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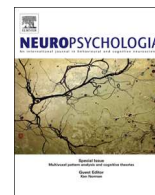
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## Neuropsychologia

journal homepage: [www.elsevier.com/locate/neuropsychologia](http://www.elsevier.com/locate/neuropsychologia)

## Neurophysiological differences in inhibitory control between adults with ADHD and their peers

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## ARTICLE INFO

## Article history:

Received 30 January 2013

Received in revised form

21 June 2013

Accepted 25 June 2013

Available online 4 July 2013

## Keywords:

Attention-deficit/hyperactivity disorder

(ADHD)

Adults

EEG

Inhibitory control

## ABSTRACT

Inhibitory control allows individuals to suppress prepotent responses and resist irrelevant stimuli, and is thought to be a core deficit in Attention-deficit/hyperactivity disorder (ADHD). Whereas numerous studies have investigated neural mechanisms underlying inhibitory control deficits in children with ADHD, less is known about underlying mechanisms in young adults with ADHD. This study explores the neural correlates of inhibitory control in college students with ADHD—a population that, despite comparatively high educational attainment, still shows marked functional impairments in academic, social, and occupational functioning. Participants were 54 college students with ADHD and 29 typically developing peers. Specifically the fronto-centrally located N2 and the centro-parietal P3 event-related potential (ERP) components were hypothesized to show decreased amplitudes for the ADHD group due to their known association with inhibitory control. Dense array electroencephalography (EEG) data was collected during a Go/nogo task. Results show lower accuracy rates for the ADHD group and significant reductions in P3 amplitude as well as a trend for reduced N2 amplitude in nogo trials where subjects successfully inhibited a response. Notably, nogo N2 and P3 amplitudes correlated with the number of ADHD symptoms: namely, smaller amplitudes were associated with more symptoms. We conclude that when compared to their typically developing peers, relatively high functioning adults with ADHD still show a deviant neural signature. These results contribute to the growing literature of adult ADHD and increase our understanding of the neural correlates of inhibitory control associated with ADHD.

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## 1. Introduction

Current empirical data suggests that attention-deficit/hyperactivity disorder (ADHD) is best conceptualized as a neurobiological disorder (Kieling, Goncalves, Tannock, & Castellanos, 2008). However, the diagnosis remains based upon evidence of significantly impairing, persistent, and age-inappropriate behavioural symptoms of inattention or hyperactivity/impulsivity, or both (American Psychiatric Association [APA], 2000). Approximately 5% of children under the age of 18 have ADHD (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Willcutt, 2012a) and according to a meta-analysis it occurs in about 4.5% of adults (Kessler et al., 2006; Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). While its symptoms and negative effects were initially believed to remit in adolescence, longitudinal research clearly indicates that this is not the case (Biederman, Petty, Evans, Small,

& Faraone, 2010). Problems in cognitive processes often remain, and in fact the arrival of young adulthood brings about new areas of potential impairment such as problems in self-reliance and self-monitoring (Gropper & Tannock, 2009; McGough et al., 2005; Torgersen, Gjervan, & Rasmussen, 2006).

At the same time, ADHD predicts a highly dispersed pattern of impairment across academic, behavioural, social, and affective domains for youth and adults alike. College students with ADHD display an academic profile that differs from most individuals with ADHD. Academically speaking, they are a relatively “high functioning” group in comparison to a large proportion of individuals with ADHD who are at 2- to 4- fold risk for academic underachievement, a two-fold risk for grade repetition, and a high school drop-out rate of 32–40% (e.g., Barkley et al., 2002; Barry, Lyman, & Klinger, 2002; Hinshaw, 1992; Loe & Feldman, 2007; Rapport, Scanlan, & Denney, 1999; Todd et al., 2002). College students with ADHD have attained relatively good academic outcomes, appear to be minimally impaired on tests of neuropsychological function compared to their peers, and manifest comparatively low levels of comorbidity compared to clinical samples of young adults with ADHD (Heiligenstein, Guenther, Levy, Savino, & Fulwiler 1999; Weyandt & DuPaul, 2006). Nonetheless, college students with ADHD struggle to successfully

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complete college and university level programs (DuPaul, Weyandt, O'Dell, & Varejao, 2009; Frazier, Youngstrom, Glutting, & Watkins, 2007). As such, these resilient individuals who appear to have overcome their neurobiological and environmental risk factors and gained entrance to post secondary education, are an important subgroup to study and understand.

A better theoretical understanding of the mechanisms underlying the difficulties associated with ADHD, and the college student subpopulation in particular, may inform the development of interventions designed to increase academic achievement and educational attainment in this population. Deficient inhibitory control, such as inhibition of a prepotent response or an ongoing response, has been postulated to be a core deficit in ADHD (Barkley, 1997). Evidence for deficits in inhibitory control in children with ADHD is growing and has come from various sources including stop-signal (see Lipszyc & Schachar, 2010, for meta-analysis) and Go/nogo tasks (Durstun, 2003; Gow et al., 2012; Inoue et al., 2012; Liddle et al., 2011). Although less research on inhibitory control has been conducted with adults with ADHD, a meta-analysis by Hervey, Epstein, and Curry (2004), using various neuropsychological measures, concludes that the domain of inhibitory control is impaired in adult ADHD.

However, the specificity of this deficit of inhibitory control at a syndromic or symptom level seems weak. Many other psychopathologies, including Tourettes syndrome, Obsessive Compulsive Disorder, and Schizophrenia, also demonstrate associated impairments on tasks requiring inhibitory control (for reviews, see: Lipszyc & Schachar, 2010; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). Moreover, meta-analyses find that individuals with ADHD have executive function (EF) impairments in many domains other than inhibitory control, such as attention and memory (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Frazier, Demaree, & Youngstrom, 2004; Hervey et al., 2004; Willcutt et al., 2008).

These findings make it difficult to directly attribute ADHD symptomatology to deficits in a specific EF. One reason for this difficulty could be that psychological research often measures the behavioural outcome of a cognitive deficit and not the mechanisms underlying it. For example, the wide array of EF deficits could be explained by a central deficit in the allocation of attention with downstream negative effects on other processes or EFs, such as working memory and inhibitory control that are dependent upon it. Moreover, performance likely depends on a complex interaction of multiple EFs acting together in a very close time span of several milliseconds.

To combat the sole reliance on behavioral outcome measures, neuroimaging methods, like electroencephalography (EEG), are commonly employed due to their high temporal resolution and tradition of interpreting event-related potentials (ERP, averaged electrophysiological recordings time-locked to an event). These techniques allow for a more refined investigation of when the deficit occurs, which may provide a better understanding of the behavioural sequelae that are observed. Although ERP research using the Stop-signal paradigm with individuals with ADHD exists; studies using Go/nogo tasks with adults with ADHD are infrequent. Go/nogo and stop-signal paradigms differ in that Go/nogo tasks rely on the ability to restrain a response initiation while providing a direct measure of inhibition accuracy; whereas, stop-signal tasks rely on the ability to cancel an ongoing response, from which a derived behavioral measure of inhibition latency is computed. Evidence suggests that both tasks are tapping into distinct, yet related, inhibitory processes at a behavioral (Schachar et al., 2007; Soreni, Crosbie, Ickowicz, & Schachar, 2009) as well as a neural level (Swick, Ashley, & Turken, 2011). The purpose of this study is to characterize the neural profile underlying Go/Nogo performance in college students with ADHD.

The nogo N2 and P3 are the most commonly studied ERP components in Go/nogo tasks associated with inhibitory control. The N2 is a frontocentral negativity typically measured 200–400 ms post stimulus on a successful inhibition trial (i.e., Nogo trial). The precise meaning of its activity is still under debate. The N2 has been linked to response inhibition (Falkenstein, Hoormann, & Hohnsbein, 1999), conflict monitoring and response selection (Donkers & van Boxtel, 2004; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003), as well as a combination of both processes (Lavric, Pizzagalli, & Forstmeier, 2004). Regardless of whether the N2 activation can, at least in part, reflect the actual inhibition of prepotent responses, there has been mounting support for the N2 component as a signature for conflict monitoring (Enriquez-Geppert, Konrad, Pantev, & Huster, 2010; Huster, Enriquez-Geppert, Pantev, & Bruchmann, 2012; Smith, Smith, Provost, & Heathcote, 2010). Most estimations of the neural source of the N2 point to the anterior cingulate cortex (ACC) (Bekker, Kenemans, & Verbaten, 2005; Bokura, Yamaguchi, & Kobayashi, 2001; Crottaz-Herbette & Menon, 2006; Kropotov & Ponomarev, 2009; Woltering, Granic, Lamm, & Lewis, 2011): a region strongly implicated in conflict monitoring and attentional control (Carter & Van Veen, 2007; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004).

When compared to their typically developing peers, children and adolescents with ADHD generally have lower N2 amplitudes in Go/nogo tasks measuring inhibitory control (Brandeis et al., 2002; but see Fallgatter et al., 2004; Gow et al., 2012; Overtom et al., 1998; Yong-Liang et al., 2000), and lower nogo N2 amplitudes have been correlated with poorer self-regulation in ADHD (Wiersema & Roeyers, 2009). The few studies using a Go/nogo paradigm to compare inhibitory control in adults with ADHD and healthy controls, found no group differences in N2 amplitudes (Helenius, Laasonen, Hokkanen, Paetau, & Niemivirta, 2011; Wiersema, van der Meere, Antrop, & Roeyers, 2006). However, these studies had small sample sizes, and may not have had enough statistical power to detect group differences in N2.

The nogo P3 occurs after the N2 and is a positive waveform typically measured between 300 and 600 ms post stimulus on successful Nogo trials. The nogo-P3 has been associated with the P3a and novelty P300 in the literature (see Polich, 2007, for review) but in this manuscript we shall refer to it as the P3. Whereas the N2 is associated with a monitoring function, the P3 in Go/nogo tasks is increasingly seen to be an index of the inhibition of a motor response, the evaluation thereof, or both (Bruin, Wijers, & Van Staveren, 2001; Dimoska, Johnstone, & Barry, 2006; Enriquez-Geppert et al., 2010; Huster et al., 2012; Smith, Johnstone, & Barry, 2008). Much like the N2, the P3 has also been source localized to the ACC, but there is evidence that a wider network is involved that includes regions such as the inferior parietal lobe, involved in attentional control; and the supplementary motor cortex, involved in motor control (Crottaz-Herbette & Menon, 2006; Huster, Westerhausen, Pantev, & Konrad, 2010; Kropotov & Ponomarev, 2009; Mulert et al., 2004; Simmonds, Pekar, & Mostofsky, 2008).

Research comparing groups of individuals with and without ADHD using Go/nogo tasks have generally shown reductions in P3 amplitudes at centroparietal sites for children and adolescents (Brandeis et al., 2002; Fallgatter et al., 2004; Gow et al., 2012; Overtom et al., 1998) as well as adults with ADHD (Wiersema et al., 2006, note: parietal P3; Helenius et al., 2011). The decreased P3 amplitudes for individuals with ADHD may suggest that fewer attentional resources are allocated to inhibitory control and related evaluative processes.

In sum, studies generally report reduced N2 and P3 amplitudes for ADHD participants in Go/Nogo tasks. These findings are in line with other tasks of inhibitory control, such as Stop Signal, Flanker, and Continuous Performance tasks, that have also found reduced



N2's (Albrecht, Banaschewski, Brandeis, Heinrich, & Rothenberger, 2005; Dimoska, Johnstone, Barry, & Clarke, 2003; Liotti et al., 2007, Liotti, Pliszka, Higgins, Perez, & Semrud-Clikeman, 2010; Prox, Dietrich, Zhang, Emrich, & Ohlmeier, 2007) and P3's (Bekker et al., 2005; Dhar, Been, Minderaa, & Althaus, 2010; Fallgatter et al., 2004, 2005; Liotti et al., 2007, 2010; McLoughlin et al., 2010; Overtom et al., 2002; Valko et al., 2009).

The current study aims to contribute to the literature on neural deficits in inhibitory control in young adults with ADHD. Specifically, we focused on this understudied population of college students with ADHD, and their typically developing peers. Furthermore, we tested whether these deficits occurred in hypothesized neural indices associated with inhibitory control. We predict an attenuated N2 and P3 indicative of deficits of inhibitory control.

## 2. Methods

### 2.1. Participants

Sixty-five students (current enrollment in post-secondary program) with ADHD (32 male, 33 female; mean age=25,  $sd=5.8$ ) were recruited from University Student Services and 32 healthy controls were students recruited through campus advertisements (14 male, 18 female; mean age=25,  $sd=4.9$ ). Inclusion criteria for the ADHD group were (1) a previous diagnosis of ADHD, and (2) registration with university or college Student Disability Services, which requires supporting documentation of a confirmed diagnosis of ADHD. All participants completed the Adult ADHD Self Report Scale (ASRS; Kessler et al., 2005) to assess current symptoms of ADHD. Exclusion criteria (for control and clinical group) were (1) uncorrected sensory impairment, (2) major neurological dysfunction and psychosis, and (3) current use of sedatives or mood altering medication other than stimulants prescribed for ADHD.

Participants were classified into either the ADHD or comparison group. We used the ASRS-A and the cognitive failures questionnaire (CFQ; Broadbent, Cooper, Fitzgerald, & Parkes, 1982) in support of our classification (see measurement sections, for detailed description of these scales). All participants who were classified as a member of the ADHD group had evidence of a DSM-IV diagnosis of ADHD as provided by the university student disability services. However, if these participants had fewer than 3 screen-positive scores on their current ASRS-A and were not receiving medication for treatment of ADHD, they were excluded from analysis. This was the case for 6 participants. Conversely, if a participant in the comparison group had 4, or more, screen-positive scores on the ASRS-A and they scored 50 or higher on the CFQ, they were also excluded from the analysis. This was the case for 2 participants. In addition, 6 participants had to be discarded because they had trial counts lower than 10, which left us with a final sample of 83 participants, of whom 54 were classified as ADHD (mean age=25.1,  $sd=5.2$ ; 28 male, 26 female) and 29 were classified into a typically developing comparison group (mean age=25.2,  $sd=5.2$ ; 13 male, 16 female). Groups did not differ significantly in age ( $p=.91$ ), or sex ( $p=.54$ ) as shown by *t*-tests and Chi-square tests, respectively.

Amongst our final sample, 25 participants with ADHD (46.3%) were being treated with medication. Of those 25 participants, 20 were using stimulants only, 1 was using anti-depressants only, and 4 were using a combination of stimulants and antidepressants. For medical and ethical reasons, participants were not asked to stop or change their medication treatment when visiting the lab for assessment.

The present study was approved by the institutional Research Ethics Board (protocol reference #23977) and all participants provided informed written consent prior to the start of the study.

### 2.2. Procedure

Before the task started, participants provided basic demographic information and completed various questionnaires and neuropsychological tasks, described below. Participants were then seated in a comfortable chair and fitted with a 129-channel EEG net (Electrical Geodesic, EGI). EEG data acquisition started after impedances for all channels were reduced to below 50 k $\Omega$  in accordance with standard data collection procedures (Ferree, Luu, Russell, & Tucker, 2001; Kappenman & Luck, 2010). Data was collected using a 0.1–1000 Hz bandpass hardware filter and a 500 Hz sampling rate and referenced to electrode Cz.

### 2.3. Go/nogo task

The Go/nogo task was presented using E-Prime software (Psychological Software Tools, Pittsburgh, PA). Participants were instructed to press a button on a response pad as fast as possible whenever a letter appeared on the screen (the Go

condition) and to withhold responding whenever a letter was repeated a second time in succession (the Nogo condition). Participants completed a practice block of 21 trials before the actual task consisting of 21 Nogo's (72 Nogo's, 33%) started.

Stimuli appeared pseudorandomly on the screen for at least 450 ms, or less when a subject pressed the button earlier in the case of a correct Go or an incorrect Nogo. After a successful Go or Nogo, an 800 ms clear-time was introduced until the next stimulus appeared to avoid unwanted intertrial effects (e.g., a previous waveform conflating a subsequent one). Error feedback was provided by a red bar appearing in the center of the screen for 300 ms following incorrect responses to motivate participants to keep pressing as fast as they could, and as a result, build up a strong response tendency. In the case of an incorrect Go (e.g., participants failed to respond to the go stimulus), a 500 ms clear-time was introduced after the error bar appeared. Incorrect Nogo's (e.g., participants responded when they should not have done so) had an extra 400 ms of clear-time in between the response and the error feedback, after which another 500 ms of clear-time followed. Average accuracy and reaction time were displayed approximately every 36 trials.

### 2.4. Clinical measures

Each participant completed a number of standardized questionnaires and tasks to assess current levels of behavioral, cognitive and social emotional functioning. All of these measures have been used in previous research and have solid psychometric properties.

The Adult ADHD Self-Report Scale (ASRS v1.1) is a reliable and valid screener for evaluating current ADHD symptoms in adolescents and adults (Adler et al., 2012; Kessler et al., 2005; Hines, King, & Curry, 2012). The ASRS v1.1 consists of eighteen questions (nine probe for inattention, and another nine probe hyperactivity/impulsivity) based on the criteria used for diagnosing ADHD in the DSM-IV-TR. Six of these items, the ASRS Part A (ASRS-A), have been found to be most predictive of symptoms consistent with a diagnosis of ADHD and a sum score will be used in the current paper. The two subscales, all consisting of 9 items each, were also used in order to examine the domains of inattention and hyperactivity. Subtypes were not investigated considering recent conclusions drawn in the field about their poor temporal stability questioning their validity (Willcutt et al., 2012b).

The cognitive failures questionnaire (CFQ) measures self-reported failures in perception, memory, and motor function in everyday life. Twenty-five questions ask subjects to rank how often these mistakes occur (Broadbent et al., 1982). Previous research has established its reliability and validity in quantifying the distractibility of individuals. CFQ scores show temporal stability with good test-retest consistency even after 16 months (Broadbent et al., 1982). Moreover, self-report scores on the CFQ correlate highly with those derived from ratings by spouses and CFQ scores predict attention performance in laboratory settings (Forster & Lavie, 2007; Tipper & Baylis, 1987).

The Symptom Assessment-45 (SA-45; Maruish, 1999) The SA-45 is a brief assessment of psychiatric symptomatology. It is based on the well-validated longer version (SCL-90-R) to create a brief, yet thorough, measure of psychiatric symptomatology. It is often used as a screening tool, to help in diagnosis, to develop treatment plans, and to measure outcomes. Its items cover symptoms of obsessive compulsivity, psychoticism, somatization, sensitivity to others, hostility to others, paranoid ideation, phobias, and depression.

### 2.5. EEG data processing

Using Netstation (Electrical Geodesic, EGI), data was filtered off-line using a 0.1–30 Hz finite impulse response (FIR) bandpass filter. Correct Go and Nogo data were segmented into epochs from 400 ms before to 1000 ms after stimulus onset. Correct Nogo trials that did not have a correct Go trial preceding, and following them, were removed from analysis because they may have reflected attentional lapses or chronic non-responding.

Segments containing artifacts were removed using automatic algorithms for the detection of eye blinks, eye movements, as well as large drifts, and spikes in the data. Eye blinks were detected when the vertical eye channels exceeded a threshold of 150  $\mu V$  (max–min) within a 160 ms (moving) time window within each trial after running a 20 ms moving-average smoothing algorithm across the entire trial period. Eye movements were detected when horizontal eye channels exceeded a threshold of 100  $\mu V$  (max–min) over a 200 ms time window. Channels were automatically marked bad when they exceeded a transition threshold of 200  $\mu V$  over the entire segment (max–min). Segments containing more than 20% bad channels were automatically removed. Also, all segments were visually inspected by a trained research assistant, who was blind to the hypotheses. Bad channels were replaced by values interpolated from remaining channels, using spherical splines.

Groups did not significantly differ in segment count for either the Nogo trials (ADHD: mean ( $sd$ )=35.8 (13.2); Comparison: mean ( $sd$ )=36.2 (10.8);  $p=.89$ ) or the Go trials (ADHD: mean ( $sd$ )=95.3 (22.8); Comparison: mean ( $sd$ )=91.9 (33.2);  $p=.63$ ). Data from six subjects were discarded for not meeting our cutoff criterion of more than 9 segments (for rationale, see Olvet & Hajcak, 2009). After averaging the trials, all channels that were referenced to Cz during recording were

re-referenced to an average reference. Data were baseline corrected using 200 ms preceding the Nogo or Go stimulus onset.

ERP components during correct Nogo conditions were automatically coded and manually checked for accuracy, and then adjusted, by trained research assistants blind to the hypotheses. The N2 was scored on stimulus-locked waves as the largest peak negative deflection with a frontocentral topography (averaged across values recorded at FCz, Fz, and Cz) between 200 and 400 ms after nogo stimulus onset. The P3, a long waveform, was determined by taking the mean activation across parietal sites between 300 and 600 ms after the stimulus (average across values recorded at Pz, P3, and P4). Time windows and sites for hypothesized components were determined a priori based on the grand average waveform, previous lab experience (e.g., unpublished pilot studies), and a review of the literature of Go/Nogo tasks similar to ours (e.g., Davis, Bruce, Snyder, & Nelson, 2003; Wiersema et al., 2006), and may therefore differ from their typical assessment. We also obtained the P1 and N1 components to investigate early perceptual effects. The early occipital P1 and N1 were scored at occipital sites as the most positive peak deflection between 80 and 160 ms post-stimulus and the most negative peak deflection between 130 and 230 ms, respectively. (Averaged across values recorded at Oz, O1 and O2). Effects of medication and trial count were covaried out of each ERP analysis and also assessed separately for each analysis in the supplements.

## 2.6. Statistical analysis

For the statistical analyses, ANOVA's and *t*-tests were conducted to compare the ADHD and comparison groups. Due to the strong hypothesis driven nature of the study, no correction for multiple comparisons was performed with respects to temporal and spatial correlates of the EEG analysis. Data points of ERP as well as behavioral variables with standard deviations larger than three were removed from the analysis as outliers. Partial  $\eta^2$  values were computed to ascertain effect size. According to Vacha-Haase and Thompson (2004),  $\eta^2 = .01$  corresponds to a small effect,  $\eta^2 = .10$  corresponds to a medium effect, and  $\eta^2 = .25$  represents a large effect.

## 3. Results

### 3.1. Symptom and neuropsychological tests

As expected, ANCOVA's controlling for medication showed significantly higher ASRS-A scores for the ADHD group for current ADHD symptomatology as measured by the ASRS-A,  $F(1, 76) = 136.74$ ,  $p < .001$ ,  $\eta^2 = .64$ , and cognitive failures, as measured by the CFQ,  $F(1, 70) = 47.33$ ,  $p < .001$ ,  $\eta^2 = .40$ . No group differences were found on an auditory working memory task as measured by the WAIS Digit Span ( $p = .32$ ). Table 1 shows the means and standard deviations of the ASRS, CFQ, and the WAIS digit span, as well as the subscales of the SA-45.

**Table 1**

Mean and Standard deviations of the ASRS (ADHD Self Report Scale), CFQ (cognitive failures questionnaire), WAIS Digit Span, and SA-45 subscales (Symptom Assessment-45) for the ADHD and the Comparison group. Last column represents the *p*-values for the group differences.

	ADHD	Comparison	<i>p</i> -values
ASRS-A***	4.59 (.94)	1.12 (1.42)	.000
ASRS-Hyperactivity***	21.74 (5.64)	9.52 (6.67)	.000
ASRS-Inattention***	26.64 (4.34)	12.36 (5.46)	.000
CFQ-total***	57.48 (11.88)	33.26 (14.47)	.000
WAIS Digit Span (SS)	9.22 (2.89)	9.88 (2.24)	.32
SA-45			
Anxiety***	9.43 (3.58)	6.72 (2.13)	.001
Depression**	10.46 (4.99)	8.16 (3.52)	.01
Obsessive Compulsive**	13.96 (5.00)	10.04 (3.69)	.005
Somatization	8.69 (4.00)	7.88 (3.84)	.22
Phobic anxiety	6.06 (2.14)	5.20 (0.82)	.10
Hostility	6.70 (3.05)	5.72 (1.28)	.17
Interpersonal sensitivity	9.46 (4.08)	7.84 (3.50)	.30
Paranoid Ideation	8.17 (3.46)	6.92 (2.57)	.18
Psychoticism	5.87 (1.85)	5.92 (1.41)	.98

\* $p < .05$ .

\*\* $p < .01$ .

\*\*\* $p < .001$ . ANCOVA's controlled for medication.

Furthermore, as typical of adult ADHD populations (Kessler et al., 2006), the ADHD group also showed significantly more internalizing problems than the comparison group, as shown by differences in the SA-45 subscales of anxiety ( $p = .001$ ), depression ( $p = .01$ ), and OCD ( $p = .005$ ).

### 3.2. Go/nogo behavioural outcomes

ANCOVA's, controlling for medication, showed that the ADHD group performed significantly worse in the Go/nogo task on Go accuracy,  $F(1, 77) = 4.28$ ,  $p = .04$ ,  $\eta^2 = .05$ , and the Nogo accuracy,  $F(1, 77) = 4.73$ ,  $p < .03$ ,  $\eta^2 = .06$ . No differences were found for measures related to reaction time. Table 2 shows the mean and standard deviation for accuracy on Go- and Nogo trials, the Go-reaction time, and a measure of the intra-individual variability (IIV) of reaction time.

### 3.3. ERP

The amplitude of the nogo N2 was attenuated in the ADHD group compared to controls, although the difference did not reach standard levels of significance,  $F(1, 79) = 2.77$ ,  $p = .10$ ,  $\eta^2 = .03$ . As is common in the literature, we also investigated the relationship between the nogo N2 and the go-N2 and found larger magnitudes for the nogo N2 for both groups. However, no group differences were found when examining the nogo-go difference-waveform ( $p = .30$ ) (see supplemental Fig. 1 for the N2 difference-waveforms).

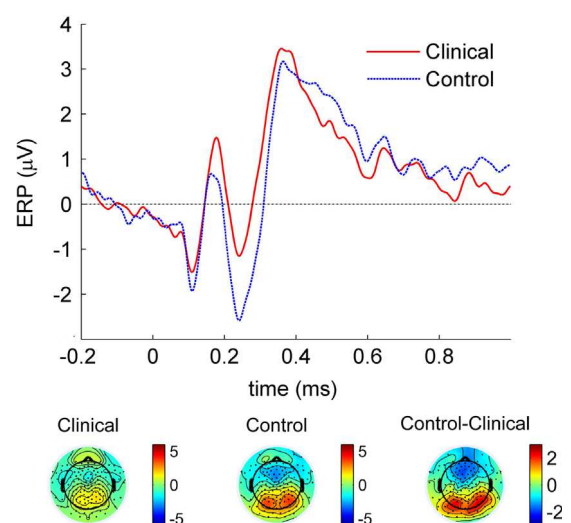
**Table 2**

Mean and Standard deviations for Go and Nogo accuracy, as well as the reaction time (in milliseconds—Go trials) and the intra-individual variability (IIV) for the ADHD and the Comparison group.

	ADHD	Comparison	<i>p</i> -value
Accuracy Go (%) *	90 (5)	91 (4)	.04
Accuracy Nogo (%)*	60 (18)	64 (14)	.03
Reaction time (ms)	317.44 (32.99)	311.82 (20.51)	.19
Reaction time (IIV)	96.05 (15)	94.34 (14.15)	.14

\*\* $p < .01$ , \*\*\* $p < .001$ . ANCOVA's controlled for Medication.

\* $p < .05$ .



**Fig. 1.** (A) N2 waveform for the ADHD group (red) and the Comparison group (blue) averaged across sites Cz, FCz, and Fz. (B) Topoplot showing average activation across the scalp between 220 and 270 ms for the ADHD group, the control group, as well as a difference plot subtracting the ADHD group activation from that of the control group. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

As expected, a significant attenuation was found for the P3 amplitudes in the ADHD group,  $F(1,79)=4.34$ ,  $p<.04$ ,  $\eta^2=.05$ . No significant differences were found for the early occipital, P1 ( $p=.97$ ) and the N1 ( $p=.79$ ) components. Furthermore, no Latency differences were found for clearly defined components such as the P1 ( $p=.76$ ), N1 (.34), and N2 ( $p=.54$ ). Figs. 1 and 2 show the waveforms and topoplots for the N2 and P3, respectively. The topoplots reveal that, as expected, the N2 shows a fronto-central distribution whereas the P3 is more central-posterior. Differences between groups were also generally largest in those areas for each respective component.

Table 3 shows the means and standard deviations of the ERP amplitudes and latencies for each component.

### 3.4. Relationships between ERP and behavioural measures

Relations between ERP components and behavioural performance measures during the Go/nogo task were investigated using data from all participants combined. The N2 correlated negatively with Nogo accuracy in the task,  $r(79)=-.281$ ,  $p<.01$ , suggesting that poor response control is associated with smaller amplitude N2's. The correlation between N2 and nogo accuracy was significant for the ADHD group ( $p=.001$ ), but not for the comparison group ( $p=.60$ ).

Investigating relations between ERP components and the questionnaires across groups revealed a positive correlation

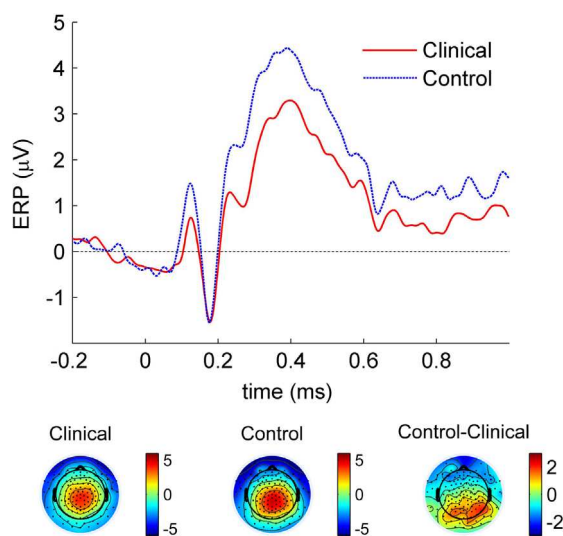
between ASRS-A and the N2, albeit not significant,  $r(79)=.239$ ,  $p=.06$ , suggesting that smaller N2 amplitudes were linked to more ADHD symptoms. There was a significant negative correlation between the P3 amplitude and the ASRS-A,  $r(79)=-.285$ ,  $p=.01$ , meaning that smaller P3's were associated with more self-reported ADHD symptoms. The P3 correlated with the inattentive symptoms of the ASRS,  $r(79)=-.25$ ,  $p=.03$ , but not with the hyperactivity/impulsivity symptoms ( $p=.28$ ).

### 3.5. Effects of medication

Our study design did not randomize, or systematically control for medication use: as a consequence, no strong conclusions can be drawn concerning the effects of medication. These analyses merely function to provide additional transparency and information in support of the main analysis.

To more completely describe the effects of medication in our sample, we split the sample into three groups: an ADHD-no meds group ( $n=29$ ; mean age=25.0,  $sd=5.13$ ; 62.1% male), an ADHD-meds group ( $n=25$ , mean age=25.24,  $sd=5.06$ ; 40% male), and a comparison group ( $n=29$ , mean age=25.24,  $sd=5.13$ ; 44.8% male). Table 4 shows the means and standard deviations for the psychological battery, clinical questionnaire, as well as the behavioural data.

ANOVA's were run to test for group differences for each of the psychological battery and behavioural performance variables. Significant group differences were found for the ASRS, and its subscales, as well as the CFQ. Although pairwise comparisons showed no difference between the ADHD participants on versus off medication (all  $p$ 's  $>.90$ ), both subgroups differed significantly from the comparison group. Similarly, while significant group differences were also found for the SA-45 anxiety,  $F(2, 79)=7.24$ ,  $p\leq.001$ ; depression,  $F(2, 79)=3.82$ ,  $p<.05$ ; and obsessive



**Fig. 2.** (A) P3 waveform for the ADHD group (red) and the Comparison group (blue) averaged across sites Pz, P3 and P4. (B) Topoplot showing average activation across the scalp between 250 and 500 ms for the ADHD group, the control group, as well as a difference-plot subtracting ADHD group activation from that of the control group. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

**Table 4**  
Mean and Standard deviations for the psychological test data (e.g., ASRS, CFQ, and WAIS), the clinical questionnaire (SA-45), and the behavioral data during the Go/Nogo (e.g., Go and Nogo accuracy as well as the reaction time (in milliseconds—Go trials) and the intra-individual variability (IIV) measure for the reaction time for the ADHD-nomeds, ADHD-meds, and the Comparison group.

	ADHD-no meds (n=29)	ADHD-meds (n=25)	Comparison (n=29)
ASRS-A***	4.69 (0.97) <sub>a</sub>	4.48 (0.92) <sub>a</sub>	1.12 (1.42) <sub>b</sub>
ASRS- Hyperactivity***	20.93 (5.50) <sub>a</sub>	22.64 (5.77) <sub>a</sub>	9.15 (6.79) <sub>b</sub>
ASRS-Inattention***	26.89 (3.64) <sub>a</sub>	26.36 (5.09) <sub>a</sub>	11.88 (5.87) <sub>b</sub>
CFQ***	58.26 (12.70) <sub>a</sub>	56.57 (11.04) <sub>a</sub>	33.26 (14.47) <sub>b</sub>
WAIS-Digit Span (SS)	9.21 (2.86) <sub>a</sub>	9.24 (2.99) <sub>a</sub>	9.88 (2.24) <sub>a</sub>
SA-45			
Anxiety***	10.00 (4.24) <sub>a</sub>	8.76 (2.52) <sub>a,b</sub>	6.72 (2.13) <sub>b</sub>
Depression*	11.48 (5.47) <sub>a</sub>	9.28 (4.16) <sub>a,b</sub>	8.16 (3.52) <sub>b</sub>
Obsessive Compulsive**	13.69 (5.01) <sub>a</sub>	14.28 (5.07) <sub>a</sub>	10.04 (3.69) <sub>b</sub>
Somatization	9.21 (4.17) <sub>a</sub>	8.08 (3.81) <sub>a</sub>	7.88 (3.84) <sub>a</sub>
Phobic Anxiety	6.03 (2.54) <sub>a</sub>	6.08 (1.61) <sub>a</sub>	5.20 (0.82) <sub>a</sub>
Hostility	6.72 (3.03) <sub>a</sub>	6.68 (3.13) <sub>a</sub>	5.72 (1.28) <sub>a</sub>
Interpersonal Sensitivity	8.97 (4.32) <sub>a</sub>	10.04 (3.79) <sub>a</sub>	7.84 (3.50) <sub>a</sub>
Paranoid ideation	8.10 (3.81) <sub>a</sub>	8.24 (3.07) <sub>a</sub>	6.92 (2.57) <sub>a</sub>
Psychoticism	5.93 (2.17) <sub>a</sub>	5.80 (1.44) <sub>a</sub>	5.92 (1.41) <sub>a</sub>
Accuracy Go	0.89 (0.05) <sub>a</sub>	0.91 (0.06) <sub>a</sub>	0.91 (0.04) <sub>a</sub>
Accuracy Nogo	0.55 (0.16) <sub>a</sub>	0.66 (.18) <sub>a</sub>	0.64 (0.14) <sub>a</sub>
Reaction time	322.25 (33.39) <sub>a</sub>	311.84 (32.30) <sub>a</sub>	311.82 (20.51) <sub>a</sub>
Reaction time (IIV)	100.08 (12.03) <sub>a</sub>	91.35 (16.92) <sub>a</sub>	94.34 (14.15) <sub>a</sub>

Note. Means that do not share subscripts differ by the following  $p$  values in the footnotes.

\*  $p\leq.05$ .

\*\*  $p\leq.01$ .

\*\*\*  $p\leq.001$  according to Bonferroni.

**Table 3**  
Means and standard deviations for ERP amplitudes as well as the latencies.

	ERP Amplitudes (µV)		ERP latencies (ms)	
	ADHD	Comparison	ADHD	Comparison
P1	2.69 (2.85)	3.24 (3.13)	119.55 (21.60)	120.57 (16.19)
N1	-2.86 (3.34)	-2.27 (3.74)	171.25 (21.15)	165.79 (19.14)
N2	-2.36 (2.78)	-3.79 (3.37)	252.38 (30.79)	259.38 (34.66)
P3	2.99 (1.94)	4.25 (2.17)	N/A	N/A



compulsive subscales,  $F(2, 79)=6.18$ ,  $p < .01$ , pairwise comparisons revealed no differences amongst the medicated and unmedicated ADHD subgroups. However, significant group differences existed between the unmedicated ADHD subgroup and the comparison group for anxiety and depression subscales of the SA-45. Moreover, both the medicated and unmedicated ADHD subgroups differed significantly from the comparison group on the obsessive compulsive subscale. No main effect of group was found for the WAIS-DS. The behavioural data showed a significant main effect of Group for Nogo accuracy,  $F(2,77)=3.554$ ,  $p < .05$ . The pairwise comparisons showed a pattern whereby the ADHD group not taking medication tended to perform worse than the ADHD group on medication ( $p=.057$ ) as well as the comparison group ( $p=.098$ ), albeit nonsignificant. No significant main effect of Group was found for Go-accuracy, Go-reaction time and the intra-individual variability in Go-reaction time, although there was a pattern in each of the variables showing the comparison and ADHD medication group to be similar, and outperforming the ADHD group without medication.

When investigating the ERP's of interest, there was a significant main effect of group for the P3 amplitudes,  $F(2, 79)=3.73$ ,  $p < .05$ . Pairwise comparisons showed a pattern whereby the comparison group differed from both the medicated and nonmedicated ADHD groups, who did not differ. A similar pattern could be found for the N2, albeit nonsignificant. Table 5 shows these results.

#### 4. Discussion

The current study set out to investigate the neurophysiological correlates of inhibitory control in college students with ADHD during a Go/nogo task. As hypothesized, we found that when compared to healthy controls, college students with ADHD showed a reduced P3 amplitude and signs of reductions in N2 amplitude, reflecting an altered neurophysiological pattern during inhibitory control. This neural signature correlated with ADHD symptoms as measured by ASRS across the whole sample and the ADHD group in particular. Therefore, this neural deficit may relate to the manifestation of ADHD symptoms in adults.

Our ADHD sample showed a pattern of decreased N2 amplitude during response inhibition processing in fronto-central sites. These results are in line with previous studies on children with ADHD (Brandeis et al., 2002; Gow et al., 2012; Overtom et al., 1998; Wiersma & Roeyers, 2009; Yong-Liang et al., 2000) but not others (see, Fallgatter et al., 2004). The N2 likely reflects conflict monitoring/evaluative processing (Carter & van Veen, 2007; Enriquez-Geppert et al., 2010; Huster et al., 2012; Ridderinkhof et al., 2004; Smith et al., 2010), rather than response inhibition per se. Because this effect was not statistically significant we should interpret this relationship as suggestive and we conclude that our hypothesis was only partly supported.

The N2 also correlated with nogo accuracy performance, as opposed to go accuracy, suggesting a role in inhibitory control. Interestingly, this effect seemed to be driven by the ADHD group and not the controls. It is possible that individuals in the

comparison group already had a more optimal neural processing, as shown by their presumed larger N2's, which may not have elicited enough neural variation in relation with their behavioral performance, explaining the absence of a significant positive correlation. This interpretation may strengthen the notion, which was only partially supported based on a direct group comparison, of N2-differences between ADHD and control group. The lack of a significant N2 reduction in the presence of significant correlations with NoGo Accuracy may suggest that a larger sample size may have been required to achieve a significant effect, or may be required in the future to reach a firm conclusion concerning N2 amplitude changes in college students with ADHD.

When compared to their college peers, the students with ADHD also showed attenuated P3 amplitudes at central-posterior sites, which is consistent with previous findings in studies on children, adolescents, and adults with ADHD (Brandeis et al., 2002; Fallgatter et al., 2004; Gow et al., 2012; Helenius et al., 2011; Overtom et al., 1998; Wiersma et al., 2006). The P3 is considered to be an index of response inhibition processing and/or the evaluation thereof (Bruin et al., 2001; Dimoska et al., 2006; Enriquez-Geppert et al., 2010; Huster et al., 2012; Smith et al., 2008). Neural activity at many brain regions, e.g., the ACC, parietal lobe, supplementary motor cortex, etc., may underlie this ERP component (Crottaz-Herbette & Menon, 2006; Huster et al., 2010; Kropotov & Ponomarev, 2009; Mulert et al., 2004; Simmonds et al., 2008). The reduced P3 amplitudes during response inhibition found in this and previous studies suggest that neural deficits presented in children with ADHD persist into adulthood, including high-functioning young adults with ADHD attending post-secondary education.

Similarly, a recent fMRI study comparing adults with ADHD and healthy controls, found reduced activation in fronto-parietal executive networks and other regions, during a Go/nogo task measuring response selection and attentional control (Mulligan et al., 2011). However, fMRI studies lack the temporal specificity that EEG offers. The current study, for example, suggests that ADHD symptoms not only affected the motor inhibitory or later evaluative processing, P3, but may also be suggestive of affecting the earlier monitoring processing N2.

No group differences were found in the early perceptual components such as the P1 and N1. Because of the relatively large sample size, this is unlikely due to issues related to statistical power. Therefore, at least for this task and sample, college students with ADHD demonstrated similar perceptual processing compared to healthy controls. However, because the perceptual processing demands were relatively low in this task (i.e., just detecting whether the letter is repeated or not), this result may not generalize to other tasks where perceptual processing demands are high.

In the present study, the ADHD group had a lower Nogo accuracy rate than the comparison group. Interestingly, this effect seems to be driven by those ADHD subjects who were not using medication. It is possible that in the ADHD group the effects of medication improved performance by increasing their vigilance and attentiveness to the task. Considering the measurable effects

**Table 5**

Means and standard deviations for ERP amplitudes, as well as the latencies, for the ADHD-nomeds, ADHD-meds, and the Comparison group.

	ERP Amplitudes ( $\mu V$ )			ERP latencies (ms)		
	ADHD no meds ( $n=29$ )	ADHD meds ( $n=25$ )	Comparison ( $n=29$ )	ADHD no meds ( $n=29$ )	ADHD meds ( $n=25$ )	Comparison ( $n=29$ )
P1	3.09 (3.07)	2.24 (2.57)	3.24 (3.13)	122.21 (19.28)	116.56 (23.99)	120.57 (16.19)
N1	−2.33 (3.70)	−3.47 (2.82)	−2.27 (3.74)	171.07 (19.73)	171.44 (23.05)	165.79 (19.14)
N2	−2.25 (2.21)	−2.49 (3.36)	−3.79 (3.37)	254.79 (34.18)	249.58 (26.75)	259.38 (34.66)
P3	3.11 (1.95)	2.85 (1.96)	4.25 (2.17)	N/A	N/A	N/A

of medication on an already relatively high-functioning sample of individuals with ADHD, we interpret these findings as an indication of response control deficits persisting well into adulthood. Indeed, impaired response control is consistent with previous findings on children with ADHD (Durstun, 2003; Gow et al., 2012; Inoue et al., 2012; Liddle et al., 2011; Lipszyc & Schachar, 2010), and in support of previous studies on adults with ADHD (Hervey et al., 2004).

In conclusion, we found that young, relatively high functioning college students with ADHD show different neurophysiological processing patterns during inhibitory control. Our hypotheses were confirmed that the differences in neural response occurred within a 200–600 ms time window, and at specific sites suggestive of difficulties with response monitoring, active motor inhibition, or the evaluation thereof. The results of this study therefore provide evidence that even this seemingly resilient group of young adults with ADHD who are enrolled in post-secondary education, display abnormal neurophysiological functioning suggestive of a less efficient allocation of attention in the service of inhibitory control.

## Acknowledgements

We would like to thank Dr. Marc Lewis for the use of his Canadian Foundation for Innovation (CFI #482246)—funded EEG lab. This research was financially supported by Dr. Rosemary Tannock's Canada Research Chair program and the Canadian Institutes of Health Research operating Grant (CIHR # 245899, Tannock & Lewis).

## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.neuropsychologia.2013.06.023>.

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