psychiatry*. 1999; 40:265–274. [PubMed: 10188709]
- Greene RW, Beszterczey SK, Katzenstein T, Park K, Goring J. Are students with ADHD more stressful to teach? Patterns of teacher stress in an elementary school sample. *Journal of Emotional and Behavioral Disorders*. 2002; 10:79–89.
- Holmbeck GN. Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from the child-clinical and pediatric psychology literatures. *Journal of Consulting and Clinical Psychology*. 1997; 65:599–610. [PubMed: 9256561]
- Huang-Pollock CL, Mikami AY, Pfiffner L, McBurnett K. ADHD subtype differences in motivational responsivity but not inhibitory control: Evidence from a reward-based variation of the stop signal paradigm. *Journal of Clinical Child and Adolescent Psychology*. 2007; 36:127–136. [PubMed: 17484686]
- Huang-Pollock CL, Nigg JT. Searching for the attention deficit in attention deficit hyperactivity disorder: The case of visuospatial orienting. *Clinical Psychology Review*. 2003; 23:801–830. [PubMed: 14529699]
- Krueger RF, Caspi A, Moffitt TE, Silva PA. The structure and stability of common mental disorders (*DSM–III–R*): A longitudinal-epidemiological study. *Journal of Abnormal Psychology*. 1998; 107:216–227. [PubMed: 9604551]
- Kuntsi J, Oosterlaan J, Stevenson J. Psychological mechanisms in hyperactivity: I Response inhibition deficit, working memory impairment, delay aversion, or something else? *Journal of Child Psychology and Psychiatry*. 2001; 42:199–210. [PubMed: 11280416]
- Lambek R, Tannock R, Dalgaard S, Trillingsgaard A, Damm D, Thomsen PH. Validating neuropsychological subtypes of ADHD: How do children with and without an executive function deficit differ? *Journal of Child Psychology and Psychiatry*. 2010; 51:895–904. [PubMed: 20406332]

- Lauth GW, Heubeck BG, Mackowiak K. Observation of children with attention-deficit hyperactivity (ADHD) problems in three natural classroom contexts. *British Journal of Educational Psychology*. 2006; 76:385–404. [PubMed: 16719970]
- Lijffijt M, Kenemans JL, Verbaten MN, van Engeland H. A meta-analytic review of stopping performance in Attention-Deficit/Hyperactivity Disorder: Deficient inhibitory motor control? *Journal of Abnormal Psychology*. 2005; 114:216–222. [PubMed: 15869352]
- Logan, GD. On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In: Dagenbach, D.; Carr, T., editors. *Inhibitory processes in attention, memory, and language*. San Diego, CA: Academic Press; 1994. p. 189-239.
- Logie RH, Della Sala S, Laiacina M, Chalmers P, Wynn V. Group aggregates and individual reliability: The case of verbal short-term memory. *Memory & Cognition*. 1996; 24:305–321. [PubMed: 8718765]
- Luman M, Oosterlaan J, Sergeant JA. The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review*. 2005; 25:183–213. [PubMed: 15642646]
- Marco R, Miranda A, Melia A, Muller U, Butler L, Gabriels I, et al. Delay and reward choice in ADHD: An experimental test of the role of delay aversion. *Neuropsychology*. 2009; 23:367–380. [PubMed: 19413450]
- Martinussen R, Hayden J, Hogg-Johnson S, Tannock R. A meta-analysis of working memory impairments in children with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2005; 44:377–384. [PubMed: 15782085]
- Milich R, Balentine AC, Lynam DR. ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology: Science and Practice*. 2001; 8:463–488.
- Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A. The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*. 2000; 41(1):49–100. [PubMed: 10945922]
- Nigg J, Willcutt EG, Doyle AE, Sonuga-Barke EJS. Causal heterogeneity in Attention-Deficit/Hyperactivity Disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry*. 2005; 57:1224–1230. [PubMed: 15949992]
- Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*. 1996; 37(1):51–87. [PubMed: 8655658]
- Reynolds, C.; Kamphaus, R. *Behavioral assessment system for children: Manual*. 2. Circle Pines, MN: American Guidance Service; 2004.
- Sagvolden T, Aase H, Johansen EB, Russell VA. A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences*. 2005; 28:397–468. [PubMed: 16209748]
- Sagvolden T, Aase H, Zeiner PI, Berger D. Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavioural Brain Research*. 1998; 94(1):61–71. [PubMed: 9708840]
- Sagvolden T, Sergeant JA. Attention deficit/hyperactivity disorder: From brain dysfunctions to behaviour. *Behavioural Brain Research*. 1998; 94(1):1–10. [PubMed: 9708834]
- Sattler, JM. *Assessment of children: Cognitive foundations*. 5. San Diego, CA: JM Sattler; 2008.
- Schachar R, Taylor E, Wieselberg M, Thorley G, Rutter M. Changes in family function and relationships in children who respond to methylphenidate. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1987; 26:728–732. [PubMed: 3667503]
- Scheres AD, Dijkstra M, Ainslie E, Balkan J, Reynolds B, Sonuga-Barke E, et al. Temporal and probabilistic discounting of rewards in children and adolescents: Effects of age and ADHD symptoms. *Neuropsychologia*. 2006; 44:2092–2103. [PubMed: 16303152]
- Scheres A, Dijkstra M, Ainslie E, Balkan J, Reynolds B, Sonuga-Barke E, et al. Temporal and probabilistic discounting of rewards in children and adolescents: Effects of age and ADHD symptoms. *Neuropsychologia Special Issue: Advances in developmental cognitive neuroscience*. 2006; 44(11):2092–2103.

- Sergeant J, Geurts H, Huijbregts S, Scheres A, Oosterlaan J. The top and the bottom of ADHD: A neuropsychological perspective. *Neuroscience and Biobehavioral Reviews*. 2003; 27:583–592. [PubMed: 14624803]
- Shaffer D, Fisher P, Lucas C, Dulcan M, Schwab-Stone M. 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*. 2000; 39(1):28–38. [PubMed: 10638065]
- Sheslow, D.; Adams, W. Wide Range Assessment of Memory and Learning—Second edition: Administration and technical manual. 2. Wilmington, DE: Wide Range; 2003.
- Solanto MV, Abikoff H, Sonuga-Barke E, Schachar R, Logan GD, Wigal T, et al. The ecological validity of delay aversion and response inhibition as measures of impulsivity in AD/HD: A supplement to the NIMH Multimodal Treatment Study of AD/HD. *Journal of Abnormal Child Psychology*. 2001; 29:215–228. [PubMed: 11411784]
- Solanto MV, Gilbert S, Raj A, Zhu J, Pope-Boyd SB, Stepak B, et al. Neurocognitive functioning in AD/HD, predominantly inattentive and combined subtypes. *Journal of Abnormal Child Psychology*. 2007; 35:729–744. [PubMed: 17629724]
- Sonuga-Barke E. Psychological heterogeneity in AD/HD—A dual pathway model of behaviour and cognition. *Behavioural Brain Research*. 2002; 130(1–2):29–36. [PubMed: 11864715]
- Sonuga-Barke E. The dual pathway model of AD/HD: An elaboration of neuro-developmental characteristics. *Neuroscience & Biobehavioral Reviews*. Special Issue: Mesocorticolimbic Branches and ADHD Variants. 2003; 27:593–604.
- Sonuga-Barke E. Causal models of Attention-Deficit/ Hyperactivity Disorder: From Common simple deficits to multiple developmental pathways. *Biological Psychiatry*. 2005; 57:1231–1238. [PubMed: 15949993]
- Sonuga-Barke E, Auerbach J, Campbell SB, Daley D, Thompson M. Varieties of preschool hyperactivity: Multiple pathways from risk to disorder. *Developmental Science*. 2005; 8:141–150. [PubMed: 15720372]
- Sonuga-Barke E, Bitsakou P, Thompson M. Beyond the dual pathway model: Evidence for the dissociation of timing, inhibitory, and delay-related impairments in Attention-Deficit/ Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2010; 49:345–355. [PubMed: 20410727]
- Sonuga-Barke E, Sergeant J, Nigg J, Willcutt E. Executive dysfunction and delay aversion in Attention Deficit Hyperactivity Disorder: Nosologic and diagnostic implications. *Child and Adolescent Psychiatric Clinics of North America*. 2008; 17:367–384. [PubMed: 18295151]
- Sonuga-Barke E, Taylor E, Sembi S, Smith J. Hyperactivity and delay aversion: I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry*. 1992; 33:387–398. [PubMed: 1564081]
- Sroufe A, Rutter M. The domain of developmental psychopathology. *Child Development*. 1984; 55(1): 17–29. [PubMed: 6705619]
- Thorell L. Do delay aversion and executive function deficits make distinct contributions to the functional impact of ADHD symptoms? A study of early academic skill deficits. *Journal of Child Psychology and Psychiatry*. 2007; 48:1061–1070. [PubMed: 17995481]
- Tsujimoto S, Kuwajima M, Sawaguchi T. Developmental fractionation of working memory and response inhibition during childhood. *Experimental Psychology*. 2007; 54(1):30–37. [PubMed: 17341012]
- van den Bergh F, Spronk M, Ferreira L, Bloemarts E, Groenink L, Olivier B, et al. Relationship of delay aversion and response inhibition to extinction learning, aggression, and sexual behaviour. *Behavioural Brain Research*. 2006; 175(1):75–81. [PubMed: 16965826]
- Wahlstedt C. Neuropsychological deficits in relation to symptoms of ADHD: Independent contributions and interactions. *Child Neuropsychology*. 2009; 15:262–279. [PubMed: 19089681]
- Wahlstedt C, Thorell LB, Bohlin G. Heterogeneity in ADHD: Neuropsychological pathways, comorbidity and symptom domains. *Journal of Abnormal Child Psychology*. 2009; 37:551–564. [PubMed: 19016322]

- Waldman I, Nigg J, Gizer I, Park L, Rappley M, Friderici K. The adrenergic receptor α -2A gene (*ADRA2A*) and neuropsychological executive functions as putative endophenotypes for childhood ADHD. *Cognitive, Affective & Behavioral Neuroscience*. 2006; 6(1):18–30.
- Wechsler, D. Wechsler Intelligence Scale for Children—Fourth edition: Technical and interpretive manual. San Antonio, TX: Harcourt Brace; 2003.
- Willcutt EG, Doyle AE, Nigg JT, Faraone SV, Pennington BF. Validity of the executive function theory of Attention-Deficit/Hyperactivity Disorder: A meta-analytic review. *Biological Psychiatry*. 2005; 57:1336–1346. [PubMed: 15950006]
- Williams BR, Ponesse JS, Schachar RJ, Logan GD, Tannock R. Development of inhibitory control across the life span. *Developmental Psychology*. 1999; 35(1):205–213. [PubMed: 9923475]

TABLE 1**Participant Demographic Information**

Variable	Control^a	ADHD^b	F(1, 90)	Effect Size (η^2)
Gender (M:F)	20:26	32:13	$\chi^2 = 8.34^{**}$	
Age (in Months)	124.56 (14.95)	121.38 (17.07)	0.73	0.10
FSIQ	109.13 (13.91)	106.80 (10.79)	3.22*	0.01
Total Inattention Symptoms	0.89 (1.18)	8.09 (1.04)	495.80***	0.92
Total Hyperactivity Symptoms	0.41 (0.65)	4.57 (3.04)	289.82***	0.48
Conduct Problems	45.02 (1.86)	54.67 (8.01)	60.34***	0.42
Anxiety	47.67 (7.07)	54.54 (9.00)	8.47***	0.16

Note: Data reported as mean (standard deviation). ADHD =Attention Deficit Hyperactivity Disorder; FSIQ =Full Scale Intelligence Quotient.

^a
 $n = 46$.

^b
 $n = 45$.

*
 $p < .05$.

**
 $p < .01$.

 $p < .001$.

TABLE 2

Experimental Task Results

Experimental Measure	Control ^a	ADHD ^b	F(1, 90)	Effect Size (η^2)
WM Composite	11.68 (2.02)	9.66 (1.98)	24.76***	0.22
C-DT (% Large)	0.64 (0.28)	0.55 (0.28)	2.06	0.02
SSRT	287.78 (95.61)	389.21 (131.10)	17.16***	0.16
MRT	770.43 (300.61)	738.45 (106.40)	0.50	0.04
SD Hits	197.64 (65.62)	219.62 (46.66)	3.43	0.01

Note: Data reported as mean (standard deviation); *p* values obtained through analyses of variance without covariates. ADHD =Attention Deficit Hyperactivity Disorder; WM =Working Memory Composite; C-DT =Choice-Delay Task; % large =percentage of choice for large reward; SSRT =Stop Signal Reaction Time; MRT =Mean Reaction Time of Hits; SD Hits = Standard Deviation of MRT.

^a
n =46.

^b
n =45.

p <.001.

TABLE 3**C-DT-SSRT and C-DT-WM Relationships**

Step and Variable	β	t Test (p Value)	R	R ²
1 ADHD Status	.379	3.88 ***	.469	
2 SSRT	-.166	1.70	.530	.027
1 ADHD Status	-.431	4.68 ***	.469	
2 WM	.251	2.73 **	.530	.061 **

Note: C-DT =Choice-Delay Task; SSRT =Stop Signal Reaction Time; WM =Working Memory Composite; ADHD =Attention Deficit Hyperactivity Disorder.

**
 $p < .01$.

 $p < .001$.