Executive Functioning Impairment in College Students with High ADHD Symptomatology

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## Abstract

We investigated if college students with high ADHD symptomatology differed from peers with low symptomatology on an electronic version of the Trail Making Test and a version of Posner's Orienting Paradigm. We recorded participants' eye movements throughout the tasks to measure shifts of attention. Analyses revealed that participants with high symptomatology did not differ from those with low symptomatology in either the time spent completing the Trails task or in the total number of fixations they made during each trial. Furthermore, participants with low symptomatology made more total eye fixations and initial saccades toward the cue than participants with high symptomatology on the orienting task. Our study has important implications for understanding the etiology of ADHD. Executive Functioning Impairment in College Students with High ADHD Symptomatology

When completing everyday tasks, we must continuously direct our attention to items of interest while filtering out those distracting items that are not important. The effortful process of managing cognitive resources is known as executive functioning. Although executive functioning is important for understanding the ease with which all individuals complete cognitive tasks, the study of executive processes is especially important for understanding the cognitive impairments associated with attention deficit hyperactivity disorder (ADHD) in young adults (Weyandt & DuPaul, 2006). In a series of experiments, we recorded the eye movements of college students with high ADHD symptomatology and those with low symptomatology while they completed computerized versions of the Trail Making Test and Posner's Orienting Paradigm. We used both paradigms to investigate if eye fixations and saccades revealed impairment in executive functioning in college students with high ADHD symptomatology.

#### ADHD Symptomatology in College Students

Although ADHD is often first diagnosed in elementary school when children begin to have problems completing academic tasks, the disorder has far reaching implications for individuals into adolescence and adulthood. The primary symptoms of ADHD are inattention and hyperactivity/impulsivity. Individuals may receive diagnoses of ADHD predominantly inattentive type, ADHD predominantly hyperactive-impulsive type, or ADHD combined type depending on the symptoms they experience (American Psychiatric Association, 2000). Inattention symptomatology includes difficulty organizing tasks and difficulty sustaining attention during tasks, whereas hyperactivity-impulsivity symptomatology includes fidgeting and interrupting others (American Psychiatric Association, 2000). For diagnosis of each subtype, an individual must have six or more of the symptoms within a category, have had symptoms prior to the age of seven, and have experienced impairment in two or more settings.

Many individuals continue to experience ADHD symptoms into adulthood. Consequently, one major concern regarding the identification of ADHD in adults is whether the diagnostic framework that psychologists use for children is applicable for the adult population. In a large study of college students from Italy, New Zealand, and the United States, DuPaul and colleagues (2001) investigated if ADHD symptomatology in college students fit the bidimensional structure of inattention and hyperactivity-impulsivity that clinicians use for diagnosis in children under the DSM-IV-TR. Researchers assessed ADHD symptomatology with the Young Adult Rating Scale (YARS). The YARS consists of 17 items from the DSM-IV that assess either inattention or hyperactivity-impulsivity in addition to 7 items that assess other difficulties that college students with ADHD might experience (DuPaul et al., 2001). Factor analysis revealed that items for both American and New Zealand college students loaded onto inattention and hyperactivity-impulsivity factors, such that the bidimensional diagnostic system that clinicians use for children explained adult reports of ADHD symptomatology. Therefore, ADHD symptomatology in adults is likely a continuation of the cognitive deficits present in childhood rather than the development of new impairments.

Although symptomatology in adults fits a bidimensional diagnostic system, symptoms of inattention are more likely to persist into adulthood than symptoms of hyperactivity or impulsivity (Biederman, Mick, & Faraone, 2000). Although hyperactivity-impulsivity symptoms may decline with age, many individuals continue to experience subclinical and clinical inattention symptoms into adulthood (Biederman et al., 2000). In a meta-analysis of nine studies that assessed ADHD symptomatology both in childhood and at a later follow-up session,

Hill and Schoener (1996) found that the percentage of individuals with diagnosed ADHD decreased as age increased. Diagnoses decreased according to an exponential function such that the number of individuals who retained ADHD symptoms decreased by approximately 50 percent every five years (Hill and Schoener, 1996). Biederman, Mick, and Faraone (2000) found that although many participants fell below the criteria of symptomatology necessary for ADHD diagnosis by the age of 20, the majority of participants continued to experience ADHD symptoms. Furthermore, hyperactivity and impulsivity symptoms declined at a faster rate with increasing age than inattention symptomatology (Biederman et al., 2000). Taken together, these studies suggest that although ADHD symptomatology may decline with age, many young adults continue to experience subclinical and clinical deficits regarding attention.

## Executive Functioning in Individuals with ADHD

A cognitive deficit in response inhibition might serve as the basis of inattention and hyperactivity/impulsivity impairment in ADHD (Barkley, 1997; Nigg 2001). Nigg (2001) suggests that a primary deficit associated with ADHD is the inability to inhibit impulsive behaviors. One form of executive functioning is the intentional inhibition of a response in order to achieve an internal goal. Not thinking about previous poor performance on a test while taking a subsequent examination with the goal of concentrating on the new material is an example of executive impulse control. Individuals with ADHD may engage in impulsive behaviors and have difficulty limiting attention to important stimuli because they are unable to inhibit unwanted shifts of attention. Recent research using a stop-signal paradigm supports the disinhibition model of ADHD (Schachar, Mota, Logan, Tannock, & Klim, 2000). Children with ADHD, conduct disorder, comorbid ADHD and conduct disorder, and normally developing children pressed corresponding keys to X and O stimuli that appeared on a computer screen. Researchers told the children to stop their responses when they heard an auditory signal. Schachar and colleagues (2000) found that children with ADHD took approximately 70 ms longer than children with no psychopathology to stop in response to the tone. Impairment in response inhibition therefore likely serves as a primary component of inattentive as well as hyperactive and impulsive symptomatology.

Measures of executive functioning such as the Wisconsin Card Sorting Task, the Stroop Color-Word Test, and the Trail Making Test (parts A and B) provide support for Barkley's (1997) model that the executive process of inhibition is a locus of impairment for children and adults with ADHD. In a longitudinal study, Hinshaw and colleagues (2007) found that girls who met the diagnostic criteria of ADHD demonstrated a deficit in executive functioning across a variety of neuropsychological assessment measures when compared to healthy peers as both children and adolescents. Furthermore, Hinshaw and colleagues found that after controlling for IQ, there were no differences in neuropsychological test performance between the inattentive and hyperactive-impulsive subgroups of ADHD. Other research suggests that executive functioning deficits extend beyond the teenage years into young adulthood for individuals with a childhood history of ADHD (Biederman et al., 2007; Halperin, Trampush, Miller, Marks, & Newcorn, 2008). Biederman and colleagues (2007) found that the majority of participants who had executive functioning deficits as adolescents continued to have impairments as young adults. Furthermore, 25 percent of the participants with ADHD who did not have an executive functioning deficit in adolescence did have one as a young adult, thus suggesting that clinicians should be aware of the development of executive function impairments with maturity (Biederman et al., 2007). These studies provide support for the understanding of response inhibition as a primary component of ADHD across development.

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Although some executive impairments that are present in children such as working memory may decrease as symptomatology diminishes during maturation, deficits in response inhibition are present both in adults with clinical and subclinical degrees of symptomatology (Halperin, Trampush, Miller, Marks, & Newcorn, 2008). Halperin and colleagues (2008) administered a variety of neuropsychological tests to individuals who had symptoms of ADHD both as adolescents and as young adults (persisters), those who had symptoms as adolescents but not as young adults (remitters) and a control group who had never experienced symptomatology associated with ADHD. The time between the original administration of the neuropsychological tests and the follow-up was approximately nine years (Halperin et al., 2008). Participants completed the Stroop Color-Word Test as a measure of inhibitory control, the Continuous Performance Test (CPT) as a measure of response inhibition, and the Wechsler Adult Intelligence Scale, Third Edition (WAIS-III). The WAIS-III yielded verbal comprehension, perceptual organization, working memory, and processing speed index scores. Researchers found that persisters had significantly lower working memory scores than controls. Both persisters and remitters differed from controls on the response variability measure of the CPT, thus supporting previous research that symptoms of response inhibition continue even after individuals' fall below the symptom threshold for diagnosis (Biederman et al., 2000). However, remitters did not differ from controls in working memory scores, thus suggesting that working memory deficits associated with ADHD decrease as inattention and hyperactive-impulsive symptomatology decrease. Consequently, working memory may serve as a mechanism through which individuals with ADHD are able to compensate for their symptoms as they mature (Halperin et al., 2008). Similarly, remitters, but not persisters, had significantly lower word reading scores on the Stroop Task than control participants. These findings suggest that although working memory may serve as a mechanism through which individuals with ADHD compensate for inattention and hyperactivity symptomatology, impairment in inhibition persists even if symptomatology is below the level necessary for diagnosis.

The potential for differences in executive abilities based on gender and ADHD subtype remains a topic of debate among researchers. In a study of children with diagnosed ADHD and no comorbid disorders, Houghton and colleagues (2009) investigated if gender or ADHD subtype mediated the influence of ADHD symptomatology on a wide range of executive functions such as response inhibition, set shifting, visual search, and planning. Researchers found no significant interactions between subtype and gender for performance on the executive functioning tasks or main effects for gender in any of the assessment measures. However, there was a significant interaction such that only children with the combined symptomatology subtype of ADHD differed from controls on performance for the Wisconsin Card Sorting Task and the Color-Word section of the Stroop Test (Houghton et al., 2009). These findings provide limited support for the hypothesis that significant differences in executive functioning between patients and controls are only present for individuals with both inattentive and hyperactive-impulsive symptomatology.

## Neural Networks Implicated in Impaired Executive Functioning

Based on structural magnetic resonance imaging (MRI) and neuropsychological assessment, researchers have hypothesized that the etiology of executive functioning deficits in adults with ADHD stems from frontal lobe dysfunction (Dinn, Robbins, & Harris, 2001; Makris et al., 2007). Dinn, Robbins, and Harris (2001) suggest that the orbitofrontal cortex and the dorsolateral prefrontal cortex may be implicated in the cognitive deficits associated with ADHD. Participants completed the Object Alternation Test (OAT), the Stroop Color-Word Test and a Go/No-Go task. The OAT tests participants' ability to learn and follow a rule to determine where an object is hidden, whereas the Stroop-Color-Word Test and the Go/No-Go task assess word interference and behavioral inhibition, respectively. Researchers found that only participants with the predominantly hyperactive-impulsive subtype of ADHD demonstrated a performance deficit on the object alternation and Go/No-Go tests. Conversely, individuals with the predominantly inattentive subtype did not differ from control participants on the object alternation test or the Stroop task but did show a deficit on the Go/No-Go task. Lastly, participants with the combined subtype had deficits on both the Stroop and on the Go/No-Go tasks. These results suggest that although all participants with ADHD demonstrated some frontal lobe impairment, there were subtle differences between the types of executive functioning deficits that each diagnosis group experienced.

Neuroimaging research has similarly shown that patients with ADHD have structural abnormalities in brain areas implicated in executive functioning and attention (Makris et al., 2007). Makris et al. (2007) used magnetic resonance imagery to examine structural differences in cortical thickness between adults with ADHD and normally functioning peers. Researchers found that adults with ADHD had cortical thinning of the neural networks that are implicated in executive functioning and attention such as the dorsolateral prefrontal cortex (DLPFC), the cingulate cortex, and the inferior parietal lobule (IPL). Researchers also found bilateral cortical thinning of the lateral superior and middle superior gyri and the orbital frontal cortex in participants with ADHD (Makris et al., 2007). Lastly, individuals with ADHD had right anterior cingulate cortex (ACC) and left posterior cingulate cortex thinning (Makris et al., 2007). The DLPFC, ACC, and IPL are all implicated in attention and executive function, thus suggesting

that structural neurological differences between adults with ADHD and controls may account for the cognitive impairments in attention and executive abilities (Makris et al., 2007).

## Overview of the Current Study

College students provide an ideal population for understanding the cognitive impacts of ADHD symptomatology in young adults. The transition to the demands of college work may present difficulties for students who had previously successfully compensated for their symptomatology as adolescents. Recent estimates regarding the prevalence of ADHD symptomatology in college students suggests that two in five students with disabilities fit the diagnostic criteria for ADHD or another learning disability with approximately 42 percent of these students being female (Henderson 1999; Guthrie, 2002). Heiligenstein and colleagues (1999) found that college students with ADHD on average had a lower mean grade point average, were more likely to be on academic probation, and reported more problems involving depression, anxiety, interpersonal relationships, and substance use than control participants. Furthermore, Rucklidge, Brown, Crawford, and Kaplan (2007) found that adults with ADHD demonstrated impaired psychosocial functioning such as depression, anxiety, and maladaptive attributional styles. Executive functioning deficits in individuals with ADHD are also independent of IQ, thus reducing the likelihood that the selective college admissions processes might prevent the presence of executive functioning impairment in college-aged samples (Brown, Reichel, & Quinlan, 2009). In a study of the cognitive implications for high IQ adults with ADHD, Brown, Reichel, and Quinlan (2009) found that more than 70 percent of their sample with ADHD and high IQ self-reported having a significant impairment on at least four of the five clusters of executive function symptoms on the Brown ADD Scale. This study importantly suggests that executive functioning impairments are also prevalent among

individuals with high IQs (Brown et al., 2009). Taken together, these studies reveal the importance of studying executive functioning impairments in college students to inform treatments and prevent negative outcomes such as poor psychosocial functioning.

In a series of experiments we investigated if college students with high ADHD symptomatology demonstrated impairment in executive response inhibition in two cognitive tasks. In the first experiment, we measured eye movements during the completion of a computerized version of the Trail Making Test parts A and B. We next investigated the impact of ADHD symptomatology on response inhibition with a modified Posner Orienting Paradigm. Previous researchers have found that individuals with high ADHD symptomatology demonstrate impaired performance on both the Trail Making Test and visual orienting tasks (Feifel, Farber, Clementz, Perry & Anllo-Vento, 2004; Muller, Gimbel, Keller-Pliebnig, Sartory, Gastpar, & Davids, 2007). Through the use of eye tracking, we measured shifts of attention during both cognitive tasks to understand the executive impairments associated with ADHD that contribute to poor neuropsychological functioning.

Experiment 1: Executive Functioning Impairments in Visual Search

Children with ADHD have difficulty completing serial visual search tasks as well tasks that require effortful cognitive processing (Mullane & Klein, 2008; Murphy, 2002). The Trail Making test is well suited for investigations of response inhibition impairment during visual search (Murphy, 2002). The task consists of a visual search test with low cognitive demands and a visual search test that requires executive planning. In the low cognitive demand task, (part A) participants connect numbered targets in increasing numerical order (1-2-3-4 ...) as quickly as possible until they reach the final target. Participants are therefore required to shift attention throughout the image to locate the appropriate next target. In the executive functioning test (part B), participants connect targets that have numerical or alphabetic labels. They must alternate between connecting numbers and letters throughout the task (1-A-2-B-3-C ...), therefore requiring that they hold the previous target in working memory while they switch to the alternate classification system. The Trail Making Test is therefore a useful assessment measure of shifts of attention during visual search for conditions with varying cognitive demands.

Individuals with ADHD demonstrate impairment in both part A and part B of the Trail Making Test in comparison to peers with no history of psychopathology or neurological damage (Muller et al., 2007; Murphy, 2002). Muller and colleagues (2007) found that adult patients were slower to complete both part A and part B of the Trail Making Test than control participants. There was a significant interaction such that patients had a larger deficit in comparison to control participants when the task required switching between numbers and letters than when there were only numbers (Muller et al., 2007). Similarly, Murphy (2002) found that adults with diagnoses of ADHD demonstrated a deficit in completing both versions of the Trail Making Test. These results suggest that increased completion times on the Trail Making Test might be the result of inefficient visual search abilities in addition to a deficit in executive functioning (Murphy, 2002).

## Overview of Experiment 1

In the current study, participants completed computerized versions of both parts A and B of the Trail Making Test while researchers recorded eye movements. We used eye fixations as a measure of where participants directed their attention during the task. Consequently, a large number of fixations would suggest that the participant made many shifts of attention. Based on behavioral research that has documented executive functioning deficits associated with ADHD symptomatology, we hypothesized that participants in the high symptomatology group would

take longer to complete all versions of the Trail Making Test than control participants (Halperin et al., 2008; Hinshaw et al., 2007; Murphy, 2002). Additionally, we hypothesized that there would be a significant interaction such that participants with high symptomatology would have a larger performance deficit in comparison to participants with low symptomatology when completing the trial that required switching between numbers and letters than when completing the numbers only trial. Additionally, we predicted that participants with high ADHD symptomatology would make more fixations when completing both versions of the Trails Task than control participants due to an inability to inhibit shifts of attention and poor visual search abilities (Muller et al., 2007; Murphy et al., 2002). Lastly, we hypothesized that high symptomatology participants would have a greater increase in the number of eye fixations in relation to participants with low symptomatology for part B than in part A of the Trails Task.

#### Method

## *Participants*

Twenty-nine students (8 men, 21 women) ranging in age from 20 years to 22 years (M = 21.3, SD = 0.61) at a small liberal arts college in upstate New York completed the study. Participants received either a gift certificate to a campus café or extra credit in a psychology course as compensation. Participants were Caucasian (86.2 percent), Hispanic (10.3 percent), or did not report an ethnicity (3.4 percent). Twenty-five participants reported right hand dominance, whereas 4 participants reported left hand dominance. All but one participant reported English as a native language.

Researchers used the College ADHD Response Evaluation (CARE) to determine eligibility to participate in the study. Men who scored in the 93rd percentile or above on the inattention subscale and the 77th percentile or above on the hyperactivity/impulsivity subscale of the CARE were eligible for the high symptomatology group. Similarly, women who scored above the 94th percentile on the inattention subscale or above the 74th percentile for the hyperactivity/impulsivity subscale were eligible for the high symptomatology group. The low ADHD symptomatology group consisted of men and women who scored below the 50th percentile on both the inattention and hyperactivity/impulsivity subscales. These criteria allowed for adequate group sizes that reflected the range of symptomatology within the college population. All participants completed the CARE assessment during first-year orientation as part of a larger research project on ADHD and college transition. Consequently, the assessment of symptomatology took place approximately 2-3 years prior to beginning of the current study. Researchers sent an email to all eligible participants that described the study. The email provided an overview of the experiment and asked participants to refrain from taking medication for 12 hours prior to their scheduled appointment. The final sample consisted of 19 low symptomatology participants (6 men, 13 women) and 10 high symptomatology participants (2 men, 8 women). All participants confirmed that they had not taken any stimulant medication within the last 12 hours at the time of testing.

## Apparatus

Researchers recorded participants' eye movements with an Applied Science Laboratories (ASL) Eyetracker 6000. The eye tracking apparatus was head-mounted and used an adjustable mirror to reflect an image of the participant's cornea to a small camera on the helmet. Researchers could view the retinal and corneal reflections from the eye tracker on a small television. Additionally, a monitor displayed the location where the participants were looking in real time to facilitate calibration. All recordings were of the left eye. Researchers also recorded changes in head position throughout data collection with a flock of birds magnetic system to

correct the eye movement data for any changes in head position. The flock of birds consisted of a transmitter positioned directly behind the participant's head and a sensor that was attached to the helmet. The eye tracking apparatus collected data at a sampling rate of 12.5 samples per second and transmitted the data to a receiver. The data then passed to a PC with a 2.79 GHz processor for later analysis. ASL software used the data from the flock of birds to adjust the calibration of the eye tracking apparatus in real time.

Participants sat approximately 50.00 cm from a 52.0 cm X 32.5 cm LCD computer monitor at a resolution of 1920 X 1200 pixels. An alternate PC ran a program in Inquisite to control the presentation of stimuli. In order to sync eye movement data with the participant responses and the presentation of stimuli, the PC running Inquisite sent xdat codes to the PC recording the eye movement data. The computer then time-stamped these signals and added the code to the eye tracking output to differentiate trials in future analyses.

#### Materials

*College ADHD Response Evaluation (CARE)*. The CARE is a self-report measure of ADHD symptomatology for young adults ranging in age from 17 to 23 years (Glutting et al., 2002). Participants rated the degree to which 59 items such as "I talk excessively" described them for the last several months. Participants made all ratings on a 3-point scale (0 = disagree, 1 = undecided, 2 = agree). The CARE consists of 44 items that assess ADHD symptomatology, 13 items that assess comorbid symptomatology, and 2 additional items related to the DSM-IV diagnostic criteria. Completion of the CARE took participants approximately 15 minutes. We scored the 44 ADHD assessment items to compute factor scores for inattention, hyperactivity, and impulsivity. We then added the factor scores to compute each participant's total ADHD symptomatology score. The maximum score for the inattention subscale was 42, whereas the

maximum scores for hyperactivity and impulsivity were 22 and 26, respectively. Consequently, the maximum total symptomatology score was 90. Previous standardization of the test revealed that the factor scores can effectively discriminate between students with and without diagnoses of ADHD, have high levels of internal consistency and test-retest reliability, and have a positive association with other assessment measures of ADHD in adulthood (Glutting, Monaghan, Adams, & Sheslow, 2002).

*Trail Making Test.* Participants completed two practice trials and two experimental trials of a computerized version of the Trail Making Test. Targets in part A of the task had numerical labels, whereas targets in part B had numerical or alphabetic labels. The practice trial for part A had numbers ranging from 1 to 8, whereas the experimental trial had numbers ranging from 1 to 26. Similarly, the practice for part B had numbers ranging from 1 to 4 and letters ranging from A to D, whereas the experimental part B trial had numbers ranging from 1 to 13 and letters ranging from A to M. Researchers labeled the beginning and ending targets in all trials "Begin" and "End," respectively, to allow participants to know when to stop looking for an additional target. Researchers constructed the stimuli so that the targets were evenly spaced throughout the left and right visual field and so that the trail between targets would not pass through other targets. Each target was circular and had a diameter of 2.6 cm, thus creating a visual angle of approximately 2.98 degrees.

#### Procedure

Upon entering the lab, participants completed a consent form and removed any metal that might interfere with the equipment. After a researcher had answered any questions regarding the experiment, participants sat facing a computer monitor with a standard computer mouse on the table in front of them and the flock of birds receiver directly behind their heads. Researchers told the participants to sit so that they could comfortably reach the mouse. A researcher then explained the nature of the task and emphasized that the participant should connect the targets in sequential order as quickly and accurately as possible. The participant completed a paper and pencil version of the traditional Trails part A task in which they used a pencil to connect numbered targets in sequential order. The researcher explained that in the computerized version, the participant should click on each target with the left mouse button. After clicking on the last target, the participant looked at the bottom right corner of the computer monitor and clicked anywhere on the screen with the right mouse button to end the trial.

After the participant understood the directions, a researcher placed the head-mounted eye tracking apparatus on the participant and turned on all equipment. Researchers eliminated all ambient light and positioned the eye tracker's mirror so that the software recognized reflections of the retina and cornea. To calibrate the eye tracking apparatus, participants looked at 9 points on a 3 X 3 numbered grid while the computer recorded the position of the eye fixations. To check the accuracy of the calibration, a researcher had the participant look at each location on the grid and used the software to watch the eye movements to each point in real time on a separate computer monitor. When the apparatus was calibrated properly, a researcher told the participant to click the left mouse button to begin the first trial.

Participants first completed the practice part A trial followed by the experimental part A trial. The participant fixated for 5 seconds at the beginning of each trial at a central plus sign. After 5 seconds the plus sign disappeared and the trial image appeared on the screen. The first target was always in the center of the image where the fixation point had been. Prior to all trials other than the Trails A practice, the calibration grid appeared and researchers recorded eye movements to each corner as references for later calibration correction. If a participant made an

error during the trial, the researcher asked the participant to go back to the last correctly identified target and continue. There was a brief pause between each trial as the researcher switched the stimulus program.

After the experimental part A trial, the researcher explained that the next trials would require alternating between targets with numbers and targets with letters. The participant then drew a line between targets in a paper version of Trails part B. After making sure that the participant understood the task, the researcher allowed the participant to continue with the final two computer trials, using the same procedure as in part A and beginning with the practice trial. After finishing the last trial, the participant completed a brief demographic form. Researchers also provided compensation and answered any questions that the participant had about the study. The entire procedure took approximately 20 minutes.

## Results

#### **Overview** of Analyses

We first investigated if participants with high ADHD symptomatology took longer to complete the experimental trials of Trails part A and B than participants with low symptomatology. In addition to examining time differences for the entire sample, we also analyzed the latency data for women separately. Since there were only 2 men in the high symptomatology condition, we were unable to run an analysis of trial latencies excluding women. We then used the total number of eye fixations as a measure of the degree to which participants shifted their attention during each trial. We defined a fixation as a location on the computer screen where the participant looked for greater than 100 ms. After computing the total number of fixations for each participant, we conducted statistical analyses to determine if participants with high symptomatology differed from peers with low symptomatology in the number of fixations that the participant made per trial. We conducted all statistical analyses at an alpha level of .05.

## Trial Latencies

We first investigated if there were significant differences in the time participants spent completing each trial based on trial type and level of symptomatology. We defined the time between the onset of the trails image and the last mouse click on a target as the time participants spent completing each trial. We excluded three participants from the latency analysis because they ended the task before clicking on all targets. We conducted a repeated measures ANOVA with two levels of trial type (Trails A and Trails B) and two levels of symptomatology (low and high). In support of our hypotheses, there was a significant main effect of trial type,

F(1, 24) = 52.84, p < .001, such that participants took longer to complete trials with numbers and letters than trials with only numbers (see Figure 1). Contrary to our predictions, there was no significant main effect of symptomatology, F(1, 24) = 0.01, *ns*. There was no time difference collapsed across trial type between participants with high symptomatology and those with low symptomatology. Lastly, the interaction between trial type and symptomatology also failed to reach statistical significance, F(1, 24) = 2.34, *ns*.

We next analyzed if women differed in trial latencies based on trial type and level of symptomatology. A repeated measures ANOVA with two levels of trial type (Trails A and Trails B and two levels of symptomatology (low and high) revealed that as in the analyses for all participants, there was a significant main effect of trial type, F(1, 19) = 54.95, p < .001. Women completed part A more quickly than part B (see Figure 2). There was no significant main effect of symptomatology participants did not differ in latency from low symptomatology participants. Unlike our analyses for men and

women together, there was a significant interaction for women, F(1, 19) 4.48, p < .05. We conducted independent groups *t*-tests to determine the nature of the interaction. Although there were no significant differences between low symptomatology and high symptomatology women for part A t(19) = -0.20, *ns*, women with high symptomatology took longer to complete part B than women with low symptomatology, t(19) = -2.23, p < .05.

Researchers next examined if the total number of eye fixations that participants made per trial differed based on trial type and level of ADHD symptomatology. In addition to the three participants who did not complete the task correctly, we excluded an additional participant due to technical difficulties with the eye tracking apparatus during data collection. A repeated measures ANOVA with two levels of trial type (Trails A and Trails B) and two levels of ADHD symptomatology (low and high) revealed that there was a significant main effect of trial type, F(1, 23) = 14.41, p < .01. Participants made more fixations in Trails B than in Trails A (see Figure 3). Contrary to our hypotheses, there was no significant main effect of symptomatology, F(1, 23) = 0.21, *ns*. Lastly, there was no significant interaction between trial type and level of symptomatology, F(1, 23) = 1.38, *ns*. When we ran the ANOVA with men excluded from the data analysis, we found the same pattern of results.

## Discussion

In the current study, we used eye tracking to measure shifts of attention during the completion of a visual search task to investigate if college students with high ADHD symptomatology demonstrated an impairment regarding the inhibition of unwanted shifts of attention. In support of our hypotheses, all participants took longer to complete part B of the Trail Making Test than part A. This finding suggests that the numbers and letters version of the task was more difficult and required greater executive planning for accurate completion.

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However, contrary to our hypotheses, when all participants were in the analysis, students with high ADHD symptomatology did not differ in the amount of time spent completing the task from peers with low ADHD symptomatology for part A or part B of the task. We had predicted that the greater cognitive demands of part B of the task would be more difficult for students with high ADHD symptomatology than those with low symptomatology. When completing part B, the participant must hold the previous number or letter in working memory as they switch to the opposite target type. Consequently, we predicted that students with high symptomatology would have difficulty both inhibiting the tendency to search for a target that was of the same type as the previous target and in holding the magnitude of the previous target in working memory. In partial support of our hypotheses, there was a significant interaction of symptomatology and trial type on trial latency when researchers excluded men from the analyses. Women with high symptomatology took longer to complete only part B trials than peers with low symptomatology. Although this finding is in accordance with our predictions, we hypothesized that both men and women with high symptomatology would have longer trial latencies for part B as a result of the high executive functioning demands of the task. A possible explanation of this finding is that the Trail Making Test is more sensitive to cognitive impairments in women with high ADHD symptomatology than men with high symptomatology. However, due to the large imbalance of men and women in the symptomatology groups of the current study, future research is needed to clarify this finding.

We next used the total number of fixations that each participant made during a trial as an indicator of the number of shifts of attention while completing the task. In support of our hypotheses, when collapsed across symptomatology conditions, participants made significantly more fixations for part B of the Trail Making Test than for part A. Our results therefore suggest

that participants both took longer and made more shifts of attention when the task required active switching between finding numerical and finding alphabetical targets. However, contrary to our predictions, there was no significant main effect of symptomatology or interaction of trial type and symptomatology for the total number of fixations that participants made per trial. Just as participants with high ADHD symptomatology did not differ from low symptomatology participants regarding trial latencies, students with high symptomatology did not shift their attention more often than participants with low symptomatology during either trial type.

One possible explanation for the lack of significant symptomatology differences in trial latencies and the total number of fixations per trial might be that our sample consisted of students at a selective liberal arts college. In order to function in a competitive academic environment, the students with high ADHD symptomatology in our sample have likely developed strategies to compensate for their symptoms of inattention. Although previous research suggests that executive functioning impairment associated with ADHD is independent of measures of intelligence such as the SAT, the Trail Making Test might not be sensitive to the level of impairment present in students at a selective school (Brown et al., 2009). An alternate explanation is that global measures of attention such as our use of the total number of fixations per trial might not be sensitive to low levels of cognitive impairment. Participants with high ADHD symptomatology might have trouble shifting attention under specific circumstances such as making eye movements from a target on the right side of the screen to a target on the left side, but not show an overall global difference in the number of fixations. Future research is therefore needed to investigate if there are significant differences between symptomatology groups regarding the location of fixations.

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Our findings do not support previous research regarding cognitive deficits associated with ADHD in adults and children. Both Murphy (2002) and Muller et al. (2007) found that adults with ADHD took longer than participants with no history of psychopathology to complete both part A and part B of the Trail Making Test. Conversely, we found that there were no significant latency differences for either part of test. Our study differed from the previous literature in our decision to recruit participants from a selective liberal arts college. Murphy and Muller et al. recruited adults who met the diagnostic criteria for ADHD. We used ADHD symptomatology as the eligibility criteria rather than diagnoses. Although we did not ask participants if they have received a diagnosis of ADHD or any other psychological disorder, it is likely that despite having the highest level of ADHD symptomatology in our sample, many participants in our high symptomatology group would not have had impairment severe enough to meet the diagnostic criteria of the DSM-IV. Therefore, the level of executive functioning impairment in our sample might be less than that in previous studies. Additionally, a difference between the current study and previous research is that we used a computerized version of the Trail Making Test rather than the traditional paper and pencil assessment measure. Since completing college coursework often requires locating information on a computer display, most participants in our sample were likely very familiar with the cognitive demands of completing similar tasks. Conversely, the paper and pencil version is less similar to everyday tasks and could therefore be more difficult and place a greater cognitive demand on participants.

A limitation of our study was that participants were free to use the mouse to move the cursor anywhere on the screen when searching for targets. In the traditional paper and pencil assessment measure, participants must keep the pencil on a target while looking for the next target. Conversely, in our experiment, participants were free to move the cursor across the

screen as they searched for targets because rather than drawing a line, participants in our study only had to click on the targets in the appropriate order. The ability to move the cursor while searching could have facilitated participants' ability to search effectively with less distraction than if they had to shift their attention without the movement of a cursor. In future use of the computerized Trail Making Test, movements of the cursor should draw a line as in the paper and pencil version to prevent participants from making movements other than those that connect targets. Another limitation of our study was that researchers interrupted participants when they had incorrectly selected a target. To standardize the procedure for informing participants of errors, future computerized versions of the Trail Making Test should provide an auditory signal if participants make an incorrect target selection.

In our first experiment we investigated if college students with high ADHD symptomatology demonstrated impaired visual search and executive functioning on a computerized version of the Trail Making Test. Contrary to our predictions, there were no significant differences in either trial latencies or in the total number of fixations that participants made per trial between high and low symptomatology participants when the analyses included both men and women. We next used a modified version of Posner's Orienting Paradigm to examine if there were symptomatology differences in the inhibition of prepotent eye movements toward new stimuli.

Experiment 2: Response Inhibition During An Orienting Task

Although we did not find significant differences in shifts of attention between high and low ADHD symptomatology students on a computerized version of the Trail Making Test, we next investigated if the symptomatology groups differed in their abilities to inhibit prepotent responses to a visual cue. The orientation of attention in response to cues may be the result of top-down or bottom-up processes. Cues that appear in individuals' peripheral vision encourage a reflexive eye movement toward the cue (Feifel et al., 2004). This tendency to direct attention toward new information is automatic and therefore is a bottom-up process. Conversely, the ability to purposefully shift attention without the appearance of a stimulus in the to-be-attended location requires effortful cognitive processing and therefore is a top-down process. Successful completion of everyday tasks requires the ability to both inhibit shifts of attention toward peripheral cues that are not task important and to actively shift attention toward locations that are in accordance with an individual's goals. Based on models of impaired response inhibition in ADHD, we investigated if students with high ADHD symptomatology had difficulty inhibiting prepotent responses to peripheral cues in relation to students with low ADHD symptomatology (Nigg, 2001).

A deficit in inhibitory mechanisms might contribute to symptoms of visual inattention (Feifel et al., 2004; Laurie, Nigg, & Henderson, 2006; Munoz, Armstrong, Hampton, & Moore, 2003). Feifel and colleagues (2004) had participants make eye movements from a central fixation point either toward a peripheral target (prosaccade trial) or to the opposite visual field of a peripheral target (antisaccade trial). Unmedicated adults with ADHD made more saccades in anticipation of the cue on prosaccade trials and made more directional errors on antisaccade trials than control participants. Loe, Feldman, Yasui, and Luna (2009) similarly found that even when receiving instructions to remain fixated on a central point, adults with ADHD broke fixation in response to the brief appearance of peripheral targets more often than control participants. Furthermore, in an additional study regarding saccadic eye movements in patients with ADHD, Munoz, Armstrong, Hampton, and Moore (2003) found that participants with ADHD had longer reaction times and slower peak eye movement velocities than participants with no psychopathology on a prosaccade task. The same participants with ADHD also had greater difficulty inhibiting saccades toward a distractor during an antisaccade task and had greater difficulty fixating on a single point than control participants (Munoz et al., 2003). Taken together, these findings suggest that the core inattentive symptoms of ADHD may result from impairment in response inhibition.

Although individuals with ADHD demonstrate impairment in the ability to inhibit unwanted saccades, ADHD is not associated with a change in magnitude or pattern of the attentional blink paradigm in relation to healthy participants (Laurie, Nigg, & Henderson, 2006; Mason, Humphreys, & Kent, 2005). During the attentional blink task, participants view a rapid stream of stimuli and respond whenever they see a target stimulus. Researchers have termed the inability of all individuals to detect a second target if it is between 180 and 450 ms after an initial target the attentional blink (Raymond, Shapiro, & Arnell, 1992). The attentional blink therefore is a useful paradigm for testing if the ability to filter stimuli differs between individuals with high ADHD symptomatology and those with low symptomatology. Laurie and colleagues (2006) found that adults with ADHD do not demonstrate a different pattern or magnitude of the attentional blink phenomenon than control participants, thus dissociating attentional filtering ability from response inhibition. Similarly, Mason, Humphreys and Kent (2005) found that there was no difference between children with ADHD and those without psychopathology regarding the time until recovery after the attentional blink. These findings support models that place response inhibition rather than the ability to filter information as the primary deficit in ADHD (Nigg, 2001).

In addition to experiencing difficulty for making accurate eye movements, students with high ADHD symptomatology might differ from control participants regarding the speed with which they make saccades. Abrams, Meyer, and Kornblum (1989) told participants to make saccadic eye movements from a fixation point to a target displayed on a computer monitor as accurately as possible. After the participant initiated the eve movement in response to a tone, the fixation point and target disappeared from screen. Researchers found that there was a linear relationship between the accuracy and the velocity of saccades such that the standard deviation of the saccade endpoint increased as velocity increased (Abrams, Meyer, & Kornblum, 1989). In a similar study of children with ADHD, Stigchel and colleagues (2007) found that participants with ADHD had longer movement latencies, spent longer searching for targets, and made more saccades to locations that did not contain either a target or a distractor during a saccadic eve movement task. Stigchel et al. also examined symptomatology as a continuous variable and found that latency of movement and total search time were associated with inattentive symptomatology, whereas the proportion of saccades directed at locations other than the target or distractor was positively related to hyperactivity/impulsivity symptomatology. The number of saccades toward the distractor did not differ between the ADHD and control groups, thus suggesting that children with ADHD do not differ from controls in saccades toward irrelevant distractors. Taken together, these findings suggest that although there is a speed and accuracy trade-off for healthy participants, individuals with ADHD take longer to locate targets, make saccades with longer movement latencies, and make more eye movements to areas without salient or distracting information than peers with low symptomatology.

Impairment of the fronto-striatal network could account for deficits in response inhibition among adults and children with ADHD (Booth et al., 2005). Booth and colleagues (2005) found that decreased activation in the fronto-striatal network was associated with an inability to maintain appropriate behaviors and to inhibit inappropriate behaviors for children with ADHD. Participants received a function magnetic resonance imagery (fMRI) scan while completing both a selective attention task and a response inhibition task. During the selective attention task participants pressed one of two buttons to indicate whether a target object was present. The response inhibition task consisted of a standard Go/No-Go task such that participants responded with a button press for any stimulus in Go trials but only for target stimuli in No-Go trials (Booth et al., 2005). Control participants had greater activation in the right middle frontal gyrus and the left and right cingulate than participants with ADHD when completing the selective attention task. Researchers found that during the response inhibition task, control children had significantly greater activation in the precentral gyrus, bilateral caudate body, right caudate head, right inferior frontal gyrus, and right thalamus (Booth et al., 2005). The large difference in activation between controls and children with ADHD provides further support that response inhibition is a primary cognitive deficit associated with ADHD.

Some adults and children with ADHD experience difficulty attending to objects in their left visual fields, thus suggesting that the etiology of ADHD might involve right hemisphere dysfunction (Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995; Manly, Cornish, Grant, Dobler, & Hollis, 2005). Manly and colleagues (2005) had elementary school teachers complete the Strengths and Weaknesses of ADHD-symptoms and Normal behavior scale (SWAN) as a measure of attention for students in their classrooms. Based on teacher ratings, researchers divided the children into a good attention group and a bad attention group (Manly et al., 2005). The children then completed a line bisection task in which they marked the midpoint of a 200 mm black horizontal line for five trials. Researchers gave all participants percentile scores ranging from the farthest right (first percentile) to the farthest left (100th percentile). Participants who were below the tenth percentile made up the left inattention group, whereas participants who were between the 90th and 100th percentiles became the right inattention group (Manly et al., 2005). The left-inattention group scored significantly worse on the SWAN attention scale, hyperactivity scale, and composite ADHD scale than peers with line bisections in the normal range. Left-inattention participants also performed worse than the control group on three measures of sustained attention (Manly et al., 2005). Lastly, participants in the right inattention group did not differ from the participants with normal line bisections on any of the measures of ADHD, thus suggesting that neglect associated with ADHD is confined to the left visual field.

In support of evidence for right hemisphere dysfunction in ADHD, other research suggests that unlike children with no history of psychopathology, children with ADHD do not have a cost in reaction time for invalidly cued trials in the left hemisphere during a covert orienting task (Carter et al., 1995). Researchers have frequently used Posner's Covert Orienting Paradigm to investigate impairment in the ability to shift attention without making eye movements in response to cues that are valid, invalid, or neutral (Alvarez & Freides, 2004; Carter et al., 1995; Collings & Kwasman, 2006). During the task participants remain fixated on a central point while a cue signals that a target will likely appear in a particular location on the screen (Posner, Walker, Friedrich, & Rafal, 1987). Participants then covertly direct their attention and respond as quickly as possible once a target stimulus appears. Cues may be exogenous, appearing at the cued location and thus eliciting a prepotent response, or endogenous, not at the to-be-cued location and therefore requiring effortful cognitive processing to follow the cue's instruction. Furthermore, cues may validly signal the location of a target, invalidly predict the location, or cue both potential target locations. Carter and colleagues had participants remain fixated on a central point while either a target box became brighter to serve as an exogenous cue

or an arrow appeared at the fixation point that served as an endogenous cue. The cues validly predicted the appearance of a subsequent target for most trials. Researchers found that although children without ADHD had longer reaction times in response to an invalid endogenous cue, children with ADHD only demonstrated a cost in longer reaction times for invalidly cued right targets regardless of whether the cue was endogenous or exogenous. These findings support Manly and colleagues (2005) in suggesting that a right hemisphere brain deficit might contribute to impaired visual processing in the left visual field.

Although Carter et al. (1995) found that participants with ADHD only had a deficit in reaction time for trials with invalidly cued right targets, other research suggests that participants with the predominantly inattentive subtype of ADHD show impairments in covert orienting for both right and left targets when there is a long period of time between the cue disappearance and target onset (Collings & Kwasman, 2006). Within a sample of children with the predominantly inattentive subtype of ADHD, the combined subtype, and control participants, only those participants with predominantly inattentive symptoms had a deficit in reaction time for responding to peripheral targets (Collings & Kwasman, 2006). Furthermore, as the period of time between the disappearance of the cue and the onset of the target increased, participants with the inattentive subtype had significantly increasing reaction times. Individuals with inattentive symptomatology might therefore have more trouble covertly shifting attention when the time between cue offset and target onset, the stimulus onset asynchrony (SOA), is long.

# Overview of the *Experiment 2*

In our second experiment, we investigated if students with high ADHD symptomatology demonstrated impaired response inhibition in relation to students with low symptomatology on a modified version of Posner's Covert Orienting Paradigm. Unlike Posner's traditional task,

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participants in the current study were free to move their eyes after the onset of each trial's cue. Each trial began with a central fixation point with a single box to the left and a single box to the right of the central point. To serve as a cue, the left box, right box, or both boxes together next became boldly outlined. Participants received instructions prior to beginning the task that in the majority of trials, the cue would appear on the opposite side of the screen from where a target would later appear. Following the cue, a target appeared in one of the boxes and the participant responded as quickly as possible with a key press on a standard computer keyboard. In addition to varying the validity of the cue, researchers varied the SOAs such that there were short, medium, and long delays between the disappearance of the cue and the onset of the target. This period of time is the stimulus onset asynchrony (SOA). We hypothesized that all participants would take longer to respond on valid cue trials than invalid or neutral cue trials since the majority of cues were invalid. We also predicted that there would be a significant interaction such that students with high ADHD symptomatology would have a greater cost in reaction time only for trials with the longest SOA (Carter et al., 1995; Collings & Kwasman, 2006). We predicted that participants with high symptomatology would make more fixations throughout trials than those with low symptomatology because of difficulty fixating on a single point (Loe et al., 2009; Munoz et al., 2003). Furthermore, we hypothesized that participants with high ADHD symptomatology would have difficulty inhibiting the prepotent response to the appearance of the cue such that they would make more initial saccades in the direction of the cue than control participants.

#### Method

## Participants

Forty-two students (18 men, 24 women) from a small liberal arts college ranging in age from 18 to 23 years (M = 19.4, SD = 1.37) participated in the study in exchange for extra credit in a psychology course or a \$5 gift certificate to a campus cafe. Researchers recruited participants through email, telephone, and psychology courses. The symptomatology assessment and eligibility criteria were the same as in the first experiment. Due to technical errors during data collection, we were unable to analyze the data of 19 participants. In the final sample there were 13 participants with low ADHD symptomatology and 10 participants with high ADHD symptomatology.

## Apparatus

Researchers recorded participants' eye movements with the same ASL eye tracking device as in experiment 1. Participants sat approximately 50.00 cm from a 33.00 cm X 24.00 cm color CRT computer monitor at a resolution of 1024 X 768 pixels. Participants made all responses with a standard computer keyboard. A Macintosh G4 computer with a 2.7 GHz processor running Psyscope controlled the presentation of stimuli. In order to sync eye movement data with the presentation of stimuli, researchers connected the Macintosh to the PC. The data acquisition program recorded the signals from the Macintosh in the eye movement data file so that researchers could later select the trial types of interest for data analysis. *Materials* 

*Posner Orienting Paradigm*. Throughout all trials, participants viewed an image that consisted of a square box to the left and a square box to the right of a central plus sign (see Figure 4). Each square had side lengths of 4.2 cm, thus creating visual angles of approximately

4.80 degrees. Furthermore, the squares were positioned 5.5 cm from the central fixation point. Each trial began with 500 ms during which there was no cue or target present on the screen. After the initial period, either one or both of the squares became outlined for 500 ms. The outlined box served as a cue for the location of a subsequent target. The cues were in the location of the target (valid), in the location opposite of the target (invalid), or in both potential target locations (neutral). The stimulus presentation program randomly assigned the SOA to 100, 250, or 800 ms. The target appeared on the screen until the participant made a response with the keyboard or timed out after 300 ms. In addition to valid, invalid, and neutral trials, there were also catch trials during which no target appeared. Lastly, there was a second period of 200 ms during which the participant viewed the image without cues or a target. The inter-trial interval was always 750 ms. Fifty percent of trials had an invalid cue, 10 percent of trials had a valid cue, and 13.3 percent of trials had neutral cues. The remaining trials were catch trials (26.7 percent).

## Procedure

Upon entering the lab, participants provided informed consent and received compensation for participation. The researcher then brought the participant to the testing apparatus and sat the participant on a stool positioned so that the participant's face would be approximately 43.00 cm from the computer monitor. A researcher placed the eye tracking apparatus on the participant's head and then tightened the helmet until the device was securely in place. The researcher then calibrated the eye tracking apparatus with the same procedure as in experiment 1.

Participants completed the orienting task as part of a larger study on cognitive impairments associated with ADHD symptomatology in college students. Participants read and

received verbal instructions prior to the orienting task to press any key on the keyboard as quickly as possible after the appearance of a target. Furthermore, researchers told participants that the majority of trials would have invalid cues regarding the position of a subsequent target. Each trial began with a central fixation point and two boxes located to the left and right of the central point. Next, one of the boxes lit up to cue the participant. Lastly, an asterisk either appeared in one of the boxes or failed to appear in either box. Participants pressed any key on the keyboard as quickly as possible after the appearance of the asterisk. Participants completed 180 trials with a break after trial number 60 and trial number 120. The orienting task took participants approximately 20 minutes to complete. After completion of the study, researchers thanked the individuals for their participation and answered any questions regarding the study.

### Results

## **Overview** of Analyses

We first tested if participants with high symptomatology differed from those with low symptomatology regarding reaction times after the appearance of the target. The reaction time was the period between the onset of the target and the participant's button press. We next examined the eye movement data. Based on our primary interest in response inhibition, all eye tracking data analyses are from trials with invalid cues. As in the first experiment, we defined a fixation as any point on the computer screen that the participant looked at for more than approximately 100 ms. We computed fixation durations, the latency between target onset and the first fixation, the total number of fixations per trial, and the number of initial saccades in the direction of the invalid cue. We conducted all data analyses at an alpha level of .05.

## **Reaction Times**

To test for reaction time differences, we conducted a 2 X 3 X 2 repeated measures ANOVA with two levels of symptomatology (low and high) and three levels of trial type (valid cue, invalid cue, and neutral cue) and three levels of SOA (100 ms, 250 ms, and 800 ms). Contrary to our hypotheses, the main effect for level of symptomatology did not reach statistical significance, F(1, 21) = 0.94, ns. Participants with high ADHD symptomatology (M = 365.259, SE = 27.74) did not have significantly different reaction times than peers with low ADHD symptomatology (M = 400.97, SE = 24.33) in response to the onset of the target. In support of our hypotheses, there was a significant main effect of trial type, F(2, 42) = 13.21, p < .001. We conducted post hoc paired group *t*-tests with a modified Bonferroni procedure to determine the nature of the relationship between trial type and reaction time. As illustrated in Figure 5, when collapsed across symptomatology level, participants had longer reaction times in response to valid cues than in response to both invalid cues and neutral cues. There was no significant difference between mean reaction times for invalid cue trials and neutral cue trials. The main effect of SOA also failed to reach statistical significance, F(2, 42) = 0.11, ns, such that reaction times were not significantly different across all participants for 100 ms (M = 380.75, SE = 19.18), 250 ms (M = 384.13, SE = 21.86), and 800 ms (M = 384.46, SE = 15.79) SOAs. Lastly, all of the interactions failed to reach statistical significance, F's < 1.74, p's > .05. Eve tracking Analyses

We conducted independent groups *t*-tests to examine if there were symptomatology differences in fixation durations, the time between target onset and the first fixation, the total number of fixations per trial, and the total number of initial saccades in the direction of the invalid cue. There were no significant differences in fixation duration between participants with

high ADHD symptomatology (M = 0.63, SD = 0.26) and those with low ADHD symptomatology (M = 0.55, SD = 0.29) for invalidly cued left target trials, t(28) = -0.78, *ns*. Additionally there were no significant differences for fixation duration between participants with high symptomatology (M = 0.68, SD = 0.33) and those with low symptomatology (M = 0.54, SD = 0.32) for invalidly cued right target trials, t(28) = -1.15, *ns*.

There were also no significant differences in the amount of time between target onset and the first fixation between participants with high ADHD symptomatology (M = 0.53, SD = 0.21) and those with low symptomatology (M = 0.44, SD = 0.17) for invalidly cued left targets, t(28) = -1.43, ns. The comparison of the time until the first fixation for invalidly cued right targets also failed to reach statistical significance, t(28) = -1.45, ns, such that participants with high symptomatology (M = 0.59, SD = 0.28) did not differ from participants with low symptomatology (M = 0.46, SD = 0.19).

We next conducted an independent groups *t*-test to investigate if high and low symptomatology participants differed regarding the total number of fixations they made during invalid cue trials. Contrary to our predictions, participants with low ADHD symptomatology made more fixations on average for trials with an invalidly cued left target than participants with high ADHD symptomatology, t(28) = 2.16, p < .05 (see Figure 6). However, the difference between participants with high and low symptomatology in the total number of fixations for trials with an invalidly cued right target did not reach statistical significance, t(28) = 1.67, *ns*, despite having the same trend.

Lastly, we conducted an independent groups *t*-test to determine if participants differed in the number of initial saccades that they made toward invalid cues. Contrary to our hypotheses, we found that participants with low symptomatology made significantly more initial saccades

toward the cues, thus demonstrating lower response inhibition than participants with high symptomatology, t(28) = 2.76, p < .05 (see Figure 7).

## Discussion

In the second experiment, we recorded the eve movements of college students with high or low ADHD symptomatology during completion of a modified version of Posner's Orienting Paradigm. We first investigated if reaction times in response to the target's onset differed based on both the type of the cue as well as the participant's level of ADHD symptomatology. In support of our hypotheses, we found that collapsed across symptomatology conditions, participants had longer reaction times following valid cues than following invalid or neutral cues. Prior to the beginning of the experiment, researchers told the participants that the majority of cues would be invalid. Consequently, our results suggest that both participants with high ADHD symptomatology and those with low symptomatology primarily focused their attention in the visual field opposite the location of the cue. Participants had to orient their attention from the side opposite of the cue to the validly cued side after the onset of the target, thus possibly leading to an increase in reaction time. Although we predicted that participants with high symptomatology would have a larger deficit in reaction time for trials with a long SOA, the reaction times of high symptomatology participants did not vary as a function of SOA. A possible explanation of this finding is that unlike the traditional orienting task, our task had a majority of invalid cues. Participants with high ADHD symptomatology might be faster to orient toward an unexpected valid cue after a long SOA than an unexpected invalid cue due to the prepotent tendency to orient toward the side of the original cue.

Although there were no differences in reaction times between high and low ADHD symptomatology participants, our analysis of the eye fixation data revealed that contrary to our

predictions, participants with low symptomatology made both more fixations for trials with an invalidly cued left target and made more initial saccades toward cues than participants with high symptomatology. We predicted that participants with high ADHD symptomatology would make more fixations because they would have difficulty attending to a single location during the task. Furthermore, we predicted that participants with high symptomatology would have greater difficulty than participants with low symptomatology in inhibiting a prepotent saccade toward the cue. A possible explanation for our finding is that individuals with low symptomatology were able to more efficiently and quickly shift their attention between the two visual fields and consequently did not need to inhibit the prepotent response toward the cue. Conversely, our participants with high symptomatology might have developed a compensatory ability to inhibit prepotent responses because of an awareness that they have trouble focusing attention and consequently cannot afford to make eve movements toward a probably invalid cue. As in experiment 1, our participants' status as students at a selective liberal arts college suggests that they have likely developed compensatory mechanisms to overcome their symptoms and perform at a comparable level as their peers with low symptomatology. The ability to block the prepotent response toward the cue might therefore serve as a compensatory mechanism that accounts for the lack of difference in reaction time between participants with high and low ADHD symptomatology. Although participants with low symptomatology made more fixations for invalid trials with a left target and made more initial saccades in the direction of the cue, there were no significant symptomatology differences in average fixation durations or the time between cue onset and the first fixation.

Our results do not support previous research that has used Posner's Covert Orienting Paradigm with populations of adults and children with ADHD (Alvarez & Friedes, 2004; Carter et al., 1995). Carter and colleagues (1995) found that individuals with ADHD had longer reaction times than control participants when they received either an exogenous or an endogenous invalid cue for a target in the left visual field. Although we were unable to analyze reaction times to each visual field separately because of missing data, our global reaction time analysis found no significant differences between participants with high ADHD symptomatology and those with low ADHD symptomatology. Furthermore, our findings do not support research on response inhibition deficits associated with ADHD (Collings & Kwasman, 2006; Feifel et al., 2004; Loe et al., 2009; Munoz et al., 2003). Feifel and colleagues (2004) found that participants with ADHD made more eve movements toward a cue during an antisaccade task, thus reflecting an inability to inhibit the prepotent response to orient attention toward a new stimulus. Similarly, Collings and Kwasman (2006) found that participants with ADHD took longer to respond to targets in non-cued areas than control participants. Contrary to these studies, we found that participants with low symptomatology made more initial saccades than high symptomatology participants in the direction of the cue even though they knew that the majority of cues would be invalid. A possible reason why the low symptomatology participants made more saccades toward the invalid cue in our design is that although researchers told participants that the majority of cues would be invalid, the task did not have explicit directions to orient attention away from the cue. In antisaccade tasks, researchers tell the participant to orient attention to the opposite visual field from the cue. Participants in our study did not need to inhibit eye movements toward the cue to follow the instructions of the task. It is possible that only the high symptomatology participants inhibited eye movements toward the cue as a compensatory mechanism for their increased level of inattention.

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Although our study has produced interesting findings, there are a few limitations. A limitation of our second experiment is that due to missing data, we were unable to investigate if there were differences in reaction times for targets in the left and right visual fields. Based on previous research, both reaction times and the number of initial fixations toward cues might be different for trials with targets in the left visual field than for trials with right visual field targets (Collings & Kwasman, 2006; Makris et al., 2007). Furthermore, we only analyzed the eye tracking data for invalid cue trials in the current study. Future research should investigate if low symptomatology participants make significantly more fixations than participants with low symptomatology across other trial types than invalid cue trials. A final limitation of our study is that the orienting task took approximately 20 minutes to complete and was very repetitive. Furthermore, the orienting task was last in a larger battery that participants completed as part of a study on cognition and ADHD. Participants with high ADHD symptomatology might have had greater difficulty than participants with low symptomatology focusing on the task in later trials due to fatigue. Consequently, it is possible that high symptomatology participants made fewer fixations than those with low symptomatology because of reduced motivation.

In our second experiment we investigated if college students with high ADHD symptomatology demonstrated impairments in response inhibition with a modified version of Posner's Orienting Task. Contrary to our hypotheses, we found that participants with low ADHD symptomatology made more fixations per trial and made more initial saccades toward cues than participants with high ADHD symptomatology. Theses findings suggest that college students with high ADHD symptomatology do not have a deficit in response inhibition during a probabilistic cuing paradigm in relation to peers with low symptomatology. Conversely, participants with high symptomatology in the current study demonstrated a superior ability than individuals with low symptomatology for inhibiting prepotent responses toward new stimuli.

### General Discussion

We investigated if college students with high ADHD symptomatology demonstrated deficits in executive functioning across two cognitive tasks. Previous research suggests that impairment in response inhibition might account for inattention and hyperactivity/impulsivity symptoms in children and adults with ADHD (Barkley, 1997; Nigg 2001). We administered a computerized version of the Trail Making Test part A and part B as well as a version of Posner's Orienting Paradigm where the majority of the cues invalidly predicted the appearance of a target to college students with either high or low self-reported ADHD symptomatology. Contrary to previous studies that have found that assessment tools of executive functioning are sensitive to impairment in children and adults with ADHD, we found no significant differences in either the time to complete the Trail Making Test or the total number of fixations per Trails trial based on level of symptomatology (Halperin, 2008; Hinshaw, 2007). Furthermore, when completing the orienting paradigm, college students with high symptomatology made significantly fewer initial saccades toward invalid cues than those with low symptomatology, thus suggesting that the students with ADHD were better at inhibiting prepotent responses than peers with low symptomatology. Our findings therefore suggest that college students with high ADHD symptomatology might be able to compensate for any impairment in executive functioning that could negatively impact performance when searching for objects or preventing the unwanted orientation of attention toward new stimuli.

Future research on executive functioning impairment in adults with ADHD should further investigate if the neural mechanisms implicated in shifts of attention differ between individuals

with high ADHD symptomatology and those with low symptomatology. Esterman, Chiu, Tamer-Rosenau, and Yantis (2009) have recently found that patterns of brain activity within the medial superior parietal lobule (mSPL) continuously vary in accordance with different executive control processes. Participants covertly attended to one of two rapid serial visual presentation (RSVP) streams. Imbedded in a stream of distractor letters were cues to either covertly switch attention from to the opposite RSVP stream, to switch a categorization rule, or to continue with the same stream and categorization rule. Participants made categorizations based on either parity or magnitude of target stimuli. Using pattern classification, researchers found that neural activity within the mSPL differed based on the type of cognitive control (switching spatial location of attention, switching classification) the participant engaged in at any moment. Although there has been research regarding structural abnormalities in the attention network of the brain in individuals with ADHD, future studies should use event-related multivoxel pattern classification to investigate if similar groups of neurons are active in different types of cognitive control between participants with high symptomatology and those with low symptomatology. Understanding the neural mechanisms involved in specific types of executive processes would improve our understanding of the etiology of ADHD and could provide neurological evidence for why some assessment measures of executive functioning reveal significant impairment for individuals with ADHD while others do not.

The study of executive functioning deficits associated with ADHD has important implications for both academic and occupational success (Biederman et al., 2006; Stavro, Ettenhofer, & Nigg, 2007). Biederman and colleagues (2006) administered a variety of neuropsychological measures to both adults with ADHD and those with no history of psychopathology. Researchers classified participants as having an executive functioning deficit if they demonstrated scores 1.5 standard deviations below the mean or if they were in the poorest seventh percentile on two or more of the neuropsychological assessment measures. In comparison to participants with ADHD but no executive functioning dysfunction, participants with ADHD and executive functioning impairment scored lower on every measure of academic performance (Biederman et al., 2006). Furthermore participants with both ADHD and executive functioning impairments were more than two times than participants with only ADHD to have received special help in school and to have repeated a grade. Similarly, Stavro, Ettenhofer, and Nigg (2007) used latent variable analysis to investigate how executive functioning and adaptive functioning were related to ADHD symptomatology. Stavro and colleagues administered a battery of neuropsychological tests and three measures of adaptive functioning. Structural equation modeling revealed that inattention symptomatology accounted for adaptive impairment, whereas hyperactive-impulsivity accounted for little unique variance. The authors concluded that a deficit in executive functioning is the primary mechanism through which inattentive symptomatology leads to adaptive impairment. Both studies suggest that the impairments in executive functioning associated with ADHD symptomatology may negatively impact an individual's quality of life. Therefore, future research regarding executive functioning impairments in adults with high ADHD symptomatology has important implications both for understanding the etiology of the disorder and for providing specialized treatment services for those individuals with severe executive functioning impairment who are most at risk for academic and social failure.

In the current study, we used eye tracking to investigate if college students with high ADHD symptomatology demonstrated executive functioning impairment on two cognitive tests. We found no significant symptomatology differences in the time necessary to complete a visual search task, the total number of fixations during a visual search task, or in reaction time to the appearance of targets during an orienting task. Furthermore, we found that students with low ADHD symptomatology made more initial saccades toward invalid cues than participants with high symptomatology. Studies of executive functioning impairment in college students with ADHD symptomatology such as ours have important implications for improving treatment services for young adults.

## References

- Abrams, R. A., Meyer, D. E., & Kornblum, S. (1989). Speed and accuracy of saccadic eye movements: Characteristics of impulse variability in the oculomotor system. *Journal of Experimental Psychology: Human Perception and Performance*, 15, 529-543.
- Alvarez, J. A., & Freides, D. (2004). Research on Attention Deficit Hyperactivity Disorder Using the Covert Orienting Paradigm of Posner. *Developmental Neuropsychology*, 26, 627-645.
- American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions:Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65-94.
- Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *The American Journal of Psychiatry*, 157, 816-818.
- Biederman, J., Petty, C., Fried, R., Fontanella, J., Doyle, A. E., Seidman, L. J., & Faraone, S. V.
  (2006). Impact of psychometrically defined deficits of executive functioning in adults with attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *163*, 1730-1738.
- Biederman, J., Petty, C., Fried, R., Doyle, A. E., Spencer, T., Seidman, L. J., Gross, L., Poetzl, K., & Faraone, S. V. (2007). Stability of executive function deficits into young adult years: A prospective longitudinal follow-up study of grown up males with ADHD. *Acta Psychiatrica Scandinavica*, *116*, 129-136
- Booth, J. R., Burman, D. D., Meyer, J. R., Lei, Z., Trommer, B. L., Davenport, N. D., Li, W., Parrish, T. B., Gitelman, D. R., & Mesulam, M. M. (2005). Larger deficits in brain

networks for response inhibition than for visual selective attention in attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry*, *46*, 94-111.

- Brown, T. E., Reichel, P. C., & Quinlan, D. M. (2009). Executive function impairments in high IQ adults with ADHD. *Journal of Attention Disorders, 13,* 161-167.
- Carter, C. S., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Asymmetrical visual-spatial attentional performance in ADHD: Evidence for a right hemispheric deficit.
- Collings, R. D., & Kwasman, A. (2006). Visual orienting deficits among boys with ADHDinattentive type. *Individual Differences Research*, *4*, 111-122.
- Dinn, W. M., Robbins, N. C., & Harris, C. L. (2001). Adult attention-deficit/hyperactivity disorder: Neuropsychological correlates and clinical presentation. *Brain and Cognitition*, 46, 114-121.
- DuPaul, G. J. Schaughency, E. A., Weyandt, L. L., Tripp, G., Kiesner, J., Ota, K., & Stanish, H. (2001) Self-report of ADHD symptoms in university students: Cross-gender and cross national prevalence. *Journal of Learning Disabilities*, *34*, 370-379.
- Esterman, M., Chiu, Tamber-Rosenau, & Yantis. (2009). Decoding cognitive control in human parietal cortex. *Proceedings of the National Academy of Science*, Published online before print.
- Feifel, D., Farber, R. H., Clementz, B. A., Perry, W., & Anllo-Vento, L. (2004). Inhibitory deficits in ocular motor behavior in adults with attention-deficit/hyperactivity disorder. *Biological Psychiatry*, 56, 333-339.
- Glutting, J. J., Monaghan, M. C., Adams, W., & Sheslow, D. (2002). Some psychometric properties of a system to measure ADHD among college students: Factor patter, reliability

and one-year predictive validity. *Measurement and Evaluation in Counseling and Development, 34*, 194-209.

- Guthrie, B. (2002). The college experience. In K. Nadeau & P. Quinn (Eds.), *Understanding Women With AD/HD* (pp. 288-312). Silver Spring, MD: Advantage.
- Halperin, J. M., Trampush, J. W., Miller, C. J., Marks, D. J., & Newcorn, J. H. (2008).
  Neuropsychological outcome in adolescents/young adults with childhood ADHD: Profiles of persisters, remitters, and controls. *Journal of Child Psychology and Psychiatry*, 49, 958-966.
- Henderson, C. (1999). College freshman with disabilities: A statistical profile. Washington, DC: HEATH Resource Center.
- Heiligenstein, E., Guenther, G., Levy, A., Savino, F., & Fulwiler, J. (1999). Psychological and academic functioning in college students with Attention Deficit Hyperactivity Disorder. *Journal of American College Health*, 47, 181-185.
- Houghton, S., Douglas, G., West, J., Whiting, K., Wall, M., Lansford, S., Powel, L., & Carroll,
  A. (1999). Differential patterns of executive function in children with attention-deficit
  hyperactivity disorder according to gender and subtype. *Journal of Child Neurology*, *14*, 801-805
- Hill, J. C., & Schoener, E. P. (1996). Age-dependent decline of attention deficit hyperactivity disorder. *The American Journal of Psychiatry*, *153*, 1143-1146.
- Hinshaw, S. P., Carte, E. T., Fan, C. F., Jassy, J. S., & Owens, E. B. (2007).
  Neuropsychological functioning of girls with attention-deficit/hyperactivity disorder followed prospectively into adolescence: Evidence for continuing deficits? *Neuropsychology*, 21, 263-273.

- Laurie, A. C., Nigg, J. T., & Henderson, J. M. (2006). Attentional versus motor inhibition in adults with attention-deficit/hyperactivity disorder. *Neuropsychology*, *4*, 430-441.
- Loe, I. M., Feldman, H. M., Yasui, E., & Luna, B. (2009). Oculomotor performance identifies underlying cognitive deficits in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child Adolescent Psychiatry*, 48, 431-440.
- Manly, T., Cornish, K., Grant, C., Dobler, V., & Hollis, C. (2005). Examining the relationship between rightward visuo-spatial bias and poor attention within the normal child population using a brief screening task. *Journal of Child Psychology and Psychiatry*, 46, 1337-1344.
- Makris, N., Biederman, J., Valera, E. M., Bush, G., Kaiser, J., Kennedy, D. N., Caviness, V. S., Faraone, S. V., & Seidman, L. J. (2007). Cortical thinning of the attention and executive function networks in adults with attention-deficit/hyperactivity disorder. *Cerebral Cortex*, 17, 1364-1375.
- Mason, D. J., Humphreys, G. W., & Kent, L. (2005). Insights into the control of attentional set in ADHD using the attentional blink paradigm. *Journal of Child Psychology and Psychiatry*, 46, 1345-1353.
- Mullane, J. C., & Klein, R. M. (2008). Visual search by children with and without ADHD. *Journal of Attention Disorders*, *12*, 44-53.
- Muller, B. W., Gimbel, K., keller-Pliebnig, A., Sartory, G., Gastpar, M., Davids, E. (2007).
- Munoz, D. P., Armstrong, I. T., Hampton, K. A., & Moore, K. D. (2003). Altered control of visual fixation and saccadic eye movements in attention-deficit hyperactivity disorder. *Jounral of Neurophysiology*, 90, 503-514.
- Murphy, P. (2002). Cognitive functioning in adults with attention-deficit/hyperactivity disorder. *Journal of Attention Disorders*, *5*, 203-209.

- Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? Psychological Bulletin, 127, 571-598.
- Posner, M. I., Walker, J. A., Friedrich, F. A., & Rafal, R. D. (1987). How do the parietal lobes direct covert attention. *Neuropsychologia*, 25, 135-145.
- Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1992). Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology: Human Perception and Performance, 18*, 849-860.
- Rucklidge, J., Brown, D., Crawford, S., & Kaplan, B. (2007). Attributional styles and psychosocial functioning of adults with ADHD. *Journal of Attention Disorders*, *10*, 288-298.
- Schachar, R., Mota, V. L., Logan, G. D., Tannock, R., & Klim, P. (2000). Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*, 28, 227-235.
- Stavro, G. M. (2007). Executive functions and adaptive functioning in you adult attentiondeficit/hyperactivity disorder. *Journal of the International Neuropsychological Society*, 13, 324.
- Stigchel, S. V., Rommelse, N. N., Deijen, J. B., Geldof, C. J., Witlox, G. J., Oosterlaan, J., Sergeant, J. A., & Theeuwes, J. (2007). Oculomotor capture in ADHD. *Cognitive Neuropsychology*, 24, 535-549.
- Weyandt, L. l., & DuPaul, G. (2006). ADHD in college students. *Journal of Attention Disorders*, 10, 9-19.

# **Figure Captions**

*Figure 1*. Mean time to complete a trial as a function of trial type and ADHD symptomatology *Figure 2*. Mean time to complete a trial as a function of trial type and ADHD symptomatology for women.

*Figure 3*. Mean total number of fixations per trial as a function of trial type and ADHD symptomatology.

Figure 4. Sample invalid cue trial with a right target.

Figure 5. Mean reaction times as a function of cue validity and ADHD symptomatology.

*Figure 6*. Mean total number of fixations for high and low ADHD symptomatology participants for an invalidly cued left target trial. The top figure displays the means for the total number of fixations during invalidly cued left target trials. The bottom figure displays the means for the total number fixations during invalidly cued right target trials.

*Figure 7*. Mean number of initial saccades in the direction of the invalid cue for high and low ADHD symptomatology participants.







Trial Type



300 ms maximum





