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CONSISTENTLY INCONSISTENT: UNDERSTANDING INTRA-INDIVIDUAL VARIABILITY IN ADHD

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ABSTRACT

STUDY 1 examined patterns of reaction time (RT) variability in children with and without ADHD under different motivational contexts. Seventeen children with ADHD and 20 typically-developing controls completed a go-no/go task that included baseline and motivational conditions. Children with ADHD were less accurate than non-ADHD controls in both conditions and responded more slowly than non-ADHD controls when motivational incentives were introduced. Fast-Fourier transform (FFT) analyses indicated that children with ADHD were more variable than non-ADHD controls at both high and low frequencies, but that group differences were greatest at low frequencies. Motivational incentives did not impact patterns of variability. Results are consistent with previous studies suggesting that low-frequency patterns of RT variability characterize task performance of children with ADHD. Lack of impact of motivational incentives implicates trait, rather than state, factors in determining patterns of variability.

STUDY 2 examined physiological correlates of RT variability in typically-developing children. Twenty-six typically-developing children completed a go-no/go task while physiological measures of attention were collected. Variability of RTs, as measured by standard deviation of RT (SDRT), was marginally negatively correlated with baseline respiratory sinus arrhythmia (RSA). Although the correlation between electroencephalogram (EEG) beta activity on go trials and SDRT was not significant, the effect size for the correlation was large. Results provide physiological corroboration of theories suggesting that RT variability in children reflects attentional lapses, specifically difficulties with orienting.

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INTRODUCTION

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 6% (APA, 2000). Multiple negative outcomes are associated with ADHD, including poor academic performance and grade retention (Biederman, et al., 2004), as well as problems in parent-child (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Schachar, Taylor, Wieselberg, Thorley, & et al., 1987), teacher-child (Greene, Beszterczey, Katzenstein, Park, & Goring, 2002), and peer relationships (Dumas, 1998; Erhardt & Hinshaw, 1994).

The core behavioral symptoms of the disorder include difficulties with sustained attention, hyperactivity, and impulsivity. In addition, many children with ADHD have deficits in executive functions (EF) as compared to typically-developing children (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Rucklidge & Tannock, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), and EF deficits have long been recognized as one of the most prominent cognitive features of the disorder. However, more recent studies have revealed significant inter-individual variability in performance on these tasks, such that no more than half of children with ADHD have EF impairments (Nigg, 2000; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). A variety of possible explanations exist for the failure to find consistent EF deficits in children with ADHD, most predominantly those proposing *inter*-individual variability due to the existence of multiple pathways to the disorder (Nigg, 2000; Sonuga-Barke, 2002). Yet another non-mutually exclusive possibility is that greater than normal *intra*-individual variability in performance (Berwid, et al., 2005; Castellanos, et al., 2005; Klein, Wendling, Huettner, Ruder, & Peper, 2006; Oades & Christiansen, 2008) has made it difficult to reliably identify the presence of cognitive deficits. Importantly, effect sizes for measures of reaction time (RT) variability (range $\eta^2 = .132\text{-}.339$) are often larger than effect sizes for specific executive function deficits in working memory, sustained attention, or response inhibition (range $\eta^2 = .018\text{-}.154$; Klein, et al., 2006; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Martinussen, et al., 2005; Willcutt, et al., 2005).

Taken together, results suggest that the most consistent performance deficit in ADHD may, in fact, be inconsistency. Accumulating evidence also suggests that this cognitive marker may be linked to the biological mechanisms underlying the disorder. Measures of RT variability correlate with many of the behavioral symptoms of ADHD, more so even than do traditional measures of cognitive deficits, such as commission or omission errors on a continuous performance test (Epstein, et al., 2003; Oades & Christiansen, 2008). Greater than average performance variability has also been found in non-ADHD members of twin pairs (Bidwell, Willcutt, DeFries, & Pennington, 2007) and in young children identified as being at risk for ADHD (Berwid, et al., 2005). Finally, findings that performance variability is associated with the presence of the DAT1 allele, which has been implicated in ADHD, (Bellgrove, Hawi, Kirley, Gill, & Robertson, 2005), and that variability can be reduced by treatment with stimulant medications (Epstein, et al., 2006), suggest that performance variability may be an important marker of biological dysfunction in the disorder.

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). This, in addition to the well-documented variability in symptom presentation within subtypes, has led to recent efforts to identify behavioral markers that can be used to sub-divide children into more homogeneous etiological groups (e.g. ADHD with EF deficits and ADHD without EF deficits; (Aron & Poldrack, 2005; Biederman, et al., 2004; Crosbie, Pérusse, Barr, & Schachar, 2008; Doyle, Faraone, et al., 2005; Doyle, Willcutt, et al., 2005; Nigg, et al., 2005; Waldman, et al., 2006). Because of its strong association with the behavioral symptoms of ADHD and possible link to an ADHD genotype, intra-individual variability has been identified as one possible endophenotype of the disorder that may provide insight into the etiology of ADHD (Bidwell, et al., 2007; Castellanos, et al., 2005).

If this is the case, it behooves us to understand which factors contribute to *intra*-individual variability in performance and, thus, which factors may contribute to the *inter*-individual variability in patterns of behavior and impairment for children with this disorder. One possibility is that intra-individual variability is a stable aspect of cognitive processing for children with ADHD that reflects inefficient recruitment of neural circuitry underlying cognitive performance and response execution. However, a wide variety of etiological theories of ADHD have also posited that dysfunctional reward processing may lead to the behavioral symptoms of the disorder (for review see Luman, Oosterlaan, & Sergeant, 2005). These include suggestions that children with ADHD are less sensitive to rewards in general (Haenlein & Caul, 1987), more easily frustrated by failure to receive anticipated rewards (Douglas & Parry, 1994), and more reliant on extrinsic rewards to guide behavior (Carlson, Booth, Shin, & Canu, 2002; Luman, et al., 2005). Most recently, attention has focused on the possibility that children with ADHD may be less sensitive specifically 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 (1994, 2003) has additionally suggested that children with ADHD who experience less reward from reinforcers presented after periods of delay become delay-averse, which leads to poor task engagement. On cognitive tasks, this lack of task engagement would be reflected in inaccurate and highly variable responding. Given the variety of potential motivational explanations for high levels of variability in children with ADHD, another possibility is that greater than expected variability results from inconsistent motivation to do well on tasks.

The current study aims to assess patterns of variability under differential reward conditions to further our understanding of the boundaries of this phenomenon in ADHD, which may in turn contribute to understanding underlying etiological factors. With a better understanding of the factors that contribute to the development of ADHD, the field will be better able to identify children at risk who would benefit from prevention programs, develop more specific treatments, and predict children's response to treatment.

STUDY 1

Patterns of Intra-individual Variability

A first step to improving our understanding of the clinical and etiological significance of performance variability is to clarify patterns within this variability. As Castellanos et al. (2005) have noted, if performance fluctuations are random and unrelated to underlying physiological mechanisms, then they have little utility for better understanding the disorder. However, evidence to-date suggests that these fluctuations do, in fact, follow a predictable pattern. In particular, Leth-Steensen, Elbaz, & Douglas (2000) and Hervey et al. (2006) have both demonstrated that RT distributions for children with ADHD are best described in terms of an ex-Gaussian distribution, in which a large number of abnormally long RTs create an extended tail at the upper end of the distribution. Further, several studies using time-series analyses have documented that these long RTs occur in a predictable time course, such that children with ADHD responded with especially long RTs approximately once every 20 seconds (or at a driving frequency between .05Hz and .075Hz; Castellanos, et al., 2005; Di Martino, et al., 2008). This specific frequency pattern uniquely contributes to predicting ADHD diagnosis beyond gross measures of RT standard deviation (Di Martino, et al., 2008).

Although high intra-individual variability defined in terms of RT standard deviation (SDRT) appears to occur on many cognitive tasks, it is especially prevalent on tasks with high working memory demands (Klein, et al., 2006), and patterns within this variability have only been well-explored on tasks with high-working memory demands. For this reason, it is unclear whether specific patterns would extend to other tasks or cognitive processes in which working memory demands are reduced. Go/no-go tasks, used extensively in studies of childhood ADHD, are thought to tap inhibition and sustained attention, both of which have been hypothesized to be important areas of cognitive deficit for children with ADHD (Barkley, 1997; Losier, McGrath, & Klein, 1996), but are low in working memory demands. Because go/no-go tasks require a large number of responses (as opposed to traditional continuous performance tests of sustained attention, which only require response to rare targets), they are optimally suited for looking at frequency patterns of variability within data. Johnson et al. (2007) examined patterns of variability in a go/no-go task and found that children with ADHD had more variability in both low and high frequency bands. However, the authors did not specifically quantify driving frequencies. In addition, the particular structure of their go/no-go task required children to respond to a fixed sequence of digits, so that the occurrence of no-go trials was predictable throughout the task, drawing into question the extent to which it truly tapped either attention or inhibition for the children in their sample.

Although some initial studies suggest that, for children with ADHD, atypically long RTs may occur in a predictable time course roughly once every 20 seconds, the current study extends this work by examining whether these patterns are specific to tasks with high working memory demands or are also seen on a go/no-go task with low working memory demands.

Motivation and Intra-individual Variability

Currently, it is not clear whether intra-individual variability in RTs reflects a stable characteristic of cognitive processing or whether it is state or context dependent (e.g. indicative of inconsistent effort or motivation). Evidence suggests that children with ADHD are more reliant than non-ADHD controls on extrinsic motivators to monitor and guide their performance (Carlson, et al., 2002; Luman, et al., 2005). This motivational style could contribute to a lack of task engagement and result in significant intra-individual variability in the absence of extrinsic rewards. In addition, recent research suggests that children with ADHD are less sensitive to the reinforcing properties of *delayed* rewards (Kuntsi, Oosterlaan, & Stevenson, 2001; Sagvolden, et al., 1998; Solanto, et al., 2001; Sonuga-Barke, et al., 1992), and that this insensitivity to delayed rewards results in poor task engagement even on tasks where rewards are not explicitly being offered (Sonuga-Barke, 2002). If this is the case, then performance variability on a wide variety of tasks may be related to a lack of motivation or poor task engagement.

An integrated theory that accounts for both differences in neural efficiency and the impact of external behavioral contingencies is found in the default-mode interference hypothesis (Sonuga-Barke & Castellanos, 2007). This theory is based on the premise that spontaneous low-frequency activity in neural networks is typically associated with resting states, and this activity is suppressed when a person is faced with a specific task or challenge so that task-relevant brain activity can guide behavior (Castellanos, et al., 2005; Raichle & Snyder, 2007; Shulman, et al., 1997). According to the default-mode hypothesis, children with ADHD do not adequately suppress this resting-state activity, and so it exerts a greater than expected influence on their task performance, resulting in periodic attentional lapses and long RTs (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Castellanos, et al., 2005; Raichle & Snyder, 2007; Shulman, et al., 1997). Although the overall difficulty an individual may have suppressing this resting-state neural activity is thought to be a relatively fixed trait, the degree to which the activity is suppressed on any given task is hypothesized to be affected by motivation to do well on the task. That is, although it may be more difficult for children with ADHD to suppress this activity, which results in patterns of abnormally long RTs, the default-mode hypothesis suggests that with additional motivation, children with ADHD should be able to suppress this activity and show patterns of RTs similar to those of non-ADHD children.

Given the importance of understanding the effects of extrinsic motivation on variability, several studies have demonstrated general improvements in response variability for both children with ADHD and for non-ADHD controls when rewards or other motivational incentives are offered (Andreou, et al., 2007; Crone, Jennings, & van der Molen, 2003; Slusarek, Velling, Bunk, & Eggers, 2001; van Meel, Heslenfeld, Oosterlaan, Luman, & Sergeant, 2005). However, if children with ADHD and non-ADHD controls both respond equally to motivational incentives, then any initial differences in performance are maintained, and evidence is weak that children with ADHD were initially performing poorly due to poor effort. In contrast, the best evidence in support of a motivational hypothesis would be a significant Reward x Diagnosis interaction, in which the performance of children with ADHD improves to a greater degree than controls with equal motivation. Among studies that have examined the impact of motivation on RT variability, only two (Andreou, et al., 2007; Slusarek, et al., 2001) were able to document a significant Reward x Diagnosis interaction.

With recent research on patterns of variability suggesting that children with ADHD are more variable than non-ADHD control in specific frequency patterns, one possible explanation for a failure to find significant Reward x Diagnosis interactions is that standard deviations are not specific enough to detect group differences. That is, because standard deviations include variability due to both low and high frequency changes, differences occurring specifically at low frequencies may be difficult to detect, and so examining the effects of reward on specific frequency bands may provide a more sensitive measure to detect change.

Another factor that may contribute to contradicting findings of the effects of reward on variability is that some, but not all, of the studies used combinations of both reward and punishment contingencies within the same experimental tasks (Andreou, et al., 2007; Slusarek, et al., 2001). Although both reward and punishment rely on externally applied contingencies that motivate behavioral change, differences may exist in the processing of positive feedback (reward), negative feedback (punishment), or both. In fact, several studies have demonstrated that physiological response to reward differs between children with and without ADHD but that groups do not differ in their responses to punishment (Crone, van der Veen, et al., 2003; Iaboni, Douglas, & Ditto, 1997; Luman, Oosterlaan, & Sergeant, 2008). Further complicating the picture, van Duijvenvoorde et al. (2008) have provided psychophysiological evidence of a developmentally normative tendency for children younger than 13 to respond more strongly to positive than to negative feedback, and even to show poorer performance when punishment is used. Deterioration of performance in punishment conditions has also been replicated in groups of children with ADHD (Crone, van der Veen, et al., 2003).

Taken together, these studies suggest that isolating the effects of reward and punishment on behavioral variability is an important step to understanding factors that contribute to this variability. In addition to isolating contributions of different types of external behavioral contingencies, it is unclear whether general reductions in variability that may occur in reward conditions are related to changes in specific frequency bands, which has implications for our understanding of the neural substrates hypothesized to underlie this variability.

Hypotheses

Hypothesis 1

If larger than average fluctuations in RTs occur in a predictable time course for children with ADHD (as opposed to randomly throughout the task), then Fast Fourier Transform (FFT) analysis will identify greater power under the curve for children with ADHD than for non-ADHD controls at the largest peak(s) within the spectra (e.g. at 0.05 Hz & .075Hz; Castellanos, et al., 2005).

Hypothesis 2A

If children with ADHD and non-ADHD controls show similar decreases in variability in motivational conditions (main effect of Reward), this would suggest that lack of effort is not a specific contributor to RT variability in ADHD.

Hypothesis 2B

If variability of the RT sequence during motivational conditions has reduced power specifically in the .05Hz-.075Hz band (Group x Reward x Frequency interaction effect), this would be consistent with hypotheses that an underlying pattern of neural activation contributing to poor task performance results from a lack of engagement with the task.

Hypothesis 2C

Variability as indexed by SDRTs may be reduced for children with ADHD in reward conditions (Group x Reward interaction effect), even if variability in a specific frequency band is unchanged. This would imply that the underlying deficit is not related to lack of motivation, but that with additional motivation children with ADHD may find ways to partially compensate during task performance.

Method

Participants and Diagnostic Procedures

Children with (n=17) and without ADHD (n=20) who were between the ages of 8 and 12 were recruited through fliers and advertisements distributed in Centre County and Mifflin County, Pennsylvania. A parent or legal guardian provided written consent for each child, and the child's written assent was also obtained. All families first participated in an initial phone screen, and children were excluded if: (a) they did not speak English as a first language; (b) they had significant visual or hearing impairments that would interfere with their ability to complete testing; (c) if parents reported the presence of mental retardation, autism, or psychosis; or (d) if they were prescribed non-stimulant psychotropic medications (e.g. Strattera, antidepressants, antipsychotics) that could not be safely discontinued 24 hours prior to testing.

Diagnoses were determined in a multi-tiered process. For each child, both a parent/legal guardian and teacher completed the Behavioral Assessment System for Children, 2nd edition (BASC-2), the Conners' Rating Scale-Revised (CRS-R), and ADHD Rating Scale- IV (ADHD-RS-IV; see descriptions of screening questionnaires below). Based on these rating forms, children were screened into the study as *possible* ADHD-group participants if the parent *and* teacher rated them above the 85th percentile on any of the scales related to inattention, hyperactivity, oppositional behavior, or aggression. Children were screened into the study as *possible* controls if both the parent and teacher rate them below the 70th percentile on all of these indices.

Final diagnoses were determined by the primary caregiver's report on the Diagnostic Interview Schedule for Children, 4th edition (DISC-IV) following the DSM-IV criteria. That is, if diagnostic criteria for ADHD was met on the DISC-IV, including age of onset, chronicity, and impairment criteria, following DSM-IV field trials, ADHD subtype was determined using an "or" algorithm. In this algorithm, a symptom was counted as present if either the parent (on the DISC) or the teacher (on the ADHD-RS) endorsed it.

To be included as a non-ADHD control, children who were screened in using questionnaires as *possible* controls also had to have three or fewer attention symptoms *and* three or fewer hyperactivity symptoms using the "or" algorithm described above.

No exclusions were made for comorbid diagnoses because this would 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; Donnelly, Reimherr, Young, & Ginsberg, 2006).

Children completed a three-hour testing session that was part of the standard data collection procedure for a larger study, which included a 2-subtest short form (Block Design and Vocabulary) of the Wechsler Intelligence Scale for Children, 4th edition (WISC-IV), a tracking version of the Logan stop task, and the computerized go/no-go task with motivational incentives (see description of tasks below for more details on all measures used). Children were excluded if their estimated Full Scale IQ was below 80.

Subject Payment

Both teachers and families were compensated for their participation in the study. Teachers were paid $5.00 for their time completing the questionnaires. Families were compensated a total of $30.00 for completion of the questionnaires and their participation in the three-hour visit that was part of the larger study. Children who participated in the study were also offered monetary reward for correct responses during two blocks of the experimental task with the maximum possible payment totaling $6.50 ($0.05/correct response * 130 total responses). This reward amount was slightly greater than average amounts ($0.03/response) that have been previously demonstrated to produce behavior change and psychophysiological response in children with and without ADHD (e.g. Börger, et al., 1999; Crowell, et al., 2006; Slusarek, et al., 2001).

Description of Measures

BASC-2 (Reynolds & Kamphaus, 2004). The BASC-2 questionnaire covers both adaptive and problem behaviors for children between the ages of six and twenty-one. The child version of the form is for use with children ages six to eleven, and the adolescent version of the form is for children 12-21 years old. The BASC-2 has norms for both teacher and parent ratings of child behaviors. For the teacher ratings the test-retest reliabilities for the scales used in the current study range from .81- .93. For the parent ratings reliabilities range from .75- .92.

CRS-R (Conners', 2004). The CRS-R is an age- and gender-normed questionnaire for use with children three to seventeen years old. The items on the ratings scale correspond with the DSM-IV-R criteria for diagnosing ADHD. Parents completed the long-form of the questionnaire (80 items) and teachers completed a short form (28 items) of the questionnaire. For parents, test-retest reliabilities for each of the Conners' scales range from .57- .72. The test-retest reliabilities for teachers are from .47-.86.

ADHD-RS-IV (DuPaul, Power, Anastopoulos, & Reid, 1998). Each item on this scale reflects one of the DSM-IV criteria as closely as possible. Focusing on the last six months of behavior, parents and teachers rate each item on 4-point Likert scale from "never or rarely" to "very often." The test-retest reliabilities for teachers' ratings range from .88-. 90 and for parents from .78- .86.

*Diagnostic Interview Schedule for Children-IV (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000)***.** The DISC-IV is a standardized diagnostic interview to assess for presence of 30 different psychiatric disorders that commonly occur in childhood. Questions are based on the DSM-IV and ICD-10 criteria for each disorder. Test-retest reliabilities for the most common disorders on the parent-report version of the scale are: *ADHD*-.79, *ODD*- .54, *CD*- .43, *GAD*- .65, *MDD*- .66.

Wechsler Intelligence Scale for Children, 4th edition (Wechsler, 2003). The WISC-IV is a standardized and valid test of intelligence normed on a sample of 2,200 children between the ages of six and sixteen. All children completed five subtests on the WISC-IV during their visit. Subtests included Matrix Reasoning, Digit Span, Vocabulary, Letter-Number Sequencing, and Arithmetic. An estimated FSIQ for each child was calculated based on the Vocabulary and Matrix Reasoning subtests. The estimate obtained from this short-form of the WISC-IV correlates with FSIQ, r = .87. The test-re-test reliability of this short-form of the WISC-IV is .93. The Working Memory Index (WMI), which is a composite score based on the Digit Span and Letter-Number Sequencing subtests, was also calculated for each child. The test-retest validity of the WMI is r=.89.

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 1000 ms. On 75% of trials ("go" trials), children were asked to indicate with a key press whether an "X" or an "O" (keys "." and "," respectively) appeared in the center of the screen. Stickers were placed over the appropriate symbols. Children were given 2300 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. 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.

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 re-calculated 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 5 blocks of 40 experimental trials with optional rest periods between blocks. Reliability (α) of SSRT between the five blocks of the task was 0.85.

Experimental Go/No-go Task. All children completed a go/no-go task programmed in Eprime 2.0 and presented in four blocks of 65 trials. Each block lasted

162 seconds. Children were required to press a button on the keyboard as quickly as possible to visually presented go stimuli (80% of trials) and withhold responses to visually presented no-go stimuli (20% of trials). Stimuli were 10 letters in two of the blocks (no-go trial was “K”) and 10 numbers in the other two blocks (no-go trial was “5”). All stimuli were presented on screen for 300ms. A fixed 2700ms ISI followed the presentation of the stimulus, during which time responses were recorded and feedback was provided (see Figure 1). In order to allow as much natural variability in RTs as possible, children were allowed to respond on go trials up to 2000ms after presentation of the stimulus. Feedback was provided on screen for 500ms immediately following the response during the 2700ms ISI. If a response was not registered by the 2000ms time limit, then it was marked as an incorrect response and feedback to this effect was provided for the standard 500ms. Children performed relatively accurately on the task and so this affected only between 0-11% of trials (or between 0-21 RT) for each individual child over the course of the entire task. Blocks of 162 seconds allow frequency resolution down to .012 Hertz (81 seconds per cycle) and an ISI of three seconds allows frequency resolution up to .20 Hertz (5 seconds per cycle).

The first two blocks of the task were considered “Baseline” blocks, for which no incentives were offered to improve performance. On these blocks children only received feedback about whether their responses were correct (green circle) or incorrect (red square). The second two blocks were “Reward” blocks during which monetary incentives were provided for correct responses. Children earned $0.05 for each correct go or no-go response. Again, the use of monetary incentives in this amount has previously been found to be salient enough to produce behavioral changes in children with and without ADHD who were a similar age to children in the current study (e.g. Börger, et al., 1999; Crowell, et al., 2006; Slusarek, et al., 2001). After each trial in the Reward blocks, children received feedback, as well as their total earned reward amount. The use of numbers versus letters in the reward block was counterbalanced within groups. Dependent variables analyzed for the current study include mean RT (MRT) on go trials (correct responses only), standard deviation of RT (SDRT) on correct go trials, as well as the time series sequence of RTs.

Although the lack of counterbalancing reward and baseline blocks presents a possible limitation within the study design, previous research suggests that long periods of non-reward that occur after a period of reinforcement lead to negative affect and increased skin conductance responses, and that children with ADHD may be especially sensitive to these effects (Brenner, Beauchaine, & Sylvers, 2005; Douglas & Parry, 1994; Latzman, Knutson, & Fowles, 2006; Sagvolden, et al., 1998; Sonuga-Barke, 2003). For this reason, counterbalancing conditions would be likely to prevent data from the two baselines being aggregated for data analysis. One concern about a lack of counterbalancing is that children with ADHD may have deficits in sustained attention that would naturally lead to declines in performance or increases in variability across tasks. While this possibility cannot be fully addressed within the context of this study, the short breaks between blocks, including time spent talking with the examiner (when directions are given) removed many of the demands on sustained attention that would be present in more traditional go/no-go tasks.

Data Analysis

Hypothesis 1

Mean Variability. Between-group comparisons of MRTs and SDRTs for correct responses were compared in one-way analysis of variance tests (ANOVA).

Patterns of Variability. Fast Fourier Transform (FFT) analyses were used to determine if RT variability for either children with ADHD or non-ADHD controls is driven by fluctuations at a specific frequency. FFT analyses require that the number of observations be a power of two, and so the sequence of RTs was padded with zeros to achieve this (Bloomfield, 2000). RTs for the no-go responses and errors of omission were extrapolated with linear interpolation based on the responses directly proceeding and following the no-go trial. Any child who made more than 20% omission errors during the task would have been excluded from analyses; however, no child made this number of omission errors. The time series of response times in each condition was concatenated and then subjected to frequency domain analysis. Although detrending is often used to remove linear trends that violate assumptions of stationarity within the data, detrending was not applied to the current dataset. Detrending removes low frequency variability that can be attributed either to changes in the MRT or to low-frequency fluctuations over the course of the task, and is therefore not always recommended when examining low-frequency patterns in reaction times (Bloomfield, 2000; Johnson, et al., 2007). Further, detrending is intended to reduce variance within the time-series, however, in the current dataset, detrending resulted in increased variance for both children with and without ADHD. When detrending leads to increased variability, it is recommended that it not be applied to the data (Anderson, 1996).

From the resulting spectra, the frequency of the largest relative maximum was identified at .055Hz, which is consistent with previous research (Castellanos, et al., 2006;

Johnson, et al., 2007). Power at .055Hz in the baseline condition was then compared across groups in a one-way ANOVA test. A 2 (high v. low frequency) x 2 (ADHD v. Non-ADHD Controls) ANOVA was also performed on the baseline data to determine whether increased power was specific to low frequency patterns.

Hypothesis 2A & 2B

Reward & RT Variability. SDRTs and power at the .055 Hz frequency were compared between groups and conditions using 2 x 2 (Group x Reward) ANOVA.

Power analysis

For main effects of variability, the sample size provided adequate power (power= .80) for detecting effect size equal or greater than η^2= 0.11, which is consistent with previous effect sizes for between group differences in variability (Klein, et al., 2006; Lijffijt, et al., 2005). For proposed interaction effects, the sample size allowed for the adequate detection (power= .80) of effect sizes as small as η^2= 0.04. Because the sample size in the current study is only able to detect relatively large effects, η^2 values will be considered in addition to p values in the interpretation of results. Effect sizes obtained in the current study will be used to identify areas for future research and larger studies.

Results

Sample Characteristics

Children with ADHD were rated as significantly more hyperactive and inattentive than controls by both parents and teachers on measures with direct correspondence to DSM-IV symptoms of ADHD (CRS-R Parent Long Form, CPRS-R Teacher Short Form; all $p < .001$). Both parents and teachers also rated children with ADHD as more oppositional than controls (all $p < .05$) and parents rated children with ADHD as marginally more anxious that non-ADHD controls (p= .08). The ADHD and control groups did not differ in estimated full scale IQ or age (all $p > .858$; see Table 1). Four of the children in the ADHD group were currently taking medications to manage symptoms of ADHD (one taking short-acting and three taking long-acting stimulant medications). For all children taking stimulant medication, a parent/guardian confirmed that they had not taken medications for at least 24 hours prior to testing.

Task Performance

Use of either letters or numbers for the reward blocks of the task was counterbalanced within group to ensure that equal numbers of children with and without ADHD were represented in each condition. Task performance, as indicated by overall accuracy and accuracy specifically in the reward block did not differ based on order of stimuli presented (all $p > .688$).

Between-subjects ANOVA indicated a significant group difference in task accuracy in which children with ADHD were less accurate than non-ADHD controls in both reward and non-reward blocks, $F(1,36)= 15.99$, $p < .001$, $\eta^2= .308$ (see Table 2). Repeated measures ANOVA comparing mean accuracy in the non-reward and reward blocks indicated no significant main effect of Reward, $F(1,36)= 2.43$, $p= .128$, $\eta^2= .063$, and no Reward x Group interaction, $F(1,36)= 0.09$, $p= .764$, $\eta^2= .003$. The lack of a significant main effect or interaction effect indicates that neither group's accuracy was significantly impacted by reward, which may be due to a ceiling effect in that all children achieved high levels of accuracy even in the non-reward blocks of the task (92% accuracy for children with ADHD and 96% accuracy for non-ADHD controls).

Between-subjects tests revealed a trend in which children with ADHD responded somewhat more slowly than non-ADHD controls, $F(1,36)= 3.00$, $p= .092$, $\eta^2= .077$ (see Table 2). In repeated measures ANOVA comparing RT in non-reward and reward blocks, neither the main effect of Reward nor the Reward x Group interaction were significant (all $p > .327$). However, given the small sample size, one-way ANOVA tests were also conducted and indicated a trend toward children with ADHD being slower than non-ADHD controls in the reward block, $F(1,37)= 3.09$, $p= .087$, $\eta^2 = .08$, but not in non-reward blocks. Between-subjects tests indicated that children with ADHD were not more variable than non-ADHD controls, $F(1,36)= 0.68$, $p= .414$, $\eta^2= .019$, and repeated-measures ANOVA indicated no main effect of Reward on variability and no Reward x Group interaction (all $p > .395$).

Speed (MRT) and accuracy were not correlated in either the reward or non-reward blocks (all $p > .271$), indicating no speed-accuracy trade-off occurring in either condition.

FFT Analyses

In visual examination of the FFT spectra for children with ADHD, a relative maximum was observed at 0.055 hertz (see Figure 1). Given that a peak at this frequency is consistent with previous research (Castellanos, et al., 2005; Di Martino, et al., 2008), power at this frequency was used as a dependent variable in the all analyses. For comparison, 0.10 hertz was used as a measure of high-frequency variability, which corresponds changes in RT over roughly five trials.

Between-subjects tests examining low-frequency power in the baseline blocks indicated that children with ADHD showed more power at .055 Hz than children without ADHD, $F(1,32)= 5.25$, $p= .029$, $\eta^2= .141$. Repeated measures ANOVA comparing baseline power at low and high frequencies for each group indicated a significant main effect of frequency in which all children showed greater power at low frequencies than high frequencies, $F(1,36)= 8.73$, $p= .005$, $\eta^2= .195$. Although the interaction effect was non-significant, a medium effect size for the Frequency x Group interaction suggests a trend in which non-ADHD controls had only a small difference in power between low frequency and high frequency variability, while children with ADHD showed a much greater difference between power at low and high frequencies, $F(1,36)= 2.54$, $p= .121$, $\eta^2= .073$ (see Figure 2).

When effects of reward were examined using repeated-measure ANOVA, there was no main effect of Reward on power at low or high frequencies, $F(1,36)= 0.63$, $p= .434$, $\eta^2= .019$, and no Reward x Frequency interaction, $F(1,36)= 0.84$, $p= .368$, $\eta^2= .025$, indicating that introduction of monetary rewards did not impact patterns of variability. No significant Reward x ADHD or Reward x ADHD x Frequency interactions were observed, $F(1,36)= 0.32$, $p= .860$, $\eta^2= .001$ and $F(1,36)= 0.48$, $p= .828$, $\eta^2= .002$, respectively, indicating no differential impact of reward based on whether or not a child had ADHD (see Figure 3).

Regression Analysis

Given previous research suggesting that intra-individual variability is most prominent on tasks with high working memory demands, regression analyses were conducted to explore the relationship of low-frequency variability to task performance and executive functioning. In all analyses, ADHD status and power at 0.10 hertz in the non-reward blocks were entered in the first step of the regression (to control for group differences in low-frequency power and associations with variability generally) and power at .055 hertz in the non-reward blocks was entered into the second step of the regression. Power at 0.055 hertz was not related to overall task accuracy or specifically to accuracy in the non-reward blocks (all $p > .410$, all $R^2\Delta < .015$). Low-frequency power in the non-reward blocks predicted a child's performance on the Working Memory Index of the WISC-IV, $R^2\Delta = .118$, $p = .047$, but it did *not* predict performance on a task measuring response inhibition (Logan stop task; see Table 2 for information about working memory and stop task performance). Together, the specific relationship of low-frequency power to working memory is consistent with previous suggestions that excessive low-frequency power may particularly impact tasks with high working memory demands.

Discussion

Traditional analyses of the go/no-go task used for the current study indicated that children with ADHD were less accurate than non-ADHD controls. Children with ADHD also responded more slowly than non-ADHD controls, with a trend towards especially slow responding in the reward as compared to the non-reward blocks. Slower responding is often interpreted as poorer performance on cognitive tasks, which would suggest that children with ADHD's performance deteriorated in the reward condition. However, MRTs are affected by a variety of internal processes, including a not only cognitive and motor speed but also the conservativeness of response criterion. For example, children who respond conservatively may have slow RTs because they wait to be certain of their response, even if their cognitive processing speed and motor speed are within normal limits. This is described as the speed-accuracy trade-off (SATO), and presents a problem for interpreting MRTs (Sergeant & van der Meere, 1990). With a SATO framework in mind, children with ADHD slowing their RTs in the reward block may indicate that they are attempting a more conservative response strategy (i.e. attempting to improve their performance). Despite slowing down, children with ADHD were not able to improve their accuracy in the reward blocks and no correlations between speed and accuracy were observed in either condition. However, even children with ADHD demonstrated greater than 90% accuracy in the non-reward blocks, and so a ceiling effect may have limited prevented detection of significant differences in accuracy between conditions or relationships between speed and accuracy.

Children with ADHD were not more variable than non-ADHD controls based on SDRT, the traditional measure of variability. However, SDRT collapses both slow patterns of change in RTs and trial-by-trial variability in RTs into a single measure. Several previous studies suggest that the greatest differences in variability between children with ADHD and non-ADHD controls may be at low frequencies (Castellanos, et al., 2006; Castellanos, et al., 2005; Di Martino, et al., 2008; Johnson, et al., 2007). Consistent with previous research, FFT analyses and the resulting power spectrum indicated that variability at .055 Hz made the largest contribution to overall patterns of variability for children with ADHD. Further, although children with ADHD showed more variability than non-ADHD controls at both high and low frequencies, a trend for the Group x Frequency interaction indicated that the greatest difference between groups was at low frequencies, and the effect size for the group difference at .055 Hz was large.

The default-mode hypothesis suggests that children with ADHD have difficulty suppressing low-frequency neural oscillations associated with a resting state in order to fully engage in a task, and that these low-frequency patterns are reflected in low-frequency variability in RTs (Castellanos, et al., 2006). In this context, the Group x

Frequency interaction found in the current study in which children with ADHD had especially high levels of low-frequency variability as compared to non-ADHD controls, supports the hypothesis that children with ADHD may not fully transition from a resting state to an actively engaged state when completing tasks.

Default-mode theory also suggests that both biological traits and contextual states can impact the degree of resting state activation; however, this hypothesis has not been formally tested in either behavioral or physiological studies. In the current study, the introduction of monetary rewards did not impact the degree of low-frequency variability for either children with or without ADHD. One explanation is that children were not motivated by amount of money offered for each response; however, the amount of reward used in this study exceeded amounts previously shown to produce behavioral and physiological change in children with and without ADHD (Börger, et al., 1999; Crowell, et al., 2006; Slusarek, et al., 2001). Further, the fact that children with ADHD slowed their RTs in the reward block suggests that they were adjusting their strategy in response to the reward. Thus, motivation appears to exert relatively little impact on patterns low-frequency variability in RTs, possibly suggesting that trait rather than state factors are the primary determinants of how well a child can shift from a resting to an actively engaged state.

Children with ADHD showed the greatest power at .055 Hz, which is consistent with previous studies (Castellanos, et al., 2005). The relative height of the peak at .055 Hz was consistent with other studies that have used go/no-go tasks; however, the peak was much less pronounced than in studies using tasks with high working memory demands (Castellanos, et al., 2005; Di Martino, et al., 2008). Power at .055 Hz significantly predicted children's performance on the Working Memory Index from the WISC-IV above both diagnostic status and high-frequency variability, but did not predict performance on a Logan stop task that measures motor response inhibition. No strong theory currently exists to explain why resting state activity would most strongly affect working memory tasks. One possibility is that the appearance of a stimulus in a go/no-go task is a natural alerting cue so that there is relatively little demand for a child to maintain active engagement on his/her own. In contrast, in working memory tasks, the child is responsible for independently maintaining information in mind with no external prompts to aid them. In the absence of external cues, children with ADHD may have a particularly difficult time maintaining active task engagement. Alternatively, the difference in low-frequency power between tasks with low- and high- working memory demands may actually reflect the fact that working memory tasks are often more difficult than go/no-go tasks. Additional studies that control for difficulty while manipulating working memory demands will be necessary to disentangle these possibilities.

Limitations & Future Directions

The current study represents a first step in identifying contextual factors that contribute to patterns of variability for children with ADHD. The current study relied on previous research to identify an amount of monetary reward that was large enough to motivate children with and without ADHD; however, there is no validation to confirm that the reward manipulation worked. In future studies, measurement of physiological responses specific to reward sensitivity, such as pre-ejection period, may help validate reward manipulations, as well as contribute to the field's understanding of reward sensitivity in children with ADHD.

In addition, the current study specifically used only reward, rather than reward and punishment, to incentivize behavioral change. To provide a full understanding of how contextual factors do or do not contribute to patterns of variability, future studies should test multiple motivational conditions, including both reward and punishment. In addition, tangible reinforcers are not the only type of motivational incentive that can be used, and in real-world contexts social reinforcement is one of the most powerful reinforcers available. Future studies may use social reinforcement contingencies to better understand the effects of this type of reinforcement on the behavior of children with ADHD.

Finally, although patterns of RT variability are hypothesized to be related to underlying neural oscillations, this has not been specifically tested in this or other studies. For this reason, assessing behavioral patterns of RT variability while simultaneously collecting physiological measures of low-frequency neural oscillations (e.g. with EEG) and/or activation in the default networks believed to underlie RT variability (e.g. with fMRI) will be critical for determining whether these mechanisms account for patterns of RT variability.

STUDY 2

Attention and Intra-individual Variability

An underlying premise of Study 1 is that RT variability (SDRT) is an indicator of attention, with increased RT variability indicating trial-to-trial variability in attention. This assumption is consistent with previous theory identifying RT variability as an index of phasic alertness, which is an individual’s readiness to respond at on any single trial (Huang-Pollock, Nigg, & Halperin, 2006; Mirsky, Pascualvaca, Duncan, & French, 1999). However, relatively little research has been conducted to validate RT variability as an index of attention by relating it to physiological indices.

Evidence that is available to validate RT as an index of attention is primarily from studies in healthy adults which indicates that behavioral indices of inattention, such as omission errors and slower than average reaction times, are associated with reduced BOLD response in top-down attentional control areas, including the right prefrontal cortex (Weissman, Roberts, Visscher, & Woldorff, 2006). Converging evidence from adults with traumatic brain injury also indicates that damage to the right prefrontal cortex is associated with attentional lapses and increased RT variability (Rueckert & Grafman, 1996; Wilkins, Shallice, & McCarthy, 1987; Zahn & Mirsky, 1999). Further, Hahn, Ross, & Stein (2007) found that for adults, RT on any specific trial of a simple RT task was related to level of activation in the anterior and posterior cingulate cortices and the superior temporal gyrus, which they interpreted as evidence of RT variability being an index of attentional control.

Although evidence from adult samples suggests that RT variability is associated with changes in BOLD activity in areas of the brain believed to be responsible for attentional control, it is less clear whether the same mechanisms account for RT variability in children. Factor analysis of performance on executive function tasks indicates that executive functions are highly related but separable processes in adult samples (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). However, this differentiation of executive functions appears to develop across childhood, such that children's scores on tests of working memory and response inhibition are more highly correlated at age 5 than at age 10 (Tsujimoto, Kuwajima, & Sawaguchi, 2007). The reason for this increased differentiation is unclear. It is possible that synaptic pruning that occurs throughout childhood and adolescence (Diamond, 2002) results in more specialized, specific neural networks. It is equally likely that younger children's performance on a wide variety of tasks may be influenced by broad factors such as motivation or ability to regulate behavior, causing the scores to be highly related regardless of the underlying process assessed. In either case, this pattern makes it difficult to unilaterally assume that variability in RT is specifically a measure of attention in children, even if this relationship is valid in adult populations.

In addition, immature fine motor development may play a larger role than attention in children's RT variability. The contribution of motor immaturity to RT variability is of particular concern because children with ADHD frequently exhibit problems in fine motor control (Piek, et al., 2004; Pitcher, Piek, & Barrett, 2002) that might present as slower and more variable RT and thus be falsely interpreted as evidence of attentional deficits. Given the myriad of alternative processes that could explain RT variability in children, it is critical to clarify the cognitive processes underlying this behavioral measure.

In addition to concerns about generalizing findings from adult samples to children, the current adult literature linking RT variability to attention has done little to clarify which aspects of attention contribute to RT variability. In the clinical literature "attention" is often used as a non-specific term that can refer to anything from being attentive to details in an assignment to startle in response to a loud sound. Cognitive models of attention can be used to better specify which of the multiple processes that are subsumed under the "attention" label may contribute to RT variability. Posner's model of attention, in particular, outlines three components that can be behaviorally and physiologically differentiated from each other (Fan, McCandliss, Sommer, Raz, & Posner, 2002): alerting, orienting, and executive control (Posner, 1992; Posner & Petersen, 1990).

Using Posner's model, alerting is defined as the ability to achieve and maintain an "alert" state in which the individual is ready to respond to the presentation of a stimulus (Posner & Petersen, 1990). In the Attention Network Task (ANT), designed to provide separate behavioral measures of each of the three types of attention described in his model, alerting is measured by comparing reaction times in cued and uncued response conditions with the assumption that individuals who are best at achieving an alert state will show the most benefit from having a cue that a target is about to occur (Fan, et al., 2002; Rueda, et al., 2004). Of note, although Posner argues that vigilance, or maintaining an alert state over time, is supported by the same neuroanatomical networks that support alerting, the short interval between the cue and the target in his task prevent specific testing of the vigilance system (Huang-Pollock, et al., 2006).

Posner & Peterson (1990) define orienting as an individual's ability to direct their attention towards the most relevant areas of the environment based on previous sensory cues. Stated another way, orienting refers to an individual's ability to use information from the environment to strategically select and attend to additional information. In the ANT, orienting is measured by comparing reaction times in conditions in which a cue provides information about where a target will occur to conditions where the cue does not provide this information. Individuals who are better at orienting are hypothesized to benefit the most from the congruent cue. Finally, in Posner's model, the executive control network is recruited in cases in which conflict between two response options needs to be resolved, and is assessed as the difference in reaction times between trials with congruent and incongruent cues.

Although RT variability could be created by variability in any of the three processes described, it has most often been considered a measure of alerting (Huang-Pollock, et al., 2006; Mirsky, et al., 1999). Specifically, standard deviation of RTs has been described as a measure of phasic alertness (i.e. the ability to achieve an alert state at any given moment), and increases in RT standard deviation over the course of a long task are thought to reflect vigilance (i.e. the ability to maintain the alert state over time). This conceptualization is consistent with findings that children with ADHD (Drechsler, Brandeis, Földényi, Imhof, & Steinhausen, 2005) and patients with traumatic brain damage (Stuss, Murphy, Binns, & Alexander, 2003) show higher RT variability than controls even on simple RT tasks, which place demands on alerting but do not require orienting based on cues or conflict resolution. Taken together, previous studies suggest that if RT variability in children does reflect an aspect of attention, it may be best characterized as an index of alerting. However, convergent validity through simultaneous assessment of behavioral and physiological measures of alerting has not been conducted with children.

Cardiac Indices of Attention

Measures of cardiac functioning are some of the least invasive physiological measurements that can be obtained, making them ideal for research with children, and several measures of cardiac functioning have been used as indices of attentional control (Berntson, Quigley, & Lozano, 2007). Although early work identified aspects of cardiac functioning, particularly heart rate variability, as correlates of performance on cognitive measures of attention, this work lacked a theory explaining why autonomic function and cognitive processes such as attention would be linked (Porges, 1992). Porges (1992) described a theoretical model for how and why cardiac measures are related to aspects of selective attention and attentional capacity. He noted broadly that central and peripheral nervous system activities are linked, and he specifically highlighted that the vagus nerve has afferent and efferent connections to both the heart and brainstem, providing a feedback loop between central nervous system functioning and the peripheral nervous system. More recent work in neuroimaging has supported Porges' model of central nervous system and cardiac functioning and has further provided evidence of a dependent relationship between vagal withdrawal and activity in the prefrontal cortex, emphasizing the interdependent nature of peripheral nervous system responses and higher-level cortical functioning (Thayer & Lane, 2009).

At rest, vagal control inhibits heart rate, helping to maintain a slow, steady pace (Berntson, et al., 1997; Berntson, et al., 2007). However, when environmental changes are introduced, vagal withdrawal results in increased heart rate and suppression of "rest-and-digest" processes, which allows the individual to achieve an "attentive" state (Porges, 1992). Within this framework, cardiac function can be used as an index of attention because reciprocal responding in the central and peripheral nervous systems aids an individual in processing sensory information in the environment and organizing a response. Porges specifically noted that a person's capacity to attend should be related to their *basal* vagal activity because individuals with low vagal control over the heart would have less ability to withdraw vagal input in response to environmental demands (Porges, 1992). In support of this model, research has demonstrated correlations between cardiac functioning and tasks measuring attention, such as continuous performance tasks and visual cancellation tasks (Duschek, Muckenthaler, Werner, & Reyes del Paso, 2009; Hansen, Johnsen, & Thayer, 2003; Hickey, Suess, Newlin, Spurgeon, & et al., 1995; Porges, 1972, 1973; Suess, Porges, & Plude, 1994). In addition, a dose-dependent relationship has been found between drugs that suppress vagal activity and continuous performance measures of attention (Dellinger, Taylor, & Porges, 1987).

Although researchers to-date have generally not specified the specific component of attention that is supported by vagal withdrawal, two facts suggest that vagal withdrawal may be most important when an individual needs to achieve and maintain an *alert* state. Porges' (1992) specified that vagal withdrawal can occur in response to exogenous cues (e.g. when a person hears a loud noise) or in response to endogenous cues (e.g. a desire to pay attention to a long, boring computer task you have been asked to complete). Although Porges' himself used the term "orienting" to describe the attentional process that vagal withdrawal is associated with, he described the process of achieving a state in which an individual is prepared to process environmental information, which is akin to Posner's description of alerting. Further, orienting and executive control as described in Posner's model both occur on a time scale measured in milliseconds (Fabiani, Gratton, & Federmeier, 2007), whereas vagal withdrawal and the effects on heart rate and the peripheral organs occur in a time scale measured in seconds (Berntson, et al., 2007). Given this difference, vagal withdrawal is not likely to be a primary contributor to orienting or executive control but could play a large role in alerting.

Quantifying vagal control over cardiac functioning can be accomplished relatively simply because vagal input is the primary determinant of well-described rhythmic changes in heart rate in time with the breathing cycle, known as respiratory sinus arrhythmia (RSA; Berntson, et al., 2007). These rhythmic changes in heart rate are measured using time-series analyses and defined power in a high-frequency band (with the specific band determined by a person's age and respiratory pace and depth (Berntson, et al., 1997; Berntson, et al., 2007). Changes in RSA are mediated by input from the vagus nerve, and strong vagal control of the heart at rest is associated with high baseline RSA (Berntson, et al., 1997; Berntson, et al., 2007). Although other measures of cardiac function, such as mid-frequency variability in heart rate, have also been used to index various components of attention, RSA was recently demonstrated to account for the greatest proportion of variance in performance on a cancellation task that placed high demands on alerting (Duschek, et al., 2009), which is consistent with Porges' model positing that vagal influences are critical determinants of this aspect of attention.

One possible approach to validating reaction time variability as an index of alerting in children would be to correlate trial-by-trial changes in reaction times to trial-by-trial changes in RSA. However, as described above, vagal influences on RSA are relatively slow when compared to the speed of cognitive processing, and so this approach is not practical. Instead, baseline RSA can be used as a measure of a person's capacity for alerting that should be negatively correlated with RT variability (SDRT), such that higher baseline RSA is associated with lower SDRT.

EEG Indices of Attention

Event-related electroencephalogram (i.e. event-related potentials; ERP) is a measure of changes in neural activity that occur in response to a specific event, and event-related changes in neural firing provide an another possible physiological index of components of attention (Fabiani, et al., 2007). Traditionally, research using ERPs has examined the latency and/or peak activity of an average waveform that is determined by averaging all neural activity occurring within a specified time window (Fabiani, et al., 2007). However, recent research has emphasized that the average waveform results from overlapping signals that oscillate at different frequencies (Karakas, Erzengin, & Basar, 2000; Schnitzler & Gross, 2005; Womelsdorf & Fries, 2007). Further, neural oscillations at specific frequencies, which occur when electrical activity becomes synchronized across multiple neuroanatomical areas, may represent a mechanism for communication within neural networks (Bressler & Menon, 2010; Schnitzler & Gross, 2005). For this reason, understanding the oscillatory pattern in response to a stimulus may be a more specific way to describe relationships between cognitive functioning and neural activity than looking at average ERP waveforms. EEG recordings are more difficult to collect in children than are measures of cardiac functioning because EEG recordings are more susceptible to movement artifacts and because placement of a large number of electrodes on the head (as opposed to only a small number on the torso as is needed to measure cardiac functioning) is relatively more invasive. Nonetheless, EEG measures provide a way of quantifying aspects of attention *during* task completion that is complimentary to cardiac measures of baseline functioning.

Previous research using the ANT task and Posner's model of attention has suggested that alerting may be related to changes in theta-, alpha-, and beta-band activity following presentation of a warning cue (Fan, et al., 2007); however, in the same study changes in activity in these bands was also reported for trials requiring response conflict resolution, indicating that beta activity may not be specific to alerting. Additional research examining EEG correlates of performance on a vigilance task, which places demands on the alerting network, indicates that adults show greater beta activation on correct as compared to incorrect trials (Arruda, Amoss, Coburn, & McGee, 2007; Bearden, Cassisi, & White, 2004).

Providing more specific evidence of a relationship between beta activity and alerting, Basile et al. (2007) examined changes in neural activity that occurred in the inter-stimulus interval between a cue and a target. They found that the most prominent change was an increase in beta band activity that was specific to the post-cue, pre-target interval. In the paradigm used by Basile et al., the cue served as a warning that a target would appear but provided no spatial information about the target location, and so the attention component being measured in the interval between the cue and the target corresponds most closely to Posner's alerting component of attention. The time specificity of changes in beta during the inter-stimulus interval following a cue suggests that increases in beta activity may relate specifically to alerting.

In addition to predicted relationships between baseline measures of RSA and SDRT, a significant correlation between SDRT and beta activity occurring during correct go trials of a go/no-go task would lend strong support to the interpretation of RT variability as an index of alerting. Understanding these cognitive processes in typically-developing populations will be an important first step in characterizing possible atypical variability in these measures that may be present for children with ADHD.

Hypotheses

Hypothesis 3A & 3B

If RT variability is a measure of alertness, then SDRT on a go/no-go task should be negatively correlated with baseline measures of RSA such that higher baseline RSA is associated with lower SDRT. In addition, SDRT should be negatively correlated with beta band activity during correct go responses. If RT variability does not specifically reflect alerting, then neither RSA nor beta band activity should predict SDRT.

Method

Participants

Children (n=26) who are between the ages of 9 and 16 were recruited for a larger study through fliers and advertisements distributed in Centre County, Pennsylvania. A parent or legal guardian provided written consent for each child, and the child's written assent was also obtained. Children were excluded from participation if their parent reported: (a) the presence of any DSM-IV-TR diagnosis or (b) a history of seizures or a seizure disorder, as well as if the child (c) had significant visual or hearing impairments that would interfere with their ability to complete testing or (d) were prescribed non-stimulant psychotropic medications (e.g. Strattera, antidepressants, antipsychotics) that could not be safely discontinued 24 hours prior to testing.

Subject Payment

Families were paid $25.00 for their participation in the 2-hour testing session. In addition, children received a toy or gift card valued at $15.00.

Procedure

Children attended a single 2-hour testing session during which time they completed a go/no-go task (described in more detail below) while EEG and autonomic data, including heart rate and impedance cardiography were collected.

Motivational Go/No-Go Task. The go/no-go task was adapted from a motivational go/no-go task used by Dr. Jim Stieben and colleagues (e.g. Stieben, et al., 2007). The task requires participants to press a button for each stimulus presented but to avoid pressing the button when a stimulus is repeated twice in a row. Stimuli in the current version were characters from the *Mr. Men and Little Miss* series (for example see Figure 5).

The task was presented in 5 blocks of 110 trials. In all five blocks children won points for correct responses and lost points for incorrect responses; however, in three of the blocks (first, third, and fifth) stimulus duration and point algorithms were manipulated to ensure that children won points and in the other two (second and fourth) to ensure that children lost points (see detailed description below). At the beginning of the game, children were shown two sets of prizes, one set of “small” prizes (plastic figurines worth < $0.50 each) and another with “large” prizes (toys and gift cards worth ~$15.00 each). They were instructed that to earn the large prizes, they needed to earn a high number of points, more than the “average child their age.” Children were allowed to look at the prizes and to pick one that they might want at the end. The error rate for no-go trials was maintained between 40-60% by dynamic adjustment of the stimulus duration based on the child’s level of performance. On each correct no-go trial that was preceded by a correct go trial, the stimulus presentation time was decreased, whereas on each incorrect no-go trial the stimulus presentation time was increased. The specific duration increases and decreases depended on block. In the “win points” blocks, the stimulus time was either increased or decreased by 50ms based on the child’s response. In “lost points” blocks, stimulus presentation time was increased by only 30ms for incorrect responses and presentation time was decreased by 60ms after correct responses. These dynamic adjustments were meant to ensure similar level of challenge and number of errors for all children. The floor and ceiling times for stimulus duration were 300 and 1,000 ms, respectively.

Children received performance feedback in the form of a red box that appeared on the screen each time they made an incorrect response. In addition, every 20 trials their accumulated points were displayed on the screen. Points were added for correct no-go responses and deducted for response errors on both go and no-go trials. The algorithm for adjusting points in the “win points” blocks was +50 for correct responses and -10 for errors. In “lose points” blocks the algorithm was +15 for correct responses and -55 for incorrect responses.

For two reasons, the analyses for the current study were conducted using the behavioral and physiological responses during the first block of the task only. First, as described in Study 1, children and adolescents may respond differently to reward and punishment conditions, and so collapsing responses across these conditions would introduce an unintended confound in interpreting results. Second, examination of RTs and SDRTs revealed that children became less variable in their responding over the course of the five blocks of the task; however, the cause of this decrease in variability was unclear. It is possible that there was a practice effect such that children became less variable as they adjusted to the task. However, because the dynamic adjustments to the stimulus presentation duration intended to maintain accuracy between 40-60% also affected the time allowed for a child to respond, it may have artificially eliminated long reaction times. That is, as the task shortened the stimulus presentation duration, what may have been recorded in the first block as a long reaction time would instead be recorded as an omission error. Given this confound, and supported by examination of the RTs and SDRTs, which were substantially below those typically observed in standard go/no-go paradigms, the first block was selected for analysis because early in the task children's RTs and SDRTs should be the least impacted by the adjustment of the stimulus presentation duration.

Electrophysiological Data Collection and Analysis. EEG data were acquired using the NetStation 4.2 data acquisition program and a 128-channel Geodesic Sensor Net (Electrical Geodesics, Eugene, Or) with Cz as a reference channel. Impedances were maintained at <50kΩ. Signals were amplified with a 16-bit analog-to-digital converter at a sampling rate of 500 samples per seconds.

Data pre-processing and analysis was completed using Brain Vision Analyzer 2.0 software (© Brain Products GmbH). Bad channels were removed from analysis and channels were re-referenced to an average of all remaining channels. A 50Hz low-pass filter was applied to the raw EEG and correction for eye movements and artifact rejection were completed using automated detection algorithms available in Analyzer 2 with visual confirmation of results. Data were segmented based on stimulus presentation (stimulus-locked) and only correct go trials were included in analysis. Baseline correction for each trial was made based on activity 50ms prior to the stimulus presentation. Finally, fast-Fourier transform algorithms available in Analyzer 2 were used to calculate power in the beta range (defined as 13 Hz-30 Hz; Fabiani, et al., 2007)) during each trial for each participant. Each child's beta activity was then calculated by averaging across trials.

Respiratory sinus arrhythmia (RSA). The ECG was recorded with Ag/AgCl spot electrodes in a modified Lead II configuration with active leads over the distal right collarbone and the lower left rib. Data were sampled and stored for analysis at 500 Hz using a Mindware Ambulatory Impedance Cardiograph. Mindware software was used to automatically detect and label R waves, and the series of heartbeats was then visually inspected and artifacts were corrected. Missing beats were estimated as the mid-point of the two nearest beat to preserve the time series.

The time series was then divided into 30 second segments, and RSA for each segment was quantified using automatic Fast-Fourier Transform (FFT) algorithms available in the Mindware HRV software package as the power in the high frequency band from 0.24 Hz to 1.04 Hz, which is the band recommended for determining RSA in children (Quigley & Stifter, 2006). Segments with less than 30 seconds of data were excluded from analyses. Baseline RSA values were determined by averaging all usable segments for each participant within the two-minute baseline prior to block 1 of the task.

Data Analysis

Hypothesis 3A & 3B

Baseline RSA scores were correlated with SDRT for all participants in a regression analysis. In a separate regression analysis beta activity was also correlated with SDRT. Because recent evidence suggests that the scalp location of beta activity during alerting his reliable within individuals but highly variable between individuals, and that maximum activity can occur either frontally or centrally(Basile, et al., 2007), beta activity at both Fz and Cz were correlated with SDRT.

Power analysis

The sample size provided adequate power (power= .80) only for detecting effect sizes at the high end of the medium range ($\eta^2 = .10$). Similar to Study 1, η^2 values will be considered in addition to *p* values in the interpretation of results due to relatively low power based on the sample size. Effect sizes obtained in the current study will be used to identify areas for future research and larger studies.

Results

Descriptive Characteristics

Sample characteristics including age, parent reported behavioral symptoms on the BASC-2, percentage of correct hits, mean reaction time (MRT), standard deviation of reaction time (SDRT), and baseline RSA are presented in Table 5. Consistent with parent report that children enrolled in the study did not carry DSM-IV diagnoses, parent-report on the BASC-2 indicated that all children in the sample scored below the 85^{th} percentile (*T*-score= 60) on an overall index of internalizing and externalizing symptoms.

Repeated measures ANOVA indicated that children demonstrated a significant decrease in RSA between baseline and block 1 of the go/no-go task, $F(1,25)= 34.51$, $p < .001$, $\eta^2 = .580$. Neither baseline RSA, MRT, nor SDRT were correlated with age (all $p > .482$); however, beta activity at Fz, $r^2 = .252$, $p = .018$, and Cz, $r^2 = .588$, $p = .010$, was significantly correlated with age (see Table 6). The group of children showing maximum beta activity at Fz was significantly younger than the group showing maximum activation at Cz, $F(1,10)= 7.64$, $p = .020$, $\eta^2 = .433$.

Relationships Among Physiological and Behavioral Measures

Baseline RSA showed a trend towards association with SDRT r^2= .125, *p*= .077, with the r^2 value indicating a medium effect size (see Figure 6). EEG data was only available for a subset of children in the sample (*n*=16). The children for whom useable EEG was available did not differ significantly from the children without useable EEG data in age, task performance (hits, MRT, SDRT), parent-reported behavioral symptoms, or baseline RSA (all $p > .404$, all $\eta^2 < .029$; see Table 5). Beta activity at Fz was not correlated with SDRT, r^2= .038, *p*= .440, nor was beta activity at Cz, r^2= -.182, *p*= .470; however, the r^2 value for the relationship between beta activity at Cz and SDRT indicated a medium effect size (see Figure 7).

Discussion

SDRT has traditionally been interpreted as reflecting attention, specifically phasic alertness, which is defined as moment-to-moment changes in attention that affect the RT for any given trial of a task. This interpretation has been supported by physiological studies in adults (Hahn, et al., 2007; Rueckert & Grafman, 1996; Weissman, et al., 2006; Zahn & Mirsky, 1999); however, the assumption has not been previously validated for children. Further, the term "attention" can refer to a wide variety of processes, any one of which could influence RT variability. The current study sought to validate SDRT as an index of the alerting as described in Posner's model of attention (Posner, 1992; Posner & Petersen, 1990) using two physiological measures of alerting: baseline RSA and beta activity during correct go trials.

Results from the current study provide partial support for SDRT as a measure of alerting. Specifically, SDRT and baseline RSA were correlated (albeit with *p*-values in the range of a trend rather than significant), which is consistent with Porges' (1992) suggestion that strong vagal control of the heart, as indicated by high baseline RSA, should provide individuals with room for physiological flexibility that will facilitate their ability to achieve an alert state. R^2 values for the correlation indicated a medium effect size for this relationship, lending weight to the interpretation of the trend as indicating a relationship between RSA and SDRT.

Although beta power, which is also believed to reflect the alerting component of attention (Arruda, et al., 2007; Bearden, et al., 2004; Fan, et al., 2007), did not correlate significantly with SDRT, the effect size for the relationship between beta activity at Cz and SDRT fell in the medium range. Only a subgroup of the total sample had usable EEG data, which greatly reduced the power to detect relationships between EEG and behavioral measures, and so the medium effect size suggests that beta activity may be related to SDRT in a larger sample of children.

Interestingly, beta activity at Fz did not correlate with SDRT and the group of children showing maximum activation at Fz was significantly younger than the group showing maximum activation at Cz. In adults, beta activity associated with alerting has previously been demonstrated have significant inter-individual variability in scalp location (Basile, et al., 2007); however, the relationship with age within the adult sample was not tested. One possible explanation of the lack of relationship between beta activity at Fz and SDRT is that alerting is not a primary determinant of SDRT for the younger children who showed maximum activity at this scalp location but is a primary determinant for older children who showed maximum activity at Cz. Given small size of the sample in the current study and the large age range, tests of this hypothesis are not possible at this time. Future studies using large groups of children within tighter age ranges will be critical for identifying the determinants of RT variability across a broad developmental range. In addition, it is not clear whether beta activity at Fz and Cz are both related to alerting but that maximum activity differs by age or whether children are completing the task using different strategies, which is reflected in different patterns of activation recorded at the scalp. Studies using tasks that dissociate alerting from other response processes in a large sample of children across a large age range will be important for disentangling these possibilities.

Finally, activity in the current study was averaged across the entire "go" trial. Although achieving and maintaining an alert state is necessary throughout the entire trial (and, in fact, the entire task), activity averaged across the entire trial likely combines activity specific to alerting with activity related to other mental activities. The short duration and inter-stimulus interval of each trial precluded using beta activation from only the pre-stimulus interval, which may have provided the cleanest measure of alerting. Future studies with longer inter-stimulus intervals may be better suited to detect relationships between EEG measures and SDRTs.

Limitations & Future Directions

Despite limitations described for the current study, it provides an important first attempt to more narrowly define the cognitive processes underlying RT variability in children. Convergent evidence from multiple behavioral and physiological indicators of alerting may be one of the strongest empirical methods for defining RT variability, and understanding the processes that contribute to RT variability in typical development will aid in our understanding of atypical cognitive development that occurs in a variety of disorders, including ADHD.

Several additional steps will be important in clarifying process that contribute to RT variability in ADHD. First, Study 1 indicates that the largest contributor to RT variability for children with ADHD is low-frequency changes in RTs. The variable inter-stimulus interval in the current study did not provide a time-series of RTs that could be submitted to frequency domain analyses; however, future studies should examine whether patterns of variability are associated with physiological measures of alerting in either typically-developing children or children with ADHD. Understanding the underlying cognitive and physiological contributors to RT variability will be critical if RT variability is to be used as an endophenotype of ADHD (Bidwell, et al., 2007; Castellanos, et al., 2005). In addition, cognitive training interventions and biofeedback treatments targeting EEG frequency bands are gaining attention as treatments for ADHD (for review see Xu, Reid, & Steckelberg, 2002). Understanding the specific cognitive processes impaired in children with ADHD and the specific physiological correlates will be critical in designing and implementing the most effective treatments for the disorder.

Table 1

Study 1: Description of Sample

Variable	Control (n=20)	ADHD (n=17)	F	η^2
Gender (M:F)	11:9	11:6	χ^2=.585	---
Age	10.79 (1.20)	10.66 (1.26)	0.10	.003
FSIQ	107.33 (11.71)	106.65 (11.59)	0.03	.001
Conners'-R Parent Ratings				
Oppositional Behavior	43.33 (3.59)	54.07 (8.84)	22.26^{***}	.418
Hyperactivity	45.50 (2.99)	61.07 (10.59)	35.70^{***}	.535
Cognitive Problems/ Inattention	44.72 (3.29)	71.07 (11.79)	82.55^{***}	.727
Anxiety	46.61 (5.75)	53.20 (14.18)	3.62^{+}	.095
Conners'-R Teacher Ratings				
Oppositional Behavior	46.72 (2.69)	55.40 (15.57)	5.43^{***}	.149
Hyperactivity	45.50 (2.87)	61.40 (14.18)	21.71^{***}	.412
Cognitive Problems/ Inattention	45.50 (4.00)	61.00 (13.26)	22.30^{***}	.418

Note. Data reported as mean (SD). ***= *p*< .001, + = non-significant trend with p< .08

Table 1

Table 2

Study 1: Task Performance Measures

Variable	Control (n=20)	ADHD (n=17)	F	η^2
Go/No-go Non-Reward Condition				
Accuracy (%)	96 (2.7)	92 (5.4)	13.20**	.292
MRT (ms)	448.55 (59.01)	466.49 (41.65)	1.12	.030
SDRT (ms)	232.33 (42.93)	237.53 (52.70)	.112	.003
Go/No-go Reward Condition				
Accuracy (%)	95 (2.6)	92 (4.4)	19.34***	.423
MRT (ms)	446.34 (50.75)	486.35 (87.99)	3.09+	.079
SDRT (ms)	228.79 (37.21)	245.35 (48.48)	1.42	.038
Logan Stop Task				
Hits (% correct)	97 (4.2)	89 (7.7)	11.67**	.302
MRT	731.72 (139.63)	687.11 (82.68)	1.11	.040
SDRT	270.94 (98.64)	394.29 (126.75)	8.47**	.239
WISC-IV Working Memory Index	107.05 (11.79)	100.59 (12.75)	2.62	.068

Note. Data reported as mean (SD). **= p< .01, + = non-significant trend with p= .08

Table 3

Study 2: Description of Sample

Variable	Complete Sample (*n*=26)	Subset with usable EEG Data (*n*=16)
Age	12.1 (2.2)	12.25 (2.4)
BASC-2 Behavioral Symptoms Index	51.19 (9.09)	50.17 (9.84)
Behavioral Responses on Go/No-go Task		
Hits (% Correct)	91.71 (10.27)	90.85 (9.06)
MRT (ms)	337.38 (41.00)	331.59 (39.99)
SDRT (ms)	135.58 (82.18)	126.65 (63.77)
Baseline RSA	6.96 (0.96)	6.83 (0.86)

Note. Data reported as mean (SD)

Table 4

Study 2: Correlations between Task Performance, Age, and Baseline RSA.

Variable	1	2	3	4	5
1. Age	---				
2. MRT (ms)	-.043	---			
3. SDRT (ms)	-.096	.075	---		
4. Baseline RSA	.121	-.135	$-.353^{+}$	---	
5. Beta (Fz)	$.545^{*}$	-.089	.194	-.175	---
6. Beta (Cz)	$.660^{**}$	-.182	.006	.063	$.736^{***}$

Note. *=- $p < .05$, ** $p < .01$, *** $p < .001$, + = non-significant trend with p= .08.

Figure 1

Study 1: Go/No-go Task Parameters

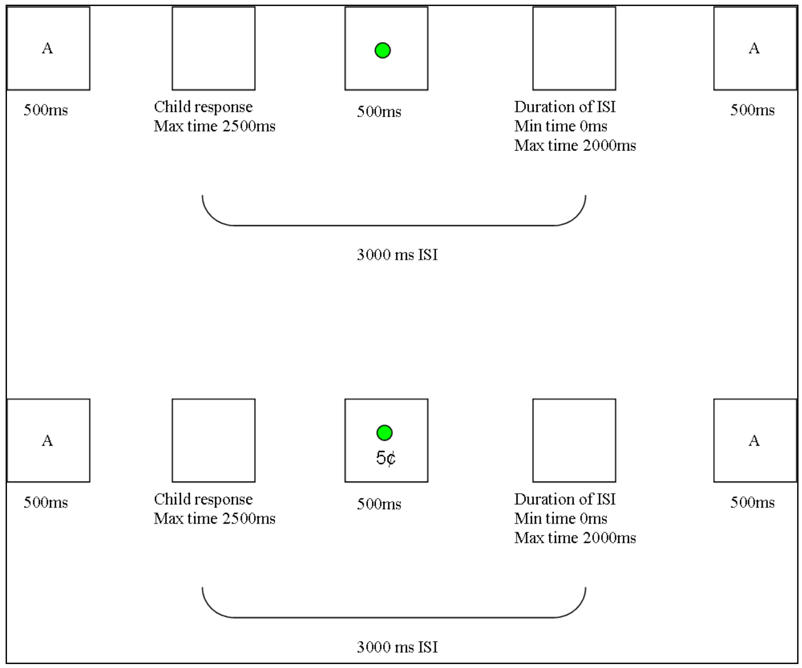


Figure 2

Study 1: Power Spectrum in Non-Reward Condition.

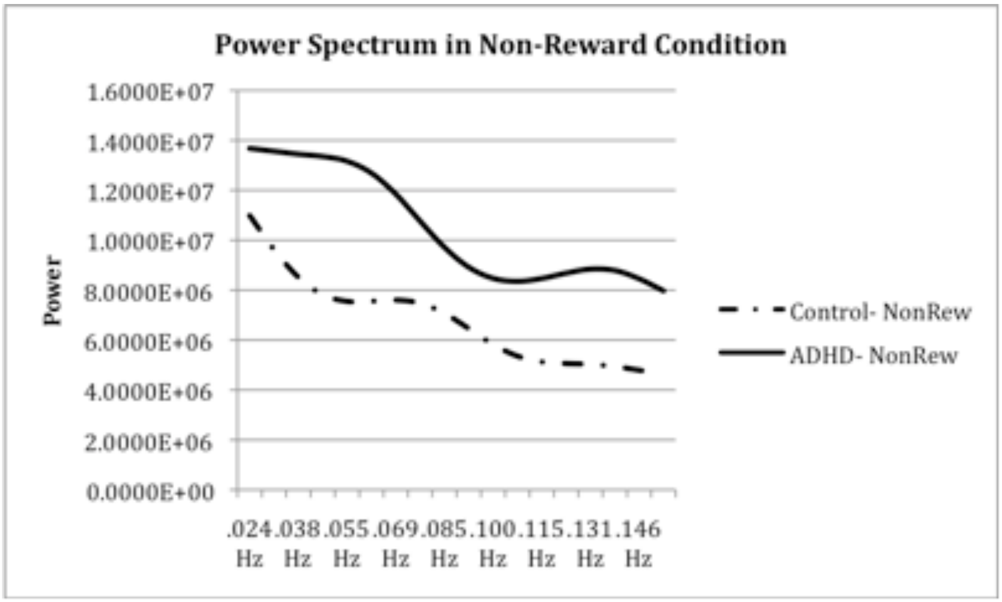


Figure 3

Study 1: Power at Low and High Frequencies in Non-Reward Block.

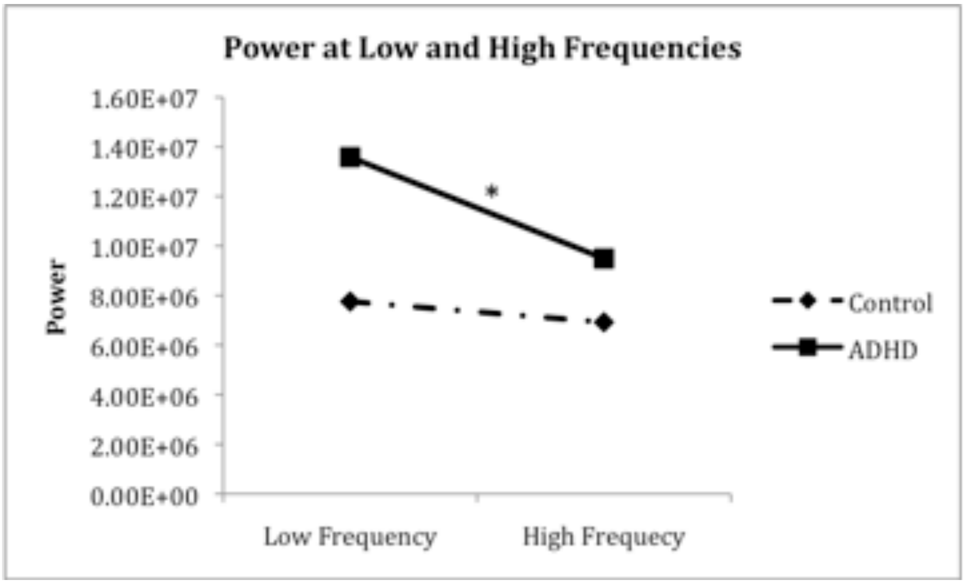


Figure 4

Study 1: Impact of Reward on Low Frequency Power.

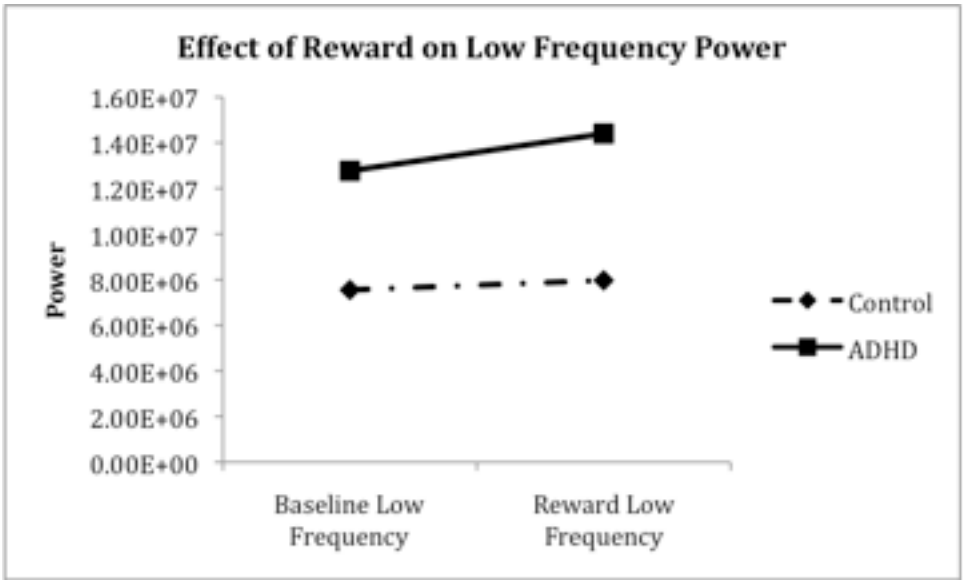


Figure 5

Study 2: Example of Little Mr. and Little Miss Figures Used in Task.

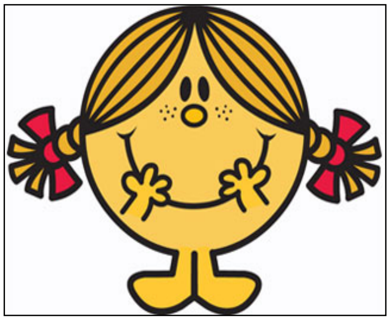


Figure 6

Study 2: Correlation Between SDRT and Baseline RSA

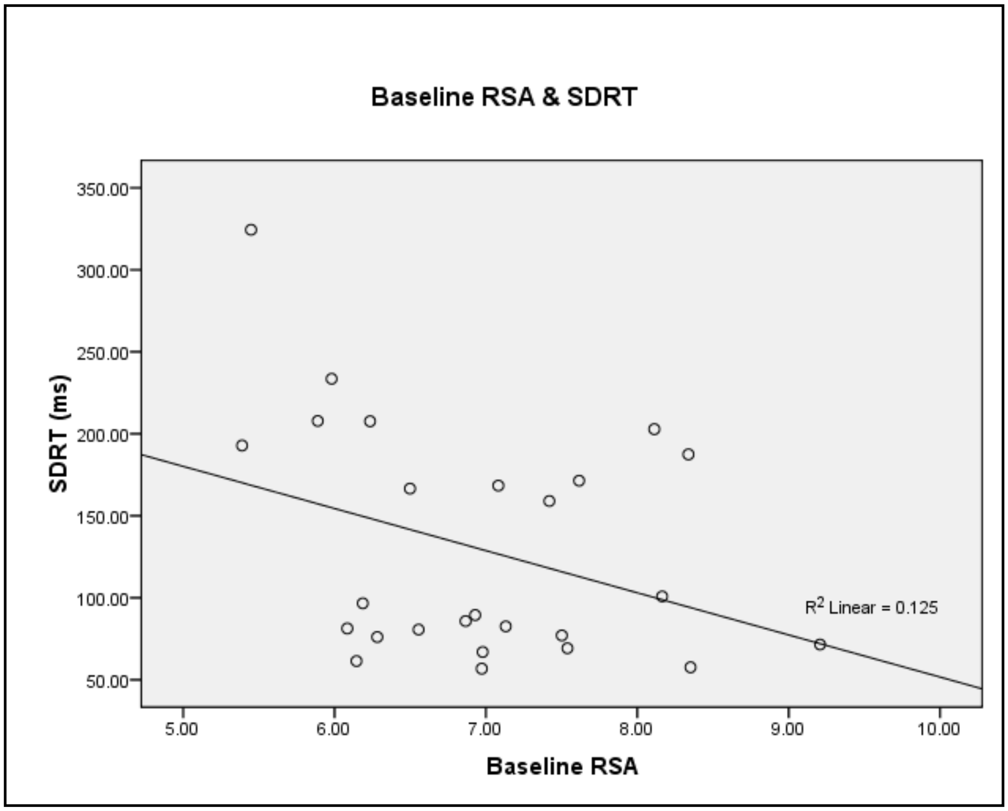
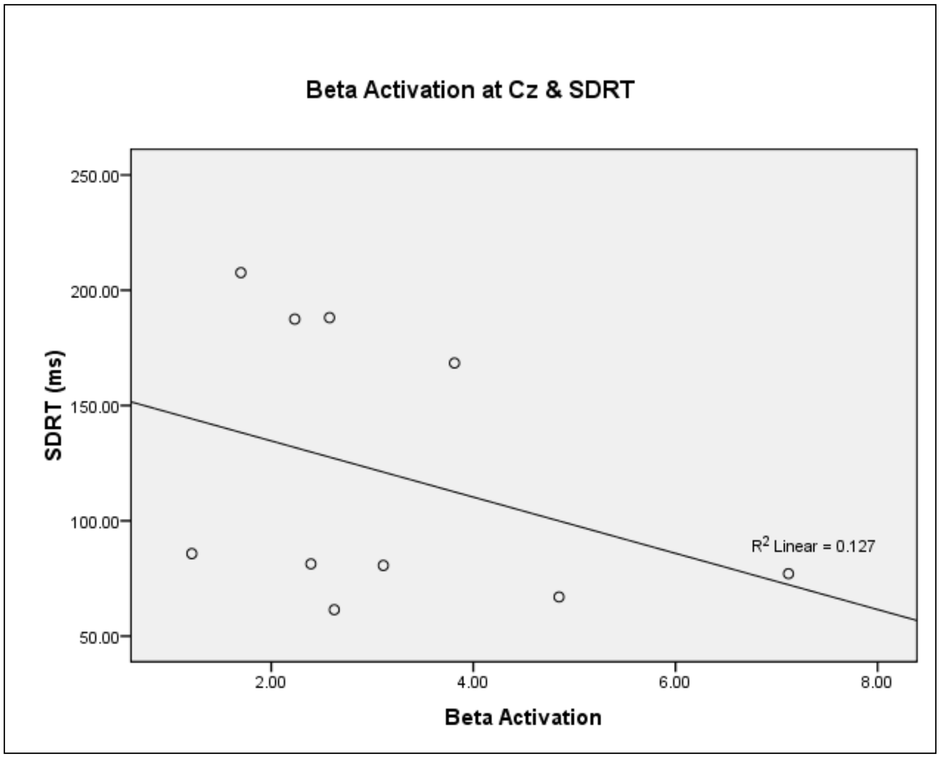


Figure 7

Study 2: Correlation Between SDRT and Beta Activity at Cz



REFERENCES

Aase, H., & Sagvolden, T. (2006). 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. Special Issue: Annual research review, 47*(5), 457-471.

Anastopoulos, A. D., Guevremont, D. C., Shelton, T. L., & DuPaul, G. J. (1992). Parenting stress among families of children with attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 20*(5), 503-520.

Anderson, O. (1996). *Time series analysis and forecasting: the Box-Jenkins approach*. London: Butterworths.

Andreou, P., Neale, B. M., Chen, W., Christiansen, H., Gabriels, I., Heise, A., et al. (2007). Reaction time performance in ADHD: Improvement under fast-incentive condition and familial effects. *Psychological Medicine, 37*(12), 1703-1715.

APA. (2000). *Diagnostic and statistical manual of mental disorders* (4th (text rev.) ed.). Washington, DC: Author.

Aron, A., & Poldrack, R. (2005). The Cognitive Neuroscience of Response Inhibition: Relevance for Genetic Research in Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry, 57*, 1285-1292.

Arruda, J., Amoss, T., Coburn, K., & McGee, H. (2007). A quantitative electroencephalographic correlate of sustained attention processing. *Applied Psychophysiology and Biofeedback, 32*, 11-17.

Baeyens, D., Roeyers, H., & Walle, J. V. (2006). Subtypes of Attention-Deficit/Hyperactivity Disorder (ADHD): Distinct or Related Disorders Across Measurement Levels? *Child Psychiatry and Human Development, 36*, 403-417.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Basile, L., Anghinah, R., Ribeiro, P., Ramos, R., Piedade, R., Ballester, G., et al. (2007). Interindividual variability in EEG correlates of attention and limits of functional mapping. *International Journal of Psychophysiology, 65*, 238-251.

Bearden, T. S., Cassisi, J. E., & White, J. N. (2004). Electrophysiological correlates of vigilance during a continuous performance test in healthy adults. *Applied Psychophysiology and Biofeedback, 29*(3), 175-188.

Bellgrove, M. A., Hawi, Z., Kirley, A., Gill, M., & Robertson, I. H. (2005). Dissecting the attention deficit hyperactivity disorder (ADHD) phenotype: Sustained attention, response variability and spatial attentional asymmetries in relation to dopamine transporter (DAT1) genotype. *Neuropsychologia, 43*(13), 1847-1857.

Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. [Journal; Peer Reviewed Journal]. *Psychophysiology, 34*(6), 623-648.

Berntson, G. G., Quigley, K. S., & Lozano, D. (2007). Cardiovascular psychophysiology. New York, NY, US: Cambridge University Press.

Berwid, O. G., Kera, E. A. C., Marks, D. J., Santra, A., Bender, H. A., & Halperin, J. M. (2005). Sustained attention and response inhibition in young children at risk for attention deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 46*(11), 1219-1229.

Bidwell, L. C., Willcutt, E. G., DeFries, J. C., & Pennington, B. F. (2007). Testing for neuropsychological endophenotypes in siblings discordant for attention-deficit/hyperactivity disorder. *Biological Psychiatry. Special Issue: Autism and attention deficit hyperactivity disroder, 62*(9), 991-998.

Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., et al. (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.

Bloomfield, P. (2000). *Fourier analysis of time series: an introduction* (2nd ed.). New York, NY: John Wiley & Sons, Inc.

Börger, N., van der Meere, J., Ronner, A., Alberts, E., Geuze, R., & Bogte, H. (1999). Heart rate variability and sustained attention in ADHD children. *Journal of Abnormal Child Psychology, 27*(1), 25-33.

Brenner, S. L., Beauchaine, T. P., & Sylvers, P. D. (2005). A comparison of psychophysiological and self-report measures of BAS and BIS activation. *Psychophysiology, 42*(1), 108-115.

Bressler, S. L., & Menon, V. (2010). Large-scale brain networks in cognition: Emerging methods and principles. *Trends in Cognitive Sciences, 14*(6), 277-290.

Carlson, C. L., Booth, J. E., Shin, M., & Canu, W. H. (2002). Parent-, teacher-, and self-rated motivational styles in ADHD subtypes. *Journal of Learning Disabilities, 35*(2), 104-113.

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.

Castellanos, F. X., Sonuga-Barke, E. J. S., Scheres, A., Di Martino, A., Hyde, C., & Walters, J. R. (2005). Varieties of Attention-Deficit/Hyperactivity Disorder-Related Intra-Individual Variability. *Biological Psychiatry, 57*(11), 1416-1423.

Chhabildas, N., Pennington, B. F., & Willcutt, E. G. (2001). A Comparison of the Neuropsychological Profiles of the DSV-IV Subtypes of ADHD. *Journal of Abnormal Child Psychology, 29*(6), 529-540.

Crone, E. A., Jennings, J. R., & van der Molen, M. W. (2003). Sensitivity to interference and response contingencies in attention-deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 44*(2), 214-226.

Crone, E. A., van der Veen, F. M., van der Molen, M. W., Somsen, R. J. M., van Beek, B., & Jennings, J. R. (2003). Cardiac concomitants of feedback processing. *Biological Psychology, 64*(1-2), 143-156.

Crosbie, J., Pérusse, D., Barr, C., & Schachar, R. (2008). Validating psychiatric endophenotypes: inhibitory control and attention deficit hyperactivity disorder. *Neuroscience & Biobehavioral Reviews, 32*, 40-55.

Crowell, S. E., Beauchaine, T. P., Gatzke-Kopp, L., Sylvers, P., Mead, H., & Chipman-Chacon, J. (2006). Autonomic Correlates of Attention-Deficit/Hyperactivity Disorder and Oppositional Defiant Disorder in Preschool Children. *Journal of Abnormal Psychology, 115*(1), 174-178.

Dellinger, J., Taylor, H., & Porges, S. W. (1987). Atropine sulfate effects on aviator performance and on respiratory-heart period interactions. *Aviation, Space, and Environmental Medicine, 58*(4), 333-338.

Di Martino, A., Ghaffari, M., Curchack, J., Reiss, P., Hyde, C., Vannucci, M., et al. (2008). Decomposing intra-subject variability in children with attention-deficit/hyperactivity disorder. *Biological Psychiatry, 64*(7), 607-614.

Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry. New York, NY, US: Oxford University Press.

Diamond, A. (2005). 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. Special Issue: Integrating Cognitive and Affective Neuroscience and Developmental Psychopathology, 17*(3), 807-825.

Donnelly, C. L., Reimherr, F. W., Young, J. L., & Ginsberg, D. L. (2006). Differential Diagnosis and Treatment of Adult ADHD and Neighboring Disorders. *Primary Psychiatry, 13*(10), 1-16.

Douglas, V. I., & Parry, P. A. (1994). Effects of reward and nonreward on frustration and attention in attention deficit disorder. *Journal of Abnormal Child Psychology, 22*(3), 281-302.

Doyle, A. E., Faraone, S. V., Seidman, L. J., Willcutt, E. G., Nigg, J. T., Waldman, I. D., et al. (2005). Are endophenotypes based on measures of executive functions useful for molecular genetic studies of ADHD? *Journal of Child Psychology and Psychiatry, 46*(7), 778-803.

Doyle, A. E., Willcutt, E. G., Seidman, L. J., Biederman, J., Chouinard, V.-A., Silva, J., et al. (2005). Attention-Deficit/Hyperactivity Disorder Endophenotypes. *Biological Psychiatry, 57*, 1324-1335.

Drechsler, R., Brandeis, D., Földényi, M., Imhof, K., & Steinhausen, H. (2005). The course of neuropsychological functions in children with attention deficit hyperactivity disorder from late childhood to early adolescence. *Journal of Child Psychology and Psychiatry, 46*(8), 824-836.

Dumas, M. C. (1998). The risk of social interaction problems among adolescents with ADHD. *Education & Treatment of Children, 21*(4), 447-460.

Duschek, S., Muckenthaler, M., Werner, N., & Reyes del Paso, G. A. (2009). Relationships between features of autonomic cardiovascular control and cognitive performance. *Biological Psychology, 81*(2), 110-117.

Epstein, J. N., Conners, C. K., Hervey, A. S., Tonev, S. T., Arnold, L. E., Abikoff, H. B., et al. (2006). Assessing medication effects in the MTA study using neuropsychological outcomes. *Journal of Child Psychology and Psychiatry, 47*(5), 446-456.

Epstein, J. N., Erkanli, A., Conners, C. K., Klaric, J., Costello, J. E., & Angold, A. (2003). Relations Between Continuous Performance Test Performance Measures and ADHD Behaviors. *Journal of Abnormal Child Psychology, 31*(5), 543-554.

Erhardt, D., & Hinshaw, S. P. (1994). 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, 62*(4), 833-842.

Fabiani, M., Gratton, G., & Federmeier, K. D. (2007). Event-related brain potentials: Methods, theory, and applications. In J. T. Cacioppo, L. G. Tassinary & G. G.

Berntson (Eds.), *Handbook of psychophysiology* (Vol. 3, pp. 85-119). New York: Cambridge University Press.

Fan, J., Byrne, J., Worden, M., Guise, K., McCandliss, B. D., Fossella, J., et al. (2007). The relation of brain oscillations to attentional networks. *The Journal of Neuroscience, 27*(23), 6197-6206.

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14*(3), 340-347.

Geurts, H., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J. A. (2005). ADHD subtypes: do they differ in their executive functioning profile? *Archives of Clinical Neuropsychology, 20*, 457-477.

Greene, R. W., Beszterczey, S. K., Katzenstein, T., Park, K., & Goring, J. (2002). Are students with ADHD more stressful to teach? Patterns of teacher stress in an elementary school sample. *Journal of Emotional and Behavioral Disorders, 10*(2), 79-89.

Haenlein, M., & Caul, W. F. (1987). Attention deficit disorder with hyperactivity: A specific hypothesis of reward dysfunction. *Journal of the American Academy of Child & Adolescent Psychiatry, 26*(3), 356-362.

Hahn, B., Ross, T. J., & Stein, E. A. (2007). Cingulate activation increases dynamically with response speed under stimulus unpredictability. *Cerebral Cortex, 17*(7), 1664-1671.

Hansen, A. L., Johnsen, B. H., & Thayer, J. F. (2003). Vagal influence on working memory and attention. [Journal; Peer Reviewed Journal]. *International Journal of Psychophysiology, 48*(3), 263-274.

Hervey, A. S., Epstein, J. N., Curry, J. F., Tonev, S., Arnold, L. E., Conners, C. K., et al. (2006). Reaction time distribution analysis of neuropsychological performance in an ADHD sample. *Child Neuropsychology, 12*(2), 125-140.

Hickey, J. E., Suess, P. E., Newlin, D. B., Spurgeon, L., & et al. (1995). Vagal tone regulation during sustained attention in boys exposed to opiates in utero. [Journal; Peer Reviewed Journal]. *Addictive Behaviors, 20*(1), 43-59.

Huang-Pollock, C. L., & Nigg, J. T. (2003). Searching for the attention deficit in attention deficit hyperactivity disorder: The case of visuospatial orienting. *Clinical Psychology Review, 23*(6), 801-830.

Huang-Pollock, C. L., Nigg, J. T., & Halperin, J. M. (2006). Single dissociation findings of ADHD deficits in vigilance but not anterior or posterior attention systems. *Neuropsychology, 20*(4), 420-429.

Iaboni, F., Douglas, V. I., & Ditto, B. (1997). Psychophysiological response of ADHD children to reward and extinction. *Psychophysiology, 34*(1), 116-123.

Johnson, K. A., Kelly, S. P., Bellgrove, M. A., Barry, E., Cox, M., Gill, M., et al. (2007). Response variability in Attention Deficit Hyperactivity Disorder: Evidence for neuropsychological heterogeneity. *Neuropsychologia, 45*(4), 630-638.

Karakas, S., Erzengin, U., & Basar, E. (2000). A new strategy involving multiple cognitive paradigms demonstrates that ERP components are determined by the superposition of oscillatory responses. *Clinical Neurophysiology, 111*(10), 1719-1732.

Klein, C., Wendling, K., Huettner, P., Ruder, H., & Peper, M. (2006). Intra-Subject Variability in Attention-Deficit Hyperactivity Disorder. *Biological Psychiatry, 60*(10), 1088-1097.

Kuntsi, J., Oosterlaan, J., & Stevenson, J. (2001). Psychological mechanisms in hyperactivity: I Response inhibition deficit, working memory impairment, delay aversion, or something else? *Journal of Child Psychology and Psychiatry, 42*(2), 199-210.

Latzman, R. D., Knutson, J. F., & Fowles, D. C. (2006). Schedule-induced electrodermal responding in children. *Psychophysiology, 43*, 623-632.

Leth-Steensen, C., Elbaz, Z. K., & Douglas, V. I. (2000). Mean response times, variability and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica, 104*(2), 167-190.

Lijffijt, M., Kenemans, J. L., Verbaten, M. N., & van Engeland, H. (2005). A Meta-Analytic Review of Stopping Performance in Attention-Deficit/Hyperactivity Disorder: Deficient Inhibitory Motor Control? *Journal of Abnormal Psychology, 114*(2), 216-222.

Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm In D. Dagenbach & T. Carr (Eds.), *Inhibitory processes in attention, memory, and language* (pp. 189-239). San Diego, CA: Academic Press.

Losier, B., McGrath, P., & Klein, R. (1996). Error patterns on the Continuous Performance Test in non-medicated and medicated samples of children with and without ADHD: A meta-analytic review. *Journal of Child Psychology and Psychiatry, 37*(8), 971-987.

Luman, M., Oosterlaan, J., & Sergeant, J. A. (2005). The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review, 25*(2), 183-213.

Luman, M., Oosterlaan, J., & Sergeant, J. A. (2008). Modulation of response timing in ADHD, effects of reinforcement valence and magnitude. *Journal of Abnormal Child Psychology, 36*(3), 445-456.

Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A Meta-Analysis of Working Memory Impairments in Children With Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(4), 377-384.

Milich, R., Balentine, A. C., & Lynam, D. R. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology: Science and Practice, 8*(4), 463-488.

Mirsky, A. F., Pascualvaca, D. M., Duncan, C. C., & French, L. M. (1999). A model of attention and its relation to ADHD. *Mental Retardation and Developmental Disabilities Research Reviews, 5*(3), 169-176.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49-100.

Nigg, J. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126*(2), 220-246.

Nigg, J., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. J. S. (2005). Causal Heterogeneity in Attention-Deficit/ Hyperactivity Disorder: Do We Need Neuropsychologically Impaired Subtypes? *Biological Psychiatry, 57*(11), 1224-1230.

Oades, R. D., & Christiansen, H. (2008). Cognitive switching processes in young people with attention-deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology, 23*(1), 21-32.

Piek, J. P., Dyck, M. J., Nieman, A., Anderson, M., Hay, D., Smith, L. M., et al. (2004). The relationship between motor coordination, executive functioning and attention in school aged children. *Archives of Clinical Neuropsychology, 19*(8), 1063-1076.

Pitcher, T. M., Piek, J. P., & Barrett, N. C. (2002). Timing and force control in boys with attention deficit hyperactivity disorder: Subtype differences and the effect of comorbid developmental coordination disorder. *Human Movement Science. Special Issue: Current Issues in Motor Control and Coordination, 21*(5-6), 919-945.

Porges, S. W. (1972). Heart rate variability and deceleration as indexes of reaction time. [Journal; Peer Reviewed Journal]. *Journal of Experimental Psychology, 92*(1), 103-110.

Porges, S. W. (1973). Heart rate variability: An autonomic correlate of reaction time performance. [Journal; Peer-Reviewed Status-Unknown]. *Bulletin of the Psychonomic Society, 1*(4), 270-272.

Porges, S. W. (1992). Autonomic regulation and attention. [Book; Edited Book; Chapter]. *Campbell, Harlene; Richardson, Rick (1992). Attention and information processing in infants and adults*, Perspectives from human and animal research.

Posner, M. I. (1992). Attention as a cognitive and neural system. [Journal; Peer Reviewed Journal]. *Current Directions in Psychological Science, 1*(1), 11-14.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. [Journal; Peer Reviewed Journal]. *Annual Review of Neuroscience, 13*, 25-42.

Quigley, K., & Stifter, C. A. (2006). A comparative validation of sympathetic reactivity in children and adults. *Psychophysiology, 43*, 357-365.

Raichle, M., & Snyder, A. (2007). A default mode of brain function: A brief history of an evolving idea. *Neuroimage, 37*(4), 1083-1090.

Rucklidge, J. J., & Tannock, R. (2002). Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry, 43*(8), 988-1003.

Rueckert, L., & Grafman, J. (1996). Sustained attention deficits in patients with right frontal lesions. *Neuropsychologia, 34*(10), 953-963.

Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., et al. (2004). Development of attentional networks in childhood. *Neuropsychologia, 42*(8), 1029-1040.

Sagvolden, T., Aase, H., Johansen, E. B., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences, 28*(3), 397-468.

Sagvolden, T., Aase, H., Zeiner, P. l., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavioural Brain Research. Special Issue: Attention deficit/hyperactivity disorder, 94*(1), 61-71.

Sagvolden, T., & Sergeant, J. A. (1998). Attention deficit/hyperactivity disorder: From brain dysfunctions to behaviour. *Behavioural Brain Research. Special Issue: Attention deficit/hyperactivity disorder, 94*(1), 1-10.

Schachar, R., Taylor, E., Wieselberg, M., Thorley, G., & et al. (1987). Changes in family function and relationships in children who respond to methylphenidate. *Journal of the American Academy of Child & Adolescent Psychiatry, 26*(5), 728-732.

Schnitzler, A., & Gross, J. (2005). Normal and Pathological Oscillatory Communication in the Brain. *Nature Neuroscience, 6*, 285-296.

Sergeant, J. A., & van der Meere, J. (1990). Additive factor method applied to psychopathology with special reference to childhood hyperactivity. *Acta Psychologica. Special Issue: Stage analysis of the reaction process: Models, methods and applications, 74*(2-3), 277-295.

Shaffer, D., Fisher, P., Lucas, C., Dulcan, M., & 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.

Shulman, G., Fiez, J., Corbetta, M., Buckner, R., Miezin, F., Raichle, M., et al. (1997). Common blood flow changes across visual tasks: II. Decreases in cerebral cortex. *Journal of Cognitive Neuroscience, 9*(5), 648-663.

Slusarek, M., Velling, S., Bunk, D., & Eggers, C. (2001). Motivational effects on inhibitory control in children with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(3), 355-363.

Solanto, M. V., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G. D., Wigal, T., et al. (2001). 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, 29*(3), 215-228.

Sonuga-Barke, E. (2002). Psychological heterogeneity in AD/HD--A dual pathway model of behaviour and cognition. *Behavioural Brain Research. Special Issue: Neurobehavioural mechanisms in ADHD, 130*(1-2), 29-36.

Sonuga-Barke, E. (2003). The dual pathway model of AD/HD: An elaboration of neuro-developmental characteristics. *Neuroscience & Biobehavioral Reviews. Special Issue: Mesocorticolimbic Branches and ADHD Variants, 27*(7), 593-604.

Sonuga-Barke, E., & Castellanos, F. X. (2007). Spontaneous attentional fluctuations in impaired states and pathological conditions: A neurobiological hypothesis. *Neuroscience & Biobehavioral Reviews, 31*(7), 977-986.

Sonuga-Barke, E., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and delay aversion: I. The effect of delay on choice. *Journal of Child Psychology and Psychiatry, 33*(2), 387-398.

Stieben, J., Lewis, M. D., Granic, I., Zelazo, P. D., Segalowitz, S., & Pepler, D. (2007). Neurophysiological mechanisms of emotion regulation for subtypes of externalizing children. *Development and Psychopathology, 19*(2), 455-480.

Stuss, D. T., Murphy, K. J., Binns, M. A., & Alexander, M. P. (2003). Staying on the job: The frontal lobes control individual performance variability. *Brain: A Journal of Neurology, 126*(11), 2363-2380.

Suess, P. E., Porges, S. W., & Plude, D. J. (1994). Cardiac vagal tone and sustained attention in school-age children. [Journal; Peer Reviewed Journal]. *Psychophysiology, 31*(1), 17-22.

Thayer, J., & Lane, R. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience and Biobehavioral Reviews, 33*(2), 81-88.

Tsujimoto, S., Kuwajima, M., & Sawaguchi, T. (2007). Developmental Fractionation of Working Memory and Response Inhibition During Childhood. *Experimental Psychology, 54*(1), 30-37.

van Duijvenvoorde, A. C. K., Zanolie, K., Rombouts, S. A. R. B., Raijmakers, M. E. J., & Crone, E. A. (2008). Evaluating the negative or valuing the
Neural mechanisms supporting feedback-based learning across development. *Journal of Neuroscience, 28*(38), 9495-9503.

van Meel, C., Heslenfeld, D., Oosterlaan, J., Luman, M., & Sergeant, J. A. (2005). ERPs associated with anticipation and evaluation of monetary reward and punishment in children with ADHD. In C. van Meel (Ed.), *PhD Thesis: Adaptive Control in ADHD* (pp. 59-80). Amsterdam: Vrije Universiteit.

Waldman, I. D., Nigg, J. T., Gizer, I. R., Park, L., Rappley, M. D., & Friderici, K. (2006). The adrenergic receptor Œ±-2A gene (ADRA2A) and neuropsychological executive functions as putative endophenotypes for childhood ADHD. *Cognitive, Affective & Behavioral Neuroscience. Special Issue: Genes, Brain, and Behavior, 6*(1), 18-30.

Weissman, D. H., Roberts, K. C., Visscher, K. M., & Woldorff, M. G. (2006). The neural bases of momentary lapses in attention. *Nature Neuroscience, 9*(7), 971-978.

Wilkins, A. J., Shallice, T., & McCarthy, R. (1987). Frontal lesions and sustained attention. *Neuropsychologia, 25*(2), 359-365.

Willcutt, 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.

Womelsdorf, T., & Fries, P. (2007). The role of neural synchronization in selective attention. *Current Opinion in Neurobiology, 17*, 154-160.

Xu, C., Reid, R., & Steckelberg, A. (2002). Technology applications for children with ADHD: Assessing the empirical support. *Education & Treatment of Children, 25*(2), 224-248.

Zahn, T. P., & Mirsky, A. F. (1999). Reaction time indicators of attention deficits in closed head injury. *Journal of Clinical and Experimental Neuropsychology, 21*(3), 352-367.

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Selected Publications, Presentations, & Posters

Karalunas, S.L. & Huang-Pollock, C.L. (*in press*). Understanding relationships between delay aversion and executive functioning. *Journal of Clinical Child & Adolescent Psychology*.

Huang-Pollock, C., Maddox, T., & **Karalunas, S.L.** (*in press*). Development of Implicit Category Learning. *Journal of Experimental Child Psychology*.

Huang-Pollock, C.L. & **Karalunas, S.L.** (2010). Working memory demands impair skill acquisition in children with ADHD. *Journal of Abnormal Psychology*, 119, 174-185.

Karalunas, S.L. & Huang-Pollock, C. *Intra-individual variability in ADHD*. Symposium talk presented at International Neuropsychology Society (INS), Acapulco, Mexico 2010.

Karalunas, S.L., Nigg, J.T., & Huang-Pollock, C.L. *Relationships among low-frequency reaction time variability, symptom domains, and functional impairment*. Poster accepted for the biennial meeting of the International Society for Research in Child and Adolescent Psychopathology, Chicago, IL, June 2011.

Karalunas, S.L. & Huang-Pollock, C. *Consistently inconsistent: effects of reward on patterns of intra-individual variability.* Poster presented at Eunethydis**,** Amsterdam, Netherlands 2010.

Karalunas, S.L. Gatzke-Kopp, L. & Huang-Pollock, C. *Understanding Intra-individual Variability in Cardiac and ERP Measures*. Poster presented at Society for Psychophysiological Research (SPR), Berlin, Germany October 2009.