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Inhibitory Processes in Adults With Persistent Childhood Onset ADHD

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The theory that attention-deficit/hyperactivity disorder (ADHD) stems from a deficit in an executive behavioral inhibition process has been little studied in adults, where the validity of ADHD is in debate. This study examined, in high-functioning young adults with persistent ADHD and a control group, 2 leading measures of inhibitory control: the antisaccade task and the negative priming task. ADHD adults showed weakened ability to effortfully stop a reflexive or anticipated oculomotor response but had normal ability to automatically suppress irrelevant information. Results suggest that an inhibitory deficit in ADHD is confined to effortful inhibition of motor response, that antisaccade and negative priming tasks index distinct inhibition systems, and that persistence of ADHD symptoms into adulthood is associated with persistence of executive motor inhibition deficits.

Childhood attention-deficit/hyperactivity disorder (ADHD) is a serious and chronic behavioral syndrome characterized by impaired attention, impulsivity, and excessive activity. A significant percentage of ADHD children show persistent symptoms into adulthood (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998). Increasingly, clinicians must assess adults for ADHD (Barkley, 1998; Weiss, Hechtman, & Weiss, 1999) without clear consensus on validity of the syndrome, the appropriate diagnostic criteria, or the active psychological dysfunction (Faraone, 2000). Further data on basic mechanisms are needed to advance assessment and intervention in adult ADHD.

ADHD in adults is viewed as a neurodevelopmental disorder with childhood onset (Barkley, 1998). Supported by extensive empirical data (Nigg, 2001; Pennington & Ozonoff, 1996), recent theories emphasize neuropsychological executive dysfunction as crucial in child ADHD, with a particular emphasis on inhibitory problems (Barkley, 1997). Neuropsychological deficits in adults with ADHD have begun to be confirmed (Corbett & Stanczak, 1999; Downey, Stelson, Pomerleau, & Giordiani, 1997), but prior studies relied on molar executive measures that do not isolate inhibitory processes (Pennington & Ozonoff, 1996).

Moreover, inhibition is not a unitary construct in terms of neural or cognitive process (Nigg, 2000). For example, effortful inhibition of motor response may have different neural correlates than cognitive suppression (Harnishfeger, 1995). On the basis of theories that emphasize motor inhibition deficits in prefrontal-basal ganglia neural circuits (Barkley, 1997), we expected ADHD deficits only in the former. In keeping with new developments in the field, we chose measures with extensive cognitive theory and data behind them, tapping (a) effortful suppression of oculomotor response and (b) relatively automatic cognitive inhibition.

The oculomotor antisaccade task is often used to study inhibition in psychopathology. Participants try to suppress (anticipatory) eye movements during a waiting period as well as reflexive (cued) eye movements in response to a sudden stimulus onset. This executive task is impaired by competing task demands and by damage to orbitoprefrontal cortex (Guitton, Buchtel, & Douglas, 1985). Distinct neural correlates are involved in oculomotor control, evading problems of peripheral motor dysmaturation that sometimes accompany ADHD. Prior oculomotor studies found that child ADHD was associated with difficulty suppressing premature responses or cue-reflex responses (Castellanos et al., 2000; Munoz, Hampton, Moore, & Goldring, 1999; Ross, Hommer, Breiger, Varley, & Radant, 1994). Two studies found the same pattern of results, but inhibition effects were shy of significance (Aman, Roberts, & Pennington, 1998; Rothlind, Posner, & Schaughency, 1991).

The negative priming paradigm was developed to study central inhibitory processes in attention (May, Kane, & Hasher, 1995). When implemented within the context of a Stroop-type paradigm, negative priming means that if the word *green* was suppressed to name the color *blue* on the initial trial, then it takes longer to name the color *green* on the next trial than if there was no relation among the color names and words on the two trials (May et al., 1995). It has been thought to represent central (executive) cognitive inhibition and thus might be impaired if ADHD reflects general failure of central inhibitory processes (Ozonoff, Strayer, McMahon, & Fillouz, 1998). This type of inhibition is distinct from the effortful suppression of motor response. Two studies of child ADHD yielded mixed results. Ozonoff et al. (1998) found a deficit in ADHD + Tourette syndrome, whereas Gaultney, Kipp, Weinstein, and McNeil (1999) found no deficit in ADHD alone.

In summary, this experiment examined ADHD inhibitory deficits using two paradigms that cover diverse kinds of inhibitory processes. In keeping with a motor inhibition model of ADHD (Nigg, 2000), we hypothesized that adults with persistent ADHD would have deficits in (a) suppression of reflexive response to a cue (antisaccade errors) and (b) effortful suppression of extraneous

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Table 1
Demographic and Behavioral Description of Groups

	Control (n = 21)		ADHD (n = 22)		
Variable	*M*	*SD*	*M*	*SD*	*p*
Males/females	9/12		10/12		.61 (*ns*)
Age (years)	21.6	2.4	23.1	4.9	.21 (*ns*)
Ethnicity (% White)	74		82		.71 (*ns*)
Family income level	4.05	0.9	3.73	1.2	.33 (*ns*)
Full-scale IQ	105.5	10.6	108.3	10.6	.39 (*ns*)
Child inattention	0.5	0.2	7.3	1.4	<.001
Child hyperactivity	0.3	0.7	6.0	2.5	<.001
Child Wender score	9.7	8.6	48.8	14.4	<.001
Adult inattention (*T*)	52.1	3.2	70.3	6.4	<.001
Adult Anx–Dep (*T*)	52.2	3.7	58.4	6.9	.001
Adult Antisocial (*T*)	54.1	4.8	57.6	9.7	.16 (*ns*)
Adult adaptive (*T*)	51.7	4.9	39.9	10.4	<.001
Adult Brown ADD (*T*)	50.1	0.2	79.0	14.8	<.001

Note. Child inattention and hyperactivity = number of respective *DSM–IV* symptoms endorsed. Child Wender score = 25 items that best identify ADHD (Ward, Wender, & Reimherr, 1993). Other adult symptom scores are from Achenbach's (1997) Young Adult Self Report, (YASR): Anx–Dep = Anxiety–Depression; Antisocial = YASR Delinquent scale; Brown ADD = Brown Attention Deficit Disorders Scales (Brown, 1996) total score. Annual family income is scaled with 1 = < $20,000, 2 = $20,000–$40,000, 3 = $40,000–$60,000, 4 = $60,000–$100,000, 5 = >$100,000. Sample ethnicity: 78% Caucasian, 5% African American, 5% Latino, 7% Asian American, and 5% other (e.g., Middle Eastern). ADHD = attention-deficit/hyperactivity disorder; *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.).

response during a waiting period (antisaccade anticipations) but (c) not in suppressing information through a central cognitive suppression system (negative priming). Nearly all prior cognitive studies of adults with ADHD, and most studies of children, failed to consider comorbid symptoms, but they are a key issue. We therefore checked whether effects were independent of comorbid subclinical aggression and anxiety/depression symptoms. Last, if disinhibition is integral to ADHD, it should be seen even in a high-functioning group; we sampled accordingly to test this hypothesis.

Method

ADHD (n = 22) and non-ADHD (n = 21) college students over age 18 were recruited through the department participant pool, a campus Disability Resource Center, a community college, and campus newspaper ads. Exclusion criteria included reading disability[1] (because it has been associated with inhibitory problems and could confound findings), IQ < 70, history of psychosis, neurological disorder, or head injury. Major depression was excluded because it can compromise validity of adult ADHD diagnosis (Barkley, 1998) and influence antisaccade performance. Current prescriptions for psychostimulant medications were in force for 48% of the ADHD sample, either Ritalin (42%) or Dexedrine (6%). Participants were tested after 24 hr off medication. We excluded those on other medications (antidepressants; n = 1). To guard against any noncompliant medication use, we rechecked results after covarying medication status and removed participants on stimulants, with no change in significance decisions for the primary hypotheses. Sample description is provided in Table 1. Demographically, groups did not differ. Results were unchanged with IQ or age covaried.

Diagnosis of ADHD was according to *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM–IV*; American Psychiatric Association, 1994) criteria for childhood combined type, with persistence of symptoms in adulthood. Normative rating scales served to establish empirically the presence of extreme attentional symptoms in adulthood. Resources were not available to obtain informant ratings.[2] Participants were assigned to the ADHD group if (a) they had previously been diagnosed with ADHD by a psychologist or psychiatrist in the community (to reduce false positives), (b) at least six inattentive and six hyperactive–impulsive symptoms were present in childhood, using an "or" algorithm to identify symptoms from the National Institute of Mental Health Diagnostic Interview Schedule for DSM–IV (DIS–IV) and a self-report symptom checklist (all but n = 3 had sufficient symptoms on the DIS–IV alone), (c) these symptoms were

[1] Reading disability was identified if Wide Range Achievement Test Word Reading < 85 and more than 1 standard deviation below full-scale IQ. Full-scale IQ was obtained with a reliable and valid seven-subtest short form (Axelrod & Paolo, 1998) of the Wechsler Adult Intelligence Scale—Revised.

[2] There are multiple issues when one attempts to establish continued ADHD diagnosis in adults, with no age-appropriate consensus diagnostic criteria extant. Our method therefore followed that of "residual ADHD" (*DSM–III–R*) or "ADHD in partial remission" (*DSM–IV*). Because the *DSM–IV* criteria cutoffs are of unknown appropriateness for adults (Barkley, 1998; Weiss et al., 1999), the use of normative rating scales adds to validity. Although self-report data can be subject to distortion, adults' self-reports of their current ADHD symptoms correspond well with ratings by observers (Downey et al., 1997), and their recollection of childhood symptoms is viewed by many clinical observers as the most valid source of information about their childhood symptoms (Barkley, 1998). However, recalled childhood symptoms are likely to be more reliably obtained by structured diagnostic interview than by rating scales. We therefore used the *DSM–IV* version of the Diagnostic Interview Schedule (DIS–IV; Robins et al., 1995), which includes an ADHD module to assess both childhood symptoms and current adult impairment from those symptoms. Interviews were administered by graduate student interviewers after they completed 16 hr of training by a trainer certified by the St. Louis DIS group. Interview fidelity was checked by having the trainer view a random selection of videotaped interviews.

present by age 7 per the structured interview, and (d) problematic symptoms continued in adulthood on the basis of $T > 65$ on the Conners Adult ADHD Rating Scale (Connors, Erhardt, & Sparrow, 1999) or Achenbach's (1997) Young Adult Self Report Attention Problems Scale.

Individuals were included in the control group if they had no history of ADHD, had $T < 64$ on the Achenbach (1997), Conners, and other ADHD instruments,[3] and endorsed fewer than 5 childhood symptoms of hyperactivity–impulsivity and inattention. Current subclinical levels of depression and anxiety, as well as comorbid aggression, were assessed with the Achenbach (1997) scales for purposes of covariance analysis.

Apparatus

Stimuli were presented on an NEC Multisync XE 15-in. (38.1 cm) monitor driven by a Hercules Dynamite Pro super video graphics adapter card. A voice key connected to a dedicated input–output (I/O) board collected vocal responses; activation of the voice key stopped a millisecond clock on the I/O board and generated a system interrupt that was serviced by software. Eye movements were recorded by an ISCAN RK-416 eye movement monitor at 120 Hz.

Antisaccade Task

The fixation display consisted of a black screen with a white cross (0.9° of visual angle) in the center, flanked by two white squares (1.3°) that were black in the middle. The onset was a luminance change from black to white in the center of the squares, 8.5° from the fixation cross. In the prosaccade condition, a target arrow appeared at the same locations on the screen as the onsets and subtended an angle of 1.1°. In the antisaccade condition, the arrow appeared in the opposite location.

Participants were seated 40 cm from the computer screen (maintained by forehead and chin rest) and were instructed to look at the center cross until the onset occurred. The preonset delay period varied randomly from 500 to 1,000 ms in 100-ms increments. In the prosaccade condition, participants were to look toward the onset; in the antisaccade condition, they were to look toward the box opposite from the onset. They pressed the top button for an up arrow and the bottom button for a down arrow. The intertrial interval was 3 s. The 40 antisaccade and 40 prosaccade trials were blocked; block order was independent of diagnosis. The initial saccade following the presentation of the onset was determined by identifying the first fixation (eye position that remained within 0.35° of its initial position for more than 20 ms) outside a central region 1.3° from the center of the screen (see Butler, Zacks, & Henderson, 1999).

Negative Priming Task

Participants were seated 60 cm from the computer screen. They were instructed to name the color of each word presented and not to correct their mistakes. Participants then saw one of four color words—RED, GREEN, BLUE, and WHITE—presented in one of four colors in the center of the computer screen. To control the effect of the previous trial on the responses to the current trial, we constructed pairs of trials, with each pair of trials containing a prime and a probe trial. The first item in each pair was the prime trial; responses to the 150 prime trials were not analyzed. The probe trials consisted of 50 facilitation (color word and color were the same), 50 interference (color word and color were different), and 50 negative priming trials. For facilitation and interference trials, the correct response was unrelated to the prime trial. The correct response on negative priming trials was the name of the color word (suppressed) on the prime trial.

On each trial, the color word was displayed until the voice key was triggered; then the screen went blank for 800 ms until the next trial. Participants had a 2-min break after the 76th, 150th, and 226th trials. The experimenter recorded errors and incorrect voice key triggers.

Results

Antisaccade Task Results

Trials were excluded from the latency and direction accuracy analyses if no saccade was made or if the response was an anticipation (a saccade before or within 100 ms of the onset); 6.6% of the control and 15.5% of the ADHD trials were eliminated. We conducted a preliminary check of correct trial reaction times (RTs) as an index of arousal or effort effects. The main analyses addressed (a) saccade directional errors and (b) fixation failures (anticipations) as indices of inhibitory ability. Raw data are in Table 2.

As expected, antisaccades took longer to initiate than prosaccades for both groups ($p < .001$). Inspection of means in Table 2 reveals that, in the prosaccade condition, the ADHD group responded nonsignificantly faster than the control group. Consistent with the increased effort required when inhibiting the reflexive response, the ADHD group was nonsignificantly slower to initiate correct antisaccades, yielding a significant interaction, $F(1, 40) = 4.32$, $p = .044$.

As expected, more saccade direction errors occurred overall in the antisaccade than the prosaccade condition, $F(1, 40) = 43.2$, $p < .0001$. Whereas both groups performed above 99% correct in the prosaccade condition, participants in the ADHD group were less accurate in moving their eyes away from the onset, resulting in a Task × Group interaction, $F(1, 40) = 4.99$, $p = .031$, $\eta^2 = .11$. The ADHD group had more errors than the control group in the antisaccade condition, $F(1, 40) = 5.71$, $p = .022$. This effect became nonsignificant with current aggressive behavior ($p = .22$) or anxiety–depression ($p = .54$) covaried. Thus errors were not specific to ADHD symptoms.

Suppressing Anticipatory Eye Movements

The ADHD group made more anticipatory eye movements in the prosaccade than the antisaccade task (19% vs. 7%), whereas controls did not (5.5% vs. 5.4%), resulting in a Task × Group interaction, $F(1, 40) = 15.2$, $p < .001$, $\eta^2 = .28$, and a simple effect of group in the prosaccade condition, $F(1, 40) = 10.7$, $p = .002$, that was robust to all covariates. A regression model showed independent associations to inhibition failure of attention problems (partial correlation; $pc = .33$, $p = .04$) but not aggression ($pc = .04$, $p = .37$) or anxiety–depression ($pc = .19$, $p = .26$). Anticipations were related to childhood *DSM–IV* inattention ($r = .43$, $p = .003$) and hyperactivity ($r = .39$, $p = .006$). This inhibitory deficit, therefore, was specific to the ADHD symptoms.

Negative Priming Results

For the negative priming analyses, trials were excluded from the RT analyses if (a) RT < 100 ms or RT > 2,000 ms; (b) the voice

[3] These other instruments were the Wender Utah Rating Scale (total score < 30) and the Brown Attention Deficit Disorder Rating Scale ($T < 64$). The reason for including these extra measures in identifying the control group was to minimize the chance of "false-positive" controls (i.e., controls with clinical ADHD symptoms by any definition of the term). These measures were not used in identifying the ADHD group because of their limited norms.

Table 2
Reaction Times and Accuracy Rates (Percentage Errors) in Negative Priming and Antisaccade Tasks With Statistical Test and Effect Size (d) for Simple Effects

	Control			ADHD				
Task	%	*M*	*SD*	%	*M*	*SD*	*p*	*d*
Antisaccade task								
Prosaccade latency (ms)		340	50		328	54	.38	0.28
Antisaccade latency (ms)		424	71		451	62	.18	0.42
Prosaccade anticipations	5		5	19		18	.002	1.04
Antisaccade anticipations	5		8	8		9	.54	0.20
Prosaccade errors	<1		<1	<1		1	.20	0.40
Antisaccade errors	9		12	18		15	.022	0.76
Negative priming task								
Negative priming (ms)		710	109		816	183	.023	0.76
Interference (ms)		689	101		792	179	.024	0.76
Facilitation (ms)		620	93		721	145	.009	0.90

Note. Errors represent trials in which the first saccade was in the wrong direction. Anticipations are saccades that occurred during the variable length waiting period prior to the onset or within 100 ms of the onset. The effect size measure *d* is the group difference divided by the mean standard deviation, and thus is in standard deviation units. ADHD = attention-deficit/hyperactivity disorder.

key had been triggered incorrectly; (c) a naming error occurred; or (d) the trial immediately followed an incorrect voice key or naming error trial. Overall, 8.7% of trials were thus excluded (0.5% of facilitation trials, 2.4% of interference trials, and 3.0% of negative priming trials). Excluded trials did not differ by group or Group × Condition (all Fs < 1.9). Negative priming occurred as expected, with slower response times to negative priming than interference trials, $F(1, 38) = 8.79$, $p = .005$ (see Table 2). The ADHD group was slower overall, $F(1, 38) = 5.7$, $p = .02$. However, negative priming effect did not differ by group: interaction $F(1, 38) = .07$, $p = .80$, $\eta^2 = .002$. Responses were faster to facilitation than interference trials ($p < .001$), and the ADHD group was slower ($p = .014$) but with no interaction ($p = .87$).

Discussion

Adults with ADHD had deficits in effortful motor inhibition (antisaccade) but not in cognitive inhibition (negative priming). On the antisaccade task, RTs to correct prosaccade trials were slightly faster in the ADHD group, suggesting that inhibition failures were not readily ascribed to problems in arousal, effort, or task engagement. Their nonsignificantly slower response onsets in the antisaccade condition may represent a strategy effect (speed–accuracy trade-off), which may have dampened the error-rate finding. The ADHD group had problems both in suppressing anticipatory saccades during the waiting period and in suppressing directional movements during the antisaccade condition, although only the former was robust to covariates.

Our results extend similar findings by Munoz et al. (1999) in ADHD children, bolstering the conclusion of a deficit in the ability to suppress unwanted eye movements. Likewise, Castellanos et al. (2000), using a different oculomotor task design, found a similar pattern in ADHD girls, with a large deficit on anticipations and a somewhat smaller deficit on reflex suppression errors. The latter parallel suggests that our results may generalize to ADHD girls. Overall, the antisaccade data lend qualified support to the theory that when problems with ADHD persist from childhood to adulthood, they may persist because of problems in an executive inhibitory control system that depends on prefrontal cortex. The effect involves motor inhibition and does not include cognitive suppression as measured here.

There was no evidence that negative priming inhibition is associated with ADHD, confirming findings of Gaultney et al. (1999) in children. The very small interaction effect size suggests that the null finding was not due to low power or small sample size. If the negative priming effect is inhibitory (see Milliken, Joordens, Merikle, & Seiffert, 1998), this type of inhibition, perhaps related to cognitive suppression processes, is spared in ADHD.[4]

Unlike prior cognitive studies of ADHD adults, we covaried subclinical comorbid symptoms. As in child studies, comorbid aggression partially but not entirely accounted for executive deficits (Nigg, Hinshaw, Carte, & Treuting, 1998). Depressive symptoms are known to affect antisaccade performance in adults, so it was unsurprising that the anxiety–depression scale partially accounted for oculomotor inhibition deficits. Further investigation of the role of anxiety is needed in more representative samples. However, the most important point was that the primary inhibitory deficit in ADHD was independent of these comorbid subclinical symptoms.

The primary qualifications to these findings concern limitations to the sample. First, the sample represents only a subgroup of individuals with ADHD. They had average to above-average IQ and were attending college; many ADHD children have below-average IQ and many do not attend college. Also, our sample was mostly female; although a high representation of female participants is not atypical of adult ADHD samples, it is atypical of most child ADHD samples. The

[4] There is debate about the core processes in negative priming (Milliken et al., 1998). If the task fails to tap inhibition, then that would be an alternative explanation for the results. However, Conway (1999) evaluated the episodic trace retrieval hypothesis of negative priming (which ascribes the negative priming effect to memory rather than attentional processes). Results suggested that a dual-mechanism model, drawing on both attentional and memory processes, best accounts for negative priming.

sample was also restricted by requiring a prior diagnosis in the community. Second, ADHD diagnoses depended on self-report. Although there is evidence for the validity of such self-reports (Downey et al., 1997), inclusion of informant reports is desirable. Also, although like most ADHD studies we verbally confirmed nonuse of stimulant medication prior to testing, some participants might deny stimulant use to gain experimental credits. Stimulants improve ADHD antisaccade performance (Aman et al., 1998), so noncompliance would work against finding group effects. Finally, comorbid diagnoses were assessed by rating scale rather than interview; specificity effects thus need further study.

In all, results may be most applicable to high-functioning ADHD and to female clients. Larger deficits might be present in a more impaired sample, so negative findings are best viewed with caution, and positive findings may underestimate the magnitude of ADHD deficits. Yet, deficits in this high-functioning group suggest that inhibitory dysfunction in relation to motor suppression may be integral to persistent ADHD, whereas central cognitive suppression may not.

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