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Reading for sound with dyslexia: Evidence for early orthographic and late phonological integration deficits

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Abstract

Deteriorated phonological representations are widely assumed to be the underlying cause of reading difficulties in developmental dyslexia, however existing evidence also implicates degraded orthographic processing. Here, we used event-related potentials whilst dyslexic and control adults performed a pseudoword-word priming task requiring deep phonological analysis to examine phonological and orthographic priming, respectively. Pseudowords were manipulated to be homophonic or non-homophonic to a target word and more or less orthographically similar. Since previous ERP research with normal readers has established phonologically driven differences as early as 250 ms from word presentation, degraded phonological representations were expected to reveal reduced phonological priming in dyslexic readers from 250 ms after target word onset. However, phonological priming main effects in both the N2 and P3 ranges were indistinguishable in amplitude between groups. Critically, we found group differences in the N1 range, such that orthographic modulations observed in controls were absent in the dyslexic group. Furthermore, early group differences in phonological priming transpired as interactions with orthographic priming (in P2, N2 and P3 ranges). A group difference in phonological priming did not emerge until the P600 range, in which the dyslexic group showed significantly attenuated priming. As the P600 is classically associated with online monitoring and reanalysis, this pattern of results suggest that during deliberate phonological processing, the phonological deficit in reading may relate more to inefficient monitoring rather than deficient detection. Meanwhile, early differences in perceptual processing of phonological information may be driven by the strength of engagement with orthographic information.

1. Introduction

Developmental dyslexia is a disorder found in both children and adults characterised by literacy abilities below those expected given their general abilities and adequate motivation. The primary cause of difficulty in developmental dyslexia (henceforth dyslexia) is disputed (see Ramus, 2003), and probably multi-factorial (Menghini et al., 2010; Pennington, 2006) however a dysfunction of phonological processing is widely thought to be at the core of the deficit (Lyon et al., 2003; Snowling, 2000). A persistent hypothesis is that dyslexic individuals have weak and/or coarsely coded phonological representations (Adlard and Hazan, 1998; Boada and Pennington, 2006; Brady, 1997; Elbro, 1996; Goswami, 2000; Hulme and Snowling, 1992; Metsala, 1997; Morais, 2003; Snowling, 2000; Swan and Goswami, 1997). These are suggested to impact reading by interfering with automatic grapheme-phonemic conversion required for skilled reading (Morais, 2003; Morais and Kolinsky, 1994).

However, a convergence of recent research has also indicated that orthographic processing may be compromised in dyslexia and contribute to difficulties with reading (e.g., Bosse et al., 2007; Maurer et al., 2007; Vidyasagar and Pammer, 2010). Vidyasagar and Pammer (2010), for example, suggest that defects in the dorsal stream of the visual system may be the core deficit in dyslexia. This hypothesis is built on numerous studies showing dyslexic group performance to be weaker for behavioural

indices of visual magnocellular function (see Laycock and Crewther, 2008; Schulte-Körne and Bruder, 2010, for recent reviews). In their perspective, focal visuo-spatial attention weaknesses affect scanning of orthographic strings resulting in poor orthographic inputs, which in turn affect grapheme-phoneme mapping. A similar perspective (Hari and Renvall, 2001; Facoetti, et al., 2006, 2008), derived primarily from observations of a prolonged attentional blink and slower spatial cued-detection in dyslexia, hypothesises that sluggish automatic engaging and disengaging of attention impairs the visual selection of graphemes, and subsequent decoding. In support of the association between attentional shifting and decoding ability, orienting performance has been found to significantly correlate with nonword reading (Facoetti et al., 2006, 2008, 2009).

Further implications of reduced orthographic sensitivity in dyslexia come from Valdois and colleagues (e.g., Bosse et al., 2007; Valdois et al., 2004; Dubois et al., 2007), who separately describe a visual attentional span (VAS) deficit. VAS refers to the number of items in a visual string that can be processed simultaneously and has been shown to be smaller in subsets of dyslexic participants and to impact reading independently from phonological problems. It is suggested that a reduced visuo-attentional window would impair whole word processing and thus particularly affect irregular word reading (Bosse and Valdois, 2009).

These hypotheses have differing implications for the point(s) at which dyslexic readers' visual word recognition ought to be affected. Recording event-related potentials (ERP) can offer insight here. ERP investigations have had a positive contribution on our understanding of the time course and stages of normal visual word recognition: from

initial processing of visual input, through orthographic analysis, phonological mapping and subsequent working memory integration (see Dien, 2009; Grainger and Holcomb, 2009). As such, ERPs present an ideal tool to enable description of the temporal course of the deficit(s) in dyslexia and clarify the case for early degraded orthographic inputs and/or phonological representations, or difficulties relating to later integrative processing.

With respect to phonological manipulations, ERP studies using visual word stimuli tend to report differences between dyslexic and control readers in the N400 range; most of which demonstrate weaker amplitude modulations to rhyme match-mismatch (Ackerman et al., 1994; McPherson et al., 1998; Rüsseler et al., 2007). However, modulations of the N400 wave are rather late to index impaired sensitivity to phonology, and are more likely to relate to the deep processing and decision-making related to integration of the phonological stimuli, rather than a marker of sublexical processes (e.g., Bentin et al., 1999; Brown and Hagoort, 1993; Chwilla et al., 1995; Connolly and Phillips, 1994). ERP studies with normal readers have shown that phonological manipulations can reliably modulate the ERP wave from 250 ms after stimulus onset in a range of tasks (masked priming: Ashby and Martin, 2008; Holcomb and Grainger, 2006; Grainger et al., 2006; rhyme and lexical decision: Bentin et al., 1999; rhyming decision: Kramer and Donchin, 1987; sentence reading: Savill et al., 2011; phoneme decision: Proverbio et al., 2004). Furthermore, amplification of the N2 peak elicited by phonological mismatch is well established: An effect referred to as the Phonological Mismatch/Mapping Negativity (PMN), typically observed when the expected final word of a sentence is replaced with a phonologically dissimilar, unexpected stimulus, has been shown with auditory stimuli (e.g., Connolly and Phillips, 1994; D'Arcy et al., 2004; Diaz

and Swaab, 2007; Newman and Connolly, 2009; Newman et al., 2003), and similar effects have been shown in visual contexts (Connolly et al., 1995; Newman and Connolly, 2004; Savill et al., 2011). The lack of studies reporting differences in phonological effects between dyslexic and normal readers in this earlier time range, within the context of reported findings within the later N400 range, seems to favour integration/working memory accounts of phonological dysfunction in a reading context, rather than degraded phonological sensitivity.

The ERP literature on visual word processing in developmental dyslexia has, however, shown early discriminatory ERP profiles between dyslexic and normal readers. These differences have been found during stages related to processing visual/orthographic input (see Coch and Mitra, 2010; Dien, 2009; Grainger and Holcomb, 2009; Hauk et al., 2006; Kast et al., 2010; Parviainen et al. 2006) prior to phonological analysis, within 150 ms of stimulus onset (Maurer et al., 2007; Helenius et al., 1999; Taroyan and Nicolson, 2009; Wimmer et al., 2002). These early group differences, found in naming, one-back and lexical decision tasks, have been shown to be letter-string specific (Helenius et al., 1999; Maurer et al., 2007), and have been observed at the word form level, e.g., absent left-lateralised P1 amplitude differences between words and pseudowords observed in dyslexic readers (Taroyan and Nicolson, 2009). Such findings of reduced orthographic activation are comparable with the varied literature implicating visual/visuo-attentional factors underlying dyslexic word processing difficulties, which share the implication of a weaker orthographic percept (Bosse et al., 2007; Facoetti et al., 2008; Hawelka et al., 2006; Hawelka and Wimmer, 2005; Jones et al., 2008; Pammer and Vidyasagar, 2005; Salmelin et al., 1996; Vidyasagar, 2004; Vidyasagar and Pammer

2010). Given the suggestion that early reduced attention or sensitivity to orthographic and/or whole word perceptual differences may interfere with later phonemic mapping (e.g., Cestnick and Coltheart, 1999; Facoetti et al., 2006, 2008; Vidyasagar and Pammer, 2010), and provide the basis of reading difficulties observed in dyslexia, it would be instructive to consider the relative contribution of orthographic and phonologic effects in phonological analysis during reading in dyslexia.

Our study was designed to disentangle sensitivity to phonological and orthographic information in dyslexic and normal readers using ERPs. The cognitive chronometry afforded by ERPs allowed us to distinguish detection and decoding from attentional and working memory processes. We used a 2 x 2 design, similar to that of Grainger et al. (2006), except we did not use masked priming. We created pseudoword-word pairs controlled for phonological and orthographic similarity in order to produce four experimental conditions, e.g., in the case of the word *horse* as target, primes could be *horce* (P+O+), *hauce* (P+O-), *horle* (P-O+) and *hiele* (P-O-, where 'P' denotes homophony and 'O' denotes orthographic neighbourhood with the paired word). To examine dyslexic readers' sensitivity to phonological manipulations, participants were asked to decide whether the presented pseudoword prime and the following target word sounded the same. This design allowed examination of the participants' overt phonological processing abilities in time, and potential interactions with orthographic processing.

We hypothesised that if poor phonological task performance is due to weak phonological sensitivity in dyslexia, dyslexic participants ought to show reduced phonological priming from the earliest point at which the ERP indexes phonological

effects. More specifically, we would expect a relatively larger N2 to P+ stimuli (that is, less N2 attenuation for weaker phonological expectations) and smaller differences between P+ and P- amplitudes than in controls. In contrast, if phonological sensitivity in reading is intact, phonological priming effects at stages of stimulus processing and discrimination (i.e., in the P2/N2 and P3 ranges) should be of similar magnitude to those found in control readers. In this situation, it is possible that processes of integration or reanalysis may instead be the source of error in phonological tasks, in which case ERPs should differ in a later time-window, i.e., that of the N400 or beyond.

Regarding orthographic effects, we reasoned that if sensitivity to orthographic information at the whole string level is decreased in dyslexia, we should observe reduced modulation by orthographic similarity from the N1 onwards in the dyslexic group. Crucially, we were interested to see the extent to which orthographic and phonological effects would interact. If, for example, orthographic sensitivity had a greater impact on phonological analysis in dyslexic than control readers, we would expect significant interactions of group with orthographic and phonological priming from the P2/N2 range onwards.

2. Results

2.1. Behavioural results

Correct response reaction times were significantly faster to homophonic than non-homophonic pairs, $F(1, 30) = 50.86$, $p < .001$. Orthographic neighbouring stimuli also resulted in faster reaction times, $F(1, 30) = 5.51$, $p < .05$, but reduced accuracy, $F(1, 30) = 6.94$, $p < .05$. Furthermore, phonological and orthographic similarity significantly interacted for both accuracy, $F(1, 30) = 111.52$, $p < .001$, and reaction time, $F(1, 30) =$

96.05, $p < .001$. This was due to decreased accuracy and increased reaction times for 'mixed' primes (i.e., P+O- and P-O+ stimuli) as compared to non-mixed ones. A trend for an interaction between group, phonological priming and orthographic priming interaction for accuracy, $p = .09$, related to the dyslexic group showing a greater relative deficit in accuracy to the mixed cues, compared to controls (see Figure 1). A significant main effect of group on reaction times indicated that responses were generally slower from the dyslexic group, $F(1, 30) = 4.76$, $p < .05$.

INSERT FIGURE 1 ABOUT HERE

2.2. Event-related potential results

2.2.1. P1

No significant effects were observed in the P1 range.

2.2.2. N1

There was a significant interaction between orthographic similarity and group on N1 mean amplitudes, $F(1, 30) = 5.18$, $p < .05$, driven by significant amplification of N1 to orthographically neighbouring (O+) stimuli in the control group only. N1 peak latencies were significantly delayed overall in the dyslexic group, $F(1, 30) = 5.99$, $p < .05$ (Peak latency: control group $M= 159$ ms; dyslexic group $M= 166$ ms)[1]. A non-significant trend for orthographic neighbour modulation of N1 latency was also observed, with O-primed words tending to elicit an earlier N1 peak ($p = .07$).

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[1] To investigate possible effects of having different peak latencies in the groups (which was the case for N1), we ran a second analysis with slightly different intervals for each of the two groups. This analysis yielded the same qualitative result as the analysis using common intervals and is not reported here.

INSERT FIGURE 2 ABOUT HERE

2.2.3. P2

No main effects were observed with respect to P2 mean amplitudes. However an interaction of phonological similarity, orthographic similarity and group significantly affected P2 amplitude, $F(1, 30) = 5.58$, $p < .05$. This interaction was driven the dyslexic group showing a significantly amplified response to P-O- primed words compared to P-O+.

A main effect of phonological similarity indicated that homophonic stimulus pairs significantly delayed P2 latencies, $F(1, 30) = 3.41$, $p < .05$. Phonological similarity also interacted with group, $F(1, 30) = 4.32$, $p < .05$, which related to only the control group showing a later peak for P+ stimuli.

2.2.4. N2

A main effect of phonological similarity showed that non-homophonic words significantly amplified the N2 peak relative to homophonic words, $F(1, 30) = 36.20$, $p < .001$. No interaction of group with phonological similarity was observed. However, mean amplitudes were modulated by a three-way interaction of group, phonological similarity and orthographic similarity, $F(1, 30) = 8.45$, $p < .01$. The source of this interaction related to the relative attenuation of P-O+ and P-O- primed words: whilst homophonic words (P+O+ and P+O- primed) elicited a significantly attenuated response relative to non-homophonic words (P-O+ and P-O- primed), P-O+ words were also significantly

attenuated relative to P-O- in the control group; the dyslexic group, on the other hand, showed similar amplification of N2 to non-homophonic words (P-O+ and P-O- primed), however they only significantly differed from P+O+ primed words, and not P+O- (see Figure 3).

N2 peak latencies were significantly shorter to homophonic words, $F(1, 30) = 12.80$, $p < .001$, and to orthographically neighbouring words, $F(1, 30) = 15.47$, $p < .001$. Furthermore, a trend for an interaction between group, phonological similarity and orthographic similarity, $p = .09$, indicated that peak latencies were significantly longer to P-O- primed words compared to all other priming conditions in the dyslexic group, whilst in the control group P-O- and P+O- peak latencies were not significantly different.

INSERT FIGURE 3 ABOUT HERE

2.2.5. P3

Phonological similarity, $F(1, 30) = 38.01$, $p < .001$, and orthographic similarity, $F(1, 30) = 4.26$, $p < .05$, significantly amplified P3 target word peaks. A three-way interaction of group, phonological similarity and orthographic similarity, $F(1, 30) = 8.17$, $p < .01$, was due to differences in group responses to P-O+ primed words: Whilst P+O+ and P+O- were significantly amplified relative to P-O+ and P-O- priming conditions in both groups, the P-O+ primed targets were significantly amplified compared to P-O- in controls only. P3 peak latencies were significantly affected by both phonological similarity, $F(1, 30) = 8.94$, $p < .01$, for which P+ primed stimuli elicited later peaks, and by an interactive effect of phonological and orthographic similarity, $F(1, 30) = 4.79$, $p <$

.05, which related to a significantly shorter P3 peak latency for P-O- primed stimuli compared to the homophonic stimuli (P+O+ and P+O-).

INSERT FIGURE 4 ABOUT HERE

2.2.6. P600

Non-homophonic, $F(1, 30) = 15.00$, $p < .001$ and orthographic non-neighbour stimulus pairs, $F(1, 30) = 14.38$, $p < .001$, elicited significantly larger P600 amplitudes than homophonic and orthographically neighbouring pairs, respectively. Participant group was found to significantly interact with both phonological similarity, $F(1, 30) = 6.05$, $p < .05$, which showed that phonological priming modulations of P600 amplitude were significant for the control group only; and with orthographic similarity, $F(1, 30) = 4.73$, $p < .05$, for which significant priming effects were also only in the control group. A further three-way interaction of group, phonological and orthographic similarity, $F(1, 30) = 4.65$, $p < .05$, indicated that the significant differences in P600 amplitude present in the controls (P+O+ significantly attenuated compared to all, and significant differences between P+O- and the P- stimuli) were absent in the dyslexic group, for whom only amplitude differences between the P-O+ and P-O- stimuli reached significance. P600 latencies were only modulated by a main effect of phonological similarity, $F(1, 30) = 11.64$, $p < .05$.

INSERT FIGURES 5 AND 6 ABOUT HERE

3. Discussion

This study aimed at dissociating phonological and orthographic priming effects during a phonological awareness task performed on letter strings by dyslexic participants and matched controls. Our main findings were (a) main effects of phonological and orthographic priming on reaction times in both the groups; (b) N1 increase by orthographic priming in the control group only; (c) a main effect of phonological priming in the N2 and P3 range in both the participant groups; (d) a set of three way interactions with group spanning the P2, N2 and P3 peaks; and (e) a reduced P600 modulation by phonological priming in the dyslexic group only. These will be discussed in turn.

3.1. Behavioural insights

The dyslexic group, as expected, performed the task significantly slower overall. Beyond this, no significant group interactions emerged in behavioural data. However, priming significantly improved performance overall and showed the expected interaction between phonological and orthographic priming such that orthographic neighbourhood facilitated recognition of phonologically primed stimuli and degraded recognition of non-matched stimuli, and vice versa for less orthographically informative cues across group. Importantly, a trend for a three-way interaction with group indicated that the significantly deleterious effect of mixed priming on accuracy was larger in the dyslexic group. In the context of a priming manipulation in which differences between all conditions were subtle and the task was designed to avoid high error, it is perhaps not surprising that reaction time or accuracy measures in isolation did not distinguish between the groups.

3.2. The use of orthographic cues: reviewing early ERP group differences

The earliest condition modulation of the ERP was found for the N1 peak, where targets primed by orthographic neighbours elicited significantly amplified peaks in the control group only. This is consistent with previously reported effects in the N1 range in controls (Hauk et al., 2009) and other orthographic variables have been shown to modulate the N1, such as written length (Assadollahi and Pulvermüller, 2003; Hauk et al., 2009), frequency (Assadollahi and Pulvermüller, 2003; Hauk and Pulvermüller, 2004; Hauk et al., 2009; Sereno et al., 1998, 2003) and lexical status, e.g., words versus consonant strings (Coch and Mitra, 2010; Compton et al., 1991; Hauk et al., 2006; Maurer et al., 2005; McCandliss et al., 1997; Sauseng et al., 2004). The absence of an N1 orthographic priming effect in our dyslexic group, along with a significantly latency delay, indicates that orthographic cues were processed less efficiently/slower in dyslexic participants. This would be consistent with previous reports of attenuated P1 or N1 to orthographic stimuli (Helenius et al., 1999; Kast et al., 2010; Maurer et al., 2007) and reduced activation in left occipitotemporal areas involved in orthographic identification and integration, as shown by functional brain imaging studies (Blau et al., 2010; Brunswick et al., 1999; Cao et al., 2006; Kronbichler et al., 2006; Liu et al., 2010; McCrory et al., 2005; Richlan et al., 2010; Salmelin et al., 1996; Van der Mark et al., 2009, 2010; Wimmer et al., 2010).

With respect to existing literature, perceptual difficulties at the word form level have been proposed to impact reading in different ways: Valdois and colleagues (e.g., Bosse et al., 2007; Lassus-Sangosse et al., 2008; Peyrin et al., 2010; Valdois et al., 2004), for instance, have suggested that subsets of dyslexic readers have a smaller attentional window impacting the scanning of letter strings, which may affect subsequent grapheme

perception and integration, and contribute to downstream phonological decoding difficulties. Poor left-to-right scanning has also been suggested as the route to impaired reading in dyslexia due to deficient processing along the dorsal visual pathway, which is suggested to degrade orthographic input and impact awareness of grapheme-phoneme correspondence (Cestnick and Coltheart, 1999; Vidyasagar and Pammer, 2010). Whilst our study was primarily aimed at addressing the interaction of orthographic and phonological information, rather than orthographic/visual word form perception per se, our data indicates that orthographic information is not accessed as readily in compensated dyslexic adults as it is in normal readers.

Further, we found significant three-way interactions of group with phonological and orthographic similarity in the P2, N2 and P3 ranges: (a) A three-way modulation affecting P2 amplitude showed a significantly amplified response in the P-O- relative to P-O+ condition in the dyslexic group only; (b) Differences in the N2 range showed the following ordering of conditions in the control group: P+O- weaker than P-O+ and P-O- and P-O+ weaker P-O-; and (c) An interaction in the P3 range induced by responses in P-O+ condition being greater than in the P-O- condition in the control group only.

Overall, three-way interactions between group, phonological, and orthographic priming may have been expected if we assume that dyslexic readers have degraded phonological representations. However, we observed significant main effects of phonological priming in the absence of a group interaction in ERP amplitudes from the N2 range through to the P3 range. We interpret this as a sign that early phonological access in our dyslexic participants may not have been functionally deficient. It must be

kept in mind that this is not a null effect since phonological priming was significant in both of our groups.

By contrast, the three-way interactions listed above seem to have arisen primarily from weaker and/or possibly qualitatively different effects of orthographic similarity in the dyslexic group. Starting with the P2, which was the earliest peak where effects of mismatch between orthographic and phonological representations might have been expected (Bles et al., 2007; Braun et al., 2009; Hsu et al., 2009; Potts, 2004), the dyslexic group showed an amplified response in the P-O- condition, perhaps because this is the point at which orthographic processing kicked in for the dyslexic participants, whereas in the case of the control group, orthographic similarity of the target word may have been resolved as early as the N1 window (cf. N1 effect which was both delayed and reduced in dyslexic participants). In other words, in the case of dyslexic participants, orientation to orthographic *dissimilarity* of the stimuli in a pair would have helped phonological discrimination but not helped the detection of homophony (i.e., no differences between P+O+ and P+O-).

The following N2 interaction may relate to reduced orthographic cueing in the dyslexic group (since P-O+ elicited weaker N2 amplitude than P-O- in the controls) and perhaps a slight phonological processing weakness since P+O- was not significantly different from the P- conditions.

The third interaction, in the P3 range, continued to show reduced orthographic similarity effects in the dyslexic group: The interaction was due to the lack of difference between P-O+ and P-O- stimulus pairs in the dyslexic group as was the case in the N2 range. In other words, orthographic similarity may have failed to capture dyslexic

participants' attention, perhaps because their focusing on phonological form may have limited distraction by orthographic information.

Together, these results show that stimuli were essentially distinguished on the basis of their phonological status. Moreover, dyslexic participants were less influenced by orthographic similarity than controls over and above phonological priming, with orthographic priming differences evident from the N1 to the P3 through the N2 range. A possible explanation is that the dyslexic group may have had weaker orthographic input at the whole-stimulus level (e.g., see literature on visuo-attention span, Valdois et al., 2004; and dorsal visual pathway hypotheses, Vidyasagar and Pammer, 2010) or managed to focus more exclusively on phonological similarity. Future studies in which phonological judgments are explicitly emphasized by the task ought to clarify whether group differences in orthographic processing are driven by reduced access of orthographic information in dyslexic readers or emerge from relative streamlining of attention to the phonological level when participants are required to focus on phonology.

3.3. P600: Late differences in phonological processing

The dyslexic group showed a significantly reduced main effect of phonological similarity within the P600 range. P600 mean amplitude was significantly attenuated in the dyslexic group for both phonological and orthographic as compared to the control group. Across groups, the strongest increase in P600 amplitude was found for the least related stimuli (P-O-), with progressively reduced amplitudes for 'P-O+' and 'P+O-', and 'P+O+' stimuli. Typically, the P600 component is triggered by linguistic incongruence that is not based on semantic integration (Kolk and Chwilla, 2007) and has been proposed

to index a process of reanalysis (Van de Meerendonk et al., 2010; Vissers et al., 2008). The reduced P600 differentiation between conditions suggests that stimulus relatedness may have been less salient and/or subject to limited reanalysis at this late reprocessing stage in the dyslexic group. Whatever difficulty or difference is driving the attenuation elicited by the dyslexic group in this task, it is unlikely to relate to sensitivity to phonological manipulations within the orthographically controlled stimuli, since no marked differences were found in the N2 and P3 ranges.

3.4. Is a reprocessing dysfunction exacerbating performance deficits in overtly phonological tasks in dyslexia?

The significant P600 attenuation possibly indexes a deficient strategic response during an overt verbal task. Recent accounts of the phonological deficit in dyslexia have focussed on working memory demand rather than perceptual deficits relating to weak phonological representations (Banai and Ahissar, 2006; Ramus and Szenkovits, 2008). For instance, a series of experiments by Ramus and colleagues targeting predictive effects of weak or fuzzy phonological representations using speech-based auditory tasks repeatedly failed to find significant differences between dyslexic and control listeners (see Ramus and Szenkovits, 2008). In the same vein, Banai and Ahissar (2006) showed that dyslexic participants only manifest phonological deficits while performing complex ordinal or parametric judgments of auditory phonological stimuli, but were not hindered in judgements of the same stimuli when they required simple (i.e., same-different) discriminations. Together the latter two studies suggest that task demand, e.g., the level of short-term memory involvement and time constraints, determines access to phonological representations and subsequent observed behavioural deficits in

phonological tasks. Our task required maintenance of the phonological form of consecutively presented pairs of stimuli, which arguably placed similar processing demands on the participants. Thus it may be that our observed P600 effects are the result of depleted working memory resources precluding phonological integration and reappraisal. Studies that have previously reported differences in P600 amplitude in dyslexic populations have attributed similar late ERP differences to conscious and strategic rather than automatic linguistic processes. Rispens and colleagues (2004, 2006), for instance, demonstrated that Dutch dyslexic participants showed no significant P600 modulation to auditorily presented sentences containing plural noun phrase structure violations. As an earlier measure of automatic syntactic parsing – an early left anterior negativity (ELAN), found approximately 200ms after violation – was unaffected, the authors suggested that their P600 differences indicated dyslexic deficits in more controlled and strategic linguistic processes involved in syntactic revision (Rispens, 2004), which may reflect some form of reprocessing failure. Within the visual word domain, studies considering the electrophysiological basis of word learning in dyslexia (Schulte-Körne et al., 2004) and recognition memory for visually-presented words (Rüsseler et al., 2003) have also shown intact early word recognition and discriminative ERP effects, with only diminished responses at the stage of conscious recollection/retrieval in the P600 range. Schulte-Körne et al. (2004), for example, studied dyslexic and control children's recognition of previously learned four-letter pseudowords and complex graphic symbols using ERPs. In the context of accurate behavioural performance and normal P300 effects, they found that the recognition ERP correlate – the P600 – was significantly attenuated specifically for the pseudowords, compared to

graphic symbols and control group responses. Whilst the authors related their finding to an impairment in visual recognition due to the limited phonological demand of the pseudowords; their results could also be interpretable as a strategic processing and/or working memory failure elicited by stimuli requiring phonological analysis in the dyslexic participants, despite intact recognition.

Some form of reduced maintenance and integration of phonological information could also account for findings of existing ERP studies using phonological tasks with visual word stimuli, which typically report reduced N400 modulations from dyslexic readers (Ackerman et al., 1994; McPherson et al., 1998; Rüsseler et al., 2007). This would fit with associations of the N400 with working memory (e.g., Gunter, et al., 2003) and decision-making processes related to stimulus integration (Brown and Hagoort, 1993; Connolly and Phillips, 1994; Chwilla et al., 1995; Holcomb, 1993). Our task did not elicit an N400 response, but instead a P600. This may be due to the differences in task demand between word rhyming judgment, in which such N400 phonological differences have typically been observed, and in this study, a homophone judgment. Kolk and colleagues (Kolk et al., 2003; Kolk and Chwilla, 2007; Van De Meerendonk et al., 2009) suggest that the N400 wave indexes lexical integration of an unexpected linguistic event (e.g., to a rhyme mismatch) but that if the unexpected event is perceptually uncertain (e.g., with complex sentences or, perhaps, brief presentations of unfamiliar pseudowords, as in the case of our homophone judgment task) integration indexed by the N400 will not occur and a veridicality check indexed by the P600 will occur instead. Thus the specificity of our P600 effect needs to be clarified: It is unlikely to be a downstream net result of weaker phonological priming in the dyslexic group, both because (a) earlier

modulations indicated similar magnitudes of phonological priming between groups and (b) the likely outcome of a weaker phonological percept would be uncertainty and thus larger, rather than significantly smaller, P600 amplitudes. If we nonetheless adopted this interpretation our data would indicate that the dyslexic group responded more confidently than the control group, which is highly unlikely. Thus the hypothesis of a performance monitoring/reanalysis deficit is more likely. The question of whether this deficient monitoring is specific to phonological task performance or indicative of a more general trend cannot be determined from this study and will require further investigation.

Converging evidence for a phonological monitoring failure comes from recent ERP studies examining dyslexic error-related negativities – a negative fluctuation typically found 100ms post an erroneous response (Horowitz-Kraus and Breznitz, 2008, 2009). Horowitz-Kraus and Breznitz (2008) reported reduced differentiation between error-related negativities and correct-related negativities from dyslexic readers compared to controls during performance of a lexical decision task, which they suggested could relate to inefficient error monitoring. The P600 effects observed here may be a pre- (or peri-) response correlate of this inefficient monitoring. Unfortunately, it was not possible to run analyses of error-related responses because of the very low rate of errors. However, future studies could address if reduced differentiation of error/correct-related negativities post-response may relate to prior atypical performance monitoring, and furthermore whether these monitoring deficits are specific to performing a phonological task.

3.5. The role of attention in manifestations of the phonological deficit?

An important consideration with our task is that attention was explicitly focused on the phonological relationship between stimuli in a pair. The only ERP difference between groups in phonological priming effects (irrespective of orthographic cues) were observed in response monitoring. It may be that we would have observed early differences in phonological priming effects if attention had not been oriented to phonology and/nor engaged in a phonological task. If this were the case, the pervasive phonological deficit would probably not be due to a significantly reduced ability to perceive phonological manipulations, but rather a relative failure in attentional capture. This would fit with the auditory ERP literature typically showing reduced phonological modulations in oddball tasks in which the oddball is task-irrelevant (Fosker and Thierry, 2004; see Bishop, 2007, for a review of MMN studies) versus normal P3 phonological modulations when participants are asked to attend to the oddball (Fosker and Thierry, 2005; Rüsseler et al., 2002). Deficits switched by attention to phonological information may explain much of the conflicting data regarding phonological processing difficulties in developmental dyslexia: Reduced voluntary orientation to phonological information, possibly exacerbated in the case of reading by reduced orthographic sensitivity and subsequently disrupted graphemic-phonemic mapping on the one hand; and limited processing capacity for deliberate phonological analysis on the other.

3.6. Conclusions

This study provides electrophysiological evidence for early sensitivity to subtle phonological manipulations of visual pseudoword stimuli, but reduced sensitivity to whole form orthographic information during phonological analysis in dyslexic readers. A

failure in stimulus integration and reprocessing, indexed by a significantly less discriminative P600 may account for the weaker performance of dyslexic participant in homophonic judgement. The phonological deficit, in pseudoword reading at least, might thus be better conceived in line with Ramus and Szenkovits' (2008) conclusion regarding the recruitment of controlled, metacognitive processes in phonological analysis. Further research should determine the specificity of reduced orthographic effects in dyslexia and clarify the role of phonology in deliberate and implicit word recognition. Tasks which selectively manipulate the focus on phonological and orthographic information and the degree of attentional demand required should help to clarify the relative perceptual and executive aspects of reading deficits in dyslexia.

4. Experimental procedure

4.1. Participants

Sixteen high-functioning developmental dyslexic adults (mean age 21.63 years; 8 males) and 16 control adults (mean age 21.19 years, 9 males) participated in our experiment that had been approved by Bangor University's Ethics Committee. Data from four control participants had previously been discarded due to technical failure or insufficient number of trials. All participants were undergraduate or postgraduate students at Bangor University and were right-handed, native speakers of English with normal or corrected-to-normal vision, and no self-reported neurological impairment or comorbid difficulties. Dyslexic volunteers were recruited from Bangor University's Dyslexia Unit and through advertisement on the University's Participant Panel; all had a diagnosis of dyslexia from an educational psychologist. The rationale for testing high-

functioning dyslexic adults is that observable deficits that persist into adulthood, even in the context of a normal reading level, can help to identify core deficits common to developmental dyslexia across abilities (see for e.g., Bruck, 1992; Gallagher et al., 1996; Ingvar et al., 2002; Jones et al., 2009; McCrory et al., 2005; Miller-Shaul, 2005; Paulesu et al., 1996; Szenkovits and Ramus, 2003; Wilson and Lesaux, 2001). Furthermore, the advantages of testing university students is that (a) they form a fairly homogenous sample with similar levels of print exposure; (b) it minimizes the likelihood of potential comorbidity, and (c) individuals are more likely to actively engage with attentionally demanding psycholinguistic tasks (see also Szenkovits and Ramus, 2003, for a similar rationale). Performance on a battery of literacy related behavioural measures, taken from the DAST (Nicolson and Fawcett, 1998), WRAT-3 (Jastak and Wilkinson, 1993) WAIS-III (Wechsler, 1997) and WIAT (Wechsler, 2005) confirmed that the individuals in the dyslexic group, although of similar academic ability and showing a reading level within the normal range, were significantly poorer than the control group across measures, with the exception of the WIAT's untimed pseudoword reading task for which differences in accuracy fell just outside of significance. Corresponding results are shown in Table 1.

INSERT TABLE 1 ABOUT HERE

4.2. Stimuli

Word stimuli were 60 four- or five-letter long English words selected from the MRC Psycholinguistic database (Coltheart, 1981). Each word (e.g., "HORSE") was paired with four pseudowords, each belonging to one of four priming conditions: P+O+, in which the prime differed from the target word by only one letter and was a homophone of the target (e.g., "horce"; mean orthographic similarity score, 0.89); P+O-, in which the

prime was homophonic to the target but had reduced orthographic overlap ("hauce"; mean orthographic score, 0.52); P-O+, in which the prime was a non-homophonic pseudoword created by changing the same letter as in the P+O+ condition ("horle"; mean orthographic score, 0.89); and P-O-, in which the prime was not a homophone of the target but had the same orthographic overlap as in the P+O- condition ("hiele", mean orthographic score, 0.52). Orthographic similarity between each prime and target was measured using *Normalized Edit Distance* (NED; see Lambert et al., 1999), for which the minimum number of edits between stimuli (i.e., substitutions, deletions or additions) is divided by the longer string length (in this study prime and targets were the same length). The NED was subtracted from one to produce a similarity rather than dissimilarity score. For instance, "HORSE" and "hauce" are separable by a minimum edit of three: substitution of 'O', 'R' 'and 'S' with 'A', 'U' and 'C': 1 - (3÷5) = 0.4. As we were interested in orthographic similarity between primes and targets, rather than effects of orthographic neighbourhood size of the stimuli, number of neighbours (NN) was controlled across conditions (Mean NN: P+O+ 3.60; P+O- 3.70; P-O+ 3.82; P-O- 3.43). Constrained bigram and trigram frequencies of each prime verified there were no significant differences in orthographic frequency across prime conditions.

Twenty-four further stimulus pairs were created as fillers to prevent a strategy of making a decision before presentation of the second stimulus based on recognition of whether the prime was a pseudohomophone and promote comparison of prime and target. Fillers consisted of six primes from each of the four conditions paired with mismatching word stimuli.

4.3. Procedure

Participants made phonological decisions to pseudoword-word pairs presented visually in sequence. They decided whether the target word sounded the same as the prime. In a given trial participants looked at a fixation cross displayed for 1000 ms, followed by a prime presented in lower case and displayed for 200 ms. The target was separated from the prime by a variable inter-stimulus interval (ISI) of 200, 260, 320, 380, 440 or 500 ms, during which the screen was blank. For a given target, the ISI was constant across conditions and frequency of ISIs was controlled across the experiment. The word target was subsequently presented in upper case for a further 200ms, and was followed by an inter-trial fixed interval of 3000 ms (see Figure 7). Participants responded by pressing one of two designated keyboard keys (yes/no response). For the duration of the task, participants were seated comfortably in a dimly lit, sound-attenuated room in front of a projector screen. All stimuli were presented at eye-level at the centre of the screen, with stimulus strings subtending a maximum visual angle of 3.7° to ensure that the word stimulus was close to foveal vision. Stimuli were presented in black Arial font, in the centre of a white background and were presented pseudo-randomly across four trials blocks, such that there was the same number of trials from each condition in each block, with rest breaks in between. Trials were presented and behavioral performance was recorded by E-Prime (Psychology Software Tools, Inc., Pittsburgh, USA). The testing session lasted approximately 15 minutes.

<div align="center">INSERT FIGURE 7 ABOUT HERE</div>

4.4. ERP Processing

The EEG was recorded with Synamps DC-amplifiers (NeuroScan, Sterling, VA, USA) from 36 Ag/AgCl electrodes, placed in an EasyCap (www.easycap.de) according

to the 10-20 system. Bipolar recordings from electrodes set above and below the left eye recorded vertical eye movement. Electrode impedance was maintained below 5 kΩ. The online reference was the left mastoid and FPz served as the ground electrode. EEG activity was band-pass filtered on-line between 0.1 and 200 Hz and sampled at 1 KHz. The EEG was re-filtered off-line with a 30 Hz band-pass zero-phase shift low pass digital filter (48 dB/octave). Eye blinks were mathematically corrected using the algorithm provided by Scan 4.3 (Neuroscan, Inc.): A model eye-blink was computed from a minimum of 50 individual blinks, and, when the variance of the model at each recording channel was below 0.001 (which was the case in every participant), the amplitude of the model was subtracted from each channel proportionally to the overall size of the eye-blink at each recording site in the continuous EEG recording, which is more conservative than the method proposed by Gratton et al. (1983). Visual inspection of the EEG identified remaining artefacts to be manually rejected. The continuous EEG was sliced into epochs ranging from -100 to 1000 ms after the onset of the target word. Epochs with voltage exceeding ±75 μV were automatically rejected. Only corrected trials were included. There was a minimum of 30 valid epochs per condition in each participant (Control group epochs: P+O+ *M*= 49.00, *SD*= 7.88; P+O- *M*= 44.19, *SD*= 8.41; P-O+ *M*= 42.63, *SD*= 9.65; P-O- *M*= 48.07, *SD*= 9.55; Dyslexic group epochs: P+O+ *M*= 48.56, *SD*= 6.79; P+O- *M*= 42.31, *SD*= 8.54; P-O+ *M*= 41.13, *SD*= 8.75; P-O- *M*= 47.44, *SD*= 8.27). Baseline correction was performed in reference to pre-stimulus activity, and individual averages were digitally re-referenced to the average of the mastoid electrodes. Individual averages defined by the prime condition were computed and used to produce grand-mean averages for the dyslexic and control group. ERP data were collected

simultaneously to behavioural data.

4.5. ERP data analysis

Main ERP components were identified based on their deflection, topography and latency. Time windows for analysis of each component were defined on the basis of the mean global field power produced by all electrodes taken together across the scalp (SEE FIG). The same windows were used in all conditions and both groups: 85–115 ms for the P1; 150–180 ms for the N1; 150-220 ms for the P2; 250-320 for the N2; 260-360 ms for the P3; and 450–670 ms for the P600. Peak detection was time-locked to the electrode of maximal amplitude for each component: O2 for the P1; P8 for the N1; FCz for the P2 and N2, CPz for the P3 and P600. Similarly, mean amplitudes were measured at electrodes chosen based on their maximum sensitivity: O1, O2, P7 and P8 electrodes for the P1; O1, O2, P7 and P8 for the N1; F3, F4, Fz, FC3, FC4, FCz for the P2 and N2; C3, C4, Cz, CP3, CP4, CPz, for the P3 and P600. Mean amplitudes and peak latencies were subjected to 2 x 2 x 2 x electrode mixed analyses of variance (ANOVAs). Within-subject factors were phonological similarity (homophonic, non-homophonic) and orthographic similarity (orthographic neighbour, non-neighbour) and electrodes and a between subjects factor of group (control, dyslexic). Behavioural data was analysed with a three-way mixed ANOVA with within subjects factors of phonological similarity (homophonic, non-homophonic) and orthographic similarity (orthographic neighbour, non-neighbour); and a between subjects factor of group (control, dyslexic).

Acknowledgements

Figure Legends

Figure 1. Mean reaction time and accuracy (Error bars represent 1 standard error)

Figure 2. Orthographic priming modulations of target N1 amplitude (Linear derivation of O1, O2, P7 and P8 electrodes).

Figure 3. The three-way interactions spanning the P2, N2 and P3 peaks. * Condition effect is $p < .05$. Dotted lines depict significant pair-wise comparisons.

Figure 4. P3 and P600 target word modulations for each prime condition (Linear derivation of C3, C4, Cz, CP3, CP4 and CPz electrodes).

Figure 5. Averaged phonological priming effects across central electrodes, depicting the diffuse P600 attenuation in the dyslexic group (P+ is P+O+ and P+O- combined; and P- is P-O+ and P-O-).

Figure 6. Orthographic and phonological priming difference waves at analysed electrode sites. A: linear derivation (LDR) of O1, O2, P7 and P8. B: LDR of F3, F4, Fz, FC3, FC4 and FCz. C: LDR of C3, C4, Cz, CP3, CP4 and CPz. Con = Control. DYS = Dyslexic.

Figure 7. A single trial. Words were presented with each priming pseudoword condition, and critical trials were interspersed with non-priming fillers. *Variable ISI (controlled across prime conditions) of 200, 260, 320, 380, 440 or 500 ms.

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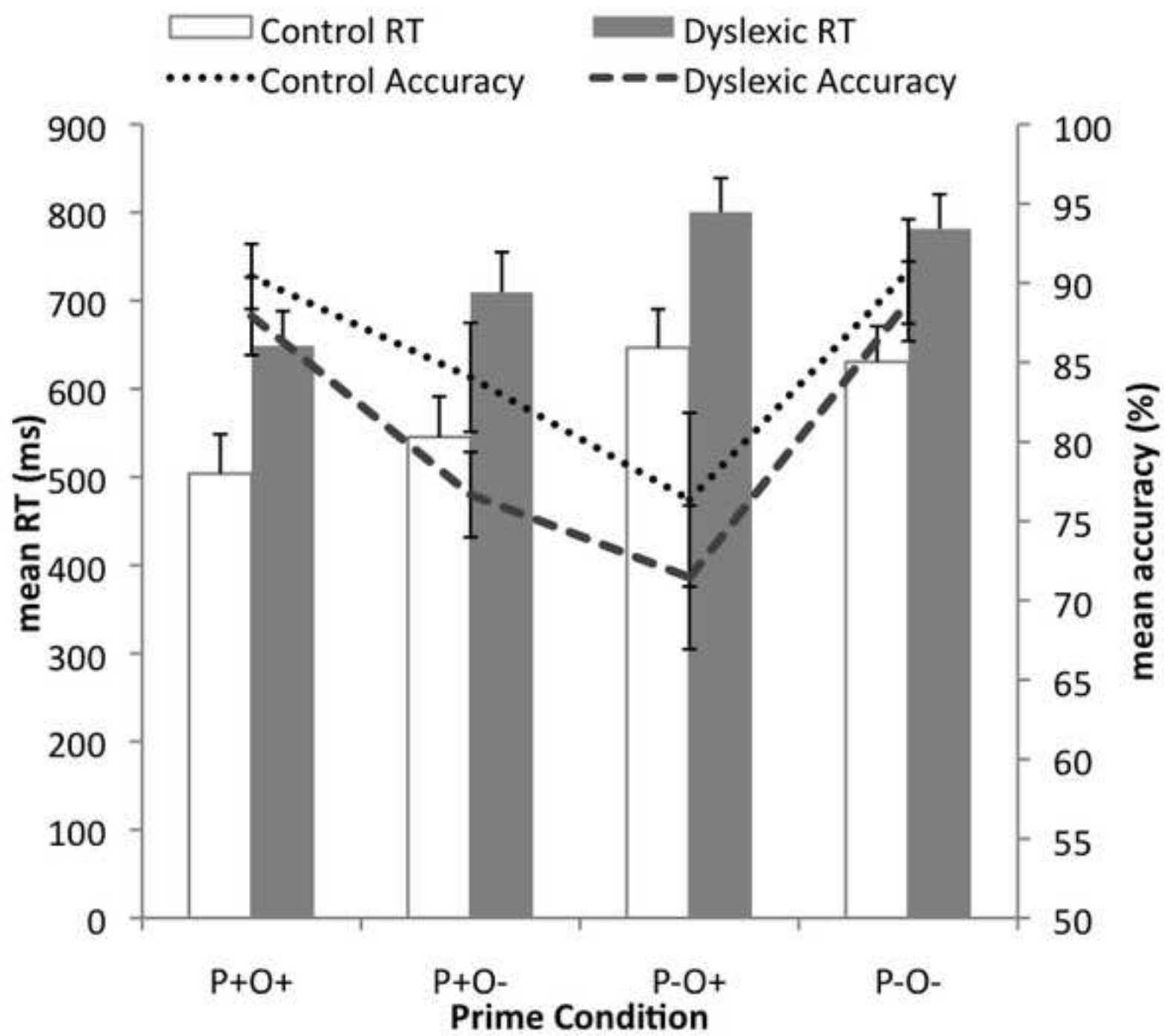**Figure 1**
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Figure 1

Figure 2
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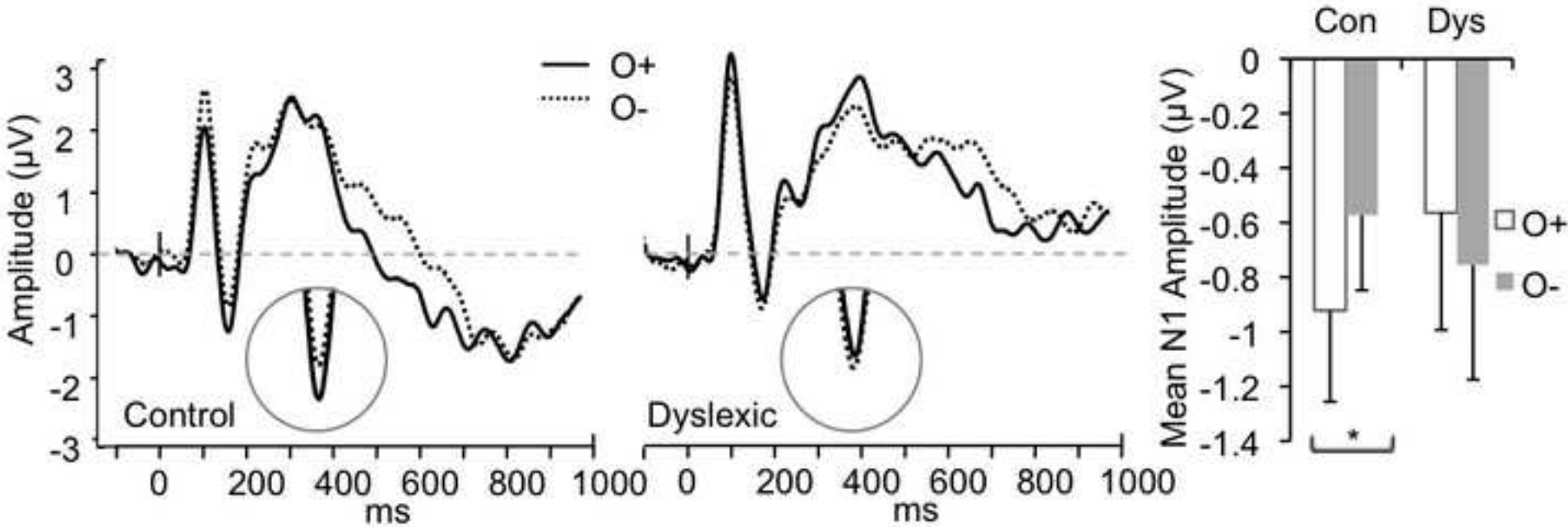
Figure 5

Legend: Phonological match (P+) — — — Phonological non-match (P-) ···········

Control Group — electrode sites: FC3, FCZ, FC4, C3, CZ, C4, CP3, CPZ, CP4. P3 and P600 markers shown at CZ.

Dyslexic Group — electrode sites: FC3, FCZ, FC4, C3, CZ, C4, CP3, CPZ, CP4. P3 and P600 markers shown at CZ.

Figure 6

1000 ms

+ 200 ms

hauce ISI*

+ 200 ms

HORSE

3000 ms
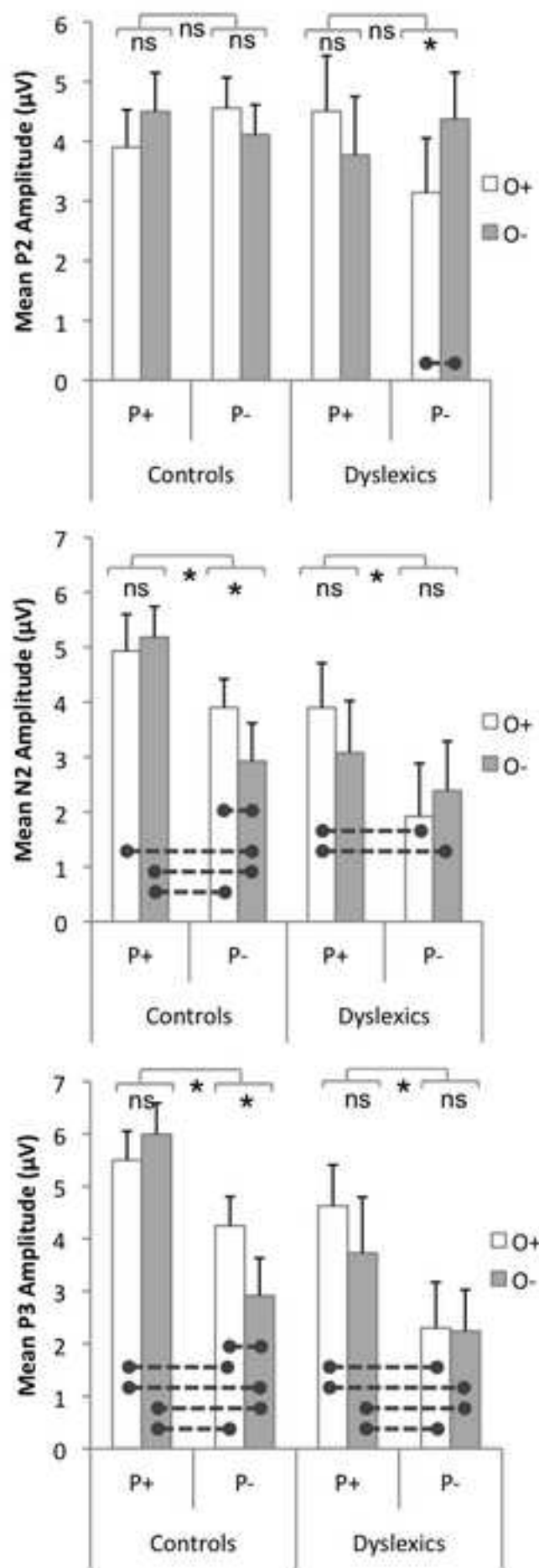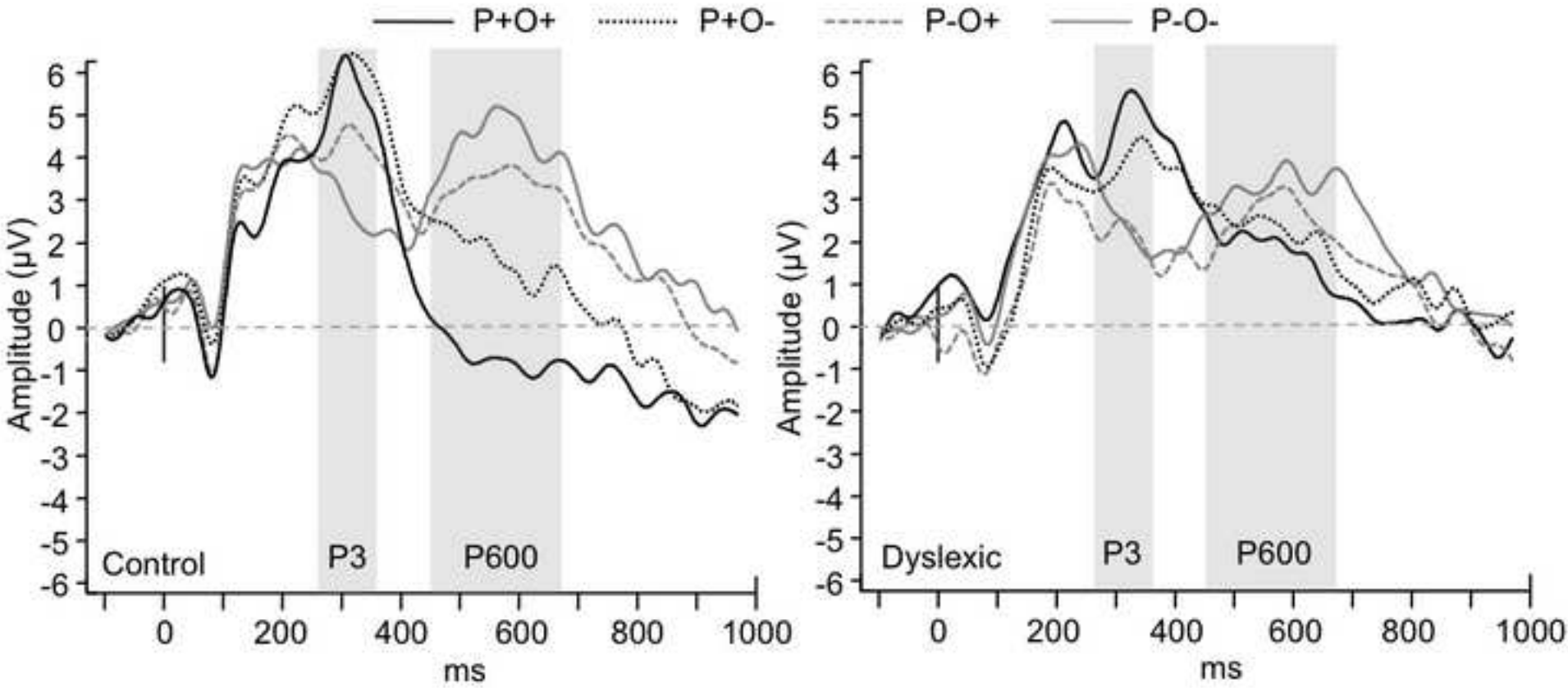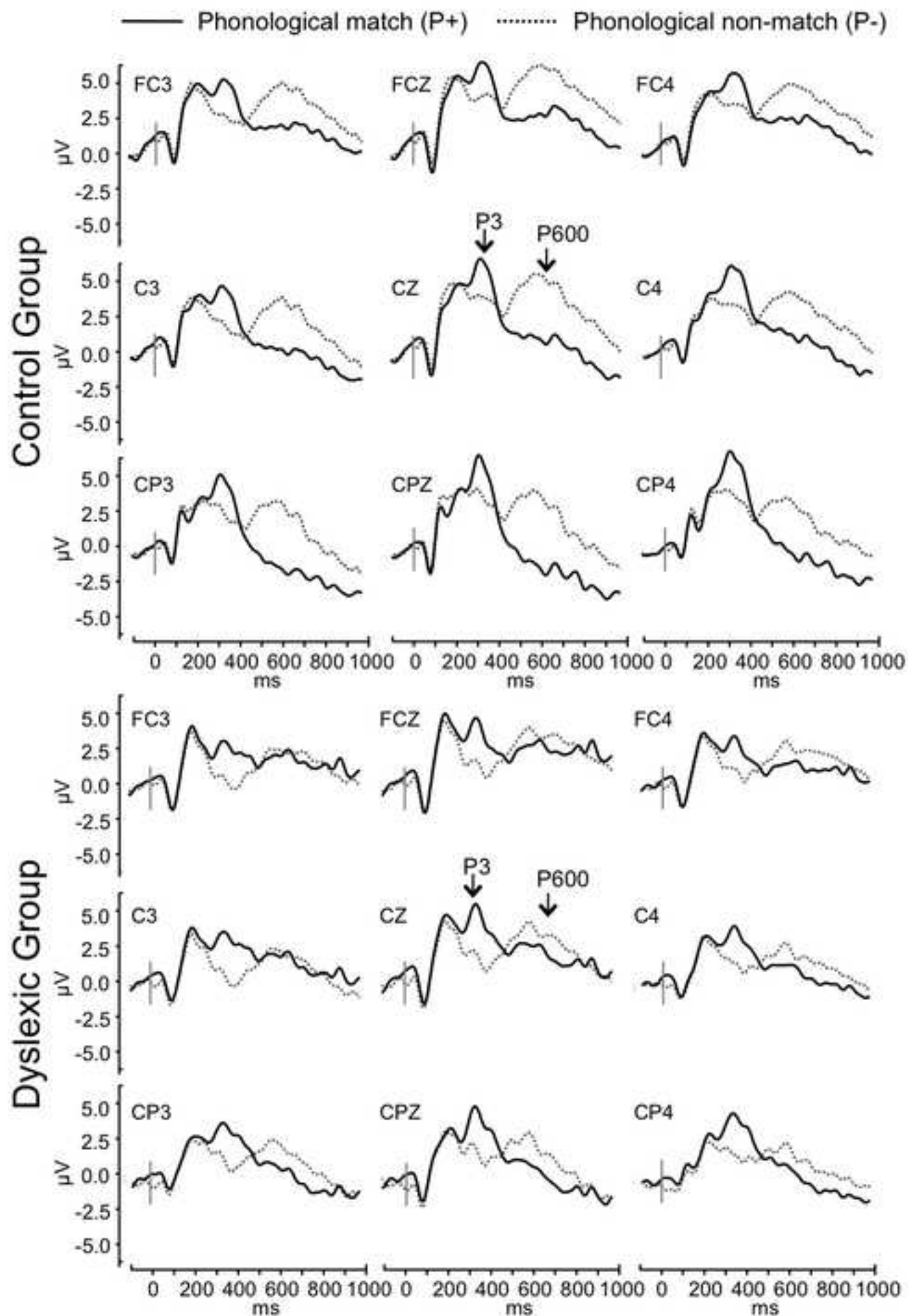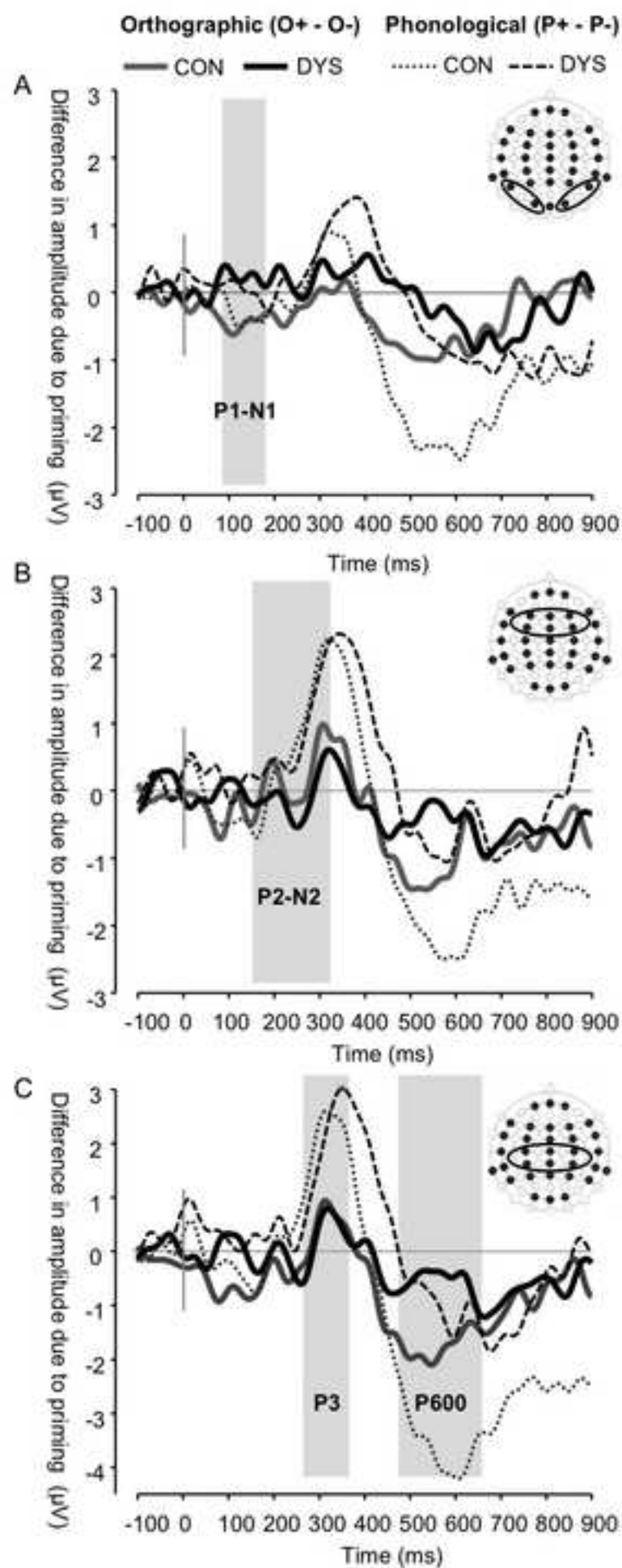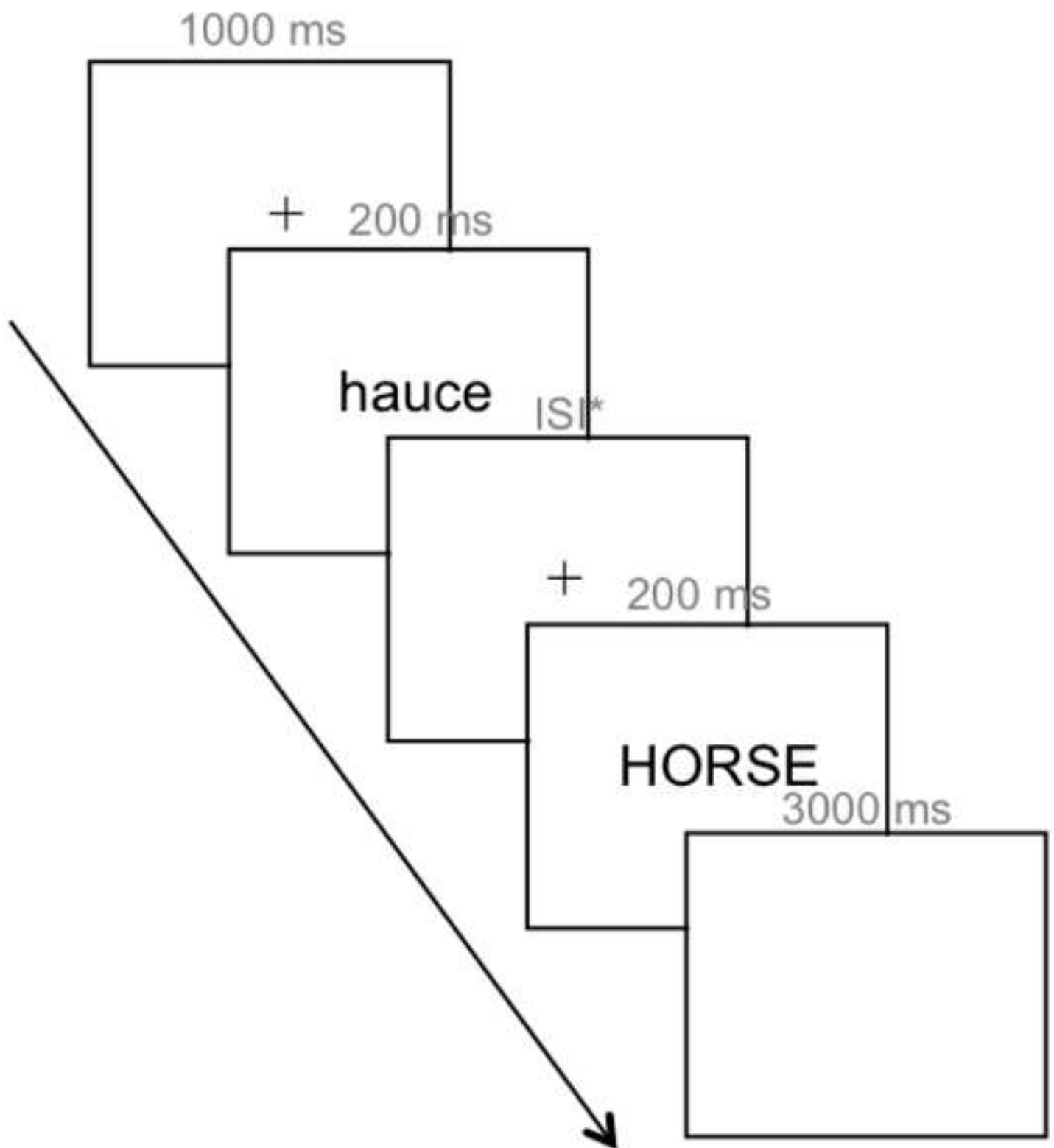
Table 1

Table 1. *Group performance on psychometric subtests*

Measure	Controls (*n*=16)		Dyslexics (*n*=16)		
	Mean	*SD*	*Mean*	*SD*	*t*
Age (years)	21.19	4.40	21.63	4.72	-0.27
DAST One-minute Reading	110.25	9.25	92.06	14.92	4.14**
DAST Nonsense Passage	93.94	4.19	88.69	6.34	2.76*
DAST Rapid Naming (s)	24.56	5.67	32.13	9.47	-2.74*
WAIS Digit Span	12.25	2.67	9.81	3.92	2.06*
WIAT Pseudoword Reading	105.63	6.51	98.44	12.32	2.06[a]
WRAT Reading	114.19	4.31	104.63	9.38	3.71**
WRAT Spelling	104.44	8.05	93.63	16.78	2.32*

Note. WAIS, WIAT and WRAT scores are age-scaled. *$p < .05$; **$p < .01$; [a]$p = .051$