

Limited Visual Working Memory Capacity in Children with Dyslexia: An ERP Study

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Abstract

Some researchers suggest that deficits in attention and working memory influence the development of dyslexia, whereas others propose that these deficits are more likely due to reduced global processing speed. The current study aimed to investigate behavioral performance in children with dyslexia compared to typically developing controls on two tasks: a visual oddball task for attention and an *n*-back task for working memory. We measured P300 event-related potentials (ERP) amplitude and latency for both tasks. Our results demonstrated reduced behavioral accuracy and P300 amplitude for the children with dyslexia compared to their typically developing peers in both the *n*-back and visual oddball tasks. We also found no differences in response time or P300 latency between these groups on either task. These findings support the idea that children with dyslexia experience deficits in cognitive processes related to working memory and attention, but do not exhibit decreased global processing speed on these tasks.

Keywords: working memory; *n*-back; oddball; dyslexia; P300 event-related potentials (ERP)

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Introduction

The ability to process and integrate text in reading is a critical skill that enables one to be successful in an academic setting (Savolainen et al., 2008; Taraban, Kerr, et al., 2004; Taraban, Rynearson, et al., 2000). Deficits in these reading abilities contribute to several negative outcomes, including poor academic achievement (Bergey et al., 2017; Chevalier et al., 2017; Snow & Strucker, 1999) and interference in career performances (Adelman & Vogel, 1990; Morris & Turnbull, 2007). Given the increased prevalence of reading disabilities in the population (Lewis, 1999; Newman et al., 2011), it is critical to further understand the neurocognitive factors related to reading difficulties.

Dyslexia, defined as a learning disorder associated with reading difficulties (Snowling & Hulme, 2012; Vellutino et al., 2004), has received considerable attention. Despite displaying adequate levels of intelligence (Tanaka et al., 2011), individuals with dyslexia experience difficulties in decoding text during reading (Snowling & Melby-Lervåg, 2016) and processing speech (Ziegler & Goswami, 2005). While it is believed that cognitive deficits likely influence the development of dyslexia (Singleton, 2002), the specific nature of which cognitive factors and how they predict impairments associated with dyslexia is still widely debated. For example, while the role of phonology-related deficits (e.g., dysfunctional verbal and auditory processes) are well documented in individuals with dyslexia (Chan, 2018; Richardson et al., 2004; Szenkovits et al., 2016), others have highlighted the role of

maladaptive nonverbal cognitive processes, such as decreased speed of attentional engagement and disengagement (Facoetti et al., 2008), a lower visual attention span (Bosse et al., 2007; Heiervang & Hugdahl, 2003), and impaired visual working memory (VWM) processes (Albano et al., 2016; Gathercole et al., 2006; Menghini et al., 2011; Pennington, 2008; Smith-Spark & Fisk, 2007; Swanson, 1994). This suggests the importance of further investigations on attention and working memory processes to better understand the nature of cognitive deficits associated with dyslexia.

Attention allows one to prioritize information that may be necessary for the completion of task-relevant goals by allocating cognitive resources necessary for greater processing of specific stimuli (Corbetta & Shulman, 2002). Enhanced processing of these stimuli leads to the subsequent transfer of this information into working memory (Awh et al., 2006). Working memory is a limited-capacity system that supports the temporary storage and manipulation of information (Cowan, 2010, 2017). The multifaceted nature of this system requires information held in working memory to be updated, revised, and replaced with newer and more relevant information (Monsell, 1996). Efficient working memory functioning is necessary for the completion of complex cognitive tasks, such as learning and reading processing. As such, individuals diagnosed with a variety of reading disorders, such as dyslexia, commonly demonstrate deficits in working memory (Brandenburg et al., 2015; Pickering, 2012; Schuchardt et al., 2008; Swanson & Alloway, 2012; Swanson et al., 2009; Vellutino et al., 2004; Wagner & Muse, 2006). Therefore, deficits in attentional and working memory related processes may contribute to the deficits observed in dyslexia.

Visual components of working memory are believed to contribute to reading processes (Magliano et al., 2016). In addition, reading and VWM processes require the engagement of overlapping brain structures responsible for multifaceted visual sensory processes, including selective attention (Mayer et al., 2007; Awh & Jonides, 2001), that enable efficient reading performance (Baddeley et al., 2019; Gathercole et al., 2006). Other evidence for the association between VWM and reading abilities can be seen by work demonstrating that reading skill training yielded increased VWM capacity (Shiran & Breznitz, 2011). Ultimately, these studies provide evidence suggesting that reduced VWM abilities may impact reading abilities, and thus contribute to the development of dyslexia.

Deficits in reading abilities in individuals with dyslexia may be maintained by a decreased storage of VWM resources necessary to complete current goal-directed tasks (Coady & Evans, 2008; Hoffman & Gillam, 2004). This reduction in VWM capacity may be caused by inefficient attentional allocation towards task-relevant information (Daucourt et al., 2019). For example, prior work has indicated that deficits in visual attention may play a role in the development of dyslexia (Saksida et al., 2016; Vidyasagar & Pammer, 2010). Thus, it is possible these individuals may experience attentional deficits, resulting in decreased storage of task-relevant information in VWM, negatively impacting overall reading performance.

Alternatively, there is a viewpoint suggesting that decreased processing speed leads to deficits in attention and working memory (Kail & Salthouse, 1994), which may result in reading disabilities. As such, it can be argued that individuals with dyslexia demonstrate cognitive inefficiency relative to their typically developing counterparts. Several other studies have reported slower reaction time for children with dyslexia compared to their peers in terms of different cognitive processing, such as phonological and orthographical processing (Breznitz, 2003, 2006), implicit learning tasks and consolidation (van der Kleij et al., 2019), visual naming (Wolf, & Bowers, 1999), and processing speed of working memory (Shiran & Breznitz, 2011). Therefore, while it is clear that individuals with dyslexia experience impairments in attention and VWM, the nature of how these processes are impacted remains unclear.

The use of electrophysiological markers may provide a useful avenue for unwrapping the association between deficits in attention and working memory processes, and the impact this has on reading disabilities (Luck, 2005). Specifically, the measurement of event-related potentials (ERP), obtained through electroencephalography (EEG) recordings, enables us to address both the speed of processing as well as fundamental underlying neurocognitive characteristics of VWM. One ERP component, the P300, has received considerable attention regarding its relationship with working memory (Brouwer et al., 2012; Christensen et al., 2012; Shiran & Breznitz, 2011) and attentional processes (Polich, 2007; Taroyan et al., 2007). Polich (2007) proposed that P300 amplitude reflects both attentional allocation towards novel or rare stimuli, and context-updating in working memory. Specifically, this positive-going ERP is often observed across various working memory paradigms

involved with the storage and maintenance of information, such as the *n*-back task, and tasks that assess attentional allocation and working memory updating, such as the visual oddball task (VOT).

The current study aimed to investigate the behavioral and neural correlates underlying both attention and VWM processes in children with dyslexia compared to typically developing controls. Specifically, we examined behavioral performance, reflected by accuracy and response times, and P300 amplitude and latency on a VWM task (*n*-back) and a visual attention task (visual oddball task). Enhanced P300 amplitude is believed to reflect the level of working memory load in the *n*-back paradigm, with larger magnitudes indicating a higher load of WM (Brouwer et al., 2012), while greater P300 on the VOT has been associated with increased attentional processing of a novel and infrequent stimulus (Polich, 2007). Prior work has also revealed decreased amplitude and increased latency of the P300 in individuals with dyslexia on an attentional task (Taroyan et al., 2007). We sought to examine which of the two perspectives (i.e., WM deficit in dyslexia vs. the global processing speed deficit in dyslexia) would explain the observed behavioral and ERP data better. The attention/WM deficit account offered the following two hypotheses. First, children with dyslexia compared to typically developing controls would exhibit lower levels of P300 amplitude and accuracy for the *n*-back task due to their lower VWM capacity, as seen in previous work (Evans et al., 2011). Second, children with dyslexia would demonstrate lower P300 amplitude and accuracy for the visual oddball task, due to their deficit in attentional allocation towards novel or rare stimuli, and context-updating in WMM. Alternatively, the global processing deficit account would offer the hypothesis that children with dyslexia would primarily show a slower reaction time and longer P300 latencies on both the visual oddball and *n*-back tasks regardless of accuracy, given their deficits in global speed of information processing.

Methods

Participants

Fifty-five children (38 males) aged 7 to 12 (mean age = 8.73, *SD* = 1.62) years were recruited for this study: 32 (mean age = 8.25, *SD* = 1.61) were evaluated as presenting dyslexia (DYS) and 23

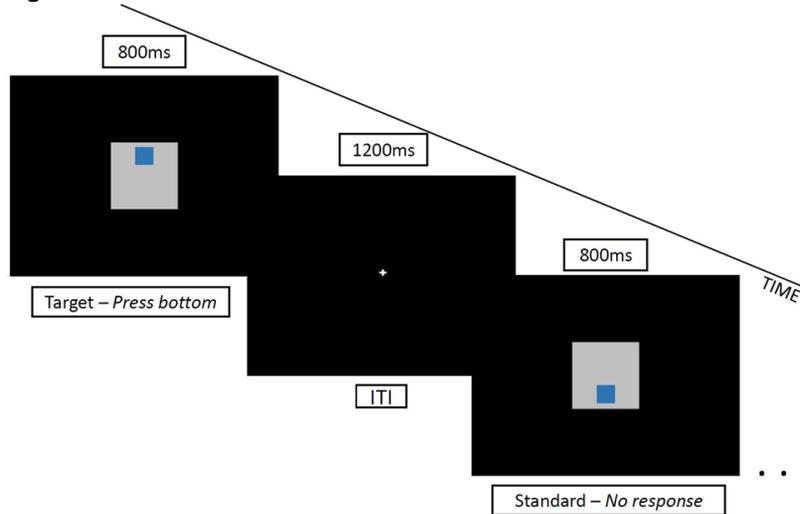
(mean age = 9.21, *SD* = 1.65) were considered as typically developing children (TDC). These children were recruited from a larger group of participants that were involved in ongoing studies in a psychological clinic in Tehran, Iran. Criteria for placement in the DYS group involved scoring two standard deviations below the mean in the Reading and Dyslexia Scale (Kormi-Nouri et al., 2012; Shokoochi-Yekta et al., 2014), currently undergoing dyslexia treatments at schools or the psychology clinic, and receiving a reading problems diagnosis with on-site psychologists at the clinic. Children in the TDC group were free from psychological or learning disabilities based on parent reports, and in-person psychological interviews conducted by on-site psychologists at the psychology clinic. Both groups of children were age-matched, and were within the average range of nonverbal IQ measured by the Raven Colored Progressive Matrices (DYS_M = 96.46, *SD* = 9.47; TDC_M = 101.73, *SD* = 12.83; Raven, 1977). Statistical analysis found no significant group difference in terms of nonverbal IQ (*p* > 0.09). Parents reported that none of the children received medication prior to the experimental session. All children had normal hearing and vision, were right-handed, and were native Persian speakers. Written consents were obtained prior to the study participation from parents and verbally from the children.

Procedure and Materials

Children completed a VOT and visual 1-back task (V1-Back) while EEG recordings were obtained. Children were seated 50 cm away from an LCD monitor used to present both tasks.

The VOT Task. The VOT is commonly used in EEG experiments to study neural responses to novel stimuli and updating of WM, usually reflected by the P300 ERP component (Polich, 2007). In this task, blue square stimuli were presented within a 10 cm by 10 cm gray field located at the center of black background. These squares would present at either the top (rare stimuli) or bottom (frequent stimuli) of the gray field for a short period of 800 ms with a 1200 ms intertrial intervals. The task constituted one block of 200 trials, with 160 frequent trials (80%) and 40 rare trials (20%). Children were instructed to respond as quickly as possible by pressing the spacebar to the frequent stimuli and withholding a response to rare stimuli (Figure 1).

Figure 1. Visual Oddball Task.

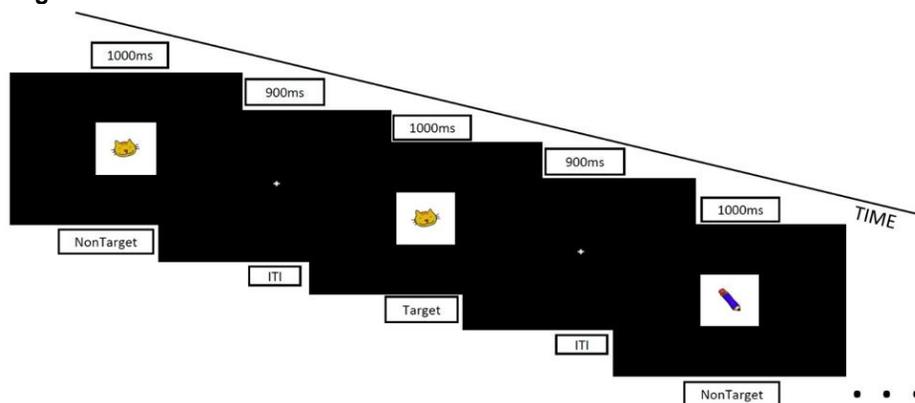


Note. Children were instructed to respond as quickly as possible by pressing the spacebar to the target stimuli and withhold a response to standard stimuli.

The V1-Back Task. The *n*-back task is used to investigate the maintenance and manipulation of information in WM (e.g., Meegan et al., 2004; Ragland et al., 2002). Watter et al. (2001) have shown that P300 amplitude increased as a function of *N*. The V1-Back task of this study consisted of 80 trials, with up to eight different cartoon stimuli (e.g., baseball, book, fish) presented individually (1000

ms) followed by a fixation cross (900 ms). A pseudorandom presentation algorithm was administrated to present the stimuli so that each stimulus had the same probability of being a target in a sequence of two trials across the task. Children were required to press the spacebar if an item shown in a given trial was the same item presented one trial prior (Figure 2).

Figure 2. Visual 1-Back Task.



Note. Children were required to press the spacebar if an object shown in a given trial was the same item presented one trial prior.

Electroencephalography Data Acquisition and Processing

Electroencephalographic (EEG) data were recorded using a Mitsar EEG device (Mitsar Co. LTD, Russia)

with a 21 Ag/AgCl electrode fitted nylon cap with the following sites according to the 10–20 International System of Electrodes (Fp1/Fp2, F3/F4, C3/C4, P3/P4, O1/O2, F7/F8, T3/T4, T5, T6, Fz, Cz, Pz).

The average value of (A1+A2) / 2 left (A1) and right (A2) ear lobe electrodes served as the reference for all channels during online recording. Impedances were kept below 5K Ω , and a 250-Hz sampling rate with a notch filter (50 Hz) was used. Electrooculography (EOG) electrodes were placed 1 cm to the left and right of the external canthi for horizontal eye movements, and an electrode under the right eye referenced to the left earlobe was used for vertical eye movements.

EEG data were cleaned and preprocessed using EEGLAB (Delorme & Makeig, 2004), and ERPLAB (Lopez-Calderon & Luck, 2014), and WinEEG software (Mitsar Co. LTD, Russia). An offline filter with a Butterworth bandpass of 0.1–50 Hz was applied prior to removal of excessive data as well as eye blink artifacts using Independent Component Analysis (ICA) methodology. A time window of –200 to 800 ms from the onset of the stimuli was segmented from the continuous EEG data with a 200 ms baseline correction applied to each bin. As recommended by Luck (2014), the segmented data was subjected to horizontal and vertical EOG artifact rejection procedures (VEOG exceeding $\pm 70 \mu\text{V}$; HEOG exceeding $\pm 40 \mu\text{V}$). Twelve subjects were removed from further analyses due to technical issues (6), and a higher percentage ($> 30\%$) of rejected trials (6). Following artifact rejection, an average of four (2%) trials from the VOT and two (2.5%) trials from the V1-Back task were removed due to excessive movements or eye blinks.

P300

The P300 component has been shown to reflect WM load in the n -back paradigm (e.g., different n -back load; Brouwer et al., 2012; Christensen et al., 2012; Evans et al., 2011; Watter et al., 2001), and greater attentional processing of rare stimuli in oddball tasks (Polich, 2007). As such, we investigated the P300 component in both the VOT and V1-Back. P300 values were quantified as the mean amplitude between 350–500 ms for V1-Back and 450–600 ms for VOT following the onset of stimuli in both tasks (Lotfi et al., 2020). We examined P300 activity from central midline (Cz electrode; Sokhadze et al., 2017) for the VOT task, and parietal midline (Pz electrode; Evans et al., 2011; Polich, 2007) for the V1-Back.

Results

Behavioral Outcomes

SPSS and R software packages were used to conduct statistical analyses. Data cleaning and handling of missing data ($< 5\%$) was done using multivariate imputations by chained equations via

MICE package of R software (van Buuren & Groothuis-Oudshoorn, 2011). In order to account for individual differences (Muthén & Curran, 1997), the data were examined using ANCOVAs with age and nonverbal IQ as covariates, and with Group (DYS or TDC) as a between-subjects factor. We examined percentage of accuracy, error rate, and reaction time (RT) as well as P300 amplitude and latency in both tasks between groups.

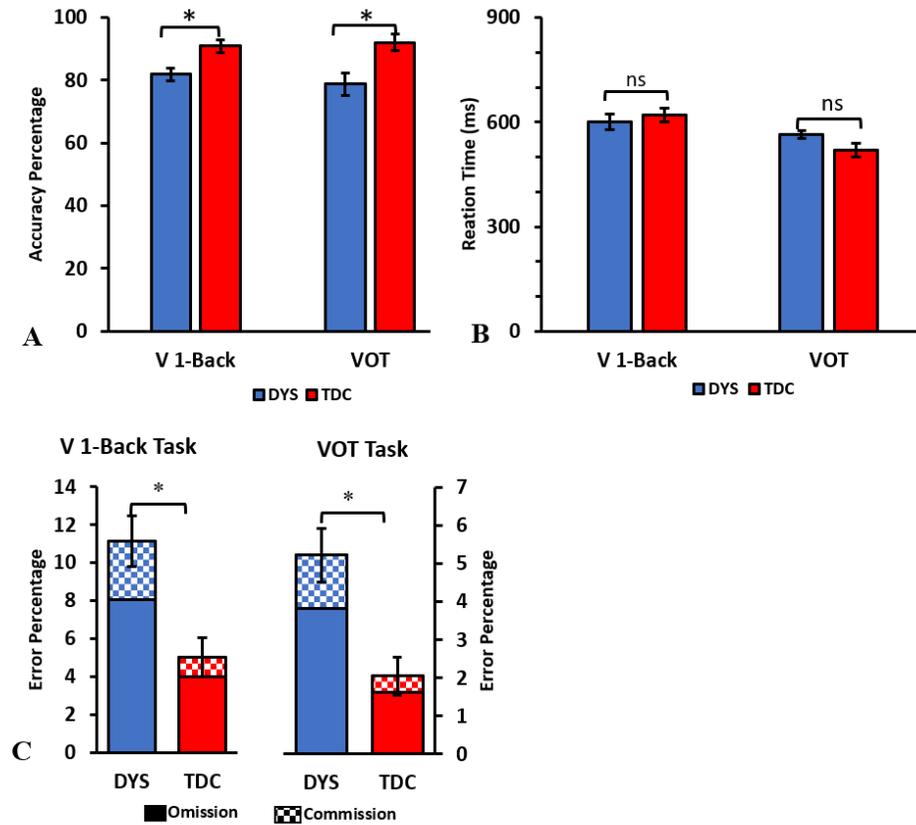
Accuracy in Behavioral Performance. On the V1-Back task, the TDC group showed a significantly higher level of accuracy than the DYS group, $F(1, 51) = 6.29$, $p < 0.05$, $\eta^2 = 0.11$ (Figure 3A). Consistently, a similar ANCOVA yielded a significant group difference with large effect size for the VOT, $F(1, 51) = 6.34$, $p < 0.05$, $\eta^2 = 0.11$ (Figure 3A), indicating that the TDC group demonstrated a significantly higher level of accuracy on VOT compared to the DYS group.

Reaction Time on the Behavioral Tasks. In terms of reaction time for both tasks, there were no significant group differences; V1-Back: $F(1, 51) = 1.34$, $p = 0.25$; VOT: $F(1, 51) = 0.87$, $p = .35$; Figure 3B. Further, ANCOVAs examining commission error rate revealed that the DYS group showed significantly larger commission error rates compared to the TDC group on both tasks; V1-Back: $F(1, 51) = 4.61$, $p < 0.05$, $\eta^2 = 0.08$; VOT: $F(1, 51) = 4.80$, $p < 0.05$, $\eta^2 = 0.09$; Figure 3C. A similar analysis showed that the DYS group showed greater omission errors in the V1-Back, $F(1, 51) = 4.71$, $p < 0.05$, $\eta^2 = 0.08$, but no group differences were observed in the VOT, $F(1, 51) = 2.18$, $p = 1.46$, $\eta^2 = 0.04$.

ERP Analyses

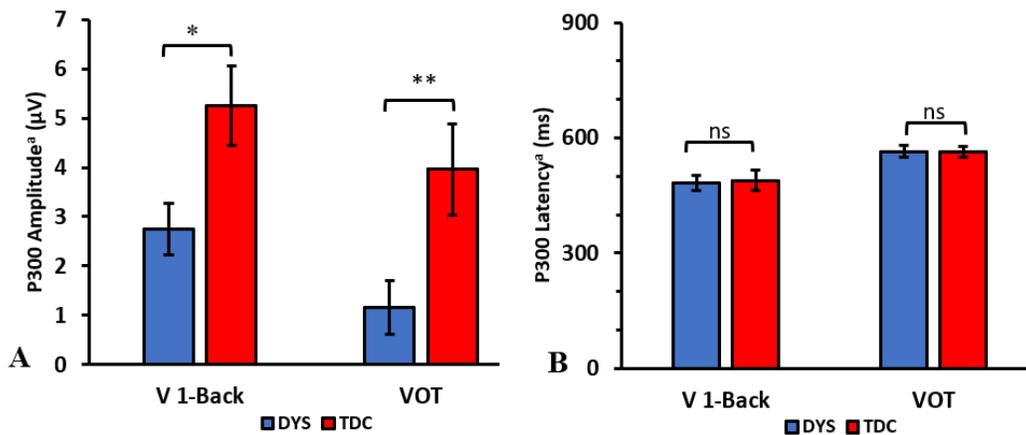
P300 Amplitude. We examined the P300 amplitude at the onset of correct target trials for the V1-Back task at the Pz electrode, and at the onset of infrequent trials for the VOT at the Cz electrode. We conducted two separate ANCOVAs to compare these variables between the DYS and TDC groups. We observed group differences across both tasks, with the TDC group showing a significant larger P300 amplitude on both the V1-Back and VOT tasks, respectively; V1-Back: $F(1, 34) = 5.11$, $p < 0.05$, $\eta^2 = 0.13$; VOT: $F(1, 39) = 5.01$, $p < 0.05$, $\eta^2 = 0.11$; Figures 4A and 5. This indicates that the TDC group had a significantly larger P300 amplitude in processing of target trials on both VOT and V1-Back tasks when compared to the DYS group. Additionally, we examined the correlation between the P3 amplitude and the accuracy of the V1-Back task between the two groups. Results showed a

Figure 3. Accuracy and Error Percentages (A & B) and RT (C) in V1-Back and VOT for DYS (Blue) and TDC (Red).



Note. * $p < 0.05$; DYS = Dyslexic Group; TDC = Typically Developing Children; VOT = Visual Oddball Task; Error bars represent SEM; as = approach significance; Checkered pattern = Omission errors; Solid color = Commission errors.

Figure 4. P3 Amplitude (A) & Latency (B) of Target Trials in V1-Back and VOT for DYS (Blue) and TDC (Red).

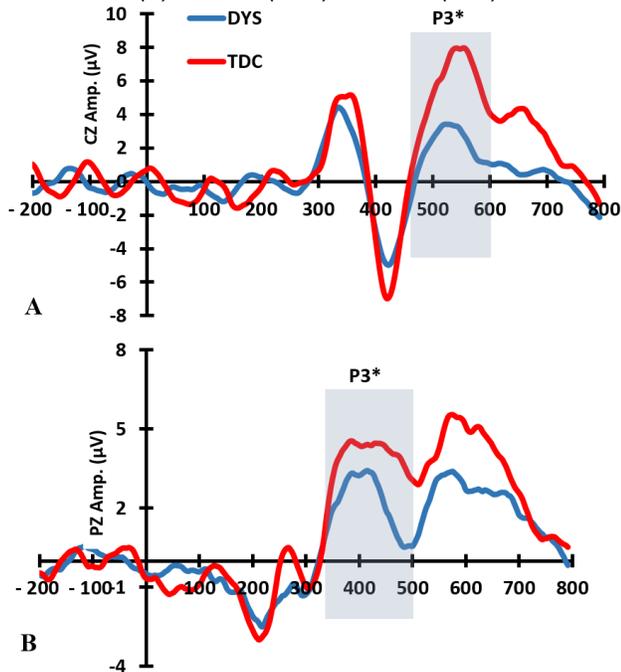


Note. * $p < 0.05$; ** $p < 0.01$; DYS = Dyslexic Group; TDC = Typically Developing Children; VOT = Visual Oddball Task; ^aP3 amplitude and latency are obtained from Pz electrode for V1-back and Cz electrode for VOT. Error bars represent SEM.

significant correlation between them, $r(43) = 0.46$, $p < 0.05$ (see Figure 6), which indicates that the amount of P300 amplitude deflection was positively associated with the rate of correct responses on the V-1Back task, however, we did not observe such a correlation between the behavioral performance and P300 amplitude for the VOT task, $r(42) = 0.128$, $p = 0.49$.

P300 Latency. We applied a similar analysis for P300 latency processing of target trials for both tasks separately. The results demonstrated that there were no group differences in P300 latency across both tasks; V1-Back: $F(1, 34) = 0.23$, $p = 0.63$; VOT: $F(1, 39) = 0.02$, $p = 0.87$; Figure 4B.

Figure 5. Average ERPs of Target Trials of VOT (A) and V1-Back (B) for DYS (Blue) and TDC (Red).

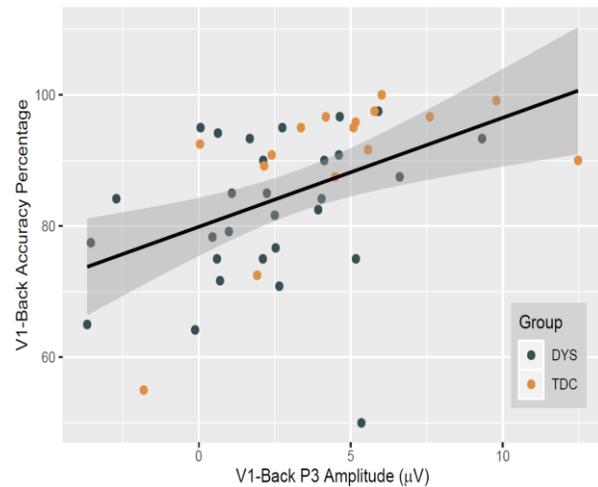


Note. * $p < 0.05$; DYS = dyslexic group; TDC = typically developing children; VOT = visual oddball task; P3 amplitude are obtained from Pz electrode for V1-Back and Cz electrode for VOT.

Discussion

Prior work has shown that when compared to their typically developing peers, children with reading problems exhibit a limited, dysfunctional phonological loop efficacy in WM performance (Pickering & Gathercole, 2001; Swanson & Howell,

Figure 6. The Correlation Plot Between the P3 Amplitude (y-axis) and the Accuracy Percentage (x-axis) of the V1-Back Task Across Both Groups.



Note. This indicates that V1-Back accuracy is positively associated with V1-Back amplitude ($r = 0.46$, $p < 0.05$).

2001; Swanson et al., 2009). Nonetheless, there is a paucity of developmental EEG studies examining the role of visual WM capacity, attentional processing (e.g., attention allocation, inhibition control), and global processing speed of information for children with dyslexia compared to their typically developing peers. Here we investigated behavioral performance and P300 amplitude reflecting VWM and attentional processing in children with dyslexia compared to typically developing counterparts on two well-established VWM and attention tasks (V1-Back and VOT) to address this gap in the literature.

In line with the WM deficit account, we found that children with dyslexia demonstrated lower VWM accuracy compared to typically developing controls on the V1-Back task. This suggests that these children exhibit behavioral deficits in maintaining visual information in WM. These results are consistent with others who have also shown that individuals with dyslexia exhibit impaired WM processes at the behavioral level (Albano et al., 2016; Brandenburg et al., 2015; Gathercole et al., 2006; Menghini et al., 2011; Pennington, 2008; Pickering, 2012; Schuchardt et al., 2008; Smith-Spark & Fisk, 2007; Swanson, 1994; Swanson et al., 2009; Vellutino et al., 2004; Wagner & Muse, 2006). It is possible that these deficits contribute to the development of reading difficulties experienced by children with dyslexia (Singleton, 2002), and serve as a potential risk factor for decreased academic

performance commonly observed in this population (Bergey et al., 2017; Chevalier et al., 2017; Snow & Strucker, 1999). Similar to V1-Back accuracy, we found significant group differences in accuracy between children with dyslexia and typically developing controls on the VOT, suggesting that attentional allocation to novel stimuli and updating in WM is attenuated in children with dyslexia compared to typically developing individuals. This finding complements the work of others who have also found attentional deficits in individuals with dyslexia (Bosse et al., 2007; Facoetti et al., 2008; Heiervang & Hugdahl, 2003; Saksida et al., 2016; Vidyasagar & Pammer, 2010). Taken together, our results suggest that children with dyslexia experience behavioral deficits on tasks including attentional allocation, working memory maintenance, and working memory updating.

Despite the differences in accuracy in the V1-Back and VOT, we did not observe any differences in response time between children with dyslexia and typically developing controls in these tasks. This finding is consistent with others who also failed to observe impaired response times in individuals with dyslexia (Evans et al., 2011), but others have reported contrasting results (Kail & Salthouse, 1994; Miller et al., 2006; Shiran & Breznitz, 2011). It is possible that the nature of the VWM and attention tasks we implemented were not sensitive enough to reflect difficulty in information processing speed that children with dyslexia might experience. Alternatively, in line with the work of others (McVay & Kane, 2012), it is likely that the cognitive impairment observed in individuals with dyslexia is more likely to impact the actual WM processes more so than the global processing speed. Overall, our behavioral results suggest that for children with dyslexia, impaired processing speed is unlikely to be the primary underlying deficit, when compared to their typically developing peers on VWM and attention tasks.

In complement with our null response time differences, we also failed to observe any significant discrepancies in P300 latency on both the V1-Back and VOT between children with dyslexia and typically developing controls. While this finding is consistent with existing data (Evans et al., 2011), it is inconsistent with others' work regarding global processing speed (Kail & Salthouse, 1994; Fawcett et al., 1993; Maciejewska et al., 2013; Miller et al., 2006; Ortiz et al., 1990; Taroyan et al., 2007). One possibility for the discrepancy between our results and others regarding latency is due to the use of age as a covariate, given that prior work suggests age

strongly influences P300 latency (Papagiannopoulou & Lagopoulos, 2017; van Dinteren et al., 2014). Therefore, we interpret the lack of group difference in the P300 latency, in addition to our null differences in response time, as further supporting the notion that children with dyslexia maintain global processing speed of information to the same degree as their peers at both the behavioral and neural level.

Consistent with our hypotheses concerning P300 amplitude, we observed reduced P300 amplitudes on both the V1-Back and VOT in children with dyslexia compared to typically developing controls. This complements prior reports showing reduced P300 amplitudes in children with reading-related impairments (Evans et al., 2011; Papagiannopoulou & Lagopoulos, 2017; Taroyan et al., 2007). Regarding the reduced accuracy in the V1-Back task, it is likely that these individuals fail to maintain information in WM, resulting in decreased behavioral outcomes. This impairment in VWM load maintenance may impact the ability to process words and letters in WM for individuals with dyslexia. Given that WM impacts functions involving multifaceted visual sensory processing, such as reading letters and words, this may suggest that individuals with dyslexia experience abnormal development of visual representation, recognition and recall of words and letters. Therefore, automatic reading fluency is not achieved, rendering semantic impairment (Giovagnoli et al., 2016). In line with this hypothesis, more recent findings of Shiran and Breznitz (2011) and Lotfi et al. (2020) have emphasized the relationship between a larger VWM capacity and reading skill improvement among dyslexic individuals, suggesting the pivotal role these processes have in impacting these individuals' performance. They argued that WM deficits in individuals with dyslexia go beyond verbal processing and can rather stem from visuospatial subsystem of the WM. This suggests that exercising WM might consequently improve the quality of reading. In line with this position, using specifically designed computerized cognitive training to target the recall span and the efficiency of visuospatial processing within WM, these researchers showed their training resulted in a significant improvement in decoding, reading rate, and comprehension of dyslexic readers (Shiran & Breznitz, 2011; Lotfi et al., 2020).

We also observed reduced P300 amplitude on rare trials on the VOT for children with dyslexia compared to their typically developing peers. Our results are consistent with others who also observed decreased

P300 on VOT (Papagiannopoulou & Lagopoulos, 2017). It is not surprising that this reduced P300 amplitude was also associated with a reduced behavioral accuracy for dyslexic children on this task. Given that P300 amplitude is believed to reflect WM updating and attentional allocation towards novel stimuli (Polich, 2007), this finding may reflect deficits in these processes for children with dyslexia compared to typically developing peers. This finding also supplements the reduced accuracy observed in these individuals for the VOT.

Overall, our results are consistent with previous studies showing limited VWM and visual attentional processes for dyslexic children both at the behavioral level (i.e., reduced accuracy) as well as underlying neural signatures (i.e., P300 amplitude), resulting in more impaired processing of stimuli. Although we did not observe a significant response time (i.e., processing speed) difference between the DYS and TDC groups in V1-Back task, it is unclear whether this would be the case if the difficulty of *n*-back task increased (namely 2-back, 3-back) which requires larger WM capacity to maintain and manipulate items. Particularly, Evans et al. (2011) reported that children with language impairments showed significant reaction time deficiency when the load of WM increased (Evans et al., 2011). Therefore, it may be the case that incorporating more challenging tasks results in greater discrepancies in performance between children with dyslexia and their typically developing peers.

Additionally, it may be possible that this reduced P300 amplitude may not merely stem from a limited WM capacity, and could represent a broader developmentally dysfunctional visual system. Numerous studies have shown that developmental dyslexia is associated with a number of broad visual-perceptual abnormalities in low spatial and high temporal frequencies (Stein, 2001), visual perception of low contrast (Stein, 2001), and eye-movement anomalies (Dusek et al., 2011; Quercia et al., 2013). Nevertheless, there is evidence showing an increase of the P300 amplitude as a function of age among typically developing children. Therefore, it is likely that the reduced P300 of children with dyslexia might change as they enter later stages of their visual development trajectory (van Dinteren et al., 2014). It is also possible that the reduced P300 for children with dyslexia was due to other compensatory effortful brain mechanisms such as exploiting mental rehearsal strategies. This is quite unlikely though, given that countless studies have reported deficits in lexical encoding and retrieval for dyslexic children compared to their typically

developing counterpart. We failed to observe a significant RT and P300 latency difference for both tasks. If this was true, this compensatory mechanism should have rendered slower RT and longer P300 latencies (van Dinteren et al., 2014). Third, we found a strong positive correlation between the magnitude of P300 and the WM capacity of V1-Back task, suggesting the possibility that the P300 amplitude deflection is modulated by the WM capacity.

This study is not without limitations. First, our V1-Back WM task was not capable of distinguishing between encoding or maintenance properties of WM. It may be that this reduced P300 reflects attenuated cognitive resources to encode or maintain degraded memoranda primarily due to resources being used to inhibit extraneous information to enter WM. In fact, our results show that the DYS group showed significantly higher rates of commission errors in both V1-Back and VOT tasks, attesting to some forms of inhibitory deficits in dyslexic children (Savage et al., 2006). Nonetheless, Huettel and McCarthy (2004) reported that VOT is associated with higher activation of the dorsolateral prefrontal cortex (dlPFC; i.e., thought to contribute to P300 magnitude), which regulates encoding, updating and activation of context-appropriate behavior, and maintaining WM. Given the lower accuracies and reduced P300 amplitudes observed for both tasks, one may infer that children with dyslexia might experience constraints on encoding, updating, and maintenance of WM. Therefore, we suggest that future studies investigate the incorporation of a WM task that distinctively taps into these properties of WM while capturing underlying neurophysiological signatures to increase validity and reliability of the result. As mentioned earlier (Evans et al., 2011), children with dyslexia might use covert compensatory effortful mechanism to offset their behavioral WM deficiencies, resulting in null findings between behavioral performance of dyslexic and typically developing children (Evans et al., 2011).

In conclusion, an abundance of evidence has highlighted impaired academic performance in individuals with reading disorders, such as dyslexia (Bergey et al., 2017; Chevalier et al., 2017; Snow & Strucker, 1999). Researchers (Albano et al., 2016; Bosse et al., 2007; Brandenburg et al., 2015; Facchetti et al., 2008; Gathercole et al., 2006; Heiervang & Hugdahl, 2003; Menghini et al., 2011; Pennington, 2008; Pickering, 2012; Schuchardt et al., 2008; Smith-Spark & Fisk, 2007; Swanson, 1994; Swanson et al., 2009; Vellutino et al., 2004; Wagner & Muse, 2006) have suggested that these

deficits may be the result of impairments in WM and attentional processes, or a more broad global processing speed impairment. Our results suggest that children with dyslexia exhibit both behavioral and neural deficits on tasks requiring WM maintenance, reflected by reduced accuracy and P300 amplitude for the *n*-back task. In addition, we identified behavioral and neural deficits in WM updating and attentional allocation processes, seen by reduced accuracy and P300 amplitude on the VOT. Finally, we found no evidence for impairments in global processing speed of information for children with dyslexia compared to their typically developing peers. These findings, using EEG, further support the notion that individuals with dyslexia, particularly children, exhibit deficits in cognitive processes related to WM and attention.

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Author Disclosure

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