# Spatial Attention and Reading Ability: ERP Correlates of Flanker and Cue-size Effects in Good and Poor Adult Phonological Decoders.

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Abstract

To investigate facilitatory and inhibitory processes during selective attention among adults with good (*n*=17) and poor (*n*=14) phonological decoding skills, a go/nogo flanker task was completed while EEG was recorded. Participants responded to a middle target letter flanked by compatible or incompatible flankers. The target was surrounded by a small or large circular cue which was presented simultaneously or 500ms prior. Poor decoders showed a greater RT cost for incompatible stimuli preceded by large cues and less RT benefit for compatible stimuli. Poor decoders also showed reduced modulation of ERPs by cue-size at left hemisphere posterior sites (N1) and by flanker compatibility at right hemisphere posterior sites (N1) and frontal sites (N2), consistent with processing differences in fronto-parietal attention networks. These findings have potential implications for understanding the relationship between spatial attention and phonological decoding in dyslexia.

Key words: dyslexia; phonological decoding; reading; flanker effect; cue-size effect; spatial attention; ERPs; N1; N2

1. **Introduction**

Phonological decoding refers to the letter-sound or grapheme-phoneme conversion process that is required to assemble a phonological code from an orthographic or visual code during reading. Developmental dyslexia is predominantly characterised by poor phonological coding ability, as assessed by nonword reading tests ([Rack, Snowling, & Olson, 1992](#_ENREF_59); [Siegel, 1994](#_ENREF_70); [Vellutino, Fletcher, Snowling, & Scanlon, 2004](#_ENREF_72)), and these difficulties continue into adulthood ([Bruck, 1993](#_ENREF_4)). Neurobiologically, dyslexia has been associated with under activation in posterior (parietotemporal and occipitotemporal) and anterior (inferior frontal gyrus) areas of the left hemisphere which are specialised for the processing of written words ([Shaywitz, Mody, & Shaywitz, 2006](#_ENREF_66)). Selective attention to individual words and letters and the suppression of those that are not the current focus of attention may be particularly important for the process of phonological decoding ([LaBerge & Brown, 1989](#_ENREF_35); [Mozer & Behrmann, 1990](#_ENREF_46); [Vidyasagar & Pammer, 2010](#_ENREF_76); [Whitney, 2001](#_ENREF_80)). Consistent with this view, in addition to phonological decoding deficits, developmental dyslexia has been associated with deficits on a range of tasks which are subserved by specialised attentional networks and there is overlap between brain areas (e.g., inferior parietal lobe and prefrontal cortex) involved in both word analysis and selective attention ([Shaywitz & Shaywitz, 2008](#_ENREF_67)).

There are three structurally and functionally distinct attentional networks in the brain: the alerting, orienting and executive control networks ([Petersen & Posner, 2012](#_ENREF_53); [Posner, Rueda, & Kanske, 2007](#_ENREF_54); [Posner & Peterson, 1990](#_ENREF_57)). While the alerting network is most important for vigilance and sustained attention, the latter two networks are particularly important for selective attention. The orienting network is comprised of two fronto-parietal networks, a ventral network specialised for stimulus-driven reorienting and a dorsal network specialised for voluntary orienting ([Corbetta & Shulman, 2002b](#_ENREF_10); [Petersen & Posner, 2012](#_ENREF_53)). In the visual modality, this network is responsible for directing attention towards particular spatial locations (on the basis of bottom-up or top-down signals) in order to enhance or inhibit processing within primary and secondary visual processing areas. In contrast, the fronto-parietal executive control network, is responsible for governing behaviour via top down control mechanisms such as maintaining task set and making adjustments according to feedback ([Petersen & Posner, 2012](#_ENREF_53)).

Dyslexia has been associated with poorer performance on visuospatial selective attention tasks (e.g., spatial cuing, visual search) which are likely to subserved by the orienting network. These findings have been argued to reflect posterior parietal abnormality or a dorsal (magnocellular) visual processing stream abnormality (see [Stein & Walsh, 1997](#_ENREF_71); [Vidyasagar, 2004](#_ENREF_74); [Vidyasagar & Pammer, 2009](#_ENREF_75); [Vidyasagar & Pammer, 2010](#_ENREF_76)). For example, it is argued that reading involves a dorsally mediated ‘spotlighting’ mechanism which moves spatial attention sequentially across the visual field to facilitate processing at particular locations within ventral visual processing areas ([Vidyasagar & Pammer, 2010](#_ENREF_76)). Given the specialisation of the right hemisphere for spatial attention, and visual field differences in attentional processing, some authors have argued for a right hemisphere locus of attentional impairment in dyslexia ([Hari, Renvall, & Tanskanen, 2001](#_ENREF_26); [Stein & Walsh, 1997](#_ENREF_71)). Importantly, these attentional deficits have been specifically associated with nonword reading deficits or poor phonological decoding ability (e.g., [Facoetti et al., 2010](#_ENREF_18); [Facoetti et al., 2006](#_ENREF_20); [Ruffino et al., 2010](#_ENREF_64)), and recent longitudinal research shows that attentional orienting ability at pre-reading predicts future reading development in primary school, suggesting a causal link ([Franceschini, Gori, Ruffino, Pedrolli, & Facoetti, 2012](#_ENREF_23)).

Dyslexia has also been associated with problems on tasks which are proposed to measure executive function ([Helland & Asbjornsen, 2000](#_ENREF_29); [Reiter, Tucha, & Lange, 2005](#_ENREF_61)). Of particular relevance to the present study, dyslexia is often associated with difficulty inhibiting distracting visual information ([Bednarek et al., 2004](#_ENREF_1); [Brosnan et al., 2002](#_ENREF_3); [Facoetti & Turatto, 2000](#_ENREF_19); [Klein & D'Entremont, 1999](#_ENREF_33); [Martelli, Di Filippo, Spinelli, & Zoccolotti, 2009](#_ENREF_42); [Moores, Cassim, & Talcott, 2011](#_ENREF_45)), which may implicate inhibitory mechanisms within the executive control network. Indeed, it has been suggested elsewhere that selective attention and attentional shifting may be necessary processes common to many executive function tasks that are traditionally used in neuropsychological assessment ([Reiter et al., 2005](#_ENREF_61)).

Few studies have examined the underlying neurobiological mechanisms associated with selective attention mechanisms in dyslexic or poor reading populations. Given the link between phonological decoding and attentional problems in dyslexia, the present study aims to explore the behavioural and electrophysiological correlates of selective attention in adults with either good or poor phonological decoding skills, as assessed by nonword reading ability. The flanker go/nogo task used in the present study allows for the examination of specific attentional mechanisms which relate to selective attention to particular spatial locations (spatial orienting and focussing) and the inhibitory processes involved in executive attentional control.

The facilitation of attention at particular spatial locations is often likened to a ‘spotlight’ or ‘zoom lens’ of a specific size ([Eriksen & St James, 1986](#_ENREF_14); [Posner, 1980](#_ENREF_55)) and separate mechanisms within the orienting network have been proposed for moving or shifting spatial attention (orienting) ([Posner, 1980](#_ENREF_55); [Posner, Snyder, & Davidson, 1980](#_ENREF_58)) and adjusting the size of attentional focus (focusing) ([Benso, Turatto, Mascetti, & Umilta, 1998](#_ENREF_2)). Research shows that attentional focussing is facilitated when visual targets are preceded by small relative to large cues (cue-size effect), implying a processing speed advantage for a narrow versus diffuse attentional focus ([Castiello & Umilta, 1990](#_ENREF_6), [1992](#_ENREF_7)).

Behavioural studies examining attentional focussing in developmental dyslexia have revealed conflicting findings. In a study using a simple dot detection task presented at fixation, normally reading children and adults showed a cue-size effect on RT at both short (99ms) and long (504ms) SOAs, and children with dyslexia only showed this effect at short SOAs ([Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000](#_ENREF_17)). It was suggested that dyslexics were unable to sustain active focussing over time and had shifted to a more distributed and less efficient visual processing mode. In a later study, Facoetti et al. (2003) investigated cue-size effects in children with dyslexia (*n*=10) and normally reading (*n*=13) children using a variable SOA of 100ms or 500ms. In this task, participants were required to discriminate and respond to the direction of a central arrow preceded by a small or large cue. Whereas control children showed cue-size effects at both SOAs, children with dyslexia showed a cue-size effect at the long (500ms) but not the short (100ms) SOA, suggesting a deficit in the automatic focussing of spatial attention ([Facoetti et al., 2003](#_ENREF_15)). The authors suggested that the conflicting findings of these two studies could be due to task difficulty, perceptual load, or the allocation of processing resources ([Facoetti et al., 2003](#_ENREF_15)).

As well as perceptual enhancement of target locations, inhibitory mechanisms act to suppress information in unattended areas of the visual field ([Cepeda, Cave, Bichot, & Kim, 1998](#_ENREF_8)). The ability to suppress irrelevant information that is not the current focus of attention can be investigated using ‘flanker’ paradigms in which participants take longer to respond to a central target stimulus that is flanked by either response incompatible relative to compatible distractor letters ([Eriksen & Eriksen, 1974](#_ENREF_13)). Eriksen and Eriksen (1974) attributed this ‘flanker effect’ to interference at the response selection stage, postulating that it reflected the time taken to inhibit an incompatible response. Other research suggests that early selection mechanisms play a role in flanker inhibition. For example, a reduction in the flanker effect is found when spatial attention is narrowly focussed on the target ([LaBerge, Brown, Carter, Bash, & Hartley, 1991](#_ENREF_36); [Yantis & Johnston, 1990](#_ENREF_83)) suggesting that flanker effects occur due to attentional leakage to flanker locations ([Yantis & Johnston, 1990](#_ENREF_83)). Similarly, greater flanker effects are found when targets are preceded by an invalid spatial cue, suggesting an interaction between orienting and executive control, such that the ability to ignore flankers is reduced when a shift in spatial attention is required ([Callejas, Lupianez, & Tudela, 2005](#_ENREF_5)). Facoetti and Molteni (2000) investigated whether attentional focussing acts as an inhibitory mechanism for suppressing distractors. The size of attentional focus was manipulated with small or large cues presented either simultaneously or 500ms prior to target onset. A reduction in the flanker effect was found for small cues in the 500ms SOA condition, suggesting that irrelevant distractor locations are inhibited when spatial attention is optimally focussed.

Several behavioural studies have examined inhibition of distracting stimuli in dyslexia using a flanker paradigm. For example, using a number identification task, Klein and D’Entremont (1999) found that adults defined as ‘poor readers’ (according to Nelson-Denny reading scores) did not show the same decrease in the flanker effect as a function of flanker eccentricity that was observed in ‘good readers’. Bednarek et al. ([2004](#_ENREF_1)) investigated the processes of alerting, orienting, and inhibition (or resolution of conflict) among Spanish dyslexics and normally reading children using a cueing task in which targets were surrounded by compatible and incompatible flankers. They found that participants with dyslexia were impaired relative to controls on accuracy and RT measures when targets were flanked by incompatible flankers, suggesting deficient executive control rather than an orienting or alerting deficit. It was argued that the origin of this effect could be either executive function mediated by the prefrontal cortex, attentional processes mediated by the posterior parietal cortex, or the magnocellular visual processing stream which has been linked to the identification of flanker stimuli ([Omtzigt & Hendriks, 2004](#_ENREF_49); [Omtzigt, Hendriks, & Kolk, 2002](#_ENREF_51)). However, further research suggests that increased interference in dyslexia is more likely to be due to crowding than to magnocellular-related surround suppression ([Omtzigt & Hendriks, 2011](#_ENREF_50); [Omtzigt, Hendriks, & Kolk, 2006](#_ENREF_52)). [Facoetti and Turatto (2000)](#_ENREF_19) found that children with dyslexia showed an asymmetric flanker effect such that the flanker effect was reduced in the left visual field and greater in the right visual field when compared to control children, suggesting problems with inhibiting information in the right visual field. This finding is consistent with the right hemisphere deficit hypothesis of developmental dyslexia and with other research suggestive of left mini-neglect in dyslexia ([Hari et al., 2001](#_ENREF_26); [Stein & Walsh, 1997](#_ENREF_71)).

It is possible to examine the neurobiological basis of attentional mechanisms by examining specific components of the ERP waveform, that are modulated by early selective attention (N1) and inhibitory (N2) processes. The posterior N1 component of the ERP waveform is modulated by spatial attention during covert orienting such that greater amplitude is observed for valid relative to invalid trials (for reviews see [Eimer, 1998](#_ENREF_11); [Mangun, 1995](#_ENREF_39)). Attentional modulation of the N1 component is also found as a function of cue-size and flanker manipulations such that amplitude is greater for small relative to large cues ([Luo, Greenwood, & Parasuraman, 2001](#_ENREF_38)) and for incompatible relative to compatible flanker trials ([Nicholls, Bruno, & Matthews, 2015](#_ENREF_48)). These N1 attention effects are argued to reflect a amplification mechanism which acts to decrease signal to noise ratio and facilitate perceptual processing of attended locations in extra-striate visual areas ([Hillyard, Vogel, & Luck, 1998](#_ENREF_30); [Hopf, Vogel, Woodman, Heinze, & Luck, 2002](#_ENREF_31); [Mangun, 1995](#_ENREF_39); [Mangun et al., 2001](#_ENREF_41)). This attentional modulation is thought to act like a spotlight to facilitate processing of information within the bounds of the current attentional focus ([Mangun et al., 2001](#_ENREF_41)). It has been argued that the attentional difficulties experienced by many people with dyslexia are related to deficient attentional spotlight mechanism (e.g., [Vidyasagar, 2001](#_ENREF_73), [2004](#_ENREF_74)), but few electrophysiological studies ([Matthews & Martin, 2009](#_ENREF_44)) have investigated the attentional modulation of the N1 component among people with dyslexia or poor phonological decoding ability.

The frontal N2 component of the ERP waveform is thought to reflect inhibitory processing within frontal areas of the executive attentional network ([Falkenstein, 2006](#_ENREF_21); [Folstein & Van Petten, 2008](#_ENREF_22)), and may also play a role in directing attention towards task relevant events ([Weissman, Gopalakrishnan, Hazlett, & Woldorff, 2005](#_ENREF_79)). N2 amplitude is typically greater for nogo relative to go trials in go/nogo paradigms, and is also greater for incompatible relative to compatible trials in flanker paradigms ([Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988](#_ENREF_25); [Heil, Osman, Wiegelmann, Rolke, & Hennighausen, 2000](#_ENREF_27); [Kopp, Rist, & Mattler, 1996](#_ENREF_34)). However, there is some debate in the literature as to whether the N2 flanker effect reflects response inhibition or a conflict detection mechanism that is mediated by anterior cingulate cortex ([Folstein & Van Petten, 2008](#_ENREF_22); [Larson, Clayson, & Clawson, 2014](#_ENREF_37)). To our knowledge, no previous study has examined modulation of the N2 component during the performance of a flanker go/nogo task.

Given that few studies have used ERPs to isolate specific attentional mechanisms and examine their relationship to phonological decoding, the aim of the present study was to examine both attentional focussing and inhibition of unattended visual stimuli among good and poor adult phonological decoders using a modified flanker task. Participants, while not necessarily dyslexic, were selected on the basis of poor phonological decoding ability (i.e., nonword reading ability). These groups were chosen due to the association between selective attention deficits and phonological decoding in dyslexia (e.g., [Facoetti et al., 2010](#_ENREF_18); [Facoetti et al., 2006](#_ENREF_20); [Ruffino et al., 2010](#_ENREF_64)). A go/nogo flanker paradigm was used rather than a two-choice response task to reduce the influence of response competition and increase the influence of perceptual and attentional processes. To investigate focusing of spatial attention, target stimuli were preceded by either large or small cues that were presented either simultaneously or 500ms prior to the target (cued).

If poor phonological decoding ability is associated with impaired attentional focussing, a greater cue-size effect would be expected for RT among poor decoders compared to good decoders, such that poor decoders would either benefit less from small focussing cues or have greater RT cost following large cues which promote a more diffuse attentional focus. This is expected to be accompanied by a reduction in the modulation of the N1 component as a function of cue-size. Furthermore if poor phonological decoders have difficulty ignoring distracting visual information, greater RT would be expected for targets flanked by incompatible relative to compatible flankers, particularly when preceded by large cues. Under these conditions, a reduction in the modulation of N1 amplitude by flanker compatibility would implicate deficient early selection within the orienting network, and a reduction in the N2 flanker effect would implicate deficient inhibitory control within the executive control network.

1. **Method**

*2.1. Participants*

Good (n=17) and poor (n=14) phonological decoders were selected from a screening sample of over 300 undergraduate Psychology students on the basis of scores in the upper and lower quartiles on the Martin and Pratt Nonword Reading Test ([Martin & Pratt, 2001](#_ENREF_43)). All participants had normal or corrected-to-normal vision and one male and one female from each group were left handed. Exclusion criteria included a history of drug, alcohol, or tobacco abuse, psychiatric or neurological disorder (including ADHD), head trauma, seizure, and those currently receiving medication. Data for one female poor decoder was excluded due to accuracy below 70% on the experimental task. The final sample consisted of 17 good decoders (10 female, 7 male) and 14 poor decoders (8 female, 6 male).

The nonword reading scores of good decoders ranged from 49-54 (out of a possible score of 54) representing a ceiling effect and the scores of poor decoders ranged from 19-43. Norms for the Nonword reading test are available for samples up to 17 years of age (Martin & Pratt, 2001). The mean nonword reading score of the good decoder group was in the 81st percentile (range 66th – 98th percentile) equating to a reading age equivalent of >17 years for all participants. The mean nonword reading score of poor decoders was in the 16th percentile (range 2nd -37th percentile) equating to an average reading age equivalent of 10-11 years (range 7 to 15 years) of this norming group.

Ravens Advanced Progressive Matrices (APM) was administered as a measure of non-verbal general intelligence ([Raven, Court, & Raven, 1994](#_ENREF_60)). Other reading measures included the Word Identification and Comprehension sub-tests from the Woodcock-Johnson (WJ) Reading Mastery Tests ([Woodcock, 1987](#_ENREF_81)), The National Adult Reading Test ([NART: Nelson & Willison, 1991](#_ENREF_47)), Reading Accuracy and Reading Rate measures from the Neale Analysis of Reading Ability and an irregular word reading test devised by the authors. Digit Span, Vocabulary, Symbol Coding and Symbol Copy sub-tests from the Weschler Adult Intelligence Scale ([WAIS-III: Wechsler, 1997](#_ENREF_78)) were also administered.

No significant group differences were found in terms of age, general nonverbal intelligence (APM), or digit span backwards (Table 1). Good decoders had significantly higher raw scores in comparison to poor decoders on the symbol coding and digit span forwards subtests and on all reading measures (non-word reading, word identification, irregular word reading, passage comprehension, reading rate, reading accuracy, and vocabulary).

*2.2. Stimuli and Apparatus*

Stimuli were presented via NeuroScan STIM Software. Each trial began with a 300ms central fixation cross, followed by a circular cue (small or large) which remained on the screen throughout the 200ms presentation of a target stimulus composed of three lowercase letters (b or d). Small cues comprised a circle around the middle letter of the target stimulus and large cues comprised a circle around all three letters. The middle target letter was a ‘b’ or ‘d’ on 50% of trials and was randomly surrounded by either compatible (same letter) or incompatible (other letter) on an equal proportion of trials. The cue and the target stimulus appeared either simultaneously (0ms SOA) or with a 500ms cue-to-target SOA. Stimuli were presented white on a black background with letters subtending 1º x 1º of visual angle at a viewing distance of 70cm, with a 1º spacing between each letter. The inter-trial interval was 1000ms. There were four blocks each comprising 200 experimental trials, two blocks for each SOA condition, with one block for each go/nogo stimulus (b or d).

Table 1

*Mean age and raw scores on reading and cognitive measures for good and poor phonological decoders.*

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | Good Decoders  (*n*=17) | | Poor Decoders  (*n*=14) | | | F-test | |
|  | *M* | *SD* | | *M* | *SD* | |  |
| Age | 21.64 | 3.85 | | 19.86 | 2.40 | | *ns* |
| APM /36 | 23.71 | 5.18 | | 21.50 | 3.84 | | *ns* |
| MP Nonword Reading /54 | 50.65 | 1.50 | | 35.14 | 6.92 | | ***\*\*\**** |
| WJ Word Identification /106 | 98.41 | 2.62 | | 88.00 | 5.14 | | \*\*\* |
| Irregular word reading /87 | 78.47 | 8.89 | | 60.71 | 7.82 | | \*\*\* |
| NART# /50 | 33.82 | 4.20 | | 18.21 | 4.23 | | \*\*\* |
| Digit Span Forwards# /16 | 11.71 | 1.72 | | 9.29 | 2.37 | | \*\* |
| Digit Span Backwards# /14 | 8.00 | 1.58 | | 7.29 | 2.30 | | *ns* |
| WJ Comprehension /68 | 61.1 | 2.5 | | 55.9 | 4.6 | | *\*\*\** |
| WAIS Vocabulary† /66 | 55.3 | 5.3 | | 42.9 | 7.3 | | *\*\*\** |
| WAIS Symbol Coding† /60 | 81.2 | 8.5 | | 74.8 | 8.4 | | *\** |
| WAIS Symbol Copy† /33 | 125.6 | 10.3 | | 115.8 | 16.7 | | *p*=.055 |
| Neale Reading Accuracy† (%) | 98.2 | 2.1 | | 88.1 | 5.6 | | *\*\*\** |
| Neale Reading Rate† (words/min) | 147.6 | 16.3 | | 110.4 | 11.6 | | *\*\*\** |

Note: \* *p*<.05, \*\* *p*<.01, \*\*\* *p*<.001, # Missing data was substituted with the mean for the group for two poor decoders and two good decoders, † Missing data was substituted with the mean for the group for five good decoders and three poor decoders.

*2.3. Electrophysiological Recording*

EEG activity was recorded with a NeuroScan system using SCAN 4.1 software and Quik-caps with sintered Ag/AgCl electrodes. EEG was recorded from 32 sites, according to the International 10-20 system referenced to linked mastoids and grounded at AFz. Horizontal electro-oculographic (EOG) activity was recorded bipolarly from the outer canthi of both eyes, and vertical EOG was recorded above and below the left eye. Electrode impedance was kept below 5 kΩ. EEG activity was sampled continuously at a rate of 1000 Hz. Continuous EEG was subjected to a zero phase-shift band-pass filter (0.15-30Hz, 24dB/Oct). Ocular artifact reduction was performed by regression and artifact averaging using NeuroScan Software. Data files were then baseline corrected and epoched (1000ms) offline commencing 100ms before stimulus onset. High and low voltage cut-offs for artifact rejection were set at ±100 μV. Correct responses were averaged for each stimulus type. The time frames for peak N1 (125-200ms) and N2 (210-300ms) amplitude were determined from grand mean averaged waveforms at posterior (O1/O2, P3/P4, P7/P8) and anterior (F3, Fz, F4) sites respectively.

*2.4. Procedure*

The study was approved the University of Tasmania Human Research Ethics Committee and all participants gave written informed consent prior to participation. Reading and cognitive measures were administered in a screening session of approximately one hour on a day prior to the two hour experimental session. Following set-up for EEG recording, participants were seated in front of a computer monitor at a viewing distance of 70cm. Four go/nogo letter discrimination tasks were completed in counterbalanced order separated by short breaks, with Go target (b or d) and SOA (0ms or 500ms) counterbalanced between subjects. Participants were asked to respond when the middle letter of the target stimulus was the Go stimulus (b or d) using a response pad and to withhold responses for the nogo stimulus (b or d). Participants were instructed to respond as quickly and as accurately as possible to all ‘go’ stimuli irrespective of cue size or flanker compatibility and to avoid excessive blinking or overt movements.

*2.5. Design and Data Analysis*

Mean RT, accuracy, and ERP waveforms were averaged across each letter type (b, d). Mean RT was analysed using a 2[Group: good decoder, poor decoder] x 2[Sex: male, female] x 2(SOA: 0ms, 500ms) x 2(Cue size: small, large) x 2(Flanker: compatible, incompatible) repeated measures ANOVA. An additional factor of 2(Response: go, nogo) was included for analysis of mean accuracy (% of correct trials). Identical analyses were conducted for electrophysiological data, with the addition of two extra electrode variables for analysis of N1 amplitude: 2(Hemisphere: left, right) x 3(Site: occipital, parietal, temporal), and one extra variable for analysis of anterior N2 amplitude: 3(Site: F3, Fz, F4). Sex was included as a factor in all analyses for control purposes but only effects in which Group interacted independently of sex are reported here due to sample size limitations. Greenhouse-Geisser corrections were applied where appropriate and significant interactions were analysed using univariate ANOVAs with Bonferroni adjusted p-values to maintain the family-wise Type 1 error rate. Partial eta-squared (ηp2) is reported as a measure of effect size.

1. **Results**

*3.1. Mean Reaction Time*

Mean RT was significantly shorter for compatible (*M*=0.368, *SD*=.033) than incompatible (*M*=0.405, *SD*=.039) stimuli, *F*(1,27)=86.16, *p*<.001 (ηp2=0.761), and for the 500ms SOA (*M*=0.383, *SD*=0.033) than the 0ms SOA (*M*=0.390, *SD*=0.039) condition, *F*(1,27)=4.26, *p*=.049 (ηp2=0.136). The main effect of Group was non-significant, *F*(1,27)=2.09, *p*=.159 (ηp2=0.072). The Cue x Flanker interaction was significant, *F*(1,27)=8.23, *p*=.008 (ηp2=0.234). Mean RT was greater for incompatible relative to compatible stimuli for both small and large spatial cues (*p*s<.001). However, mean RT was significantly shorter for small relative to large cues for incompatible stimuli, *F*(1,27)=6.11, *p*=.02 (ηp2=0.185), and tended to be shorter for large in comparison to small cues for compatible stimuli, *F*(1,27)=4.38, *p*=.046 (ηp2=0.140) (*p*>.025, Bonferroni corrected).

There was a significant SOA x Cue x Flanker x Group interaction (Figure 1), *F*(1,27)=5.59, *p*=.026 (ηp2=0.171). Separate analyses for each SOA, revealed a significant Cue x Flanker x Group interaction for the simultaneous condition, *F*(1,27)=4.97, *p*=.034 (ηp2=0.155). Planned comparisons were conducted to examine the flanker effect for each group for each experimental condition (Figure 1). There was a significant flanker effect under all conditions for good decoders (*p*s<.001). For poor decoders, the flanker effect was significant for both large cue conditions (*p*<.001), however, for small cue conditions, the flanker effect was marginally significant for the 500ms SOA (*p*=.049, *p*>.05 Bonferroni corrected), and non-significant for the simultaneous condition (*p*=.410), indicative of less RT benefit of compatible flankers when the spatial scale of attention was smaller (small spatial cue), particularly when the cue was presented simultaneously with the target.

*Figure 1.* Mean reaction time for incompatible and compatible flanker trials for good (left) and poor (right) decoders for simultaneous (0ms) and cued (500ms) conditions (error bars denote 95%CIs). \*\*\**p*<.001, \*\*, *p*<.01, \**p*<.05

To examine cue-size effects, separate analyses were also conducted for compatible and incompatible trials. For compatible trials (Figure 2), there was a significant Cue x SOA x Group interaction, *F*(1,29)=8.13, *p*=.008 (ηp2=0.219). Good decoders showed a significant Cue x SOA interaction, *F*(1,16)=5.85, *p*=.028 (ηp2=0.268), such that RT was significantly shorter for large relative to small cues for the cued task (500 ms SOA), and this RT tended to be shorter relative to poor decoders, *F*(1,29)=3.45, *p*=.073 (ηp2=0.1). Thus good decoders benefited more from the presence of compatible flankers when they fell within the bounds of the large cueing stimulus.

For incompatible trials, there was a marginally significant Cue x Group interaction regardless of SOA (see Figure 2), *F*(1,27)=3.98, *p*=.056 (ηp2=0.129), such that the effect of Cue was significant for poor decoders, *F*(1,12)=7.97, *p*=.015 (ηp2=0.399), but not for good decoders, *F*(1,15)=.136, *p*=.717 (ηp2=0.009), indicating a greater interference effect for poor decoders when incompatible flankers fell within the bounds of the large cueing stimulus. Between group differences were non-significant for both small and large cues (*p*s>.05).

*Figure 2.* Mean reaction time for good and poor decoders as a function of cue-size on compatible (left) and incompatible (right) flanker trials (error bars denote 95%CIs)

\**p*<.05, \*\**p*<.01

*3.2. Mean Accuracy*

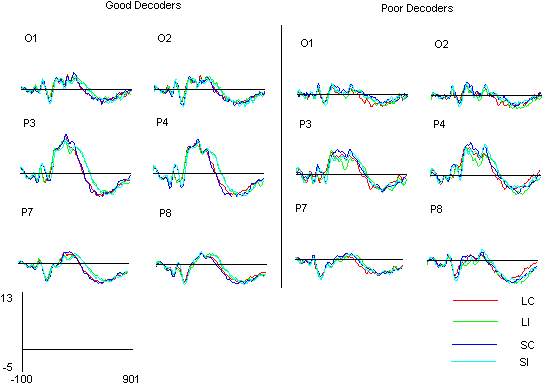
The percentage of correct trials was greater for go (*M*=98.6, *SD*=1.72) compared to nogo (*M*=93.8, *SD*=1.72) trials, *F*(1,27)=61.58, *p*<.001 (ηp2=0.695), for the 500ms (*M*=96.9, *SD*=3.35) compared to the 0ms SOA (*M*=95.5, *SD*=2.58) condition, *F*(1,27)=16.90, *p*<.001 (ηp2=0.385), for large (*M*=96.5, *SD*=2.17) than small (*M*=95.9, *SD*=2.24) cues, *F*(1,27)=5.29, *p*=.029 (ηp2=0.164), and for compatible (*M*=97.8, *SD*=1.42) compared to incompatible (*M*=94.6, *SD*=3.36) stimuli, *F*(1,27)=31.10, *p*<.001 (ηp2=0.535).

The interaction between Response, Flanker, and Group was significant, *F*(1,27)=5.53, *p*=.026 (ηp2=0.170), such that the Flanker x Group interaction was significant for nogo stimuli, *F*(1,27)=4.63, *p*=.041 (ηp2=0.146). For nogo trials, there were no significant between group differences (*p*s>.05), however, the difference between compatible and incompatible nogo trials was greater for good decoders (97% vs. 89%), *F*(1,15)=30.40, *p*<.001 (ηp2=0.670) than poor decoders (96% vs 92%), *F*(1,12)=10.43, *p*=.007, (ηp2=0.465).

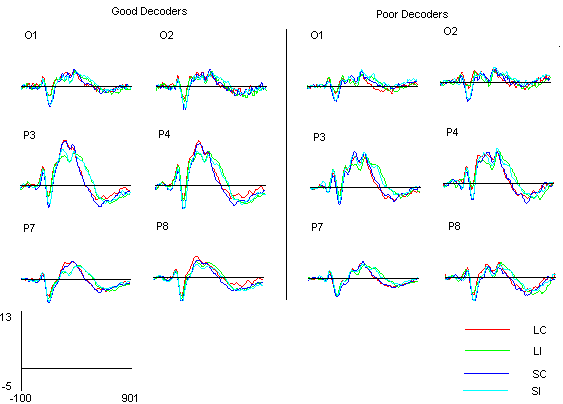
*3.3. Electrophysiological Analyses*

*3.3.1. Posterior N1 Amplitude*

Figure 3 shows grand averaged ERP waveforms for the simultaneous (0ms) and cued (500ms) conditions at occipital (O1, O2), parietal (P3, P4), and posterior temporal (P7, P8) sites as a function of Group, Flanker, and Cue. N1 amplitude was significantly greater for small (*M*=-4.82, *SD*=1.48) compared to large (*M*=-3.95, *SD*=1.85) cues, *F*(1,27)=14.82, *p*=.001 (ηp2=0.354). This was qualified by a significant Cue x Hemisphere x Group interaction (Figure 4), *F*(1,27)=5.58, *p*=.026 (ηp2=0.146). The overall effect of Cue was significant for good decoders regardless of hemisphere, *F*(1,15)=10.61, *p*=.005 (ηp2=0.414). However, poor decoders showed a significant Cue x Hemisphere interaction, *F*(1,12)=7.94, *p*=.016 (ηp2=0.398), such that the effect of Cue was significant in the right hemisphere (*p*=.011, ηp2=0.427), but not the left hemisphere (*p*=.310, ηp2=0.086). The main effect of Hemisphere was non-significant for both groups and there were no significant between group differences (*p*s>.05).



a)



b)

*Figure 3.* Grand averaged waveforms for good decoders (left) and poor decoders (right) at posterior sites for the 0ms (a) and 500ms (b) SOA conditions (LC=large cue-compatible; LI=large cue-incompatible SC=small cue-compatible; SI=small cue-incompatible).

*Figure 4.* Mean N1 amplitude for good decoders (left) and poor decoders (right) as a function of Cue-size and Hemisphere.

There was also a significant SOA x Cue x Flanker x Hemisphere x Group interaction, *F*(1,27)=7.69, *p*=.010 (ηp2=0.222). To further elucidate these effects, separate analyses were conducted for each SOA and cue-size condition. For stimuli presented with large cues in the 0ms SOA (simultaneous) condition there was a significant Flanker x Hemisphere x Group interaction (Figure 5), *F*(1,27)=13.24, *p*=.001 (ηp2=0.329). The Flanker x Hemisphere interaction was significant for both good, *F*(1,16)=14.27, *p*=.002 (ηp2=0.471), and poor decoders, *F*(1,13)=4.89, *p*=.045 (ηp2=0.273), but there was a reversal of hemispheric effects. Good decoders showed significantly greater N1 amplitude for incompatible relative to compatible flankers in the right hemisphere, *F*(1,16)=6.75, *p*<.019 (ηp2=0.297), and this tended to be greater in comparison to poor decoders, *F*(1,29)=5.02, *p*=.033 (ηp2=0.148) (*p*>.025, Bonferroni corrected). In contrast, poor decoders tended to show greater N1 amplitude incompatible relative to compatible flankers in the left hemisphere, *F*(1,13)=3.81, *p*=.073 (ηp2=0.227).

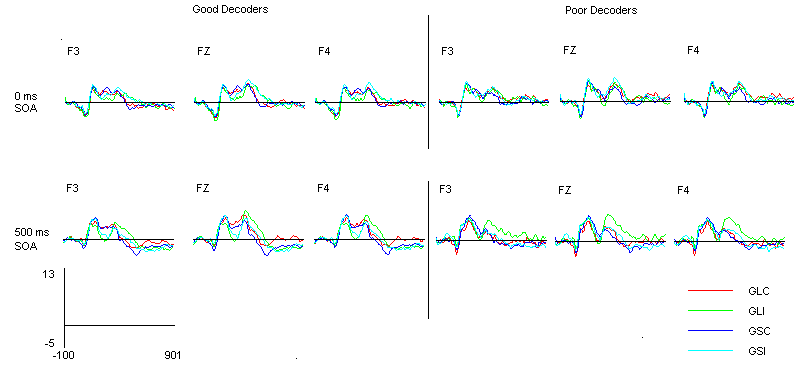
 

*Figure 5.* Mean N1 amplitude at posterior sites for good decoders (left) and poor decoders (right) for large cues in the 0ms (simultaneous) condition as a function of hemisphere and flanker.

*3.3.2. Anterior N2 Amplitude*

Figure 6 shows grand averaged waveforms at frontal sites for good decoders and poor decoders as a function of SOA, Cue, and Flanker. N2 amplitude was significantly greater overall for the 0ms SOA (simultaneous) condition (*M*=-1.90, *SD*=2.68) in comparison to the 500ms SOA (cued) condition (*M*=-0.32, *SD*=2.62), *F*(1,27)=12.32, *p*=.002 (ηp2=0.313), for incompatible (*M*=-1.58, *SD*=2.53) than compatible (*M*=-0.65, *SD*=2.46) flankers, *F*(1,27)=8.95, *p*=.006 (ηp2=0.249), and for large (*M*=-1.43, *SD*=2.69) compared to small (*M*=-0.795, *SD*=2.11) cues, *F*(1,27)=8.03, *p*=.009 (ηp2=0.229).

The Flanker x Group interaction approached significance, *F*(1,27)=3.48, *p*=.073 (ηp2=0.114), such that good decoders, *F*(1,15)=9.53, *p*=.008 (ηp2=0.389), but not poor decoders, *F*(1,12)=1.06, *p*=.324 (ηp2=0.081), showed significantly greater N2 amplitude for incompatible compared to compatible flanker stimuli. There were no significant between group differences (*p*s>.05).



*Figure 6.* Grand averaged waveforms at frontal sites for good decoders (left) and poor decoders (right) for the 0ms SOA and 500ms SOA as a function of cue-size and flanker.

Note: LC=large cue-compatible; LI=large cue-incompatible SC=small cue-compatible; SI=small cue-incompatible).

*Correlational analyses*

To examine the relationship between behavioural and electrophysiological measures flanker (incompatible minus compatible) and cue-size effects were calculated for each dependent measure (RT, N1 amplitude, N2 amplitude) and Pearson correlations were conducted (Table 2). For RT cue-size effects, the cost of small cues was calculated for compatible trials (small minus large cues) and the cost of large cues was calculated on incompatible trials (large minus small cues), with positive values indicating a greater RT costs. For N1/N2 cue-size (small minus large) and flanker effects (incompatible minus compatible), a more negative value indicates a greater effect. For good decoders, there was a strong negative relationship between nonword reading scores and RT cue-size effects on incompatible trials. In contrast, for poor decoders, there was a strong negative relationship between nonword reading scores and RT cue-size effects on compatible trials, and strong positive relationships between RT cue-size effects (compatible and incompatible) and N1 cue-size effects in both the left and right hemisphere. For RT flanker effects in the presence of a small spatial cue, there was a strong negative association with the N2 flanker effect for good decoders, and strong negative associations with the N1 cue-size effects for poor decoders. For RT flanker effects in the presence of a large spatial cue, there was a strong negative correlation with N1 cue-size effects in the right hemisphere for good decoders and no significant associations for poor decoders.

Table 2.

*Pearson correlation coefficients between behavioural, electrophysiological, and reading measures among good and poor decoders*

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | RT Cue-size effect (compatible) | RT Cue-size effect (incompatible) | | RT Flanker effect  (small cue) | | RT Flanker effect  (large cue) | |
| **Good decoders** |  |  | |  | |  | |
| N1 cue-size effect (LH) | .144 | .009 | | -.296 | | -.426 | |
| N1 cue-size effect (RH) | .107 | .011 | | -.375 | | -.615\*\* | |
| N1 flanker effect (LH) | -.452 | -.236 | | .543\* | | .324 | |
| N1 flanker effect (RH) | -.318 | -.107 | | .355 | | .248 | |
| N2 flanker effect | .377 | .379 | | -.647\*\* | | -.428 | |
| Nonword reading score | -.006 | -.545\* | | .268 | | -.151 | |
| **Poor decoders** |  | |  | |  | |  | |
| N1 cue-size effect (LH) | .674\*\* | | .590\* | | -.656\* | | .341 | |
| N1 cue-size effect (RH) | .537\* | | .631\* | | -.612\* | | .206 | |
| N1 flanker effect (LH) | -.106 | | -.261 | | .301 | | .272 | |
| N1 flanker effect (RH) | .178 | | -.422 | | .027 | | .030 | |
| N2 flanker effect | .020 | | -.197 | | .026 | | -.081 | |
| Nonword reading score | -.535\* | | -.220 | | .433 | | -.250 | |

Note: flanker effect (incompatible minus compatible trials); compatible RT cue-size effect (small minus large cues); incompatible RT cue-size effect (large minus small cues); N1/N2 cue-size effects (small minus large cues)

\**p*<.05, \*\**p*<.01, \*\*\**p*<.001

1. **Discussion**

When targets were flanked by incompatible flankers, RT costs were observed for large relative to small spatial cues, suggesting that when the spatial scale of attention is smaller and more precise, the effect of flanking stimuli is reduced ([Ericksen & St James, 1986](#_ENREF_12); [Facoetti & Molteni, 2000](#_ENREF_16)). However, when stimuli were compatible, RT tended to be shorter for large relative to small cues suggesting that facilitation effects for compatible stimuli were greatest when attention was more broadly focussed ([Callejas et al., 2005](#_ENREF_5); [Facoetti & Molteni, 2000](#_ENREF_16)). However, there were differential effects of flanker compatibility and cue-size for each group. While good decoders showed significant flanker effects across all conditions, poor decoders showed a reduction in the flanker effect for trials involving small spatial cues, particularly under the simultaneous condition. This is partially consistent with the hypothesis that poor decoders would benefit less from small spatial cues, but was largely attributable to less benefit of compatible flanking stimuli. Relatedly, good decoders also benefited more from compatible flankers when they fell within the bounds of the large cuing stimulus. In contrast, and consistent with the second hypothesis, poor decoders showed a greater cost of incompatible flankers when they fell within the bounds of a large cuing stimulus.

Broadening the scale of spatial attention increases flanker interference ([Facoetti & Molteni, 2000](#_ENREF_16)) and the ability to ignore flankers is more difficult when the focus of attention needs to be shifted ([Callejas et al., 2005](#_ENREF_5)). Thus the latter finding suggests that poor decoders were less able to either focus spatial attention, and/or suppress information from flanker locations, and is consistent with behavioural research showing that dyslexics are more receptive to flanker interference during the performance of similar tasks ([Bednarek et al., 2004](#_ENREF_1); [Brosnan et al., 2002](#_ENREF_3); [Facoetti & Turatto, 2000](#_ENREF_19); [Klein & D'Entremont, 1999](#_ENREF_33); [Martelli et al., 2009](#_ENREF_42)). While poor decoders were expected to benefit less from small spatial cues, this wasn’t necessarily predicted to be greatest on compatible trials, and may reflect less benefit from parallel processing of flanker stimuli despite the small focussing cue when compared to good decoders.

While correlational analyses should be interpreted with caution due to sample size limitations and restricted ranges, these behavioural effects were related to phonological decoding ability. For poor decoders lower nonword reading scores were associated with greater cue-size effects on compatible trials (i.e., greater RT costs for small cues). In contrast, for good decoders, lower nonword reading scores were associated with greater cue-size effects on incompatible trials (i.e., greater costs for large cues).

Notably, this is the first study to examine the electrophysiological mechanisms underlying such behavioural effects. Posterior N1 amplitude was significantly greater for small relative to large cues suggesting that it was modulated by the spatial scale of attention ([Luo et al., 2001](#_ENREF_38)). As expected, this effect was greatest for the 500ms SOA (cued) condition when the effects of spatial attention were maximised ([Castiello & Umilta, 1990](#_ENREF_6)). Good and poor phonological decoders showed hemispheric differences in the modulation of N1 amplitude as a function of cue-size. Overall N1 amplitude was modulated by cue-size in both hemispheres for good decoders but only in the right hemisphere for poor decoders. This suggests hemispheric differences in early selective attention processes, but is not necessarily consistent with the right hemisphere parietal deficit that has been proposed to underlie attentional difficulties in dyslexia ([Hari et al., 2001](#_ENREF_26); [Stein & Walsh, 1997](#_ENREF_71)). It does however suggest hemispheric differences in the focussing of spatial attention and less recruitment of left hemisphere resources which is consistent with other research in dyslexic populations. For example, functional imaging research has shown under activation of left temporo-parietal language areas during the performance of reading tasks ([Shaywitz et al., 2006](#_ENREF_66)), including evidence for increased activation following a phonological intervention ([Shaywitz et al., 2004](#_ENREF_65)).

N1 amplitude was also modulated by flanker compatibility, such that greater N1 amplitude was observed for incompatible relative to compatible flankers. The modulation of N1 amplitude by flanker compatibility is consistent with the theory that N1 amplitude reflects the gradient of visual attention ([Mangun & Hillyard, 1991](#_ENREF_40)) or the demands of visual discrimination within the focus of attention ([Vogel & Luck, 2000](#_ENREF_77)). For stimuli presented with large cues in the simultaneous condition, good decoders showed significantly greater N1 amplitude in the right hemisphere for incompatible relative to compatible flankers, and this effect tended to be greater in comparison to poor decoders. In contrast, poor decoders tended to show a flanker effect in the left rather than the right hemisphere. These hemispheric differences in focussing the spatial scale of attention to letters in the presence of incompatible distractors may underlie the greater flanker interference effect observed in RT for poor decoders and are consistent with findings from a previous study showing a reduction in N1 modulation among poor phonological decoders as a function of the spatial scale of attention in a sustained global/local attention task ([Matthews & Martin, 2009](#_ENREF_44)).

These latter findings also provide support for the right hemisphere deficit hypothesis of dyslexia (e.g., [Facoetti & Turatto, 2000](#_ENREF_19); [Hari et al., 2001](#_ENREF_26); [Stein & Walsh, 1997](#_ENREF_71)). For example, previous research has indicated that children with dyslexia show a reduction in the flanker effect in the left visual field and a strong flanker effect in the right visual field ([Facoetti & Turatto, 2000](#_ENREF_19)). Given that patients with temporo-parietal lesions show a reduction in flanker effect in the contralesional visual field, and a larger effect in the ipsilesional visual field ([Ro, Cohen, Ivry, & Rafal, 1998](#_ENREF_62)), these findings were argued to reflect a right hemisphere posterior parietal deficit. Previous fMRI research has also demonstrated reduced activation in the right parieto-occipito-temporal cortex during an attentional orienting task among dyslexic relative to normal readers ([Heim et al., 2010](#_ENREF_28)).

Taken together the differences in N1 amplitude suggest differences between good and poor decoders in terms of early selective attention processes that are mediated by the fronto-parietal orienting network. While poor decoders recruited right hemisphere resources when attentional focussing was cue-driven, they recruited the left rather than the right hemisphere to selectively attend to the target in the presence of irrelevant flankers.

There was also evidence for processing differences in the frontal executive control network. As expected, frontal N2 amplitude was modulated by flanker compatibility reflecting the inhibition of irrelevant information ([Gratton et al., 1988](#_ENREF_25); [Heil et al., 2000](#_ENREF_27); [Kopp et al., 1996](#_ENREF_34)). This N2 flanker effect was greater for good decoders suggesting less top-down inhibition of irrelevant flankers for poor phonological decoders. This may explain the greater flanker interference effects on RT found among poor phonological decoders in the present research. Previous research has also shown reduced modulation of N2 amplitude as a function of global/local processing among poor phonological decoders ([Matthews & Martin, 2009](#_ENREF_44)).

The fronto-central N2 component is thought to index cognitive control processes involved in the inhibition of conflicting information ([Folstein & Van Petten, 2008](#_ENREF_22); [Heil et al., 2000](#_ENREF_27)). Among other areas, the anterior cingulate cortex and its interaction with the dorsolateral prefrontal cortex have been implicated in flanker interference effects ([Zurawska Vel Grajewska, Sim, Hoenig, Herrnberger, & Kiefer, 2011](#_ENREF_84)) and the topographical profile of the N2 component is consistent with activity in these areas ([Yamaguchi, Toyoda, Xu, Kobayashi, & Henik, 2002](#_ENREF_82)). One theory is that the anterior cingulate monitors conflicting information and acts on the dorsolateral prefrontal cortex to increase selective attention to task relevant information and suppress task irrelevant information ([Weissman et al., 2005](#_ENREF_79); [Zurawska Vel Grajewska et al., 2011](#_ENREF_84)). Bednarek et al. (2004) suggested that the behavioural impairment exhibited by dyslexics when targets were flanked by incompatible flankers indicated a deficit in executive control rather than orienting or alerting ([see also Kelly, Best, & Kirk, 1989, for a prefrontal cortical hypothesis of dyslexia](#_ENREF_32)). However, neuroanatomical models of attention propose an interaction between fronto-parietal attentional networks involved in orienting and executive control. For example, top-down feedback projections from frontal areas, feedback projections from parietal areas, and bottom-up sensory information can all modulate early visual processing to influence attentional selection (e.g., [Corbetta & Shulman, 2002a](#_ENREF_9); [Posner, 2004](#_ENREF_56); [Posner & Peterson, 1990](#_ENREF_57); [Shipp, 2004](#_ENREF_69)). Of particular relevance, the right frontal eye fields (FEF) have been implicated in controlling the size of attention focus ([Ronconi, Basso, Gori, & Facoetti, 2014](#_ENREF_63)), and also interact with areas of the intraparietal sulcus and the superior parietal lobe as part of the dorsal goal-directed attentional network (Peterson & Posner, 2012). There is also overlap between selective attention and reading in terms of the involvement of brain areas such as the inferior parietal lobe and the prefrontal cortex ([Shaywitz & Shaywitz, 2008](#_ENREF_67)).

In the present research, selective attention was examined among a sample of adults with good and poor phonological decoding skills. While it is likely that many participants would have satisfied the criteria for a clinical diagnosis of dyslexia, the present findings cannot be directly generalised to dyslexia. However, poor decoders also showed significantly lower scores on other measures of single word reading and reading in context when compared to good decoders, with scores on non-word reading falling between the 2nd and 37th percentile of the norming group. Furthermore, while there were clear processing differences between good and poor decoders, is not possible to determine whether these effects were causally related to decoding ability or whether they represent processing strategies which may have developed to compensate for decoding abilities ([Shaywitz & Shaywitz, 2008](#_ENREF_67); [Shaywitz et al., 2003](#_ENREF_68)).

The present research has focused on amplitude modulation of specific ERP components which are thought to index specific attentional mechanisms. While the examination of ERP latencies was beyond the scope of the present research, latency analyses may provide further insight into speed of processing and is an area for future research. Similarly, examination of longer latency ERP components may shed further light on the processing differences between these groups.

Given evidence for differences in selective attention among dyslexic populations, interventions aimed at improving attentional skills may be important in the remediation of dyslexia. For example, in a recent study, a 12 hours of playing action video games was found to result in an improvement in both reading speed and attentional skills in dyslexic children ([Franceschini et al., 2013](#_ENREF_24)). The present work suggests that examination of specific ERP components could be used to determine the mechanism underlying such improvements. For example, correlational analyses, while preliminary, suggest differential relationships between behavioural performance and N1/N2 amplitude for each group.

In summary, poor decoders showed less benefit of compatible flankers (particularly when segregated by small cues), which may relate to reduced parallel processing of competing letter representations. In addition poor decoders showed a greater RT cost for incompatible stimuli preceded by large cues, indicating differences in voluntarily focussing the spatial scale of attention and suppressing information at unattended locations. The inhibition of distractors is thought to be particularly important for successful reading ([LaBerge & Brown, 1989](#_ENREF_35)) and for developing grapheme phoneme conversion skills ([Brosnan et al., 2002](#_ENREF_3)). The present study is one of few to examine the underlying electrophysiological correlates of the spatial attention difficulties observed in dyslexia. Together the N1 findings suggest hemispheric differences in attentional focussing. For cue-driven attention (cue-size effects), N1 modulation was found in both hemispheres for good decoders and only the right hemisphere for poor decoders. In contrast, when large cues were presented simultaneously with the target, and voluntary focussing was required, N1 amplitude was modulated by flanker compatibility in the right hemisphere for good decoders and in the left hemisphere for poor decoders, supporting the right hemisphere deficit hypothesis of dyslexia. There were also differences in processing in the frontal executive control network, with less modulation of the frontal N2 component among poor decoders compared to good decoders. Together these findings have important implications for understanding how specific selective attention mechanisms relate to reading ability. While the present research has specifically examined people with good and poor phonological decoding skills, further research is required to examine electrophysiological indices of attentional processes among children and adults with a diagnosis of dyslexia, particularly among those with a primary phonological decoding deficit.

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