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Separating Automatic and Intentional Inhibitory Mechanisms of Attention in Adults with Attention-Deficit/Hyperactivity Disorder

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Abstract

Researchers in the cognitive sciences recognize a fundamental distinction between automatic and intentional mechanisms of inhibitory control. The use of eye-tracking tasks to assess selective attention has led to a better understanding of this distinction in specific populations such as children with attention-deficit/hyperactivity disorder (ADHD). This study examined automatic and intentional inhibitory control mechanisms in adults with ADHD using a saccadic interference (SI) task and a delayed ocular response (DOR) task. Thirty adults with ADHD were compared to 27 comparison adults on measures of inhibitory control. The DOR task showed that adults with ADHD were less able than comparison adults to inhibit a reflexive saccade towards the sudden appearance of a stimulus in the periphery. However, SI task performance showed that the ADHD group did not differ significantly from the comparison group on a measure of automatic inhibitory control. These findings suggest a dissociation between automatic and intentional inhibitory deficits in adults with ADHD.

Keywords

ADHD; inhibition; automatic; intentional

A deficit of inhibitory control has been identified consistently as a feature of children with attention-deficit/hyperactivity disorder (ADHD; Pennington & Ozonoff, 1996). Several influential models of ADHD (Barkley, 1997; Quay, 1997) posit various forms of inhibitory control dysfunction as a central feature of ADHD. Given the centrality of this trait to the disorder, a better understanding of inhibitory control in this group may provide some insight into the nature of the cognitive deficits typical of individuals with ADHD. Many studies have examined inhibitory control deficits in children (e.g., Derefinko et al., 2008; Schachar & Logan, 1990) and early adolescents (e.g., Aman, Roberts, & Pennington, 1998) with ADHD; however, much less is known about the development of inhibitory control into adulthood.

Inhibitory control is defined by Barkley (1997) as three interconnected processes, including inhibiting prepotent responses, inhibiting ongoing responses, and interference control (Barkley, 1997). It is implicated in the control of both behavioral actions and cognitive processes (Fillmore, 2003). For example, inhibitory control represents an individual's ability

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to filter out distracters and retain task-relevant information. As part of typical cognitive development, inhibitory control is implied in other cognitive constructs and abilities, such as working memory (Ridderinkhof & van der Molen, 1997), the effective execution of goal directed behavior, and sustaining attention in the presence of distractions (Barkley, 1997). Accordingly, inhibitory control has become a topic of interest in the study of developmental psychopathology, particularly in the study of childhood behavior disorders. Along these lines, researchers have focused on the role of inhibitory deficits in the symptomatology of ADHD. Findings generally have supported impaired inhibitory control as a central trait of the disorder (Nigg, 2001; Pennington & Ozonoff, 1996). However, it is important to note that different theoretical accounts of ADHD emphasize different types of inhibitory dysfunction (e.g., executive inhibition, motivational inhibition; Barkley, 1997; Quay, 1997; for review, see Nigg, 2001). Other models of ADHD question the primacy of inhibitory deficits in the symptomatic profile of the disorder, instead suggesting that disinhibition exists downstream of other deficits (e.g., working memory, activation regulation, delay aversion; Rapport, Chung, Shore, & Isaacs, 2001; Sergeant, Oosterlaan, & van der Meere, 1999; Sonuga-Barke, 2002). Such competing theories highlight the need for research aimed at better understanding the specific deficits associated with ADHD.

Studies of inhibitory control have typically been conducted using tasks similar to the stop-signal paradigm put forth by Logan and Cowan (1984). The stop-signal task and similar tasks (e.g., cued go/no-go task) require that a participant execute a behavioral response (e.g., press a button) upon the presentation of a stimulus and inhibit a response upon the presentation of the same stimulus accompanied with a stop signal. Thus, the participant is required to suppress the tendency to execute the prepotent behavioral response. Using such tasks, researchers have attempted to elucidate the inhibitory control deficits in children with ADHD, with some evidence indicating that children with ADHD are less able to inhibit behavioral responses than comparison peers (Bedard et al., 2003; Oosterlaan & Sergeant, 1996; Shachar, Tannock, Marriott, & Logan, 1995; Shachar & Logan, 1990). However, it should be noted that these conclusions have been contested by recent meta-analytic studies that suggest that observed inhibitory deficits in children with ADHD are attributable to a more generalized deficit in attention and cognitive processing (Alderson, Rapport, & Kofler, 2007; Lijffijt, Kenemans, Verbaten, & Engeland, 2005).

More recent studies have expanded the examination to investigate how inhibitory control deficits might contribute to other symptoms of ADHD. One such area of investigation is the role of inhibitory control in the attention problems (e.g., distractibility) evident in children with ADHD. A link between the two has been shown in the developmental literature, in that inhibitory control plays a role in regulating the allocation of attention (Fillmore, Milich, & Lorch, 2009; Luna, Garver, Urban, Lazar, & Sweeney, 2004). To get at this relation, researchers have utilized tasks assessing the inhibitory control of attention, particularly through the examination of ocular responses. Hanisch and colleagues (2006) used two ocular response tasks to examine control of attention in children with ADHD: the antisaccade task and the countermanding task. The antisaccade task requires participants to suppress a reflexive saccade towards a target and instead generate a saccade in the opposite direction. The countermanding task is similar to the stop-signal task in that it requires the participant to make a behavioral response upon the presentation of a stimulus and inhibit the response upon the presentation of the stimulus in the presence of a stop-signal; however, the response required for the countermanding task is a saccade towards the target instead of a button press. Findings from Hanisch et al. supported the existence of an inhibitory deficit of attention in children with ADHD. Perhaps more importantly, this study found no evidence of a general oculomotor deficit in children with ADHD, suggesting that the attention allocation difficulties were related to inhibitory control deficits. In a similar study, Klein and colleagues (2003) examined the inhibitory control of children and adolescents with ADHD

using an antisaccade task. Again, findings of this study suggested a relation between inhibitory control deficits and the allocation of attention in individuals with ADHD. This evidence for an impairment of inhibitory control of attention in children with ADHD is important because it may account for the well documented attention problems in this group. For example, the inability to suppress the allocation of attention towards a peripheral stimulus may play a role in the susceptibility to distraction observed in children with ADHD.

Distinguishing Between Automatic and Intentional Inhibitory Control Mechanisms

Other work in the area of inhibitory control recognizes a fundamental distinction in the inhibitory control mechanisms of attention: automatic inhibitory control mechanisms and intentional inhibitory control mechanisms (Marzi, 1999; Shimojo, Hikosaka, & Miyauchi, 1999). Automatic inhibitory control mechanisms occur in a reflexive manner without awareness (i.e., automatically filtering an irrelevant stimulus without conscious processing). Conversely, intentional inhibitory control mechanisms operate under conscious control at the level of awareness (i.e., inhibiting a shift in attention towards a task-irrelevant stimulus; Godijn & Theeuwes, 2003).

Research has shown that the two mechanisms of automatic and intentional inhibitory control processes are subject to dissociation (Abroms, Gottlob, & Fillmore, 2006), such that a reduction of intentional inhibitory control does not necessarily extend to automatic inhibitory control. Hawk and colleagues (2003) found that children with ADHD showed an intentional, but not automatic, inhibitory deficit on a prepulse inhibition task. Our group (Fillmore et al., 2009) examined the dissociation between intentional and automatic inhibitory control of attention in groups of children with different subtypes of ADHD (e.g., ADHD/predominately inattentive [ADHD/PI], ADHD/combined [ADHD/C], and ADHD/C with comorbid oppositional defiant disorder [ADHD/C + ODD]). A countermanding task was used to measure intentional inhibitory control and an inhibition of return (IOR; Klein, 2000) task to measure automatic inhibitory control. All subtype groups showed a comparable deficit of intentional inhibitory control on the countermanding task. However, the ADHD/PI group showed a smaller deficit of automatic inhibitory control than the ADHD/C and ADHD/C + ODD groups on the IOR task. This suggests a dissociation between the automatic and intentional inhibitory control mechanisms in the ADHD/PI group. In a discussion of these findings, we suggested that the development of automatic inhibitory control mechanisms may be implicated in the development of intentional inhibitory control. Specifically, the normal development of automatic inhibitory control mechanisms of attention may serve as the cognitive precursors to intentional inhibitory control mechanisms that are assumed to develop later (Aksan & Koschanska, 2004). Considering the evidence supporting the dependence of intentional inhibitory control mechanisms on earlier developing systems of automatic inhibition, an examination of inhibitory control deficits in individuals with ADHD in a developmental context may prove useful. This raises an important question: Are inhibitory control deficits manifested in adults with ADHD?

Inhibitory Control Deficits in Adults with ADHD

Adult ADHD has become an increasingly popular area of study, due in part to emerging evidence that this group may experience neuropsychological deficits different from children with ADHD (Lijffijt et al., 2005). Compared to the child population, relatively little is known about the neurological and cognitive profiles of adults with ADHD; however, what evidence is available suggests continued symptomatology at least partially similar to

children with ADHD. Specifically, adults with the disorder continue to have attentional problems (Sandson, Bachna, & Morin, 2000; Hollingsworth, McAuliffe, & Knowlton, 2001), other executive functioning deficits (Nigg et al., 2005; Lovejoy et al., 1999), and working memory deficits (Murphy, Barkley, & Bush, 2001). An expanding literature has begun to identify inhibitory control deficits in this group (Carr, Nigg, & Henderson, 2006; Feifel, Farber, Clementz, Perry, & Anllo-Vento, 2004; Armstrong & Munoz, 2003; Ross, Harris, Olincy, & Radant, 2000). Moreover, meta-analytic work has shown that adults with ADHD, unlike children with ADHD, show a specific inhibitory control deficit on the stop-signal task (Lijffijt et al., 2005).

The existence of these inhibitory deficits in adults with ADHD has been documented, although some questions remain concerning the nature of this impairment. Specifically, no study to date has directly compared the automatic and intentional inhibitory control mechanisms of adults with ADHD using ocular response tasks. Several studies have addressed attention filtering using an attention blink task, finding that adults with ADHD were not impaired in automatic attentional filtering (Carr et al., 2006; Hollingsworth et al., 2001). However, it is unclear whether the attention blink task provides a clear distinction between automatic and intentional attention filtering mechanisms. Furthermore, attention filtering is at least conceptually distinct from inhibitory control mechanisms. Instead, attention filtering as measured by the attentional blink task is thought to reflect information processing speed (Duncan, Ward, & Shapiro, 1994).

The Current Study

The current study examined the nature of the inhibitory control deficits in adults with ADHD. Specifically, we examined whether the inhibitory control deficits found in adults with ADHD are contingent on the type of inhibitory control response required (i.e., automatic or intentional) with a specific focus on the inhibition of attention mechanisms. A group of young adults with and without ADHD completed two tasks assessing inhibitory control: the delayed ocular response (DOR) task and the saccadic interference (SI) task. The DOR task taps intentional inhibitory control by assessing a participant's ability to inhibit a reflexive saccade toward the sudden onset of a peripheral visual stimulus. The SI task measures automatic inhibitory control by presenting a distracter stimulus as the participant performs a saccade towards a target. This distracter stimulus draws the attention of the participant and evokes automatic inhibition to minimize the interference caused by the distracter stimulus. These two tasks were selected because both required the participant to execute a saccade along a horizontal path towards a target. Thus, the tasks required similar eye movements, but differed in the types of inhibitory responses that were involved.

We hypothesized that adults with ADHD would show an impairment of intentional inhibitory control as evident through impaired performance on the DOR task relative to the healthy comparison group. In addition, we hypothesized that adults with ADHD would show relatively less impairment in automatic inhibitory control as evident through task performance on the SI task. This prediction was based upon prior work in a number of different fields. Prior studies have demonstrated that adults with ADHD show less impairment in automatic attentional tasks relative to more consciously controlled processes (e.g., Carr et al., 2006). Developmental studies have identified age-related deficits in intentional, but not automatic, inhibitory processes (Andrés, Guerrini, Phillips, & Perfect, 2008; Kramer, Hahn, Irwin, & Theeuwes, 2000). Work in the area of psychopharmacology has shown automatic inhibitory control mechanisms to be less susceptible to the disruptive effects of alcohol and other drugs compared with intentional inhibitory control mechanisms (Fillmore & Vogel-Sprott, 2006; Holloway, 1995). Furthermore, frontal regions of the brain are involved in programming goal-directed saccades, so this region plays a unique role in

intentional inhibition (Kramer et al., 2000). Because the pathophysiology of ADHD is thought to be centered in the frontal regions of the brains, it seems likely that this population would show impairment unique to intentional inhibitory mechanisms. Taken together, this evidence suggests that automatic inhibitory mechanisms are more resilient against impairing factors than intentional inhibitory mechanisms, and that this pattern likely generalizes to adults with ADHD.

The separation of automatic and intentional inhibitory control in this population may answer a question proposed by Halperin and Shulz (2006). These authors questioned the role of the prefrontal cortex in the pathophysiology of ADHD, instead suggesting that dysfunction in this region is secondary to midbrain and hindbrain dysfunction. One prediction made by this model is that adults with ADHD should differ from healthy adults on measures of automatic processing, and these differences should be attenuated on measures involving intentional control. For the current study, this model predicts group differences in the SI task (automatic inhibitory control), but not on the DOR task (intentional inhibitory control). This prediction runs counter to the hypotheses of the current study.

Another question germane to the current study concerns the primary cognitive deficits associated with ADHD. A debate in the literature exists regarding the primacy of inhibitory deficits in the symptomatic profile of adults with ADHD. A number of theorists propose that inhibitory deficits underlie other cognitive deficits in this group, whereas others speculate that working memory deficits are primary to this disorder (Nigg, 2001; Rapport et al., 2001). The relation between working memory and inhibitory control is a difficult one to untangle, given the interactive nature of these cognitive functions. To address this, Ross and colleagues (2000) proposed a method of separating working memory and inhibitory control using the DOR task. We hypothesized that the ADHD group would show inhibitory deficits, but not working memory deficits, relative to the control group.

Method

Participants

Participants included 30 individuals with ADHD (17 men and 13 women; M age = 21.1 years, SD = 1.7) and 27 individuals with no history of ADHD (13 men and 14 women; M age = 22.0 years, SD = 1.7). Participants were recruited through advertisements (i.e., newspaper ads and posters) seeking adults for a study of neurological and motor functioning. Participation was limited to individuals who were between the ages of 19 and 30 and had no uncorrected vision problems. Individuals with past or current severe psychiatric diagnoses (i.e., bipolar disorder, schizophrenia), as determined through self-report and medical records, were not invited to participate. Demographic information is shown in Table 1.

To ensure that members of the ADHD group were actively experiencing ADHD symptoms, only the potential participants who were currently prescribed medication for ADHD were invited to participate. Members of the ADHD group reported several different prescriptions, including Adderall (n = 7), AdderallXR (n = 11), AdderallXR and Adderall (n = 5), Concerta (n = 2), Dexedrine (n = 2), Daytrana (n = 1), and Ritalin (n = 2). Furthermore, these individuals were asked to provide informed consent for the access of medical records for the purpose of confirming the diagnosis. The ADHD group included only individuals whose diagnosis could be confirmed through medical records. Participants were asked to discontinue the use of their medication for 24 hours prior to the study to insure that they were unmedicated during the testing sessions. Participants confirmed compliance with this request at the beginning of each session.

In addition to medical records, ADHD diagnosis was confirmed by meeting symptoms-based criteria on at least two of three ADHD scales, including the ADD/H Adolescent Self-Report Scale—Short Form (AASRS; Robin & Vandermay, 1996), the Conners Adult ADHD Rating Scale—Long Form (CAARS—S:L; Conners, Erhardt, & Sparrow, 1999), and an ADHD Symptom Checklist of 12 ADHD symptoms that serve as diagnostic criteria according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association, 1994). All available information pertaining to diagnostic status (i.e., symptom ratings scales, available clinical records) was reviewed by a licensed clinical psychologist with over 30 years of experience in diagnosing ADHD. This method of diagnosis confirmation has been successfully used by this research group in other studies (e.g., Weafer, Camarillo, Fillmore, Milich, & Marczynski, 2008; Weafer, Fillmore, & Milich, 2008).

The AASRS—Short Form assesses symptoms experienced within the last month; this provided confirmation that participants were currently experiencing ADHD symptoms. The utilized cutoff score for this scale was the recommended diagnostic criterion of 11 or higher. The current study used the total ADHD symptoms subscale on the CAARS—S: L, which is based on well-established DSM-IV criteria of ADHD. The items on this scale assess the presence of ADHD symptoms throughout adulthood. For the current study, the diagnostic criteria for the CAARS—S: L was a *T* score greater than 65 on the total ADHD symptoms scale. Both scales have been shown to have sufficient specificity and sensitivity in identifying individuals with ADHD. (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999; Robin & Vandermay, 1996). Furthermore, these scales were used because they focus on ADHD symptoms as they are experienced by adults. The ADHD Symptom Checklist was created using DSM-IV items that loaded highly on the ADHD symptoms factor on the Young ADHD Questionnaire-Self-Report (Young, 2004). The scale emphasizes adult ADHD symptoms and includes six symptoms of inattention and six symptoms of hyperactivity. The respondent rates the frequency of symptom occurrence as *not at all*, *sometimes*, *often*, and *very often*. A symptom rated as occurring *often* or *very often* was counted and a total symptom count of four or more was used as a diagnostic criteria.

Participants completed the 30-item Barratt Impulsiveness Scale (BIS; Patton, Stanford, & Barratt, 1995) as an additional measure of impulsivity. The BIS served to further validate group classification. Impulsivity is a core characteristic associated with ADHD; the scale was administered to verify difference in this dimension between the ADHD and control groups. This questionnaire measures impulsivity through items such as “I act on impulse” and “I consider myself always careful”. Participants indicate how frequently each statement applies to them on a 4-point Likert scale (*never*, *occasionally*, *often*, *almost always*). Possible score totals range from 30 to 120, with higher scores indicating greater total levels of impulsiveness. Group comparisons on the BIS confirmed the expected differences in impulsivity, with the ADHD group reporting greater levels of impulsivity than the control group, $t(55) = 6.32, p < .001, d = 1.69$.

All participants were screened using health questionnaires and a medical history interview. These measures assessed participants' current or past medical disorders, including serious physical disease, impaired cardiovascular functioning, chronic obstructive pulmonary disease, seizure, head trauma, CNS tumors, or histories of psychiatric disorder. Participants in the comparison group reported past or current diagnoses of depression and/or anxiety ($n = 4$) and alcohol abuse ($n = 1$). Those in the ADHD group reported past or current diagnoses of depression and/or anxiety ($n = 5$), alcohol abuse ($n = 2$), and a learning disability ($n = 1$).

Tasks

The DOR and SI tasks were operated using E-prime experimental generation software (Schneider, Eschman, & Zuccolotto, 2002) and performed on a personal computer in a fixed order. A chin rest was used to minimize head movement and maintain an eye-to-screen distance of 73.6 cm. Eye movement was recorded using a Model 504 Eye Tracking System (Applied Science Laboratory, Boston, MA, USA), which sampled eye location at 60 Hz and recorded X/Y coordinates. These coordinates were used to define fixations and saccades. Calculations of saccade distance and duration were done using fixation onset and offset times. Onsets of fixations were defined as periods of at least 100 ms in which the line of gaze had a standard deviation of less than 0.5° of visual angle. Offsets of fixation were determined by periods of at least 50 ms in which the gaze position was at least 1° of visual angle away from the initial fixation position. All sampled eye locations between the beginning and end of a fixation were averaged to produce a final fixation position.

Delayed ocular response task—The DOR task taps intentional inhibitory control of attention by measuring a participant's ability to intentionally inhibit the tendency to make a reflexive saccade toward the sudden appearance of a visual stimulus on a computer screen. Participants were seated in a darkened room and instructed to maintain focus on a fixation point. While participants were focused on the fixation point, a bright target stimulus was presented in the periphery. The sudden appearance of such a stimulus would normally elicit a reflexive saccade towards the stimulus (Peterson, Kramer, & Irwin, 2004). However, for the DOR task participants are instructed to “delay” looking at this stimulus, and instead maintain their gaze on the fixation point until it disappears. Thus, this task requires the intentional inhibition of a reflexive saccade.

A trial began with the presentation of a white fixation point (+) with a luminance of 39.6 lux presented against a black background. Participants were instructed to fixate on this point. After 1,500 ms, the target stimulus (a white circle) briefly appeared for 100 ms to the left or right of the fixation point. The fixation point then remained on the screen for a random “wait” interval (800, 1,000, and 1,200 ms). Participants were told to withhold any saccade to the target. Following the wait interval, the central fixation point disappeared and the display was blank for 1,000 ms. Participants were told to execute a saccade to the location of the target stimulus as quickly as possibly upon the disappearance of the fixation point.

The task consisted of 96 trials, and required 7 minutes to complete. Fixation points and targets were presented in five locations that were separated from each other by 4.1° of visual angle. These positions were distributed horizontally across the center of the screen, resulting in four possible visual angles between the fixation point and target (4.1° , 8.2° , 12.3° , and 16.4°). Each trial began with the presentation of the fixation point at the target location of the preceding trial. Each of the four angular distances and the direction of the saccade required between the fixation point and target locations were presented on an equal number of trials during a test, such that 24 trials were presented at each angle with 12 of these going in each direction. The three different wait intervals each occurred in 32 trials. Target locations and wait intervals were presented in a random and unpredictable sequence.

The DOR task has been used successfully as a measure of intentional inhibitory control in selective attention in research involving participants who have deficient control of attention, such as those with schizophrenia (Ross, Heinlein, Zerbe, & Radant, 2005) or ADHD (Ross, Hommer, Breiger, Varley, & Radant, 1994), those under the influence of alcohol (Abroms et al., 2006), and older adults (Gottlob, Fillmore, & Abroms, 2007a). These groups have been shown to have poor control of selective attention and typically demonstrate reduced inhibitory control against irrelevant stimulus input. Studies of DOR task performance show

that these individuals demonstrate increased inhibitory failures compared with healthy controls.

Criterion variables: Saccades on the DOR task were categorized based on when they occurred. They were classified as either premature (i.e., occurred after target presentation but before fixation point offset), proper (i.e., occurred during the 1,000-ms response period), or late (i.e., occurred after the next fixation point appeared). On the majority of trials (81%), participants executed proper saccades. The primary measure of interest was the number of premature saccades, which represented the number of inhibitory failures. The second measure of interest was RT on trials with proper saccades. This was defined as the time elapsed between the disappearance of the fixation point and the completion of a saccade towards the target region. The final measure of interest was the accuracy to which the saccade localized the position of the target. Recall that the DOR task requires the participant to hold the location of the distracter stimulus in visual working memory until the fixation point disappears, at which time the participant must make a memory guided saccade to the location of the distracter stimulus. Accuracy was defined as the angular discrepancy between the target position and the landing point of the saccade. The difference between these two locations was measured in terms of degree of absolute deviation; greater deviation scores indicated poorer accuracy of the saccades. Greater RT of proper saccades and lower overall accuracy are indicative of working memory deficits, such as those found in individuals with schizophrenia (Ross et al., 1998). Additionally, if the onset of the saccade occurred within 60 ms of the onset of the target stimulus, the saccade was considered anticipatory and excluded from any analyses (Becker, 1989). This data reduction method is consistent with prior work using the DOR task (Abroms et al., 2006).

Saccadic interference task—The SI task taps automatic inhibitory control of attention by measuring a participant's ability to filter an irrelevant stimulus while executing a saccade towards a target location. The difference in response time between trials with and without the presentation of the distracter stimulus represents the magnitude of the SI effect (i.e., the amount of interference caused by the presence of a distracter stimulus). Reingold and Stampe (2002) have discussed the rationale for this effect. These researchers suggest that the distracter stimulus interferes with saccade generation, and automatic inhibitory processes in the superior colliculus are activated in order to minimize interference from the distracter stimulus. Slower activation of the automatic inhibitory mechanism results in greater interference, which is indicated by a larger SI effect. This explanation is supported by studies implicating the superior colliculus in saccadic inhibitory processes (Munoz, Dorris, Pare', & Everling, 2000; Munoz & Istvan, 1998; Munoz & Wurtz, 1992). The SI task has been used to demonstrate impaired automatic inhibition in older adults (Gottlob, Fillmore, & Abroms, 2007b).

The SI task used in the present study required participants to execute a saccade to the location of a target once during each trial. The task consisted of 80 trials, requiring 6 minutes to complete. A trial began with the presentation of a black fixation point (+) on a grey screen. The grey screen had a luminance of 11.5 lux. Following a variable delay period of either 500 or 900 ms, a target (a black circle) was presented to the right or left of the original fixation point. Both the fixation point and target stimulus remained on the display for 1,000 ms. Participants were instructed to fixate on the focal point and make a saccade to the target as quickly as possible upon its presentation. Fixation points were presented in the center of the display and targets were randomly presented 4.1° or 8.2° to the right or left of the fixation point. Trials were separated by 1,000 ms intervals.

The distracter stimulus was a brief (33 ms) increase in illumination that occurred at one of five stimulus onset asynchronies (SOAs) after the presentation of the target. The SOAs

between the presentation of the target and the onset of the distracter stimulus included 0, 50, 100, 150, and 200 ms. These SOAs were chosen to maximize interference, based on the findings of Reingold and Stampe (2002) that maximum interference effects occur when the distracter was presented 100 ms before the execution of the saccade. The increased illumination that constituted the distracter task was created by presenting two white bars on the top and bottom third of the screen. The luminance of these bars was 39.6 lux. Targets were presented an equal number of times at all four locations (i.e., 4.1° or 8.2° to the left or right of the fixation point), with half of the trials at each location comprising distracter trials. On trials with no distracters the timing of events was otherwise identical.

Criterion variables: Saccadic RT was the primary measure of interest for the SI task. This refers to the time elapsed between the target onset and the fixation at the target location. Separate RT means were produced for trials with and without the distracter stimulus. The SI effect is reflected by the degree to which the mean RT is longer in the distraction condition compared with the no-distraction condition. For a trial to be included in the analyses, the participant must have executed a saccade that covered at least half the distance to the target location. This data reduction method is consistent with prior work using the SI task (Abroms et al., 2006). A second measure of interest for the SI task was participants' variability in saccadic RT as measured by individual standard deviation from their own mean RT. Previous research has found that ADHD is characterized by increased RT variability (Fillmore et al., 2009; Schachar, Tannock, Marriott, & Logan, 1995; Spencer et al., 2009). This increased variability is likely linked to difficulty maintaining attention (Castellanos & Tannock, 2002).

Procedure

This study took place in a laboratory setting in the university's Department of Psychology. These tasks were administered as part of a larger testing battery that included other measures of cognitive functioning. Participants first attended an individual familiarization session in which they became acquainted with the eye tracking tasks and provided background information. After providing informed consent, participants were interviewed and completed questionnaires concerning their health status, drug and alcohol use, impulsivity, and demographic characteristics. The experimenter then administered the Kaufman Brief Intelligence Scale (K-BIT) in order to assess IQ. Participants in the ADHD group provided a signed release of their medical records and were interviewed regarding any medications currently prescribed for the disorder. All participants completed the ADHD scales. Participants then completed a single session consisting of a number of cognitive tasks to ensure that they understood the procedures. Following the task completion, a testing session was scheduled. The testing session was separated from the familiarization session by a minimum of 24 hours.

The testing session began with the participant completing preliminary questionnaires (e.g., verification that participants had not taken any medication). Upon completion of these questionnaires, participants were reminded of the requirements of the tasks. The tasks were then administered to the participants. To avoid fatigue effects, participants were allowed breaks as needed between tasks. After the testing session concluded the participants were debriefed and compensated approximately \$50 per session.

Results

A chi-square analysis found that gender make-up was independent of group, $\chi^2(1, n = 57) = .608, p = .520$. Moreover, no significant gender differences were found in task performance (p 's $> .12$). Thus, gender was not included in any analyses as a factor or covariate. Groups were found to differ significantly in age, $t(55) = 1.99, p = .052, d = .53$

and IQ $t(55) = 2.26, p = .028, d = .61$. Results did not differ when age and verbal and matrices IQ on the KBIT were included as covariates in the analyses, so analyses are reported without the inclusion of these covariates.

DOR task performance

The number of premature saccades was compared between groups using a two-sample t -test. The findings showed that the ADHD group ($M = 21.57, SD = 13.85$) produced significantly more premature saccades than the control group ($M = 9.22, SD = 6.79$), $t(55) = 4.20, p < .001, d = 1.13$. There were very few late saccades per participant, with the control group ($M = 2.21, SD = 3.81$) producing fewer late saccades than the ADHD group ($M = 3.30, SD = 3.28$). This difference was not significant, $t(55) = 1.049, p = .299, d = .28$. Figure 1 compares the mean number of premature and late saccades between groups. The ADHD group ($M = 1.67$ degrees, $SD = .56$) and the control group ($M = 1.57$ degrees, $SD = .56$) had similar accuracy of proper saccades, $t(55) = .77, p = .443, d = .21$. The ADHD group ($M = 434.90$ ms, $SD = 85.09$) and control group ($M = 418.25$ ms, $SD = 73.13$) had similar reaction times on proper saccades, $t(55) = .79, p = .434, d = .21$.

SI task performance

A 2 (ADHD vs. control) $\times$ 2 (distracter vs. no distracter) $\times$ 5 (SOA) mixed design analysis of variance (ANOVA) was conducted to determine whether RT should be collapsed across SOA. Results showed a main effect of SOA (SOA means: 0 ms = 353.23 ms, 50 ms = 335.49 ms, 100 ms = 339.34 ms, 150 ms = 334.937 ms, 200 ms = 320.99 ms), $F(4, 216) = 23.88, p < .001$. However, the effect of SOA was similar between groups, as indicated by the absence of a significant group $\times$ SOA interaction, $F(4, 216) = .428, p = .788$. Because of this nonsignificant interaction—and in the interest of clarity—the remaining analyses of the SI task are collapsed across SOA.

RT on the SI task was collapsed across SOA and analyzed using a 2 (ADHD vs. Control) $\times$ 2 (distracter vs. no distracter) mixed design ANOVA. One participant in the control group was excluded from the analysis due to problems with the eye tracking equipment. As expected, a main effect was found for condition, $F(1, 54) = 53.92, p < .001, d = 2.0$, confirming that the distracter stimulus significantly slowed reaction time in both groups. There was a main effect of group on overall reaction time, with the ADHD group responding slower than the control group, $F(1, 54) = 5.94, p = .018, d = .61$. However, there was no significant interaction between group and condition, $F(1, 54) = 1.36, p = .249, d = .31$. Recall that the SI effect refers to the RT difference between distracter and no distracter trials. The absence of a significant interaction indicates that the ADHD and control groups had a comparable SI effect. See Figure 2 for RTs across group and condition.

A similar 2 $\times$ 2 ANOVA was used to examine individual variance of RT on the SI task. Individual standard deviations were calculated for each participant and a mean SD was calculated for each group. A larger SD reflected more variability in RT. The ANOVA revealed a main effect of condition on SD, $F(1, 55) = 23.23, p < .001, d = 1.35$, and significant main effect of group, $F(1, 55) = 4.02, p = .050, d = .54$. As expected, the ADHD group had greater variability in saccadic RT than the control group. There was no significant interaction between group and condition, $F(1, 55) = .83, p = .365, d = .25$. RT variance is shown in Figure 2.

Between task comparison

Results of the DOR and SI tasks would suggest that the ADHD group was relatively less impaired on the SI task. In order to examine whether this difference in impairment was significant, criterion variables on the DOR (i.e., premature saccades) and SI tasks (i.e., SI

effect) were converted to Z scores and analyzed using a 2 (ADHD vs. Control) $\times$ 2 (DOR vs. SI task) mixed design ANOVA. The ANOVA revealed a significant main effect of group, $F(1, 55) = 10.12, p = .002, d = .86$, as well as a significant group by task interaction, $F(1, 55) = 4.88, p = .031, d = .60$. This interaction indicates that the ADHD group showed a greater impairment on the DOR task relative to the SI task.

Discussion

This study examined the inhibitory control of adults with ADHD using two selective attention tasks designed to measure intentional and automatic inhibition of distracting information. The two tasks required a similar response from the participant, but evoked a different type of inhibitory mechanism. The SI task assessed automatic inhibitory control by measuring the ability to filter an irrelevant visual stimulus. The DOR task assessed intentional inhibitory control by measuring the ability to inhibit a saccade towards a stimulus presented in the periphery. The increased number of premature saccades made by the ADHD group on the DOR task suggests a deficit of intentional inhibitory control in adults with ADHD. This is consistent with prior research in this area for adults (Carr et al., 2006) and children (Derefinko et al., 2008) with ADHD. Considering SI task results along with DOR task results, it seems that the ADHD group was less impaired in automatic inhibitory control relative to intentional inhibitory control. These results indicate a dissociation between automatic and intentional inhibitory control in adults with ADHD, such that adults with the disorder are relatively less impaired, if not unimpaired, in automatic inhibitory control.

Impairment of intentional inhibitory control was evident from task performance on the DOR, with the ADHD group executing over twice as many premature saccades on average as the comparison group. The pattern of performance observed in the ADHD group has been seen in other groups, including individuals under the influence of alcohol (Abroms et al., 2006) and individuals with schizophrenia (Ross et al., 2000). It is worth noting that alternative explanations have been proposed for the inhibitory failures of individuals with schizophrenia. It is argued that the underlying cognitive deficit for this group is one of working memory, and the inhibitory control deficits are secondary to dysfunctional working memory (Ross et al., 2000; Cornblatt & Keilp, 1994; Goldman-Rakic, 1994; Park & Holzman, 1992). Conversely, researchers speculate that this pattern is reversed in individuals with ADHD, with underlying inhibitory control deficits manifesting as dysfunctional working memory (Barkley, 1997). Because of the interdependence of working memory and inhibitory control (Ridderinkhof & van der Molen, 1997), the two are difficult to tease apart, and a failure of either mechanism can result in a premature saccade on the DOR task.

The DOR task provides a unique opportunity to separate inhibitory mechanisms and working memory (Ross et al., 2000). The accuracy and RT analyses are, by necessity, limited to proper trials (i.e., trials in which the participant executed a proper saccade), and a proper trial represents the absence of an inhibitory failure. If the inhibitory failure is responsible for the visual working memory deficits, controlling for such failures should eliminate the subsequent working memory deficits. Conversely, if inhibitory control deficits are secondary to working memory dysfunction, then measures of working memory (i.e., saccadic accuracy and saccadic RT on the DOR) should reflect this dysfunction even during proper saccades. According to this rationale, no differences between groups were found on measures of working memory in the current study (i.e., saccadic accuracy and RT) when controlling for inhibitory failures (i.e., premature saccades). This replicates the findings of Ross and colleagues and supports the notion that inhibitory control deficits of attention could contribute to working memory dysfunction of adults with ADHD. These findings are

consistent with disinhibition theories of ADHD (e.g., Barkley, 1997, Nigg, 2001), which posit that most symptoms of ADHD are secondary to inhibitory control deficits. Furthermore, the observed inhibitory control deficits have implications for other symptoms of the disorder, such as distractibility and attentional problems. Given the close relation between eye movement and attention (Shimojo et al., 1999), the impaired ability to inhibit a saccadic response towards a task-irrelevant stimulus may be implicated in the difficulties maintaining attention that is characteristic of those with ADHD. Intentional inhibitory control is also important in goal directed behavior and this may explain the difficulty in this area exhibited by adults with ADHD. However, these findings should be interpreted with the caveat that DOR places minimal demands on the visuospatial working memory system. It is possible that the set-size of one stimulus per trial was not sufficient to expose differences between groups.

The current study also explored automatic inhibitory control mechanisms in adults with ADHD and found no evidence to support a deficit in this area. It should be noted that although the SI effect was larger in the ADHD group, this group difference was small ($d = .31$) and did not reach significance. Thus, it cannot be concluded from these data that adults with ADHD are unimpaired in automatic inhibitory control; however, it is clear that these adults are less impaired in automatic inhibitory control relative to intentional inhibitory control. This differs from the findings that children with ADHD/C showed significant deficits of automatic inhibitory control (Fillmore et al., 2009). There may be several possible explanations for these differences. First, it may be that developmental changes are responsible for the intact automatic inhibitory mechanisms observed among adults with ADHD. Second, the discrepancy in findings between the current study and Fillmore et al. (2009) may be due to the use of different measures of automatic inhibitory control. It is possible that the IOR task and SI task assess different constructs, and this discrepancy is responsible for the differences in results. That the IOR task assesses automatic inhibitory control has a strong theoretical and empirical backing (see Taylor & Klein, 1998). Similarly, a large body of literature supports the validity of the SI task (see Reingold & Stampe, 2002; Munoz et al., 2000). Moreover, neurological evidence suggests that both the SI effect and IOR are derived from processes in the superior colliculus (Serenio, Briand, Amador, & Szapiel, 2006; Reingold & Stampe, 2002); this region of the brain is associated with saccadic inhibition and the generation of reflexive saccades (Munoz & Istvan, 1998; Sparks, 1978). Taken together, these findings support the notion that the IOR and SI task elicit similar automatic inhibitory control responses.

Finally, a participant selection effect may have caused the discrepancy in findings between the child and adult studies. The current study utilized a sample consisting predominately of college students. Thus, it is likely that the most severely impaired individuals were not included in the sample, and automatic inhibitory control deficits may be more severe in individuals who experience severe impairment from their ADHD. However, it is noteworthy that individuals in the ADHD group were all experiencing symptoms severe enough to require medication. Related to this, participants in the study by Fillmore et al. may have been more severely impaired relative to participants in the current study, which may explain the discrepant findings. Considering the evidence, it seems likely that either participant selection effects or developmental changes explain findings of the current study pertaining to automatic inhibitory control. Further examinations of inhibitory control deficits in individuals with ADHD will be required to further clarify this finding. Specifically, longitudinal studies will be required to control for possible participant selection effects inherent in cross-sectional research.

An examination of individual RT on the SI task revealed that the ADHD group had a slower overall RT and a larger individual RT variance than the control group. This is likely due to

the more variable RT thought to be a central characteristic of ADHD (Castellanos & Tannock, 2002). Inconsistent RT between trials on stop signal tasks is generally observed in children with ADHD (Tannock, 1998), and this finding has been extended to adults with ADHD (Barkley et al., 2008; Epstein, Conners, Sitarenios, & Erhardt, 1998). The common finding that individuals with ADHD display a slower and more variable reaction time is thought to reflect a deficit of selective attention.

The evidence of a dissociation between deficits involving intentional versus automatic mechanisms might implicate dysfunction of frontal eye fields over other neural substrates of the visual system. As part of the retinotectal pathway, the generation of endogenous (i.e., intentional) saccades is thought to occur in the frontal eye fields (Guitton, Buchtel, & Douglas, 1985), whereas the superior colliculus is involved in the generation and execution of exogenous (i.e., automatic) saccades (Mohler & Wurtz, 1977). Neuroimaging studies have found abnormal functioning in the frontal eye fields of children with ADHD (Castellanos et al., 1996), and it is possible that in adults with ADHD the localization of dysfunction to the pathway regulating controlled saccadic activity limits the inhibitory control deficit to this type of eye movement. Imaging studies examining the neurological correlates of saccadic eye movements in adults with ADHD will be necessary to further address this possibility.

A recent paper by Halperin and Shulz (2006) challenges the central role of the prefrontal cortex in the pathophysiology of ADHD. Recall that a prediction of this model is that adults with ADHD should differ from healthy adults on measures of automatic processing, whereas these differences should attenuate on measures involving conscious control. The findings of the current study run counter to these predictions in that the ADHD group was less impaired in automatic inhibitory control relative to intentional inhibitory control.

Limitations

The results of the current study contribute to the understanding of inhibitory processes in adults with ADHD; however, there are several limitations. First, the use of a single ADHD group may have obscured differences between subtypes. There appear to be differences in automatic inhibitory control between subtypes of ADHD in children (Fillmore et al., 2009; Derfinko et al., 2008), and it is unknown whether this discrepancy continues into adulthood. Accordingly, it is possible that adults with ADHD/C show a different pattern of inhibitory control deficits than those with ADHD/PI. Future research should address this limitation by utilizing subtype groups. Second, the battery of ADHD scales did not include an observer report measure, as recommended by some (Barkley et al., 2008). Such external data may further validate diagnosis status. However, it is important to note that the field lacks consensus on the most appropriate strategy for diagnosing ADHD in adulthood, with research suggesting that even the conventional *DSM-IV* criteria may not be appropriate for adults with ADHD (Barkley, Murphy, & Fischer, 2008). Thus, the multi-step classification strategy utilized in the current study was developed to obtain converging evidence from multiple sources of information in order to confirm diagnostic status.

A third limitation is that eye movement do not always indicate the allocation of attention. Although attention and eye fixation are closely related, there are some circumstances in which a shift in attention does not result in a saccade toward the attended region. For example, a reflexive shift in attention may not result in an eye movement if the individual is able to quickly return his or her attention to the originally attended region (Gojin & Theeuwes, 2003). This is particularly relevant for the DOR task. It is possible that the ADHD and control groups performed similar number of premature shifts in attention, but participants in the control group were able to redirect their attention to the fixation point before a saccade was programmed. Lastly, this study was not able to rule out motivation

deficits thought to be related to ADHD (Luman, Oosterlaan, Sergeant, 2005; Sonuga-Barke, 2002) as an alternative explanation for the findings. It is possible that the ADHD group showed a greater impairment on the DOR task because they were less motivated to perform this effortful process. Conversely, the less effortful SI task may be less susceptible to motivational factors.

In sum, the present research provides new information on the specific nature of ocular inhibitory impairments experienced by adults with ADHD. A substantial number of studies have examined dissociations between different types of inhibitory control in children with ADHD (e.g., Fillmore et al., 2009), and these studies have clarified our understanding of these deficits in this population. Similar studies are required to better understand inhibitory deficits in adults with ADHD. By distinguishing between automatic and intentional inhibitory control the current study was able to show that the inhibitory impairment experienced by adults with ADHD appears greater in intentional, relative to automatic, inhibitory mechanisms.

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Figure 1. Mean number (+ SE) of premature and late saccades of the ADHD and comparison groups on the delayed ocular response task.

Figure 2. Mean saccadic reaction time (+ SE) and mean individual standard deviation (+ SE) of the ADHD and comparison groups on distracter and no distracter conditions of the saccadic interference task.

Table 1

Demographic and diagnostic information by group

	Group				<i>t</i>
	Control (<i>n</i> = 27)		ADHD (<i>n</i> = 30)		
	Mean	SD	Mean	SD	
Demographic					
Age	22.1	1.7	21.1	1.7	2.2*
Education	15.3	1.3	15.1	1.0	0.7
IQ: Verbal	107.1	6.9	103.2	10.8	1.6
IQ: Nonverbal	111.2	8.8	105.6	9.8	2.2*
IQ: Composite	110.1	6.9	104.9	10.1	2.3*
Diagnostic					
CAARS					
DSM-IA	51.0	11.1	75.9	11.5	8.3***
DSM-HI	46.4	9.7	64.8	14.8	5.5***
DSM-Tot	49.0	11.4	73.7	11.8	5.6***
Index	45.1	8.4	59.1	10.1	8.0***
DSM	0.9	1.3	5.4	2.1	9.7***
AASRS	8.1	5.6	21.4	5.8	8.8***
BIS	52.6	8.5	68.5	9.9	6.5***

Note. Group contrasts were tests by independent sample *t* tests. For all comparisons, $n = 57$, $df = 55$. Age is reported in years. Education refers to years education completed. IQ: Verbal, IQ: Nonverbal, and IQ: Composite refers to respective scaled scores of the Kaufman Brief Intelligence Test. CAARS scores are T-scores; DSM-IA is DSM-IV Inattentive Symptoms, DSM-HI is DSM-IV Hyperactive-Impulsive Symptoms; DSM-Tot is DSM-IV ADHD Symptoms Total; and Index is ADHD Index. DSM refers to symptom count on the ADHD symptoms checklist. AASRS refers to total score on the ADD/H Adolescent Self-Report Scale—Short Form. BIS refers to the total score on the Barratt Impulsiveness Scale.

* $p < .05$,

*** $p < .001$.