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Decoding ability makes waves in reading: Deficient interactions between attention and phonological analysis in developmental dyslexia

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Abstract

Whilst there is general consensus that phonological processing is deficient in developmental dyslexia, recent research also implicates visuo-attentional contributions. Capitalising on the P3a wave of event-related potentials as an index of attentional capture, we tested dyslexic and normal readers on a novel variant of a visual oddball task to examine the interplay of orthographic-phonological integration and attentional engagement. Targets were animal words (10% occurrence). Amongst nontarget stimuli were two critical conditions: pseudohomophones of targets (10%) and control pseudohomophones (of fillers; 10%). Pseudohomophones of targets (but not control pseudohomophones) elicited a large P3 wave in normal readers only, revealing a lack of attentional engagement with these phonologically salient stimuli in dyslexic participants. Critically, both groups showed similar early phonological discrimination as indexed by posterior P2 modulations. Furthermore, phonological engagement, as indexed by P3a differences between pseudohomophone conditions, correlated with several measures of reading. Meanwhile, an analogous experiment using coloured shapes instead of orthographic stimuli failed to show group differences between experimental modulations in the P2 or P3 ranges. Overall, our results show that, whilst automatic aspects of phonological processing appear intact in developmental dyslexia, the breakdown in pseudoword reading occurs at a later stage, when attention is oriented to orthographic-phonological information.

Keywords: attention, dyslexia, event-related potential, P3a, pseudohomophone.

1. Introduction

Impaired decoding of visually unfamiliar words or pseudowords is one of the key characteristics of developmental dyslexia (in an alphabetic language; see Rack, Snowling & Olson, 1992). Since pseudowords do not have an entry in the lexicon that can be directly accessed from print, pseudoword recognition requires sublexical decoding (that is, breaking down the letter string into its smaller units to map and blend their corresponding sounds). In the context of behavioural evidence indicating phonological processing weakness in dyslexia (see Vellutino, Fletcher, Snowling & Scanlon, 2004, for a review), one of the dominant hypotheses accounting for pseudoword reading deficiency is the existence of weaker and/or poorly specified phonological representations (e.g., Adlard & Hazan, 1998; Boada & Pennington, 2006; Morais, 2003; Snowling, 2000; Swan & Goswami, 1997). Degraded phonological representations are thought to impact reading in general by interfering with the acquisition and use of grapheme-phoneme correspondence (GPC) rules (Morais, 2003; Morais & Kolinsky, 1994). However, successful pseudoword decoding also requires accurate orthographic processing, intact graphemic parsing, integration of visual and phonological information both at the grapheme and/or letter string level, and for all to be present in the context of adequate attentional engagement, motivation, and working memory resources (e.g., Guthrie & Wigfield, 1999; Lepola, Poskiparta, Laakkonen & Niemi, 2005; Lien, Ruthruff, Cornett, Goodin & Allen, 2008; O'Malley, Reynolds, Stolz & Besner, 2008; Reynolds & Besner, 2006; Shaywitz & Shaywitz, 2008). It is perhaps not surprising then, that corresponding differences in orthographic processing (Bosse, Tainturier & Valdois, 2007; Hawelka, Huber & Wimmer, 2006; Maurer et al., 2007; Pitchford, Ledgeway & Masterson, 2009; Valdois, Bosse & Tainturier, 2004; Vidyasagar & Pammer, 2010), visuo-attentional

processing (Bosse et al., 2007; Facoetti et al., 2003a, 2006, 2010; Facoetti, Lorusso, Paganoni, Umiltà & Mascetti, 2003b, Facoetti, Ruffino, Peru, Paganoni & Chelazzi, 2008; Hawelka et al., 2006; Hawelka & Wimmer, 2005; Lassus-Sangosse, N'guyen-Morel & Valdois, 2008; Valdois et al., 2004; Pammer, Lavis, Hansen & Cornelissen, 2004b; Roach & Hogben 2007, 2008; Romani, Tsouknida, di Betta & Olson, 2011; Vidyasagar & Pammer, 2010), symbol-sound mapping (e.g., Ziegler, Pech-Georgel, Dufau & Grainger, 2010), and working memory involvement (e.g., Banai & Ahissar, 2006; Menghini, Finzi, Carlesimo & Vicari, 2011; Ram-Tsur, Faust & Zivotofsky, 2008; Ramus & Szenkovits, 2008) have also been reported.

On the basis of most of the latter observations, authors have challenged the primacy of the phonological deficit; often emphasising the potential role of attention. For instance, deficits in exogenous visual attention have been reported in behavioural studies of visuospatial processing, such as the Posner paradigm, in which dyslexic individuals typically show reduced reaction time (RT) advantages from valid spatial cues as compared to uncued or nonvalid stimuli (Brannan & Williams, 1987; Facoetti et al. 2003a, 2003b, 2006; Jones, Branigan, & Kelly, 2008; Roach & Hogben, 2004; 2007, 2008) and from poorer stimulus detection in attentional blink tasks (Buchholz & Aimola Davies, 2007; Facoetti et al., 2008; Ruffino, Trussardi, Gori, Finzi, Giovagnoli, Menghini et al., 2010). Furthermore, performance in these tasks have been found to correlate with pseudoword reading measures (Facoetti et al., 2006, 2008), which has led to the hypothesis that stimulus-driven engagement of attention is weaker or slower in dyslexic individuals who manifest deficient phonological awareness (e.g., Buchholz & Aimola Davies, 2008; Facoetti et al., 2006, 2010; Hari & Renvall, 2001; Ruffino et al., 2010). Reading difficulties would then arise because of impaired visuo-attentional mechanisms

controlling letter sequence scanning, affecting orthographic inputs, and impacting subsequent phonological mapping (e.g., Facoetti et al., 2006, 2008; Pammer et al., 2006; Ruffino et al., 2010; Valdois et al., 2004; Vidyasagar & Pammer, 2010). Alternatively, a general impairment in attentional filtering has been put forward, in which difficulties relate to selecting task-relevant sensory information (Roach & Hogben, 2007, 2008; see also Geiger et al., 2008).

These hypotheses of decoding dysfunction in dyslexia are based on behavioural studies mostly outside a reading context. Such studies are essential for theory development and for identifying characteristics of developmental dyslexia but are limited in their insight into the relative and interactive contributions of attention, orthography and phonology in reading. Using event-related brain potentials (ERPs), processing in dyslexia in one or more of these stages of reading can be studied with exquisite temporal resolution.

Based on results from studies with skilled readers, we know that manipulations of whole-word phonology can reliably modulate ERP waves within approximately 250 ms of stimulus onset, in the early P2-N2 ranges (Braun, Hutzler, Ziegler, Dambacher & Jacobs, 2009; see Dien 2009 or Grainger & Holcomb, 2009, for reviews). If, for example, phonological representations are impaired in dyslexia, which ought to impact reading both via deficient grapheme-phoneme decoding and subsequently weakened phonological percept, then phonological manipulations of visual word stimuli should affect dyslexic and normal readers differently in the early processing windows. Since ERP studies of visual word processing in dyslexia had not reported phonological effects earlier than in the N400 window (where differences could equally be attributed to attentional or working memory factors as much as perceptual differences), we specifically targeted phonological processing in the P2-N2 range in developmental dyslexics (Savill & Thierry, 2011b).

Dyslexic adults, engaged in judgements regarding the homophony of written words with a preceding pseudoword, displayed similar phonological modulations as normal readers in this early time range. Interestingly, reduced phonological modulations in the dyslexic group emerged later, in the P600 range, which we interpreted as a sign of deficient monitoring of phonological information rather than that of a fundamental deficit in extracting phonological information from print. Recently, we estimated a potential temporal onset for weaker phonological engagement in dyslexia in the P3a range i.e., between 350 and 450 ms (Savill & Thierry, 2011a). In a sentence reading context, we found that, unlike controls, dyslexic adults do not show a P3a response to homophones and pseudohomophones (homophonic pseudowords) of predictable sentence endings despite earlier phonological priming effects in the P2a (frontocentral P2 peak; see Potts, 2004) and N2 ranges. We interpreted this pattern as evidence of impaired attentional engagement with stimuli that are phonologically relevant but orthographically unexpected, whilst perceptual phonological processing appears intact. In the present study, we set out to test the robustness of our previous findings using a P3a-eliciting paradigm. We developed a novel visual oddball paradigm that incorporates a range of words along with pseudohomophones to test the P3a-eliciting capacity of orthographic-phonological incongruities. The rare, target stimuli were animal nouns, making up 10% of the stimuli shown (TARG). Filler stimuli were words from seven other semantic categories consisting of the same number of exemplars (totalling 70% of the stimulus set, FILL). The critical P3a-eliciting nontarget consisted of pseudohomophones of the animal names used as targets (10% of the stimulus set), i.e., pseudohomophones of targets (PSHT). Finally, we also included a rare, nontarget control condition, which consisted of pseudohomophones of one of the filler semantic categories (food words, PSHF) and

which were therefore irrelevant. We created this latter condition in order to (1) test whether the pseudohomophone status in itself would be enough to elicit a P3a (whether or not the pseudoword is a homophone of an animal name); (2) provide a measure of phonological engagement with phonologically relevant versus irrelevant pseudohomophones; and (3) provide a control condition for the phonological duplication of stimuli (since targets were phonologically duplicated too).

On the basis of our previous study (Savill & Thierry, 2011a), in which we showed that dyslexic adults fail to manifest a P3a in response to the phonological content of unanticipated orthographic forms, we predicted that PSHT stimuli would elicit a significant P3a in the control group only as compared to control conditions. Furthermore, given that visual stimuli presented in an unprimed continuous stream can modulate P2 amplitudes based on semantic manipulations (e.g., Kissler, Herbert, Winkler & Junghofer, 2009, Martin-Loeches, 2007; Moscoso del Prado Martín, Hauk & Pulvermüller, 2006), lexical properties (e.g., Costa, Strijkers, Martin & Thierry, 2009; Strijkers, Costa & Thierry, 2010), and phonological effects (Braun et al. 2009; Meng, Jian, Shu, Tian & Zhou, 2008; Kong, Zhang, Kang, Du, Zhang & Wang, 2010; Zhang, Zhang & Kong, 2009), we anticipated ERPs modulations also in the P2 range here. To see whether oddball effects in the main experiment would generalise to the processing of nonverbal stimuli, participants were also tested on a nonverbal control version of the experiment, which used a set of coloured shapes mimicking the relationships between conditions in the word version (conditions were labelled TARG-S, FILL-S, PSHT-S, PSHF-S).

2. Method

2.1. Participants

Fourteen adults with a diagnosis of developmental dyslexia (7 males; mean age 22.57 years) and 14 age-matched control adults (7 males; mean age 23.14 years) took part in the experiments approved by Bangor University's ethics committee. All were right-handed with normal or corrected-to-normal vision, reported no attentional difficulties or comorbidities and were students recruited via Bangor University's dyslexia unit or participant panel. Using student participants ensured relative homogeneity as regards reading experience, intelligence, age, and socioeconomic status; all criteria that should be controlled when comparing ERP responses between groups (Picton et al., 2000). In addition, dyslexic participants had been assessed by the Miles Dyslexia Centre at Bangor University and could conveniently be selected on the basis of residual weaknesses in measures of pseudoword reading and spelling. Performance on subtests taken from the Dyslexia Adult Screening Test (DAST; Nicolson & Fawcett, 1998), Wide Range Achievement Test (WRAT-3; Jastak & Wilkinson, 1993), Wechsler Individual Achievement Test (WIAT-II; Wechsler, 2005) and Wechsler Adult Intelligence Scale (WAIS-III; Wechsler, 1997) showed that the dyslexic group were significantly impaired compared to the control group on measures of literacy and phonological skill, but did not differ in nonverbal reasoning and digit span performance (see Fosker & Thierry, 2004, 2005 for a similar profile). Table 1 details group performance on the measures used.

INSERT TABLE 1 ABOUT HERE

2.2. Stimuli

Word task: Twenty-five English words between three and eight letters long were selected (from the MRC Psycholinguistic database) as exemplars for each of eight semantic categories (animals, food items, clothing, tools, nature, jobs, furniture items and body parts). Kucera-Francis written frequency, concreteness, imageability, length, number of syllables, orthographic neighbourhood density (OND), and constrained bigram and trigram frequencies were controlled across semantic category. Animal words were allocated as target stimuli (TARG) and the remaining semantic categories acted as filler items (FILL). Pseudohomophones (homophonic pseudowords) of the words from two of the categories were also created: animals (pseudohomophones of targets; PSHT) and food items (pseudohomophones of fillers; PSHF). Pseudohomophones did not differ in length from their exemplars and the PSHT and PSHF conditions were also controlled for their orthographic similarity to their exemplar, and for OND, and constrained bigram and trigram frequencies (<http://www.neuro.mcw.edu/mcword/>, Medler & Binder, 2005). The same proportion (28%) of animal and food pseudohomophones, 24% of animal words, and 28% of food words contained a lexical phonological neighbour (e.g., 'air' in 'bair'). Therefore, any strong lexical facilitation of phonological access afforded by such stimuli was unbiased between conditions. The low proportion of these stimuli means that, for the majority of pseudohomophones, sublexical phonological processing was necessary. The linguistic characteristics of the stimuli are summarised in the supplementary materials.

Shape task: Target stimuli were 25 unicoloured squares (TARG-S). Relevant rare nontargets were 25 bicoloured squares (PSHT-S) while the rare nontarget controls were 25 bicoloured triangles (PSHF-S). The standard fillers consisted of 25 unicoloured

shapes belonging to each of 7 geometric forms (triangle, circle, parallelogram, trapezoid, kite, pentagon, hexagon; FILL-S). Stimuli were created in Adobe Illustrator CS3 and each shape was matched for number of pixels and coloured in one of 5 isoluminant shades of blue. The second shade embedded in the rare nontarget stimuli occupied the same number of pixels in each case (see Figure 1 for sample stimuli). The final 25 colours (blues, yellows, greens, pinks and purples) were created by adjusting the hue value in 5 steps for each shape in Adobe Photoshop CS3.

2.3. Design

The experiment adapted a traditional oddball design, which was analogous for both word and nonverbal tasks. In both tasks, targets (animal words / unicoloured squares), relevant nontargets (animal pseudohomophones / bicoloured squares) and non-relevant nontargets (food word pseudohomophones / bicoloured triangles) were each presented 10% of the time. Stimuli taken from across 7 other categories (non-animal words / unicoloured non-square shapes) constituted the remaining 70%. Both word and nonverbal tasks used a repeated measures mixed design with within-subjects factors of stimulus (TARG/-S, FILL/-S, PSHT/-S, PSHF/-S) and electrode and between-subjects factor of group (control, dyslexic).

INSERT FIGURE 1 ABOUT HERE

2.4. Procedure

Participants were seated in a sound attenuated, dimly lit room and asked to keep eye and head movement to a minimum. Stimuli were centrally presented on a 40" high resolution

LED screen (75 Hz refresh rate) with word stimuli in black lower case Arial font on a white background (Word stimuli: max. visual angle of $3.44^\circ \times 0.41^\circ$; shape task: $2.79^\circ \times 3.00^\circ$). In a given trial, a blank screen was displayed for 150 ms, followed by the stimulus, which was shown for 200 ms. Participants had 1250 ms in which to make a response during which the screen was blank, and remained blank for a further 250 ms in the word version. Participants were asked to make a button press as soon as a target stimulus was presented (i.e., an animal word or single-coloured square), and to withhold a response to all other stimuli. Stimuli were presented semi-randomly using E-Prime (Psychology Software Tools, Inc., Pittsburgh, USA) such that stimulus categories were represented evenly across each block and each stimulus was shown once in both halves of the experiment. There were 500 trials in total, presented across four blocks. Task order was counterbalanced across participants.

2.5. Data Acquisition

Electrophysiological (EEG) data were continuously recorded at a rate of 1kHz from 37 Ag/AgCl electrodes placed according to the 10-20 system in an elastic cap (impedances $< 5 \text{ k}\Omega$). Recordings were in reference to the left mastoid with FPz serving as the ground electrode. Eye movement was monitored by electrodes positioned above and below the left eye. EEG recordings were filtered online between 0.1 and 200 Hz and re-filtered offline using a zero-phase shift band-pass between 0.1 and 30 Hz. Eye blinks were removed using the correction procedure provided by Edit 4.3 (NeuroScan). EEG epochs ranged from -100 to 1000 ms after the onset of stimulus. Epochs with voltage exceeding $\pm 75 \mu\text{V}$ were automatically rejected. After baseline correction in reference to pre-stimulus activity, individual averages were re-referenced to the average of the left

and right mastoids and computed for each experimental condition and averaged together according to participant group to produce grand-mean averages (30+ trials in each case. Average number of trials used given in Table 2).

INSERT TABLE 2 ABOUT HERE

2.6. Data Analysis

In both tasks, a typical P1/N1 complex was observed followed by an early frontal P2 (P150) peak and a later observable P2 at posterior sites. These were followed by a diffuse P3 peaking over central midline sites and which was visibly earlier in the shape task than the word task. Epochs for mean amplitude analyses were defined based on visual inspection of ERP modulations (Luck, 2005) and apriori expectations from previous experiments using similar stimuli (e.g., Savill and Thierry, 2011a). Windows of analysis were 80 – 115 ms for the P1; 140 – 180 ms for the N1; 140 – 200 ms for the frontal P2; from 210 – 240 ms for the posterior P2; 270 – 350 ms for the N2 and, in the case of the P3a, 370 – 670 ms in the word task and 280-580 ms in the shape task. To check whether target stimuli elicited a P3b response, statistical analyses were performed over posterior electrodes using the same time window and the same number of electrodes as for the analysis of the P3a. Peak detection was time-locked to the electrode of maximal amplitude for each component: O1 for the P1; P7 for the N1; FCz for the frontal P2 (P150) and N2, Pz for the posterior P2, Cz for the P3a and Pz for the P3b. Mean amplitudes were measured at electrodes chosen on their maximum sensitivity: O1, O2, P7 and P8 electrodes for the P1 and N1; FC3, FC4, FCz, C3, C4, Cz for the frontal P2, N2 and P3a; CP3, CP4, CPz and Pz for the posterior P2 and CP3, CP4, CPz, P3, P4 and Pz for the P3b. Greenhouse-Geisser correction of degrees of freedom was applied where relevant.

3. Results

3.1. Word Task

3.1.1. Behavioural Results: Mean reaction time and hit rates to animal words did not differ between groups ($t(26) = 0.14, p = 0.89$; control RT = 568 ± 59 ms; dyslexic RT = 566 ± 57 ms and $t(26) = 1.69, p = .10$; control accuracy = $97\% \pm 3\%$; dyslexic accuracy = $94\% \pm 6\%$). PSHT stimuli elicited significantly more false alarms as compared to PSHF and FILL, however this did not significantly differ between groups ($p = .10$; Correct rejections: PSHT: Control = 88.86%; Dyslexic = 82.86%; PSHF: Control = 100%; Dyslexic = 100%; FILL: Control = 99.98%; Dyslexic = 99.73%).

3.1.2. ERP Results: There were no significant modulations of the P1, N1, or frontal P2 peaks by the experimental conditions. The P2 over occipitoparietal electrodes was significantly modulated by experimental condition ($F(3, 78) = 9.04, p < .001$; see Figure 2). Pairwise comparisons showed that posterior P2 mean amplitudes were increased to the TARG and PSHT conditions relative to FILL and PSHF, with no difference between TARG and PSHT or FILL and PSHF, respectively. No group differences or latency effects were observed in the P2 range.

INSERT FIGURE 2 ABOUT HERE

In the P3a range, a significant effect of condition on mean amplitudes was observed over frontocentral electrodes ($F(3, 78) = 23.80, p < .001$). Pair-wise comparisons showed that

TARG and PSHT elicited larger amplitudes than FILL and PSHF conditions, with TARG eliciting greater amplitudes than PSHT. This was qualified by a main effect of group ($F(1, 26) = 4.43, p < .05$) and further by an interaction between group and condition ($F(3, 78) = 4.12, p < .05$). The group effect indicated smaller P3a amplitudes overall in the dyslexic group. The group by condition interaction showed that the P3a elicited by PSHT in control readers was significantly larger than PSHF and FILL conditions and did not differ in amplitude from TARG; whereas the dyslexic group showed no discernable separation of PSHT, PSHF and FILL, with only TARG eliciting significantly larger amplitudes (see Figure 3).

P3a latency analyses showed no condition effect but P3a peak latencies were longer overall in the dyslexic group ($F(1, 26) = 10.52, p < .01$; control = 521 ± 80 ms; dyslexic = 580 ± 77 ms).

INSERT FIGURE 3 ABOUT HERE

As expected, amplitude analyses over posterior sites showed that target words elicited a large P3b significantly larger than that elicited in all other conditions (condition main effect: $F(3, 78) = 63.42, p < .001$; Mean amplitudes: TARG = $5.70 \mu\text{V}$, PSHT = $3.54 \mu\text{V}$, PSHF = $1.34 \mu\text{V}$, FILL = $1.32 \mu\text{V}$). P3b amplitudes tended to be smaller in the dyslexic group overall, $p = .08$. The difference between the P3b elicited by PSHTs and TARG was smaller in the dyslexic group but the interaction between condition and group was not overall significant (see Figure 4). To distinguish P3a and P3b effects, an anteriority factor was included in an omnibus ANOVA on P3 (anterior and posterior sites respectively). The anteriority factor interacted with experimental condition, due to the large posterior response to TARG stimuli ($F(3, 78) = 21.70, p < .001$). Across anterior

and posterior sites, the interaction of group and condition was just short of significance, $p = .06$. This effect was driven by a three way interaction of anteriority, experimental condition and group, ($F(3, 78) = 3.68, p < .05$), indicating that critical group differences lied anteriorly, in the P3a realm.

3.2. Shape Task

3.2.1. Behavioural Results: Mean reaction time and proportion of correct hits to target squares did not differ between groups (reaction time: $t(26) = 0.62, p = .54$; control = 413 ± 56 ms; dyslexic = 402 ± 41 ms; accuracy: $t(26) = 1.38, p = .18$; control = $99.6\% \pm 0.9\%$; dyslexic = $99.0\% \pm 1.3\%$). Both groups correctly withheld behavioural responses to 100% of the PSHF-S and FILL stimuli; however this significantly differed from PSHT-S, which were correctly rejected at a rate of 97.6% overall ($F(2, 52) = 23.26, p < .001$) and significantly less so in the dyslexic group ($F(2, 52) = 11.59, p < .01$; control = 99.3%; dyslexic = 95.9%).

3.2.2. ERP Results: PSHF-S stimuli elicited larger P1 amplitudes ($F(3,78) = 18.08, p < .001$) and earlier P1 and N1 peaks ($F(3,78) = 9.67, p < .001$ and $F(3, 78) = 11.14, p < .001$ respectively) compared to the other conditions. P1 and N1 also peaked earlier to PSHF-S than FILL. Frontal P2 latencies were significantly affected by condition ($F(3,78) = 7.11, p < .001$) because the peak elicited by target stimuli was delayed. No significant amplitude modulations were observed for the N1 or frontal P2 peaks. Posterior visual P2 peak mean amplitudes were significantly modulated by condition ($F(3, 78) = 18.29, p < .001$) such that all conditions were significantly different from each

other, with PSHF-S showing the largest P2 (PSHF-S>PSHT-S>FILL-S>TARG-S). No group differences for the P2 peak were observed.

As expected, condition significantly modulated P3a amplitudes ($F(3, 78) = 36.08, p < .001$; Figure 3). Pair-wise comparisons showed that TARG-S elicited a larger P3 compared to all conditions and PSHT-S was significantly larger than PSHF-S and FILL-S. A main effect of group showed that P3a amplitudes were generally smaller in the dyslexic group ($F(3, 78) = 5.53, p < .05$). No significant group interaction or P3a latency effects were observed. In other words the pattern observed in the P3a range was not different between groups.

INSERT FIGURE 4 ABOUT HERE

TARG-S stimuli elicited a large P3b response at least twice that measured in any other condition (main effect: $F(3, 78) = 92.63, p < .001$; Figure 4). As in the word task, PSHT-S also elicited a small P3b in comparison to PSHF-S and FILL-S (Mean amplitudes: TARG-S = 5.36 μV ; PSHT-S = 2.22 μV ; PSHF-S = 0.43 μV ; FILL-S = 0.84 μV). An omnibus ANOVA including P3a and P3b electrode sites using an anteriority factor confirmed a main effect of condition on P3 amplitudes in the same direction across the scalp, $F(3, 78) = 65.34, p < .001$, but also showed that the anteriority factor interacted with condition, $F(3, 78) = 20.75, p < .001$, due to the relatively larger TARG amplitudes over posterior sites (anterior = 4.20 μV ; posterior = 5.31 μV). No other interactions or group differences were observed.

3.3. Amplitude correlations

Bivariate correlations performed on the differences in posterior P2, P3a and P3b

amplitudes elicited by PSHT relative to PSHF and TARG with our diagnostic language measures are shown in Table 3.

INSERT TABLE 3 ABOUT HERE.

4. Discussion

This study examined attentional engagement by the phonological content of orthographic stimuli in dyslexic and normal-reading adults and compared this with engagement with nonverbal stimuli, using P3a modulations of event-related potentials elicited in visual oddball tasks as the index of choice.

The results of our main experiment indicated that incorrect spellings of semantically defined target words failed to significantly engage attention in dyslexic participants over and above irrelevant filler words and irrelevant misspellings. This was manifested by the dyslexic group showing similarly attenuated P3a ERP responses to pseudohomophone targets, pseudohomophone fillers, and fillers, which were, in turn, significantly smaller than the P3 elicited by target words. This differed from normal readers, who, in the context of larger P3 amplitudes overall, showed the anticipated pattern of increased P3a amplitudes to pseudohomophone targets such that they were of the same magnitude as those elicited by target words and larger than filler conditions. Importantly, we can infer from the P3a being elicited by pseudohomophone targets and not pseudohomophone fillers that the control group response was underpinned by phonological processing rather than orthographic familiarity or lexicality. Furthermore, in normal readers these phonological effects were strongly prepotent since ERPs to pseudohomophone targets were of the same magnitude as those elicited by targets; even though they were

discriminated on an orthographic basis in a similar time window (average response time to targets = 570 ms). Thus, although dyslexic participants showed normal P3 (i.e., both P3a and P3b) responses to targets (i.e., similar amplitude increase to animal words), they showed little or no attentional engagement with the orthographically unfamiliar stimuli irrespective of their phonological relevance. Consistent with our earlier study showing that P3a differences in phonological engagement are observable with familiar lexical stimuli when they are orthographically unexpected (i.e., homophones; Savill & Thierry, 2011), these data indicate deficient attentional engagement by phonologically relevant written words in dyslexia that is not explained by reduced perceptual sensitivity. In our nonverbal control task, on the other hand, the dyslexic group showed a clear P3a response to the nonverbal equivalent of the pseudohomophone targets, and the pattern of difference between conditions was similar between groups. This shows that the P3a differences found were task-specific. Furthermore, we found significant correlations between ERP modulations in the verbal task and performance in reading, spelling and rapid naming tasks, while corresponding correlations were absent in the nonverbal shape task. Importantly, these behavioural correlations were largely limited to P3a modulations sensitive to the phonological relevance of stimuli (indexed by the difference in amplitude between target and nontarget pseudohomophones).¹ This pattern of results lends support to the hypothesis of an attentional deficit in accessing or processing the correspondence

¹ In terms of reading measures, we found significant correlations between phonological modulations of P3a amplitudes and the one-minute reading subtest from the DAST (Nicolson & Fawcett, 1998), but not the reading subtest of the Wide Range Achievement Test (WRAT-3; Jastak & Wilkinson, 1993). Since these tests tap different reading skills; the speeded DAST reading measure is effectively an index of the fluency of recoding, whereas the WRAT-3 is an untimed, graded achievement test of word familiarity and sight reading ability; it is not surprising that automatic phonological orienting processes would be more important for performance in the former rather than the latter measure of lexical recognition.

between orthographic and phonological form and/or phonologically relevant information in manifestations of reading impairment (e.g., Ramus & Szenkovits, 2008; Facoetti et al., 2006, 2008, 2010; Vidyasagar & Pammer, 2010; Ziegler et al., 2010; Savill & Thierry, 2011a).

Event-related potentials, however, also provide insights into early processing stages after stimulus presentation, enabling us to also determine whether these P3a effects are the consequence of early perceptual differences in stimulus processing or purely attention-dependent. Crucially, the P3a group differences in the word task emerged after similar ERP modulations in the P2 range for target words and pseudohomophone targets in the two groups. Specifically, the posterior P2 peak was significantly larger for targets and pseudohomophone targets as compared to filler stimuli in both the dyslexic and control groups, with no significant differences between target words and pseudohomophone targets. Since phonological access is the only route by which pseudohomophone targets and pseudohomophone fillers can be discriminated, both groups showed rapid phonological discrimination on the basis of semantic information irrespective of orthographic/lexical status. The timing of this effect (peaking at 220 ms) is comparable with several ERP studies showing early phonological extraction from orthographic stimuli (Braun et al., 2009; Hsu, Tsai, Lee & Tzeng, 2009; Kong et al., 2010; Meng et al., 2008; Zhang et al., 2009) and is consistent with most current estimates for the timing of semantically-driven lexical access (e.g., Costa et al., 2009; Moscoso del Prado Martin et al. 2006; Strijkers et al., 2010). The case for these P2 modulations in both of our participant groups reflecting rapid phonologically-mediated semantic discrimination is compelling given the variety of possible exemplars for each semantic category, and the orthographic variability of stimuli within (but controlled across) each category, unlikely

based solely on low-level physical properties. Such “normal” early phonological effects before attentional engagement are in agreement with our recent observations in dyslexic adults performing sentence reading and homophone judgment tasks (Savill & Thierry, 2011a; 2011b).

4.1. Is the attentional engagement deficit in dyslexia specific to orthographic stimuli?

Given that hypotheses of an attentional engagement deficit in dyslexia are based on observations of impaired spatial and nonspatial attentional cuing, usually with nonverbal stimuli, it is reasonable to expect dyslexic participants would also show reduced attentional capture by the PSHT-S condition in the shape task. Whilst we did not find interactions between group and other experimental factors, P3 amplitude was nonetheless reduced overall in the dyslexic group, as they were in the word task. Similar observations have been made before (Barnea, Lamm, Epstein & Pratt, 1994; Dhar, Been, Minderaa & Althaus, 2008, 2010; Holcomb, Ackerman & Dykman, 1985; Lovrich & Stamm, 1983; Meyler & Breznitz, 2005; Taylor & Keenan, 1990) but not consistently (Giorgewa et al., 2002; Silva-Pereyra et al., 2001, 2003, 2010; Stelmack, Saxe, Noldycullum, Campbell & Armitage, 1988). It is possible that the overall reduction in P3a amplitude indexes a generalized deficit in frontal attention allocation mechanisms. On the other hand, since the verbal and nonverbal tasks were qualitatively matched, the nonverbal stimulus conditions may have been too distinct to capture subtle relationships between PSHT-S and TARG-S stimuli (as hinted by the P1 range modulations). Alternatively, attentional processing may be qualitatively different for stimuli that are not words. For example, dyslexic participants have been shown to display larger nontarget P3s during an active oddball task, and larger target P3s in a passive task in an oddball

design when discriminating between two single letters, which the authors discuss in terms of altered allocation of attention in visual classification (Rüsseler, Johannes, Kowalczyk, Wieringa & Münte, 2003; see also Silva-Pereyra et al., 2010).

Nonetheless, the lack of a group by condition interaction in the shape task indicates that attentional engagement mechanisms may be particularly relevant to processing orthographic stimuli. Specific deficits in attentional processing of orthographic strings are supported by two ERP studies that tested reading-impaired (RI) children in an oddball context. First, an early ERP study (Holcomb et al., 1985) showed that unexpected visual distractors within a target detection task elicited smaller P3s in RI children when targets were words as compared to nonverbal symbol strings, and as compared to children with or without attention deficits. Second, stimulus-driven differences in P3 amplitude have similarly been observed in forced-choice semantic categorisation (Silva-Pereyra et al., 2001). In this case, when RI children had to rapidly decide whether a presented stimulus was an animal or not (presented 22% of the time), they did not show a significant P3 when the stimuli were words. They did, however, show P3 responses similar to normal readers within the same paradigm when the stimuli were line drawings. Along with our observation of significant correlations between language measures and P3a modulations in the word task, but not in the shape task, these data suggest that attentional engagement particularly interacts with visual word processing in dyslexia. Therefore, reduced responsiveness to visual words may be as much part of the deficit in dyslexia as it may be a long-term practical consequence.

Aside from this, it is unknown whether dyslexic individuals also show attentionally mediated differences in phonological access in other contexts than reading, such as speech perception or efficient retrieval of phonological codes. Indeed, our strongest

correlation between ERP responses and diagnostic measures was between phonological modulations of P3a amplitude and rapid naming performance. This indicates that the deficient engagement of attention with phonology may not be limited to the domain of reading.

4.2. Understanding the temporal dynamics of reading is key

The phonological modulations of the posterior P2 that we observed in both the groups were as early as those reported for whole-string phonology in previous ERP studies (e.g., Barnea & Breznitz, 1998; Braun et al., 2009; see Dien, 2009). These early phonological responses appear to be normal in the dyslexic group prior to the P3a differences between groups, which suggests the existence of temporally dissociable phonological processing stages and/or streams; one which involves rapid, automatic phonological access that is relatively intact in dyslexics and is separate from later decoding processes affected by deficient attentional engagement. Behavioural (e.g., Frost, 1998) and magnetocencephalography (MEG) data (Cornelissen et al., 2009; Wheat, Cornelissen, Frost & Hansen, 2010) have implicated that serial decoding processes can be preceded by an early phase of automatic phonological access. For instance, recent applications of MEG have shown lexically-driven activations in a similar time range as our P2 modulations within the left inferior frontal gyrus (IFG; BA 44, within 200 ms; Cornelissen et al., 2009; Pammer et al., 2004a), and in particular, phonologically-related oscillations in BA 44 as early as 100 ms, using pseudohomophone masked primes (Wheat et al. 2010). Interestingly, Cornelissen and colleagues (Cornelissen et al., 2009; Pammer et al., 2004a; Wheat et al., 2010) have found these IFG responses to occur shortly after activations in mid occipital gyrus (within 100 ms of stimulus onset), and prior to

modulations of both ventral occipitotemporal cortex (vOTC; associated with abstracted orthographic analysis: e.g., Binder, Medler, Westbury, Liebenthal & Buchanan, 2006; Cohen et al., 2002; McCandliss, Cohen & Dehaene, 2003; Moore & Price, 1999) and left temporoparietal regions (left TPJ, associated with phonological analysis; see Graves, Desai, Humphries & Seidenberg & Binder, 2010; Hartwigsen, Baumgaertner, Price, Koehnke, Ulmer & Siebner, 2010; Jobard, Crivello & Tzourio-Mazoyer, 2003; Levy et al., 2009; Pugh et al. 2000; Sandak, Mencl, Frost & Pugh, 2004; Stoeckel, Gough, Watkins & Devlin, 2009). Whilst our ERP data are insufficient to speculate on the precise functional significance of early phonological activations, they are consistent with intact phonological access within 200 ms of stimulus onset and later impairments involving cortical areas typically associated with reading.

TPJ dysfunction has been speculated as the neurofunctional cause of attentional engagement deficits in dyslexia (e.g., Facoetti et al., 2010; Ruffino et al., 2010). Taking together (a) our current understanding of P3 neural generators, (b) neuroimaging data in relation to phonological analysis in reading, (c) patterns of abnormal activation in developmental dyslexia, and (d) the temporal series identified by MEG, the left TPJ –and to a lesser extent the left IFG– is likely to have a key role in the generation of the P3 effect observed here.

Firstly, TPJ involvement (which MEG studies have shown activated after 300 ms during word recognition; Pammer et al., 2004a) appears to be requisite to P3 generation (i.e., both P3a and P3b; see Bledowski et al., 2004a, 2004b; Knight, Scabini, Woods & Clayworth, 1989; Linden, 2005; Soltani & Knight, 2000; Polich, 2007; Yamaguchi & Knight, 1991) with additional involvement of inferior and dorsolateral prefrontal cortex in the case of novelty effects (Clark, Fannon, Lai, Benson & Bauer, 2000; Corbetta, Patel

& Shulman, 2008; Corbetta & Shulman, 2002; Daffner et al., 2000, 2003; Kiehl, Laurens, Duty, Forster & Liddle, 2001; Lee, Park, Kang, Kang, Kim & Kwon, 2007; McCarthy, Luby, Gore & Goldman-Rakic, 1997). TPJ activation, on the other hand, is modulated by the relevance of a stimulus (e.g., its phonological relevance; e.g., Clark et al., 2000; Cristescu, Devlin & Nobre, 2006; Doricchi, Macci, Silvetti & Macaluso, 2010; Downar, Crawley, Mikulis & Davis, 2001, 2002; Fjell, Walhovd, Fischl & Reinvang, 2007; Kiehl et al. 2001; McCarthy et al., 1997; Serences et al., 2005) and, within inferior parietal cortex (IPL) in particular, is implicated in stimulus-driven attentional orienting (see Corbetta & Shulman, 2002; Corbetta et al., 2008 for reviews).

Secondly, TPJ and left posterior IFG are understood to be involved in phonological decoding during reading (e.g., Borowsky, Cummine, Owen, Friesen, Shih & Sarty, 2006; Das, Padakannaya, Pugh & Singh, 2011; Graves et al., 2010; Jobard et al., 2003; Levy et al., 2009; Pugh et al., 2000; Sandak et al., 2004). For example, left IPL is implicated in orthographic-phonological integration at the whole string level (e.g., Bitan et al., 2007; Booth, 2002; Booth et al, 2003, 2004; Booth, Cho, Burman & Bitan, 2007) and hypothesised to be part of a sublexical reading pathway *en route* to left posterior IFG (Borowsky et al., 2006, Jobard et al., 2003; Levy et al., 2009) involved in effortful phonological integration (Fiez, Balota, Raichle & Petersen, 1999; Graves et al., 2010; Mechelli, Gorno-Tempini & Price, 2003; Thierry, Ibarrola, Démonet & Cardebat, 2003).

Thirdly, there is substantial functional evidence for underactive TPJ and abnormal IFG activations during decoding and phonological tasks in developmental dyslexic readers (typically found alongside underactivation of vOTC in relation to abstract orthographic processing; Paulesu et al., 1996; Pugh et al., 2000; Rumsey et al., 1997; see Richlan et al., 2009, or Gabrieli, 2009, for reviews).

Fourth, a recent proposal that the left TPJ facilitates maintenance of verbal material in working memory (e.g., Awh et al., 1996; Baddeley, 2003; Paulesu, Frith & Frackowiak, 1993) via a mechanism of automatic orienting of attention to verbal material (see Ravizza, Hazeltine, Ruiz & Zhu, 2011; see also Chien, Ravizza & Fiez, 2003; Ravizza, Delgado, Chein, Becker & Fiez, 2004) is also consistent with our interpretation of the P3a group differences (see also Savill & Thierry, 2011a) as an index of group differences in attentional orientation to phonological information.

In sum, our P3 ERP data provide additional support for a functional link between attentional orienting mechanisms, phonological decoding, and developmental dyslexia, subtended by temporoparietal cortex involvement and the pathway to inferior frontal cortex.

4.3. How might deficient attentional engagement account for reading difficulties?

It remains unclear how attentional engagement would be critical for decoding, or how specific to reading the implicated processes in dyslexia may be. Some possible mechanisms are: (a) (inefficient) serial engagement / disengagement of attention across a letter string (see for e.g., Facoetti et al., 2008; Vidyasagar & Pammer, 2010); (b) (globally reduced) attentional orientation to the correspondence between orthographic and phonological information, or to phonological information in general, during reading (see Savill & Thierry 2011a, 2011b); (c) (impaired) attentional selection of perceptual information (see Roach & Hogben, 2008) or (d) (inefficient) working memory engagement during decoding (e.g., Berninger, Raskind, Richards, Abbot & Stock, 2008; Menghini et al., 2011; Swanson & Ashbaker, 2000). Whichever of these mechanisms best describes the interaction of attentional and reading processes in dyslexia, a potential outcome of the phonological form of a written word being less attentionally engaging for

dyslexic readers is common to all. Since this study and that by Savill & Thierry (2011a) are the first to demonstrate differences in stimulus-driven attentional processes between dyslexic and normal readers in a reading context, further investigation is needed to establish the specificity of these effects. Therefore, at this stage, we are cautious to speculate how the effects we observe extend to normal word reading. However, a general deficit in stimulus-driven phonological engagement (rather than in phonological sensitivity) would have clear implications for decoding success, and reading efficiency globally. For instance, in terms of decoding, the mechanisms by which impaired phonological representations are implicated in the reading outcome (see Snowling, 2000) equally apply if the deficit is at the attentional rather than the perceptual level, and the difficulty is in accessing phonological information rather than sensitivity to it.

Segmentation, the use of grapheme-phoneme mapping, blending, and maintenance in verbal working memory, all require attentionally mediated phonological access (importantly, with the exception of verbal working memory, these are also orthographically mediated). Therefore, sublexical reading processes would be greatly affected by reduced stimulus-driven engagement with phonological information; presumably influencing effortful decoding. Indirectly, lexical reading mechanisms (i.e., direct retrieval of a word's phonological form from the lexicon) could too be affected by reduced attention orienting in reading over time via (1) attentional constraints on the strength of the associations learnt between orthographic and phonological representations of a given lexical item, and (2) inefficient access to phonological representations on subsequent presentations. Deficient phonological engagement might therefore affect both reading routes, albeit disproportionately. Importantly, however, the attentional engagement effects we focus on here are differences in automatic phonological processing of pseudowords, in dyslexic individuals who are identifiable by their

pseudoword reading and phonological awareness difficulties. Not all reading difficulties can be explained by phonological mechanisms and not all dyslexic readers experience significant phonological impairments. Therefore, here, we have confined the discussion to phonological decoding impairment. In surface dyslexia the greatest deficits are in reading processes that rely on whole word recognition (e.g., exception words) and, in this case, phonological task performance may be unimpaired (see Valdois et al., 2004; Bosse et al., 2007). As we did not test individuals with this profile, it is unclear whether a reading deficit at the onset of attentional engagement would apply only in individuals with phonological processing difficulties. That said, existing behavioural studies indicate attentional orienting deficits that are specific to individuals with phonological decoding impairments (Facoetti et al., 2006, 2010). Therefore, based on the existing data, we suggest that pseudoword reading impairments in dyslexia stem from deficient attention to phonological information, and that the resulting lack of decoding fluency may indirectly influence broader word recognition processes.

4.4. To what extent could these data reflect differences unique to high-functioning dyslexic readers?

While there are a number of methodological and interpretational advantages to comparing performance of university students who differ only in terms of whether they are diagnosed as dyslexic, the dyslexic participants we tested might not be regarded as ‘typical’ dyslexic readers. These participants have obviously compensated to some degree for their reading difficulties and have attained a reading proficiency within a normal range. Their high-functioning status leaves the question of whether the same pattern of results would be found in dyslexic individuals who have persistently poor reading, or in children with dyslexia. That is, it is possible that intact early phonological

access seen in the P2 range in high-functioning dyslexic adults may be an index (or cause) of their relatively well-compensated reading. Clearly, the generalisability of the early phonological access we see in our dyslexic participants to other dyslexic populations needs to be investigated in future studies. Attentional orienting differences between dyslexic and non-dyslexic readers, on the other hand, are likely to remain observable similarly across levels of reading proficiency. This is because (1) it is unlikely that an attentional orienting deficit would develop only in high functioning dyslexics; and (2) there is a hypothetical link between attentional orienting differences and TPJ underactivation, whilst TPJ dysfunction is suggested to be a persistent marker of dyslexia irrespective of levels of compensation (unlike activation in left IFG, which varies with compensation level; Hoefft et al., 2007, 2011). Nonetheless, similar investigation comparing dyslexic children or adult dyslexics who are persistently poor readers with age-matched controls² will help determine whether poor readers show earlier perceptual differences from normal readers or whether decoding driven processing differences indeed emerge at the stage of attentional engagement.

5. Conclusions

In this study we use a P3-eliciting oddball paradigm to characterise attentional capture by incorrect spellings of phonologically relevant material using pseudohomophones.

Consistent with our previous results with dyslexic adults, we failed to see a deficit in rapid sublexical decoding (Savill and Thierry, 2011a). In addition, we observed (1) a failure in attentional engagement with phonologically-relevant stimuli in dyslexic

² The alternative of testing reading-matched controls is constrained by ERP modulations being markedly affected by age and cognitive ability (see Picton et al., 2000), and therefore using controls that differ in these respects may introduce several undesirable interpretational confounds.

readers, possibly underpinned by temporoparietal junction dysfunction; (2) early intact phonological access in a context of spontaneous, uncued word recognition; (3) correlations between attentional engagement and behavioural measures of reading and spelling; and (4) specificity of these effects to the case of word-like stimuli. With greater understanding of the spatiotemporal dynamics of phonological activation in reading and its interactions with orthographic processing and attention, further studies will help to unravel the determinants of reading performance in developmental dyslexia and hopefully pave the way to efficient remediation strategies.

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Figure captions

Figure 1. Experimental design: Example of 8 consecutive trials in the word task (left) and the shape task (right). Response targets were animal words in the word task and single-coloured squares in the shape task.

Figure 2. (A) Group grand-averages over occipitoparietal electrodes showing similar posterior P2 modulations in the word task. Linear derivation of electrodes P3, P4, Pz, O1, O2 and Oz; (B) Group mean amplitudes for each experimental condition averaged across posterior P2 electrodes between 210 and 240 ms post stimulus onset. Connecting lines depict significant pairwise comparisons at $p < .05$ (dotted connector, $p = .053$). TARG = Target words; PSHT = Pseudohomophones of target words; PSHF = Pseudohomophones of filler words; FILL = Nontarget filler words.

Figure 3. Group grand averages over frontocentral electrodes showing P3a modulations in the word and shape tasks. Linear derivation of electrodes FC3, FC4, FCz, C3, C4, and Cz. For word task ERPs, TARG = Target words; PSHT = Pseudohomophones of target words; PSHF = Pseudohomophone of filler words; FILL = Nontarget filler words; for shape task ERPs the respective shape equivalents of each condition are shown.

Figure 4. Group grand averages over centroparietal electrodes showing P3b modulations in the word and shape tasks. Linear derivation of electrodes CP3, CP4, CPz, P3, P4, and Pz. For word task ERPs, TARG = Target words; PSHT = Pseudohomophones of target words; PSHF = Pseudohomophone of filler words; FILL = Nontarget filler words; for

shape task ERPs the respective shape equivalents of each condition are shown.

Table 1. *Literacy and cognitive measures*

Measure	Control (n=14)		Dyslexic (n=14)		<i>t</i> (26)
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age (years)	23.14	4.50	22.57	5.35	0.31
Reading (untimed; WRAT ^a)	114.79	4.17	101.50	8.36	5.32***
One Minute Reading (DAST)	118.57	12.50	91.86	18.40	4.49***
Spelling (untimed; WRAT ^a)	108.79	7.86	94.21	10.28	4.21***
Two Minute Spelling (DAST)	36.71	2.16	28.64	4.73	5.80***
Nonsense Passage Reading (DAST)	95.79	2.75	79.07	14.13	4.35**
Phon. Segmentation (max=12; DAST)	11.21	0.80	10.14	1.70	2.13*
Pseudoword Reading (WIAT ^a)	110.29	5.58	92.29	12.56	4.90***
Rapid Naming (s; DAST)	24.75	5.09	29.91	7.47	-2.13*
Spoonerisms (max=3; DAST)	2.93	0.27	1.93	1.21	3.03**
Digit Span (WAIS ^b)	11.00	3.53	9.14	2.60	1.59
Matrix Reasoning (WAIS ^b)	14.43	1.51	13.93	2.02	0.74

Note. Standardised scores are reported where available: ^a Subtest's standard score; ^b WAIS subtest's age-scaled score. * $p < .05$; ** $p < .01$; *** $p < .001$; based on corrections for unequal variances, where necessary.

Table 2. *The average number of trials used in individual ERPs per group*

	Word Task		Shape Task	
	Control	Dyslexic	Control	Dyslexic
Target word (TARG) / shape (TARG-S) trials	42 (4.63)	38 (4.70)	43 (7.36)	43 (5.57)
Target-like pseudohomophone (PSHT) / shape (PSHT-S) trials	47 (4.29)	46 (2.64)	43 (8.67)	45 (6.11)
Nontarget-like pseudohomophone (PSHF) / shape (PSHF-S) trials	48 (4.69)	46 (3.92)	43 (8.61)	45 (5.71)
Nontarget filler words (FILL) / shape (FILL-S) trials	326 (37.19)	322 (16.74)	301 (62.82)	312 (41.90)

Note. Mean number of trials shown with standard deviation in parentheses.

Table 3. *Correlations between the standardised language measures and amplitude modulations of the posterior P2, P3a and P3b peaks.*

	TARG – PSHT			TARG-S – PSHT-S		
	Orthographic discrimination			Unicolour discrimination		
	P2 (post.)	P3a	P3b	P2 (post.)	P3a	P3b
WRAT reading	-.156	.038	-.153	-.033	-.022	.275
WRAT spelling	-.131	-.146	-.275	-.148	-.002	.082
Two min. spelling	-.248	-.024	-.129	-.264	-.324	-.004
One min. reading	-.019	-.065	-.126	-.200	-.225	.001
Pseudoword reading	-.077	-.096	-.299	.120	.042	.268
Nonsense Passage	.141	.086	-.053	-.172	-.063	.033
Rapid Naming	.081	.234	.308	.291	-.293	.231
Digit span	.426*	.033	.055	.239	-.175	.206
Matrix reasoning	.221	.400*	.365^a	.173	-.250	.246

	PSHT – PSHF			PSHT-S – PSHF-S		
	Phonological relevance			Relevance detection		
	P2 (post.)	P3a	P3b	P2 (post.)	P3a	P3b
WRAT reading	.131	.255	.179	-.142	-.264	.304
WRAT spelling	.434*	.384*	.418*	-.045	.016	.113
Two min. spelling	-.008	.339	.276	.415*	.143	.230
One min. reading	.047	.477*	.341	.234	.140	.202
Pseudoword reading	.214	.371^a	.268	-.368^a	.260	.230
Nonsense Passage	.126	.407*	.339	.028	.257	.335
Rapid Naming	-.067	-.538**	-.357^a	-.112	-.173	-.154
Digit span	-.111	.179	.129	-.351	.329	.149
Matrix reasoning	.009	-.135	-.094	.064	.013	.048

Note. Pearson r values reported; Significant and trending correlations in bold * $p < .05$; ** $p < .01$; ^a $p < .07$ (two-tailed). Note that inter-correlations between individual ERP amplitude peaks and between individual language measures, which by their nature are strongly associated, are not reported.

Figure 1
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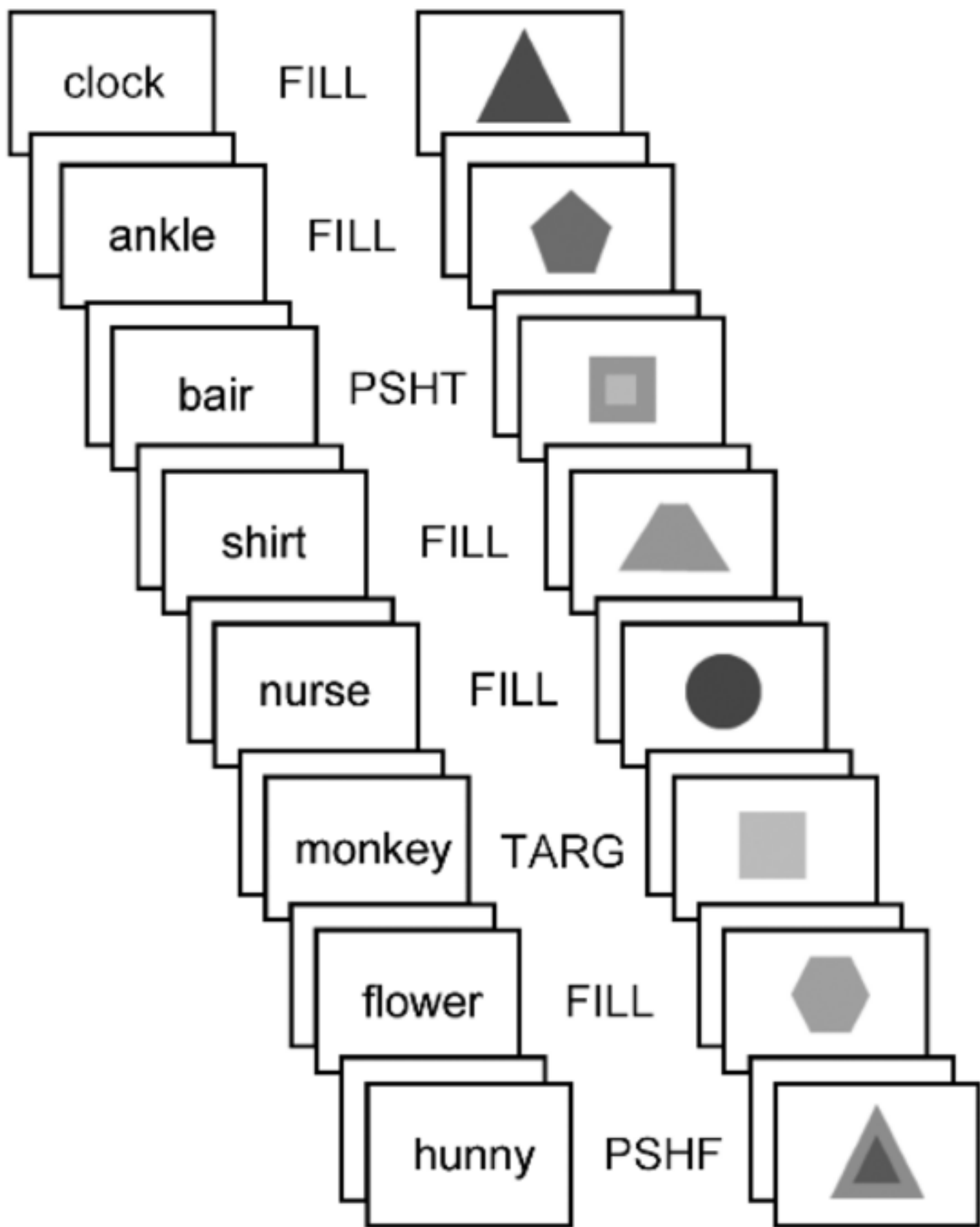


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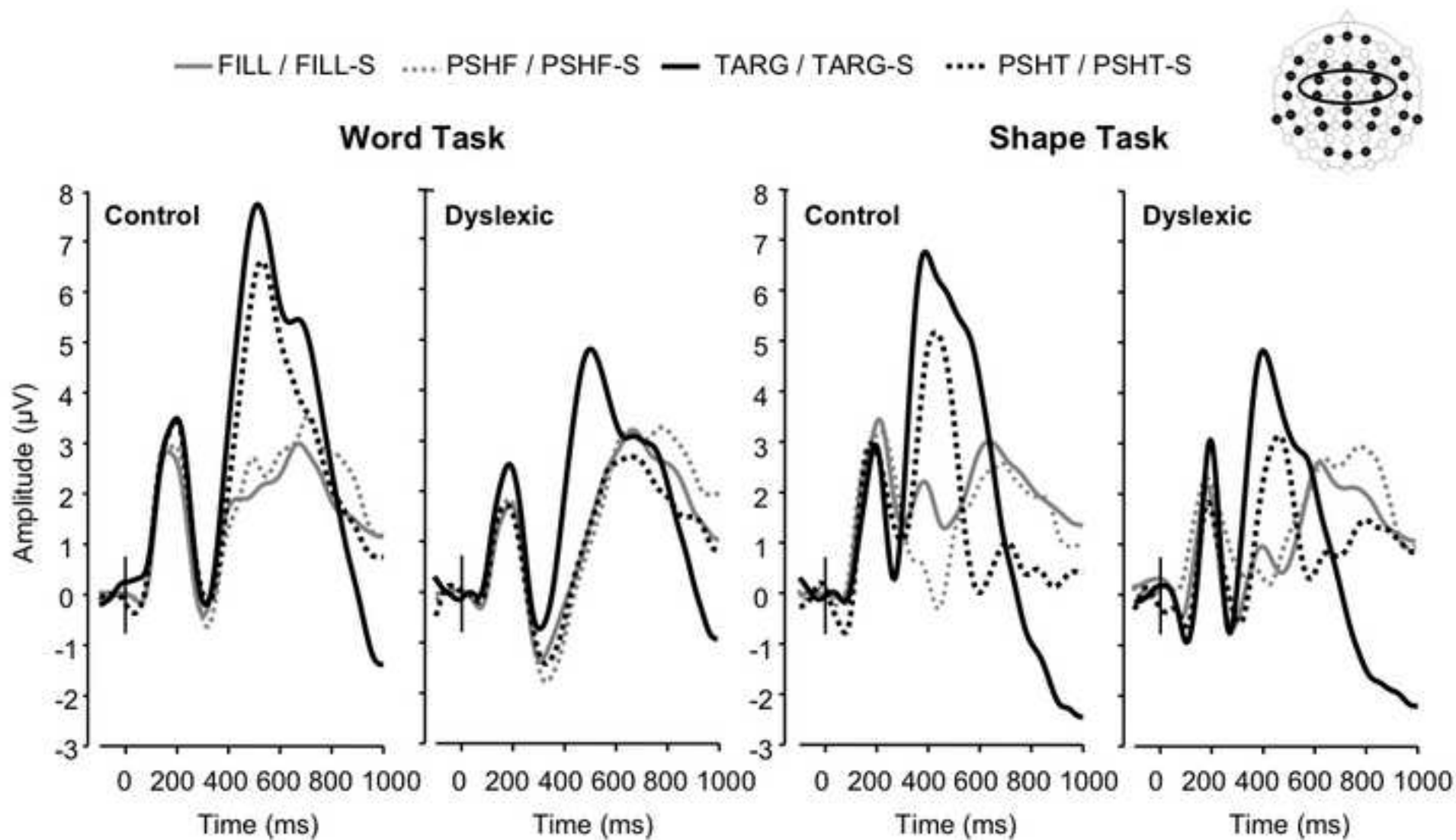


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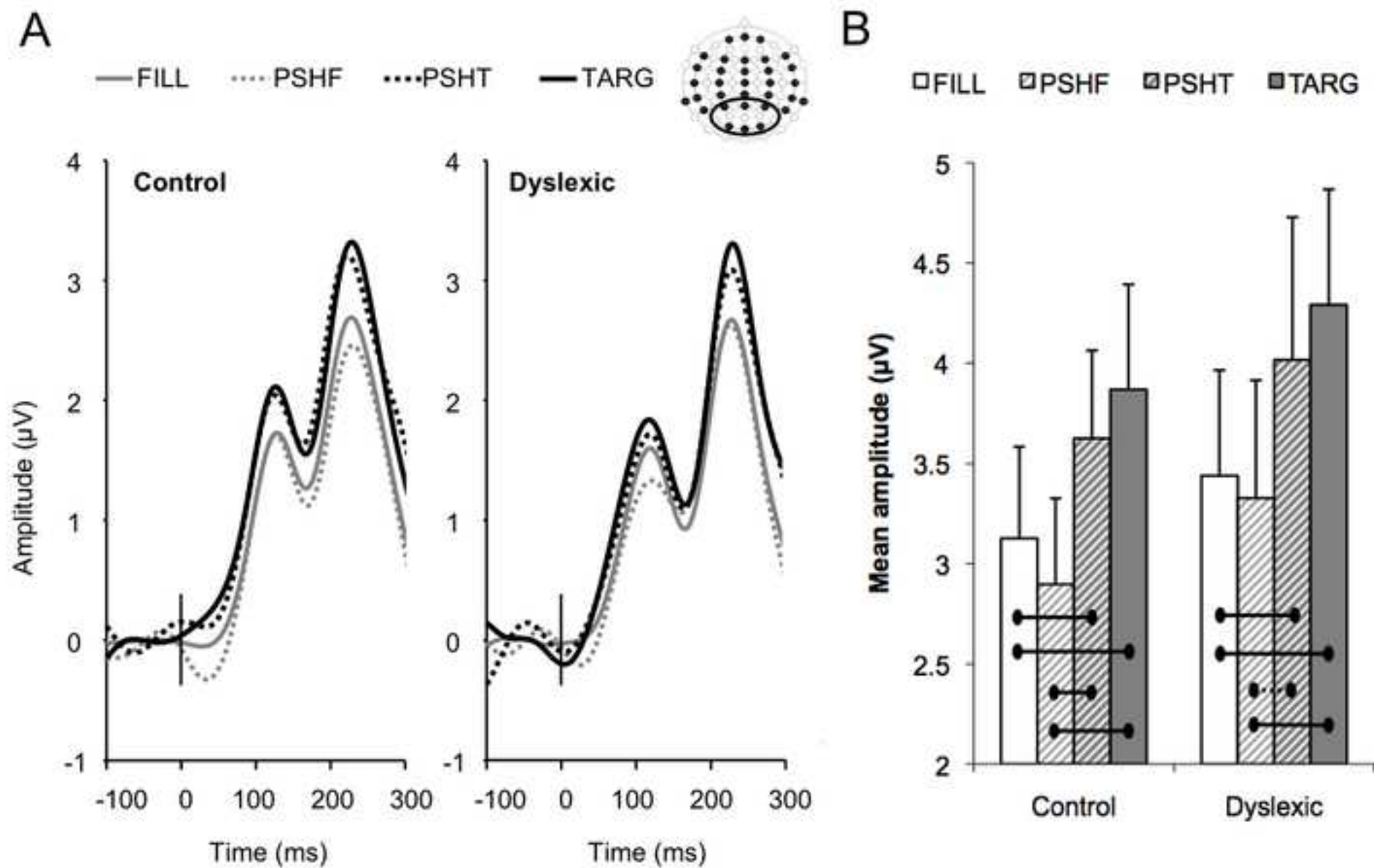


Figure 4
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