Adult ADHD and working memory: Neural evidence of impaired encoding

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**Abstract**

Objectives: To investigate neural and behavioural correlates of visual encoding during a working memory (WM) task in young adults with and without Attention-Deficit/Hyperactivity Disorder (ADHD).

Methods: A sample of 30 college students currently meeting a diagnosis of ADHD and 25 typically developing students, matched on age and gender, performed a delayed match-to-sample task with low and high memory load conditions. Dense-array electroencephalography was recorded. Specifically, the P3, an event related potential (ERP) associated with WM, was examined because of its relation with attentional allocation during WM. Task performance (accuracy, reaction time) as well as performance on other neuropsychological tasks of WM was analyzed.

Results: Neural differences were found between the groups. Specifically, the P3 amplitude was smaller in the ADHD group compared to the comparison group for both load conditions at parietal–occipital sites. Lower scores on behavioural working memory tasks were suggestive of impaired behavioural WM performance in the ADHD group.

Conclusions: Findings from this study provide the first evidence of neural differences in the encoding stage of WM in young adults with ADHD, suggesting ineffective allocation of attentional resources involved in encoding of information in WM.

Significance: These findings, reflecting alternate neural functioning of WM, may explain some of the difficulties related to WM functioning that college students with ADHD report in their everyday cognitive functioning.

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1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is the most frequently diagnosed psychiatric disorder in childhood with worldwide prevalence rates estimated at 5.3% (Polanczyk and Rohde, 2007). Longitudinal studies show that approximately 65% of children with ADHD continue to show symptoms in adulthood (Barkley et al. 2002; Faroone et al., 2006; Polanczyk and Rohde, 2007). A newly-emergent subset of young adults with ADHD – those who gain entrance into post-secondary education – is of interest because of their ongoing impairments despite educational success relative to others with ADHD (DuPaul et al., 2009). These students present various impairments in social and academic domains. For instance, they exhibit difficulties with college adjustment, social skills and self-esteem (Shaw-Zirt et al., 2005), lower Grade Point Averages, poorer academic success, and they are more likely to be placed on academic probation (Heiligenstein et al., 1999; Frazier et al., 2007). Working memory (WM), seen as one of the core deficits of ADHD (Martinussen et al., 2005; Hervey et al., 2004), could be a key factor explaining some of the functional impairments mentioned above. WM allows us to temporarily hold...
and manipulate information “on-line” for a few seconds in order to respond effectively based on that information (Baddeley, 2003). WM involves several stages of information processing: encoding, maintenance (rehearsal) and retrieval. WM has been linked with many real world activities (e.g., reading comprehension, following a conversation, problem-solving; Baddeley and Hitch, 1994) and therefore WM impairments may have pernicious effects on everyday life, especially considering the social and academic demands students face in college.

WM processing can be effectively investigated using ERP methodology due to its fine temporal resolution. Among ERP components, changes in P3 amplitudes, occurring about 300 ms after the stimulus, have been reliably associated with WM functioning. Several theories have been proposed to interpret the role of the P3 in working memory tasks (for overview, see Kok, 2001; Polich, 2007), however, there is no consensus as to what the P3 represents in the context of a WM task, especially during the encoding phase. For example, Donchin and Coles (1988) interpreted the P3 within the framework of a “context updating theory” which proposes that P3 is increased when there is a need to update, change, or refresh the current contents of WM. Researchers also focus on the intricate role of attentional processes in working memory functioning. For instance, some suggest that WM capacity is determined by an executive attentional process (Kane et al., 2007; Vogel et al., 2005), or the ‘capacity of the focus of attention’ (i.e., the scope of attention; Cowan, 2001). Ford et al. (1994) suggested that P3 amplitude reflects the effort to allocate attention (see also, Polich, 2007). Similarly, Kok (2001) proposed that the P3 reflects the ‘attentional capacity invested in the categorization of task relevant events’. In sum, these studies suggest a possible link between P3 amplitude and allocation of attentional resources that subserve working memory functioning. Thus, in the current study, we interpret P3 activation as reflecting the direction of attentional resources towards encoding information into WM, such as categorizing events or updating mental representations. However, attention acts at many levels of processing during working memory, including early perceptual processes (Rutman et al., 2010). For instance, ERP components such as the P1 are believed to represent sensory responses elicited by visual stimuli as early as 100 ms (Luck, 2005). Thus, we also measure the P1 component to ascertain whether perceptual/sensory processes are intact or altered in college students with ADHD.

A recent meta-analysis of six studies investigating the P3 component in adults with ADHD found significantly reduced P3 amplitude compared to controls (Szuromi et al., 2011). However, all of the studies used a GoNoGo paradigm: none used a WM task. Moreover, a recently published EEG study of WM functioning in adult ADHD utilized an n-back task and mainly investigated time-frequency measures (Missonnier et al., 2013). Among other findings in that study, Missonier and colleagues (2013) reported reduced low frequency oscillations (e.g., theta) in the ADH group during the period in which the P3 would have occurred (e.g., between 200 and 500 ms), suggesting that both time- and frequency domain measures reflect similar underlying neural processes.

Only one study to date has investigated the P3 component in the context of a ‘delayed match-to-sample’ WM paradigm and this involved a comparison of participants with and without Schizophrenia (Haenschel et al., 2007). In this paradigm, the to-be-remembered target stimuli were presented in a sequence that varied in length (e.g., 2 versus 3 stimuli) to measure effects of WM load. Longer sequences place greater demands on WM in terms of storage and updating information compared to short sequences. Then, after a delay period, the participant is asked whether a test-stimulus matched one of the targets. The task uses irregular shapes as stimuli to avoid verbal/semantic processing. Significantly reduced mean P1 (an early visual component) and P3 amplitudes were found in individuals with Schizophrenia compared to controls. The load effect was present only for the P1 component. The authors interpreted the reduced P1 activity as an early visual processing deficit whereas the decreased P3 amplitudes were interpreted as evidence of reduced ability in categorizing or evaluating stimuli.

To investigate WM, the present study adapted Haenschel’s WM paradigm (Haenschel et al., 2007) and used it with a sample of college students with ADHD. This paradigm allowed us to investigate the neural effects of encoding stimuli presented in sequence under different load conditions (low versus high load). Specifically, the current task required participants to allocate attention to the stimulus, register it, and then quickly shift attention to the next stimulus while keeping the previous one in mind. This process requires both earlier attention allocation to sensory/perception of the stimulus and later allocation of attention to evaluate the stimulus and update the mental representation. To the best of our knowledge, no previous study has investigated the encoding phase of WM using a delayed-match-to-sample task in participants with ADHD. A complete understanding of ADHD necessitates the study of different subpopulations of this clinical condition, which represent different levels of functioning. Our sample is unique in that it consists of college students with ADHD who are relatively high functioning and successful academically, but who continue to show impairment (DuPaul et al., 2009). A better understanding of mechanisms underlying their difficulties may inform interventions designed to increase academic achievement and educational attainment in this population.

To determine whether participants with ADHD are impaired during WM encoding, we measured P1 and P3 amplitudes, which allowed us to ascertain at which stage (early vs. later visual processing) any impairment occurred. Furthermore, to test whether any impairment might vary according to the level of WM demand, we utilized two load conditions (low, high) in the delayed match-to-sample WM task. We predicted that individuals with ADHD would show impaired WM performance and reduced P3 amplitude and that these group differences would be most pronounced under the high-load WM condition.

2. Method

2.1. Participants

A total of 32 unmedicated individuals with ADHD (47% male; aged 19–35) and 25 control participants (44% male; aged 19–32) participated in the study. Participants with ADHD were recruited from University Student Disability Services in a major urban area, via email lists and flyers. Inclusion criteria were; (1) current enrolment in a post-secondary program, (2) a previous diagnosis of ADHD, (3) registration with respective university or college Student Disability Services, which requires documented evidence of a previously confirmed diagnosis of ADHD (typically, but not invariably in elementary school), and (4) aged 19–35. Exclusion criteria were; (1) uncorrected sensory impairment, (2) major neurological dysfunction and psychosis, and (3) current use of sedating or mood altering medication including stimulant medication for ADHD. Amongst the clinical sample, 4 reported comorbid learning disability. Participants in the comparison group were recruited through campus advertisements and they were required to have no history or current presentation of mental health disorders.

All participants completed the Adult ADHD Self-Report Scale (ASRS v1.1), the Symptom Assessment–45 (SA–45; Maruish, 1999) and the Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982; Wallace et al., 2002) to quantify current symptoms of ADHD.
additional psychopathology, and associated levels of cognitive impairment, respectively. The ASRS is a reliable and valid scale for evaluating current symptoms of ADHD in adults (Adler et al., 2006). The ASRS v1.1 consists of eighteen questions based on the criteria used for diagnosing ADHD in the DSM-IV-TR. The SA-45 is a brief assessment of psychiatric symptoms. It is based on the well-validated longer version (SCL-90-R) to create a brief, yet thorough, measure of psychiatric symptoms. The CFQ measures self-reported failures in perception, memory, and motor function in everyday life. This 25-item questionnaire uses a 5-point Likert scale and has a good external validity (Broadbent et al., 1982; Wallace et al., 2002). Questions require participants to rank how often these failures in cognition occur. Both ASRS and CFQ instruments yield a total score whereby a higher score represents more ADHD symptoms and more cognitive failures, respectively (Table 1).

The study was approved by the Research Ethics Boards of the participating universities. All participants provided informed written consent before starting the study.

2.2. Tasks

2.2.1. Neuropsychological tasks measuring WM performance

Three neuropsychological tasks were used to measure WM performance. The Digit Span subtest from the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV) was used to assess verbal working memory ability (Wechsler, 2008). The Spatial Span (SSP) and Spatial Working Memory (SWM) tasks from the Cambridge Neuropsychological Testing Automated Battery (CANTAB) were used to assess visual spatial WM. The SSP required participants to remember the spatial sequence of squares flashed briefly one at a time on the screen. The scores represent the highest level at which the participant reproduces at least one correct sequence. The SWM task requires participants to find a blue token in a series of displayed boxes and use these to fill up an empty column, taking care not to return to boxes where a blue token had been previously found. The standardized z-score for total number of errors for the SWM was used.

2.2.2. The ERP delayed match-to-sample task

The delayed match-to-sample task was adapted from Haenschel et al. (2007). Thirty-six different abstract figures were used as stimuli. The task consisted of 2 WM load conditions (144 trials total): a high load condition in which a sequence of 3 stimuli was presented (72 trials) and a low load condition with a sequence of 2 stimuli (72 trials). The low load (2-stimulus) condition is shown in Fig. 1. Our task differed from that of Haenschel et al. (2007) in that we made the task self-paced to better optimize participants’ readiness on computer-paced tasks which are known to be difficult in the ADHD population (e.g., interference effects of ‘set shifting’ between trials) and thereby obtain a purer measure of working memory. Furthermore, we were also hoping to reduce the loss of trials due to artifacts. Participants initiated each trial by pressing the space bar on a keyboard, which served to optimize their readiness for the start of each trial. Trials were presented in blocks of 12 trials each of either a low or high load. The blocks of high (3 Stimuli) and low (2 Stimuli) loads were randomly distributed. Before each block, participants were shown which condition to expect (e.g., low or high load) and after each block, feedback on accuracy was provided (as the percentage of trials that were correct). After the space bar was pressed, a clear time of 600 ms was introduced before the fixation stimulus was presented for 400 ms. Each target stimulus appeared in the center of the screen for 600 ms (visual angle, 1.34) with an inter-stimulus interval of 400 ms. Two seconds after the last target stimulus appeared (delay; maintenance), a probe stimulus was shown for two seconds (target; retrieval; see Fig. 1). Participants were asked to indicate whether the target stimulus was one of the previously presented stimuli by pressing the keys labelled as ‘Same’ or ‘Different’ on the keypad using their dominant hand. Response accuracy was emphasized, but to maintain a reasonable pace of processing, participants were required to respond within a 2 second time frame, after which the trial would be terminated and scored as incorrect. However, response time was recorded. E-prime 1.2 software (Psychology Software Tools, Inc.) was used to control stimulus presentation and timing as well as to record the accuracy and latency of responses.

Trial time varied between 4 and 6 seconds for low load and 5–7 s for the high load. Since between-trial inter-stimulus intervals would vary because of the self-pacing (participant pressed space bar to start the next trial), total time for task completion was calculated and compared between groups. The analysis indicated that there were no significant differences between the two groups in terms of total time to complete the task (see Table 2).

2.3. Neural data acquisition, processing

2.3.1. ERP data

The EEG was recorded with a 128-channel Geodesic Hydrocel Sensor Net at a 500 Hz sampling rate, using Netstation stand-alone software (Electrical Geodesics Inc, Eugene, Oregon). Netstation was used to filter (FIR, 0.5–30 Hz, 60 Hz notch) and segment the data (400 ms before stimulus onset and 2000 ms for low load, or 3000 ms for high load, after stimulus onset). Segments containing artifacts were removed using automatic algorithms to detect eye blinks and eye movements, as well as large drifts or spikes in the data. Eye blinks were detected when the vertical eye channels exceeded a threshold of 150 µV (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 µV (max–min) over a 200 ms time window. Channels were automatically marked bad when they exceeded a transition threshold of 200 µV over the entire segment (max–min). Segments containing more than 20% bad channels were automatically removed. In addition, all epochs were visually inspected by trained research assistants blind to the hypotheses. Bad channels were replaced by values interpolated from neighbouring channel data using spherical splines. Results were verified manually by a research assistant, who was unaware of the study objectives or hypotheses. Because we used a relatively

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**Table 1**

Mean and standard deviations of the ASRS (ADHD Self Report Scale), CFQ (Cognitive Failures Questionnaire), and SA-45 subscales (Symptom Assessment-45) for the ADHD and the comparison group.

<table>
<thead>
<tr>
<th></th>
<th>ADHD</th>
<th>Comparison</th>
<th>P-values</th>
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<tbody>
<tr>
<td>ASRS***</td>
<td>44.97</td>
<td>18.71</td>
<td>.00</td>
</tr>
<tr>
<td>CFQ***</td>
<td>53.88</td>
<td>27.56</td>
<td>.00</td>
</tr>
<tr>
<td>SA-45</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Anxiety</td>
<td>8.41</td>
<td>6.75</td>
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</tr>
<tr>
<td>Depression</td>
<td>9.37</td>
<td>8.05</td>
<td>.21</td>
</tr>
<tr>
<td>Obsessive compulsive***</td>
<td>14.72</td>
<td>9.25</td>
<td>.00</td>
</tr>
<tr>
<td>Somatization</td>
<td>7.65</td>
<td>6.55</td>
<td>.11</td>
</tr>
<tr>
<td>Phobic anxiety</td>
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<td>5.29</td>
<td>.30</td>
</tr>
<tr>
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<td>6.48</td>
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<td>.28</td>
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<tr>
<td>Interpersonal sensitivity</td>
<td>8.67</td>
<td>7.76</td>
<td>.38</td>
</tr>
<tr>
<td>Paranoia ideation</td>
<td>7.93</td>
<td>6.62</td>
<td>.07</td>
</tr>
<tr>
<td>Psychoticity</td>
<td>5.54</td>
<td>5.62</td>
<td>.80</td>
</tr>
</tbody>
</table>

*p-values shown reflect one-way ANOVA’s.

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* p < .05.

** * p < .01.

*** * p < .001.
low high-pass filter, a linear detrending algorithm was applied to individual epochs to eliminate any left-over linear drift if it existed. The data were then averaged for each subject and stimulus, average referenced, and coded within the segment. The P1 components were scored as the maximum positivity between 80 and 160 ms after stimulus-onset for each stimulus (e.g., 3 stimuli for high load) for electrode sites 75 (Oz), 70 (O1) and 83 (O2). Similarly, P3 components were scored as the mean positivity between 250 and 500 ms after stimulus onset for electrode sites 65, 66, 84, and 90 (See Supplementary Fig. S1 for electrode map) compared to the baseline. The mean activity was chosen to represent P3 because this is a more elongated component with peaks (often several) that are less clearly defined compared to ‘perceptual’ components (see also, Luck, 2005; Woltering et al., 2011). Electrodes were selected based on the literature (e.g., see Polich, 2007, for rationale) as well as the maximal positivities in the grand average waveforms (relative to the 200 ms baseline). Latency ranges were determined based on inspection of the individual ERPs as well as Haenschel et al., 2007 study. We acknowledge that, considering the paucity of ERP studies using delayed-match-to-sample tasks, our component labeling may vary from convention used in other tasks. Before exportation, data were baseline corrected for 200 ms prior to stimulus onset, separately, for each individual stimulus. Only correct trials were used in the analysis. Averages across electrode sites were used for the P1 and P3 analysis.

2.4. Data analysis

Independent one-way ANOVAs were used to test for group differences in performance on the neuropsychological tasks. Separate repeated measures ANOVAs were conducted to test for group differences in accuracy and reaction time on the delayed match-to-sample task, with Load as the repeated measure (High load vs. Low) and Group as a between participant factor (ADHD vs. Control). By contrast, the incomplete factorial design of the Delayed-Match-To-Sample task (2 stimuli in Low Load; 3 stimuli in High Load) precluded a Group (2) x Load (2) repeated measures ANOVA to investigate effects on the P1 and P3 ERP components. Thus we used a multi-step approach to disentangle the effects of Load, Stimulus, and Group on our ERP components (P1, P3). In Step-1, to investigate the effects of Group and WM Load on the P3 and P1, we collapsed across Stimulus, and ran a Group by Load Repeated Measures ANOVA for the first and
last Stimuli separately. The last stimuli of each load would represent the situation in which a participant would hold the maximum amount of information in mind for each condition when encoding new information. This analysis involved a comparison of the second (last) stimulus of the low load with the third (last) stimulus of the high load: the second stimulus of the high load was not compared to either the first or the second stimulus of the low load as it was not conceptually equivalent to those stimuli. In Step-3, we ran separate Group by Stimulus repeated measures ANOVAs for each load. For the high load, contrasts were run to test for differences between the three stimuli. This step allowed us to investigate sequence effects of stimulus presentation, which may provide insights in the effects of neural resources being allocated during encoding with increasing amount of stimuli to be held in mind.

Two participants, whose ERP data yielded less than 10 trials, were excluded from this analysis. Data points (behavioural and ERP) with SD’s >3 were regarded as outliers and adjusted using the winsorizing technique (Tabachnick and Fidell, 2001). Three data points in ADHD group and 2 data points in control group were winsorized. No significant differences were found in trial-counts [Low load: $F(1, 49) = .258, p = .553$, High load: $F(1, 49) = .006, p = .938$] between the groups.

Partial eta-squared values ($\eta^2$) were computed to ascertain effect size (ES). According to Vacha-Haase and Thompson (2004), ES based on $\eta^2 = .10$ corresponds to a small effect, $\eta^2 = .25$ corresponds to a medium effect, and $\eta^2 = .50$ represents a large effect.

### 3. Results

#### 3.1. Behavioural data

Analysis of demographic and clinical variables confirmed that the ADHD and control groups were comparable in terms of age [$F(53) = 1.358, p = .25$] and sex [$\chi^2(1, 21) = .039, p = 1.00$]. As expected, ADHD participants reported significantly more ADHD symptoms [$F(54) = 1, p < .001$] and cognitive failures in everyday life than controls [$F(42) = 59.91, p < .001$]. The ADHD group also reported more obsessive-compulsive symptoms [$F(43) = 25.22, p < .001$] on the SA-45, compared to the Control group. Means and standard deviations are presented in Table 1.

Analysis of neuropsychological tests of WM performance revealed, as expected, that the ADHD group obtained significantly lower scores on the CANTAB-SWM [$F(1, 44) = 4.54, p = .039$], compared to the control group. ADHD group also showed poorer scores in WAIS Digit Span, albeit a non-significant difference [$F(1, 48) = 3.29, p = .076$]. Groups did not differ on CANTAB-SSP. The mean scores for the ADHD group on these two tests of WM fell in the low average range: their mean SWM z-score was .51, which corresponds to a scaled score of just below 7 (16th percentile); and their mean Digit Span scaled score was 8.31 (25th percentile). By contrast, scores for the comparison group fell well within the average range: mean Digit Span scaled score of 9.95 corresponds to the 50th percentile and the SWM z-score was .51, which corresponds to a scaled score of 10 or the 50th percentile. As predicted, analysis of the Delayed-Match-To-Sample Task, revealed a main effect of Load on performance accuracy [$F(1, 53) = 49.88, p = .000$, ES = .49]. Specifically, all participants were less accurate during the high WM load compared to the low WM load condition, indicating the success of the experimental manipulation. The groups did not differ significantly in terms of either their accuracy or response times (to the target stimulus). There was no significant Group-by-Load interaction for either response accuracy or response times. Moreover, there were no group differences in terms of intra-individual variability of response times and neither the main effect of Load nor the Group-by-Load interaction was significant. Means and standard deviations of the neuropsychological tasks and the delayed match to sample task measures are shown in Table 2.

#### 3.2. ERP data

The waveforms for each stimulus are presented in Fig. 2, in Panel A for the low load and Panel B for the high load. Table 3 shows the means and standard deviations for the ERP components for each load and for each group collapsed across stimulus. The waveforms and (difference) topoplots for the P3, collapsed across stimulus, are presented in Fig. 3, Panel A for the low load and Panel B for the high load. As can be seen in the topoplots, the difference in P3 amplitude between the ADHD and control group was observed primarily in the parietal/occipital regions (see Supplementary Fig. S2 for a t-plot).

##### 3.2.1. P1 analyses

For P1 amplitude, Step-1 analysis, which examined the effects of Group and Load collapsed across stimuli, showed a significant main effect of Load [$F(1, 53) = 8.26, p = .006, ES = .14$] with higher amplitude for high load. There was no main effect of Group or Group-by-Load interaction. Step-2 analyzed Group and Load effects separately for the first and last stimuli. For the first stimulus there was a significant Group-by-Load interaction [$F(1, 53) = 6.27, p = .015, ES = .106$] in the absence of main effects for either Group or Load. Post-hoc tests revealed that participants with ADHD had increased P1 amplitudes for the High compared to the Low load, whereas the comparison group did not show this effect ($p < .05$). For the last stimulus, a main effect of Load was present [$F(1, 52) = 5.76, p = .02$, ES = .10] but neither the main effect of Group nor the Group-by-Load interaction was significant. Step-3 analysis of P1 during the low load condition revealed a main effect of Stimulus, [$F(1, 53) = 9.31, p = .004, ES = .15$] with lower P1 amplitudes for the first stimulus compared to the last. No Group, or Group-by-Stimulus interaction effect was found. The high load also revealed a main effect of Stimulus, [$F(1, 51) = 13.61, p = .000, ES = .35$], with lower P1 amplitudes for the first Stimulus compared to the second and third. No Group or Group-by-Stimulus interaction was found. Last, the P1 did not correlate with any clinical questionnaire or behavioural measures.

For P1 latency, Step-1 analysis revealed no main effect of Load, Group, or Load-by-Group interaction for either the high or low load conditions. On the Step-2 analysis, no main effects of Group, Load, or a Stimulus-by-Group interaction were found, nor were there any significant effects for the first stimulus. On the Step-3 analysis, neither the main effects of Stimulus or Group, nor the Stimulus-by-Group interaction was significant.

##### 3.2.2. P3 Analyses

For P3 amplitude, our Step-1 analysis showed a significant main effect of Group, [$F(1, 53) = 6.30, p = .015, ES = .11$], with the ADHD group showing smaller P3 amplitudes than the control group. There was also a main effect of Load, [$F(1, 53) = 4.04, p = .049$, ES = .07], with larger P3 amplitudes for the high load confirming that our P3 index was sensitive to effects of load. No Group-by-Load interaction was found.

The Step-2 analysis tested the Group by Load effects separately for the first and last Stimuli. There was a main effect of Group for the first, [$F(1, 53) = 5.31, p = .025, ES = .09$], and last [$F(1, 52) = 5.76, p = .020, ES = .10$], stimuli suggesting the group difference was present across stimulus. There was a main effect of Load for the first [$F(1, 53) = 6.55, p = .013, ES = .11$] but not for the last stimulus, suggesting our general load effect was driven mostly by the first stimulus. Also, no significant Group-by-Load interaction was found for both first and last stimuli.
Our Step-3 analysis, testing for sequence effects of stimuli, revealed a main effect of Stimulus, $F(1, 53) = 11.76, p = .001, ES = .18$, and a main effect of Group, $F(1, 53) = 4.89, p = .031, ES = .08$, during the low load. No Group-by-Stimulus interaction was found. The high load also revealed a main effect of Stimulus, $F(1, 53) = 3.17, p = .05, ES = .11$, and a main effect of Group, $F(1, 52) = 7.63, p = .008, ES = .13$, with no Group-by-Stimulus interaction. In general, for both WM loads, responses to the first stimulus showed lower amplitudes than to the other stimuli ($p's < .05$). For the high load, pairwise comparisons showed that the second and third stimulus did not differ. The ADHD group showed lower P3 amplitudes for all three stimuli compared to the control group.

For the P3 latency, the Step-1 latency analysis indicated no main effect of Group, a marginally significant effect for Load, $F(1, 53) = 3.23, p = .078, ES = .058$, with shorter latencies for the high load, and no Load-by-Group interaction. Step-2 analysis revealed a main effect of Load for the first $F(1, 53) = 8.16, p = .006, ES = .133$, but not for the last stimulus. No main effect of Group or Group-by-Load interaction was found for either the first or last stimulus. The Step-3 analysis showed a main effect of Stimulus for the low load condition $F(1, 53) = 5.47, p = .023, ES = .093$. Post-hoc tests revealed that the ADHD group showed increased P3 amplitude for the second stimulus compared to the first, whereas the comparison group did not show this difference ($p = .03$). No main effect of Group or Group-by-Stimulus interaction was found for both first and last stimuli.

4. Discussion

The present study is the first to investigate neural changes in P3 amplitudes and latencies during the encoding phase of WM in ADHD, using a delayed match-to-sample task. The main finding was a reduced P3 amplitude for the ADHD group, regardless of WM load or stimulus order (i.e., first or last), compared to the control group. Behavioural results showed that all participants were less accurate during the high WM load condition compared to the low load condition, which attests to the success of our experimental manipulation of WM load.

Changes in P3 amplitudes during WM tasks have been associated with differences in the allocation of attention necessary for WM functioning (Donchin and Coles, 1988; Polich, 2007; Kok, 2001). Our findings of lower P3 amplitude in the ADHD group compared to the control group, independent of load or stimulus order, suggests a more general problem of attentional allocation in the encoding phase of WM rather than a more specific problem.
associated with an increasing demand on WM. The reduced P3 amplitude in college students with ADHD found in this study using a WM task is consistent with and extend findings from a recent meta-analysis on P3 in adults with ADHD (to WM encoding during a WM task), which revealed a decreased P3 amplitude recorded during Go–Nogo tasks in ADHD (Szuromi et al., 2011; and also see, Woltering et al., 2013, using a similar sample to that in the present study). Go–Nogo tasks, which traditionally are used to index response inhibition, do involve WM, albeit low-level demands, and some require constant updating of working memory.

We believe that the decreased P3 amplitude observed in individuals with ADHD can be interpreted as deficient attentional allocation, which leads to inefficient encoding of information in memory. Theoretically, the notion of P3 reflecting attentional allocation is not that different to Kok’s notion of capacity since recent insights in the field of working memory have linked attentional processes and working memory capacity closely together (Awh and Vogel, 2008). A functional relationship between P3 amplitude and attentional resource allocation has also been reported in the literature (Ford et al., 1994; Kok, 2001). Also, many studies found the P3 to be related with brain regions thought to be involved in attention, such as the parietal lobe, the temporoparietal junction, lateral prefrontal areas and the cingulate gyrus (Linden, 2005; Verleger et al., 1994). Similarly, a study that explored the cortical basis of ERP components revealed an impaired ventral attentional pathway in ADHD participants during the time of the P3 (Helenius et al., 2011). Furthermore, in their meta-analysis, Szuromi et al. (2011) interpreted the overall reduction of P3 amplitude in adults with ADHD as a dysfunction of the ventral attention network since the P3 related to target detection is thought to be generated from the temporoparietal junction. In sum, previous research suggests a possible link between P3 amplitude and attentional resource allocation. Hence, based on previous findings, we speculate that weak activation in attention pathways might play a role in the defective allocation of attentional resources during activities involving WM, accounting for the observed decreased P3 amplitudes in college students with ADHD.

Our analyses of P1 amplitude revealed no significant group effects except for one: namely that the ADHD group seems to manifest an increase in P1 amplitude from the low to the high WM load condition in response to the first stimulus only. Compared to Haenschel et al. (2007), who did find robust P1 group differences in Schizophrenia patients, our findings suggest that differences in WM encoding between the ADHD and non-ADHD peer group were due primarily to differences in attentional allocation during the WM encoding phase as opposed to early visual processing. Only one other study to date has investigated neural differences in WM between ADHD adults and their peers using EEG (Missonnier et al., 2013). Despite the use of different methodologies (n-back versus delayed-match-to-sample task, respectively; smaller sample size, and the focus on different neurophysiological measures, such as time-frequency domain), conclusions are similar. That is, both conclude that individuals with ADHD manifest differences in neural correlates linked to the attentional network involved in the allocation of attention subserving WM.

Behavioural differences between groups were found during the WM task: namely the ADHD group had slower response times to the retrieval cue, albeit marginally significant, but did not differ in response accuracy. It is possible that the slower response times in the ADHD group indicate their need for more time to make accurate decisions compared to their peers. In this sense, the longer response times may be another manifestation of slow processing speed or a compensatory mechanism. Although accuracy in our ADHD group was comparable to that of their typically developing peers, cognitive mechanisms of processing, such as the allocation of attention, could still function less optimally. It is possible that this deficit may show in contexts other than within the experimental confines of the current task. This suggestion is strengthened by other findings from this study: namely that for the ADHD group, their mean scores for WAIS Digit Span and CANTAB SWM were in low average range, whereas those of the control group were in the average range; the T-scores for the ADHD group (mean = 41.5) were in the mildly impaired range whereas those for the control group (mean = 50) were within the well-functioning range. Note that the two neuropsychological tasks not only require the maintenance but also the updating of the spatial representations, which renders their WM demands similar to those of the delayed-match-to-sample task. Furthermore, these behavioural and neuropsychological differences were found despite the fact that the participants with ADHD in this study are high functioning (mostly college students) and the task was self-paced to exclude potential effects of poor sustained attention confounding the WM result.

In light of our neural results, the lack of differences in behavioural accuracy in the delayed-match-to-sample task could suggest...
that our group differences found in neural processing indicate inefficient means of attentional allocation; a detriment that may not always be observed in behavioural accuracy. The P3 could be a neural indicator of this inefficient mechanism.

From a clinical perspective, the WM impairment (albeit modest) in these college students with ADHD compared to their college-peers may well help explain why this relatively successful subgroup continues to manifest functional impairments in academic, social functioning during college years. Furthermore, these results support findings from a recent meta-analysis suggesting long-term memory difficulties in adult ADHD may be attributable to difficulties in the encoding phase (Skodzik et al., 2013). Our findings highlight the importance of studying WM encoding and in this newly emergent subgroup of young adults with ADHD.

This study has several limitations, which should be taken into consideration when interpreting the findings. First of all, we did not have a direct measure of IQ, or scholastic ability, which limits the characterization of our sample. However, these college students must be of at least average intelligence (as suggested by the WAIS Digit Span) to be able to successfully complete high school and gain college entrance. Furthermore, we did not measure alcohol and recreational drug use in this sample of college students, which could influence allocation of attention or WM or both. Also, the current analyses were restricted to the encoding stage and further research is warranted to characterize the maintenance and retrieval stages of WM.

In conclusion, keeping in mind the aforementioned limitations, our results suggest differences in neural processing in WM with adult ADHD. Our results may suggest, based on what is known about changes in P3 amplitudes in the ERP literature, that weak activation in attentional pathways might play a role in defective attention resource allocation to updating representations in working memory. Moreover, our results concerning the P1 versus P3 suggest a temporal as well as syndrome specific neural index of adult ADHD. These findings could have important implications for understanding the cognitive difficulties ADHD adults face, as well as intervention.

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Appendix A. Supplementary data


References


Linden DEJ. The P300: where in the brain is it produced and what does it tell us? Neuroscientist 2005;11:363–79.

Luck SJ. An introduction to the event-related potential technique (cognitive neuroscience), Cambridge, MA: MIT Press; 2005.


