Is visuospatial attention different for people suffering from dyslexia?

An EEG study using Lateralized Power Spectra

T. Verwoert

Supervisors:

Dr. R. H. J. van der Lubbe

Dr. M. L. Noordzij

University Twente, Enschede, the Netherlands

Faculty of Behavioral Sciences

Department for Cognitive Psychology and Ergonomics

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Abstract

This study measured the electrocephalogram (EEG) while dyslectics and controls performed an endogenous cueing task to check for differences in visuospatial attention. A neuropsychological test battery focusing on dyslexia, and different aspects of attention was carried out as a separate measure. Event related lateralizations (ERLs), the lateralized power spectra (LPS) and the lateralized power spectra, computed on event related potentials (LPS-ERP) were analyzed and checked for group differences. The LPS analysis revealed mostly increased ipsilateral theta and alpha power on occipital and parietal sites in controls as compared to dyslectics, which might be related to a dysfunction in the dorsal/magnocellular pathway of dyslectics. The difference in lateralization between dyslectics and controls on posterior parietal sites may be linked to reversing and rotating letters or words, a common issue reported in dyslectics.

Introduction

Dyslexia is the most common learning disability that affects both children and adults. It is a disorder in which people with normal intelligence and motivation fail to read as accurately and fluently as their peers (Démonet, Taylor, & Chaix, 2004; Shaywitz & Shaywitz, 2001). The estimated amount of people that suffer from dyslexia is widely discussed and varying percentages are reported in the literature ranging from 4% to 17% (Gabrieli, 2009; Shaywitz & Shaywitz, 2001; Wijers, Been, & Romkes, 2005; Xu, Yang, Siok, & Tan, 2015). McBride et al. (2008) argued that dyslexia is a neurological disorder with a dysfunction in the left-hemisphere language network. Functional brain imaging studies showed a failure of the left hemispheric posterior brain systems in dyslectics when performing a reading task (Schlaggar & McCandliss, 2007; Shaywitz & Shaywitz, 2001; Xu, et al., 2015). A study of Wijers et al. (2005) focused on analyzing visuospatial attention using EEG data of both hemispheres and compared these using lateralizations. They found differences in lateralization between dyslectics and controls that hint

at a difference in visuospatial processing. Since the study of Wijers et al. (2005) found promising results and not much research has been done using lateralizations, this study replicated and extended the methods employed in the study of Wijers et al. (2005). To understand possible differences in language processing of dyslectics, recent literature will be discussed and a model describing language processing is introduced. Based on this literature and model, the main hypotheses concerning language processing in dyslexia will be discussed.

Language processing

A useful starting point to understand how people process language is the triangle model of the lexicon (see Figure 1). This model proposes that there are bidirectional relations between orthography (spelling), phonology (sound) and semantics (meaning) (Halligan & Wade, 2005).

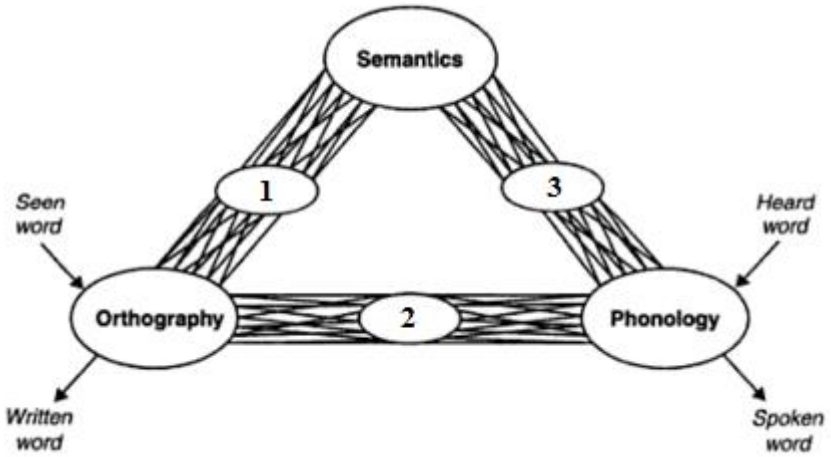

Figure 1. A graphical representation of the triangular lexicon model, which specifies bidirectional relations between orthography (spelling), phonology (sound) and semantics (meaning) when processing language. Adapted from Halligan and Wade (2005).

According to the model there are two ways in which semantic knowledge can be activated by the visual presentation of a word. When confronted with visual features (e.g., letters), the reader can transform these letters into phonemes and then interpret the meaning of the words (via routes 2 and 3 in Figure 1). Alternatively, it is possible to go to semantics directly

(route 1 in Figure 1). On the basis of this model, one might propose that dyslexia is the result from a dysfunction along either an orthographical-phonological or an orthographical-semantic pathway. The triangular model can be related to a model of Price (2000) to describe the anatomy of language processing using functional neuroimaging. The orthographical-semantic pathway (route 1) is thought to follow the following route: visual information is perceived by the eyes and via the retina it goes to the posterior fusiform gyrus (also referred to as Visual Word Form Area) and lingual gyrus, which can be related to the orthography node in the triangular lexicon model. Consequentially, to process information from orthography to semantics, posterior inferior temporal/mid-fusiform areas are involved, followed by the extrasylvian temporo-parietal area of which the latter can be linked to the semantics node of the triangular lexicon model. The orthographical-phonological pathway (route 2) is similar up to the point of reaching the posterior inferior temporal/mid-fusiform area, then is linked to the posterior superior temporal sulcus, which is related to process acoustics or phonology (Price, 2000).

Current hypotheses on dyslexia

Several hypotheses have been proposed to understand dyslexia: the magnocellular hypothesis (Stein & Walsh, 1997), the cerebellar hypothesis (Nicolson, Fawcet & Dean, 2001), the phonological theory (Bradley & Bryant, 1978) and the rapid auditory processing theory (Tallal, 1980; Ramus et al., 2003). Since the current study focuses on visuospatial attention, the first two seem most relevant and will be discussed in more detail.

The magnocellular hypothesis

The magnocellular hypothesis was first postulated by Stein and Walsh (1997), they found a connection between magnocellular cells in the visual system and dyslexia. Facoetti, Lorusso, Paganoni, Umilta and Mascetti (2003) suggested that dyslectics suffer from magnocellular dysfunction (i.e., neurons part of the visual system). This can be related to the orthographical node of the triangular lexial model, since both are based on visual input. To

understand the magnocellular hypothesis, it is important to know that visual information goes through the visual pathway and is divided by two paths, a dorsal and a ventral stream (Vidyasagar & Pammer, 2010). The dorsal stream processes movement, depth, visually guided actions, spatial localization and rapid changes (Heth & Lavidor, 2015). The dorsal stream for 90% relies on rapid input from the magno cells (Benasich & Fitch, 2012). The second, ventral, stream is thought to be concerned with recognizing and perceiving objects and gets input from primarily the parvo cells (Goodale & Milner, 1992). Parvo cells are sensitive to colors and contribute to observing in a high resolution, they are smaller and slower to respond than magno cells. Magno cells on the other hand are larger cells that are sensitive to rapid changing stimuli and moving objects (Jaśkowski & Rusiak, 2005; Stein & Walsh, 1997). Studies showed that the magno cells of dyslectics were smaller than the ones from controls, while the parvo cells were the same size (Gori, Cecchini, Bigoni, Molteni & Facoetti, 2014; Livingstone, Rosen, Drislane & Galaburda, 1991). Next to the difference in size, it was found that the lateral geniculate nucleus (LGN) of dyslectics was smaller in the left hemisphere while the right LGN was normal (Giraldo-Chica, Hegarty & Schneider, 2015). How these differences in size influence functionality is not properly understood yet. The dorsal dominated, magnocellular pathway may be fully responsible for controlling attention after the action is highly automatized, which is the case for reading (Milner & Goodale, 1995). The magnocellular pathway starts at the ganglion cells in the retina, goes through the magnocellular layer of the LGN and then goes to the occipital and parietal cortex (Gori, Molteni & Facoetti, 2016; Talcott, Hansen, Willis-Owen, McKinnell, Richardson & Stein, 1998; Qian & Bi, 2016). It is thought that in dyslectics this stream is impaired while the parvocellular dominated stream is intact (Gori et al., 2014). Importantly, the dorsal pathway is thought to play an important role in visual processing which is related to the posterior parietal cortex (PPC). The PPC is related to covert spatial orienting, visually guided movements, spatial attention and eye movements (Gori, Cecchini, Bigoni,

Moleteni & Facoetti, 2014; Jaśkowski & Rusiak, 2005). Chouake, Levy, Javitt and Lavido (2012) mentioned that a correlation exists between visual word recognition and the magnocellular pathway, they also argued that this might explain the lower reading abilities of dyslectics. Additionally, studies showed that the PPC is related with rotating and reversing visual stimuli, which is a common issue reported in dyslectics (Jaśkowski & Rusiak, 2005; Stein, 1997). Schulte-Körne and Bruder (2010) argued that the magnocellular deficit hypothesis is one of the most important theories for understanding dyslexia, evidence for this theory is primarily provided by potentials evoked using altered visual and rapid moving stimuli which are presented at low contrasts.

Cerebellar hypothesis

The cerebellar hypothesis was forwarded by Nicholson and Fawcett (1990), they indicated that dyslectics have problems automating reading and motor control. The latter is important because motor control influences speech articulation and this is thought to lead to deficient phonological representations (Ramus et al., 2003). Dysfunction in the cerebellum has been linked to multiple developmental disorders, for instance autism, attention deficit-hyperactivity disorder and dyslexia (Nicholson, Fawcett & Dean, 2001; Stoodley, 2016). Neuro-imaging studies have shown that dyslectics have anatomical anomalies within the cerebellum and an atypical symmetry of the cerebellum exists in dyslectics (Jaśkowski & Rusiak, 2005). Moreover, Stoodley (2016) argued that right-sided damage to the cerebellum is associated with poorer language outcomes and left-sided damage of the cerebellum with visuospatial attention, which both are relevant when reading. Some studies linked the magnocellular hypothesis and the cerebellar hypothesis together, which argued that one does not exclude the other and both could be true (Jaśkowski & Rusiak, 2005; Stein, 2001).

Critique on the magnocellular hypothesis

Nevertheless, there are critics of the magnocellular hypothesis like Norton, Beach and Gabrieli (2015). They argued that most neuroimaging studies concerning dyslexia have been conducted with children or adults that have had years of reading difficulty and that it is therefore impossible to determine whether the differences in brain structures are the cause of dyslexia or if these are a consequence from years of altered and reduced reading experience. They also mentioned that the reduced middle temporal activity can be solely observed in adults and argued that the reduced middle temporal activity is not visible when dyslectic children were compared with children of the same reading ability. Gori, Seitz, Ronconi, Franceschini and Facoetti (2015b) countered this argument by making a comparison between older dyslectic children and younger controls, which were matched for their reading abilities and their future reading development. Even when controlling for age and only analyzing children they found deficits in magnocellular dorsal function in dyslectics. Gori et al. (2015b) also argued that a magnocellular dorsal pathway deficit in dyslexia can be observed before the processing of orthography to phonology takes place and is visuospatial attention related. This implies that the deficit is visible before learning how to read and can be found at young age and in children among all languages. This makes the magnocellular hypothesis a promising explanation for the difficulties dyslectics have and would suggest it can be diagnosed at a very young age.

Assessing visuospatial attention

As described above there is still debate on what exactly causes dyslexia, employing new techniques like in Wijers et al. (2005) to assess the brains of dyslectics might bring more clarity. Wijers et al. (2005) proposed to study lateralizations of attentional control using electroencephalographic data (EEG) while participants were completing a cueing task. The cueing task Wijers et al. (2005) used to measure visual attention is similar to the Posner cueing task (Posner, 1980). This task could be used to assess an individual's ability to shift attention

between left and right cues, in this case in dyslectics and controls. Wijers et al. (2005) found a difference in frontal attention effects in dyslectics, controls showed an effect mostly over the right hemisphere, while the dyslectics showed an effect in both hemispheres. They did this by employing Event Related brain Potentials (ERPs) and constructing and analyzing Event Related Lateralizations (ERLs) of collected EEG data.

When analyzing ERLs, it will possibly reveal three different components. These are formulated as the early directing attention negativity (EDAN), the anterior directing attention negativity (ADAN) and the late directing attention positivity (LDAP) (Harter, Miller, Price, LaLonde, & Keyes, 1989). The EDAN is a contralateral negativity with a peak above the occopito-parietal areas (around the electrodes PO7/8 and O1/2) at approximately 200-400 ms after cue onset (Meyers, Walther, Wallis, Stokes & Nobre, 2015; Van der Lubbe & Utzerath, 2013). The EDAN is thought to show the first stage of spatial orienting, by selecting the relevant part of the cue (Van der Lubbe, Neggers, Verleger & Kenemans, 2006; Van Velzen & Eimer, 2003). The ADAN follows 400 ms after cue onset which is also characterized by greater negativity but at electrodes more anterior compared to EDAN. The ADAN is associated with activity of premotor cortex and the frontal eye fields (Amso & Sceriff, 2015; Van der Lubbe & Utzerath, 2013). It is thought that the ADAN plays a role in saccadic inhibition, because participants are told to do so while fixating in the Posner task (Van der Lubbe et al., 2006). Lastly, the LDAP represents a late positivity which is maximal above posterior sites around 500-700 ms after cue onset and is thought to be involved with modulating activity of attention (Van der Lubbe & Utzerath, 2013). Wijers et al. (2005) found the EDAN and LDAP components in their study and revealed a difference in frontal hemispheric lateralization of attentional control in the study of Wijers et al. (2005). Assessing lateralizations seemed a useful measure to assess possible attention related differences in dyslectics compared to controls and this study tried and replicate these findings with other dyslectics and controls.

We propose something similar to the study of Wijers et al. (2005), however we extended the techniques used. Both ERLs and ERPs do not account for possible changes in internally generated activity. For this reason we additionally analyzed Lateralized Power Spectra (LPS) by using wavelets on EEG, which were first proposed by Van der Lubbe and Utzerath (2013). LPS is a double subtraction method proposed by Van der Lubbe and Utzerath (2013) to assess raw EEG data and this method is comparable to the method employed to calculate Lateralized Readiness Potentials (LRP) and Event Related Lateralizations (ERLs).

Additionally, using LPS makes it possible to observe if the generated activity is of an induced, rather than an evoked nature (Van der Lubbe & Utzerath, 2013). This is done by employing yet another form of analysis, and is done by applying the LPS procedure on ERPs, which results in the LPS-ERP. Then it is possible to check if ipsi-contralateral differences are present in the LPS results while not present in the LPS-ERP results, this would suggest the activity is internally generated. If the difference is both visible in the LPS and the LPS-ERP results, this implies the activity is externally evoked. Additionally, the LPS-ERP results might reveal effects in specific time windows at certain locations that are not assessable in ERLs due to differences between participants (Van der Lubbe & Utzerath, 2013).

Aim of the present study

By studying particular aspects of non-linguistic visual processing in isolation (such as magnocellular function or eye movements), research can begin to disentangle cause from effect in developmental dyslexia. The aim of this study was to replicate and extend the study of Wijers et al. (2005) and moreover, if there was a difference in visual attention between dyslectics and controls. In this research, EEG data will be analyzed for dyslectics and a control group when performing the Posner cueing task. As was previously found by Wijers et al. (2005), we expected to find the three ERL components and a difference in frontal amplitude between dyslectics and controls. By extending the techniques used by Wijers et al. (2005), differences

in power were expected in the (posterior) parietal cortex between dyslectics and controls on the LPS since this an important area of the dorsal stream of the magnocellular system. Lastly, visuospatial attention of dyslectics and controls were assessed by comparing the results of the LPS procedure and the LPS-ERP, this revealed whether specific processes were evoked by the cue or were induced by internally generated fluctuations in activity in the brain. Next to the EEG data, behavioral data (reaction time and errors made) was collected of the participants when performing the Posner cueing task. It was hypothesized that dyslectics would be slower and less accurate than the controls on the Posner task, especially when faced with invalid trials.

Methods

Participants

A total of 26 participants (15 males and 11 females) participated in this study, all but two (one in the experimental group and one in the control) were found to be right-handed with the Annett's Handedness Inventory (Annett, 1970). The experimental group consisted of 11 participants of which 6 were male and 5 female, the control group therefore comprised of fifteen participants of whom 10 were male and 5 female. They were recruited using the SONA database to recruit local students at the University of Twente, M_{age} was 21.7. All participants were checked for visual impairments, 24 of the participants had normal or corrected-to-normal vision. None of the participants were colorblind, and had no history of neurological diseases apart from dyslexia for the experimental group. Before starting the experiment the participants signed an informed consent and the used procedures were approved by a local ethics committee at the Faculty of Behavioral Sciences at the University of Twente.

Task and stimuli

The participants were checked for dyslexia by employing a small dyslexia test battery using the DST^{NL} (dyslexia screening test), the Trail Making Test, the Bourdon-Wiersma Test, and the Balloons Test. These neuropsychological tests are clinically used to assess visual search, visual attention, attentional neglect, and task switching. In this study they served as a separate measure to check for differences in visuospatial attention of the dyslectics and controls.

The main task consisted of a variant of the Posner (1980) endogenous cuing paradigm. The task in total consisted of 672 trials, separated in blocks of 168 trials each, and these were preceded by 20 practice trials. The total duration of the task was approximately 70 minutes. An overview of a trial shown in Figure 2, a default display consisted of a white fixation point centered on the screen with a black background, together with two open light grey circles at the left and right of this fixation point in the middle.

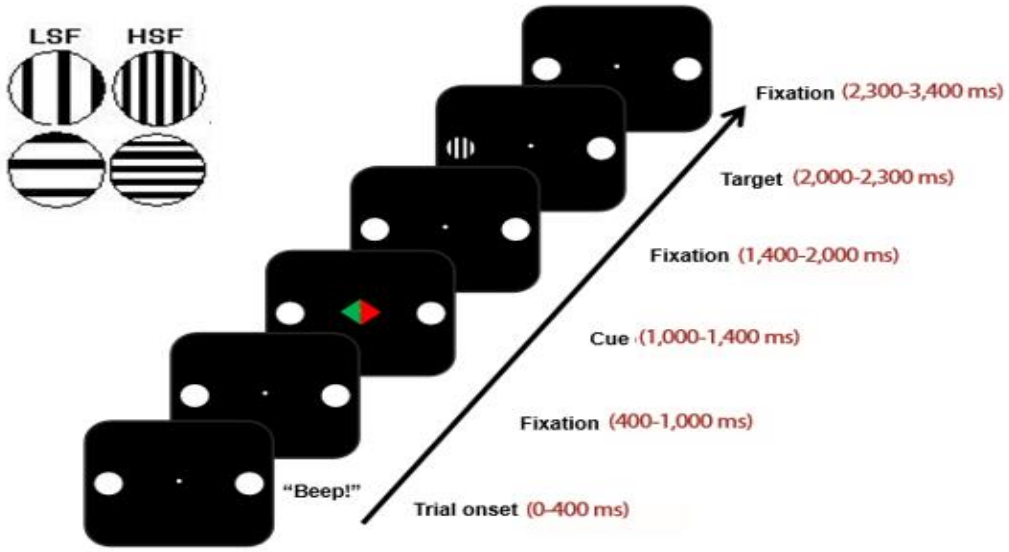

Figure 2. An example of a trial of the Posner (1980) task used. Four types of targets were used, they were either high or low in spatial frequencies (HSF or LSF) and had either a vertical or horizontal orientation. The participant had to press a key corresponding to the side the cue appeared in the smallest amount of time.

The start of a trial was indicated by an auditory signal while the fixation dot increased in size and luminance. Participants were instructed to direct their eyes towards the fixation point in the center. After 600 ms a diamond-shaped cue appeared in the center instead of the fixation dot, the cue consisted of two colored triangles pointing to the left and right circles. One of the triangles was shown in red and the other in green of which one was defined as relevant, in the first half of the task, half of the participants were informed that the circle where the green triangle pointed to, was the most probable location of the cue appearing (80% of the trials, i.e., valid trials). On 15% of the trials the target appeared on the other side (where the red triangle was pointing to, i.e., invalid trials) whereas on 5% of the trials, no target occurred (catch trials). In the second part the participants were informed that the red triangle indicated the most probable target location, this process was counterbalanced for the other half of the participants. After 400 ms of showing the diamond-shaped cue, the cue disappeared and 600 ms of fixation on the white dot followed before the actual target appearing. Several types of targets were used; they were either high or low in spatial frequencies (HSF or LSF), had either a vertical or horizontal orientation and were presented either left or right. The participant had to press the left button when a target with a horizontally striped pattern was presented in the smallest amount of time and the right button when a vertically striped target was presented. Correspondence and non-correspondence of the response side and the cue side were measured (e.g., a corresponding trial consisted of a left sided target that required a left button response and generally show faster reaction times, this effect is known as the Simon effect (Van der Lubbe, Bundt & Abrahamse, 2014)). After 300 ms of showing the target, 1.100 ms of fixation followed before starting a new trial.

Apparatus and EEG recordings

The participants were seated on a comfortable chair in a darkened room at approximately 70 cm from a 17-inch monitor. The program 'Presentation' (Neurobehavioral Systems, Inc., 2012) was used to run the Posner task which was installed on a separate computer. The left and right target buttons ("Ctrl" keys) were pressed with the left and right index fingers on a standard QWERTY keyboard.

EEG was recorded using Ag/AgCl ring electrodes placed on standard scalp sites according the extended 10-20 system at 61 locations mounted in an elastic cap (Braincap, Brain Products GmbH). Next to EEG, electro-oculogram (EOG) was applied by using a horizontal and vertical electro-oculogram (hEOG and vEOG). This was done by applying electrodes above and below the left eye and by placing them at the canthus of the left and right eye. Electrode gel and standard procedures were used to improve conductivity so electrode resistance was always under 5kΩ. The EEG and EOG were amplified using a 72-channel QuickAmp (Brain Products GmbH), this amplifier had a built-in average reference. The data collected was registered using BrainVision Recorder (Brain Products GmbH), which was installed on a separate acquisition computer.

Data processing

The neuropsychological pre-tests were scored using their standard scoring procedures. The data of the Posner task was analyzed with IBM SPSS 20 (IBM Corporation) using repeated measures. Averages of reaction time (RT) and proportion of correct (PC) were acquired with the markers from the EEG/EOG recordings. The marker data were analyzed by employing Matlab. RT and PC are a function of Cue Validity (validly or invalidly cued targets), Spatial Frequency of the target (low or high), Correspondence (corresponding or non-corresponding) and Target Orientation (horizontal or vertical). These data were analyzed by employing a repeated measures analyses of variance (ANOVA) with $\alpha \leq 0.05$.

The EEG data was processed using BrainVision Analyzer 2.0 (Brain products GmbH). The first step in data processing was to partition the data in segments from -500 to 2500 ms relative to the cue onset. Trials where eye movements were found on critical moments somewhere from 0 to 1000 ms after cue onset have been removed, the hEOG and vEOG channels were used to exclude values exceeding +/- 40 μV for the hEOG and +/- 120μV for the vEOG, where the 40 μV corresponds with a horizontal movement of approximately 2 degrees (Van der Lubbe & Utzerath, 2013; Van der Lubbe & Woestenburg, 1997). This left on average 64% of the trials. This strict procedure was done to ensure that observed effects were not due to saccades made by the participants. Furthermore, this made sure that the effects of cue validity on behavioral measures were not due to overt orienting instead of covert orienting.

EEG analyses of the cue-target interval

EEG channels containing artifacts were removed (gradient criterion: 100 μV per 1 ms, min-max criterion: -/+ 150 μV, low activity criterion: 0.1 μV for 50 ms; individual channel mode), the number of removed channels was very low (< 1%). ERPs were computed for left and right cues, then ERLs were made by employing the procedure done by Wascher and Wauschkuhn (1996). Subsequently, a wavelet analysis was carried out on single trials on all EEG channels, a Complex Morlet wavelet (c = 5) was used. For the LPS and LPS-ERP analyses seven different frequency bands were used. The frequency range was separated in seven logarithmic steps, resulting in bands covering the lower θ to upper β range, ranging from 4 till 20 Hz. The LPS and the LPS-ERP were calculated using the methods discussed in Van der Lubbe and Utzerath (2013). The LPS formula returned a value between -1 and +1, where a positive value indicated that the power within the used frequency band was larger in the ipsilateral hemisphere compared to the contralateral hemisphere. A negative value indicated the opposite; the power of the band was larger in the contralateral hemisphere compared to the

ipsilateral hemisphere. A value of zero shows there was no difference in ipsi-contralateral difference on the band in terms of power.

An analysis of all time windows (windows of 20 ms each, from 0-1000 ms after cue onset) of all electrode pairs was done to find time windows that had a significantly deviant value compared to zero. Since multiple tests were done, a correction for multi testing has been done. This was achieved by applying the procedure of Talsma, Wijers, Klaver and Mulder (2001) which resulted in a critical p-value of $\alpha = 0.006$ and will be used for the initial ERL, LPS and LPS-ERP analyses to determine which time windows were relevant for the group difference analysis. Two or more consecutive significantly deviant windows were then checked for group differences on the relevant electrode pairs and frequency bands ($\alpha \leq 0.05$). The critical value of 0.05 was chosen because the previous procedure with two consecutive time windows of $\alpha \leq 0.006$ was felt to be conservative enough to minimize the false discovery rate.

Results

Neuropsychological tests

The dyslectics were somewhat older (2.7 years) compared to the controls (M_{age} = 23.15 and 20.43 years, respectively). To control for this, age was introduced as a covariate in the analyses of the neuropsychological tests, behavioral data of the Posner task and the EEG. This showed that age differences were not responsible for observed effects and was therefore left out as a covariate in the results below. One female dyslectic participant was excluded from analysis of neuropsychological data due to previous experience with the tests used.

The scores on the dyslexia screening test (DST^{NL}) were analyzed using a multivariate test yielding a significant difference between groups $F(6, 20) = 4.8$, $p = 0.003$. The dyslectics scored especially poor on the nonsense words subtest compared to controls (75.93 vs. 127.00), $F(1, 27) = 27.6$, $p < 0.001$. An age related difference was found on the one minute writing

subtest, $F(1, 27) = 4.3$, $p = 0.049$. The test provided norm scores but since not all subtests were carried out this norm score has been corrected. Scoring lower than 38 indicated a high risk of dyslexia whereas scoring higher indicated normal psycholinguistic functioning. A difference was found in the sum of norm scores between the groups, where controls scored significantly higher than dyslectics (57.9 vs. 46.8), $t(24) = 4.2$, $p < 0.001$, $d = 1.71$. Using a one-sampled t-test the controls were found to be scoring higher than the norm of 38, $t(13) = 12.9$, $p < 0.001$, $d = 7.16$. The dyslectics were also found to score higher compared to the norm, $t(12) = -3.8$, $p = 0.002$, $d = 2.19$.

Differences between means of the Bourdon, Trail making and Balloons tests were analyzed using an ANOVA (see Table 1). Further investigation of the data yielded no significant differences between controls and dyslectics.

Table 1

Mean scores for the Bourdon and Trailmaking tests (with standard deviations)

Group	Test							
	Bourdon (in sec)	Bourdon (omissions)	Trailmaking A (in sec)	Trailmaking B (in sec)	Balloons A (in sec)	Balloons A errors	Balloons B (in sec)	Balloons B errors
Control	12.63 (2.02)	15.35 (12.07)	26.54 (7.92)	48.26 (13.00)	33.21 (20.55)	0.14 (0.54)	97.89 (36.60)	0.93 (1.82)
Dyslectic	11.60 (1.67)	16.25 (12.10)	24.92 (5.44)	58.13 (26.87)	30.48 (11.80)	0.08 (0.30)	78.20 (28.56)	2.17 (3.27)

Behavioral measures Posner task

It was found that the reaction time (RT) was faster of controls compared to dyslectics (773 vs. 903 ms), $F(1, 24) = 6.8$, $p = 0.15$, $\eta_p^2 = .22$. RT was faster on validly cued than on invalidly cued trials (808 vs. 868 ms), $F(1, 24) = 56.5$, $p < .001$, $\eta_p^2 = .70$. The RT on High Spacial

Frequency (HSF) targets was slower compared to the Low Spatial Frequency (LSF) targets (respectively 889 vs. 787 ms), $F(1, 24) = 64.1$, $p < .001$, $\eta_p^2 = .73$. No significance was found in reaction time in high or low frequency of the target between the groups. An interaction effect in reaction time has been observed between frequency, correspondence and group, $F(1, 24) = 5.04$, $p = 0.034$, $\eta_p^2 = .17$. Further analysis of this interaction revealed that when dyslectics were faced with a high spatial, non-corresponding target, they would have the highest RT.

No group difference was observed on percentage correct (PC). Further analyses on PC revealed that responses on valid cues were answered slightly more correct compared to invalid cues (83.0 vs 80.6%), $F(1, 24) = 7.6$, $p = .011$, $\eta_p^2 = .24$. HSF trials were answered significantly less correct than LSF trials, respectively 70.2 vs. 93.4%, $F(1, 24) = 92.6$, $p < 0.001$, $\eta_p^2 = .79$. No significant difference has been observed in corresponding or non-corresponding trials. An interaction effect in percentage correct was found between frequency and correspondence, $F(1, 24) = 6.6$, $p = 0.017$, $\eta_p^2 = .22$. Further analysis showed that corresponding targets of a HSF was hardest to answer correctly (66.4%), while a non-corresponding HSF target was (74.0%) was second hardest to answer correct. Their counterparts of LSF were both answered correct for over 93% of the cases.

EEG analyses

The most relevant findings of the ERL are summarized in Table 2 below and mostly consist of electrode pairs above occipital and parietal regions. The EDAN and ADAN components were less pronounced and therefore less visible but the LDAP component was highly pronounced and visible between 540 and 660 ms (see Figure 3). A group difference analysis of the ERL findings resulted in no significant differences between dyslectics and controls.

Table 2

A summary of findings of the ERL analysis when the significance criterion was crossed for at least two successive time windows ($\alpha \leq 0.006$)

ERL			
Component	Window	Maxima	t(25)
LDAP	540-660	P5/PO3	3.2* - 6.6**
	560-640	P1	3.1* - 4.0**
	560-660	P3/P7/O1/PO7	3.1* - 5.9**
	580-660	CP5/TP7/FT7	3.1* - 4.6**
	600-660	T7	3.0* - 3.0*
	620-660	CP3	3.1* - 3.1*

Note. Effects are described in contra-ipsilateral differences. ERL = event related lateralizations; LDAP = late directing attention positivity. *$p \leq 0.006$, ** $p < 0.001$.

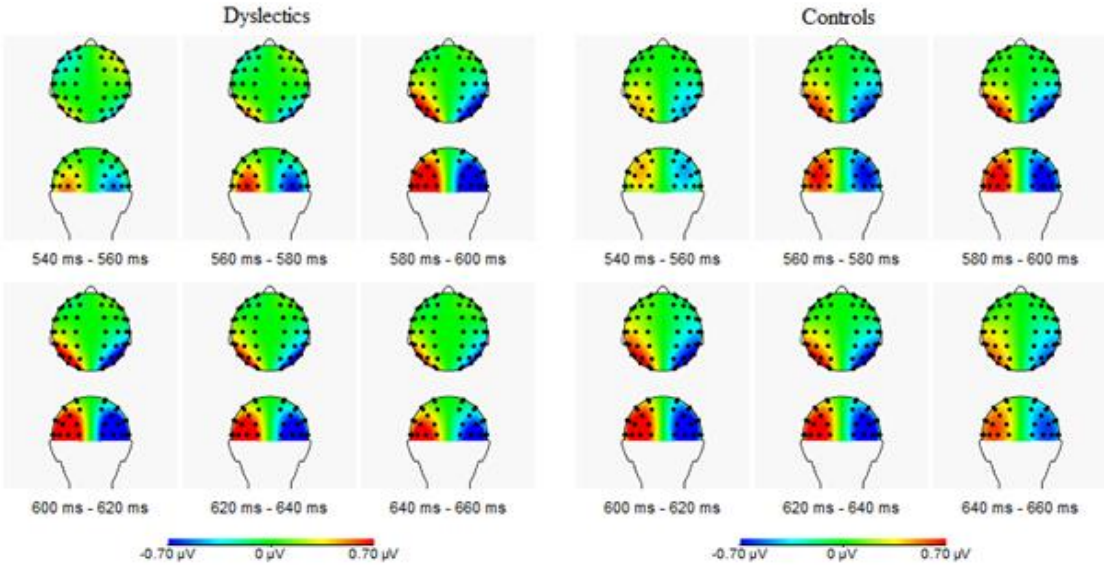

Figure 3. Topographical maps of the event related lateralizations in 20 ms windows between 540-660 ms after cue onset based on interpolation of spherical splines (fourth order), averaged separately for both dyslectics and controls. In the left hemisphere, the contra-ipsilateral difference map is displayed, whereas a mirrored ipsi-contralateral difference map is displayed for the right hemisphere.

LPS analysis

The LPS effects that were significantly deviant from 0 for two consecutive time windows ($\alpha \leq 0.006$) are shown in Appendix A. The analysis revealed significant deviant time windows on all bands and mostly occurred in the frontal, central, parietal and occipital regions. These time windows were used for a group analysis of dyslectics and controls ($\alpha \leq 0.05$), a summary of these LPS results are presented in Table 3. On the θ_2 band significant differences between the groups have been found on the P1/2 electrodes between two time windows, 600-640 ms, the controls showed increased ipsi-contralateral power compared to the dyslectics (0.029 vs. 0.003, p = .030, d = .94; 0.030 vs. 0.002, p = .018, d = 1.03). For the same band a difference has been found on the FC5/6 electrodes for three consecutive time windows between 600-660 ms. The controls showed decreased ipsi-contralateral power compared to dyslectics (-0.029 vs. -0.006, p = .041, d = .88; -0.029 vs. -0.004, p = .029, d = .95; -0.028 vs. -0.003, p = .026, d = .97). In the α_1 band differences have been found on the P7/8 pair between 900-1000 ms, where the controls showed more ipsi-contralateral power than the dyslectics (0.063 vs. 0.007, p = .024, d = .99; 0.065 vs. 0.005, p = 0.008, d = 1.18; 0.066 vs. 0.004, p = .004, d = 1.31; 0.068 vs. 0.005, p = 0.003, d = 1.32; 0.070 vs. 0.006, p = 0.005; d = 1.26). The PO7/8 electrodes also show a difference on the α_1 band (between 920-1000 ms, see Figure 4), the controls again showed more ipsi-contralateral power compared to the dyslectics (0.066 vs. 0.002, p = .005, d = 1.49.; 0.069 vs. 0.000, p = 0.002, d = 1.74; 0.072 vs. -0.000, p = 0.001, d = 1.80; 0.074 vs. 0.000, p =.001, d = 1.73). Topographical maps were made for the θ_2 and the α_1 bands of the relevant time windows, see Figures 5 and 6, respectively.

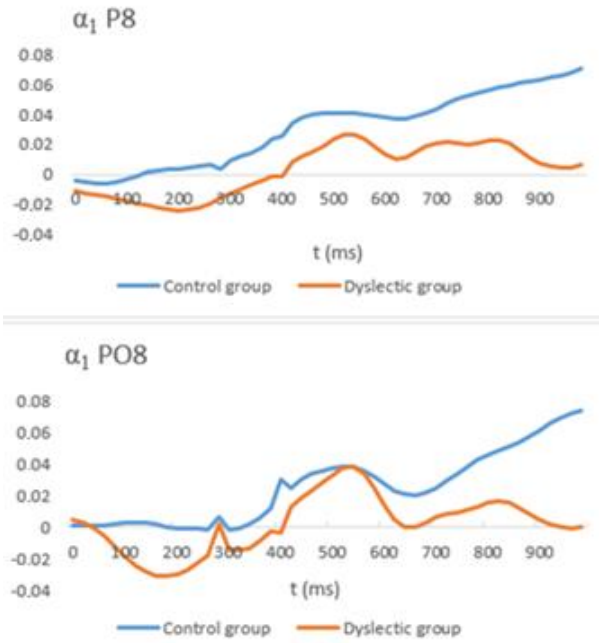

Figure 4. Grand average lateralizations of the P8 and PO8 electrodes on the α_1 band with group differences of the lateralized power spectra. On the PO7/8 electrodes there is a significant difference in ipsi-contralateral power between the groups on 920-1000 ms and on the P7/8 electrodes also an ipsi-contralateral difference can be seen between 900-1000 ms.

Table 3

A summary of findings of LPS analysis on the raw EEG when the significance criterion was crossed for at least two successive time windows ($\alpha \leq 0.006$) and after, with a significant difference between dyslectics and controls ($\alpha \leq 0.05$)

Wavelets		LPS	
Band	Window (ms)	Maximum	F(24)
θ_2	600 - 640	P2	5.3* - 6.4*
θ_2	600 - 660	FC6	4.7* - 5.6*
α_1	900 - 1000	P8	5.8* - 10.5**
α_1	920 - 1000	PO8	8.0* - 13.6**

Note. Effects are described in ipsi-contralateral differences. LPS = the lateralized power spectra. *$p \leq 0.05$. **$p < 0.005$.

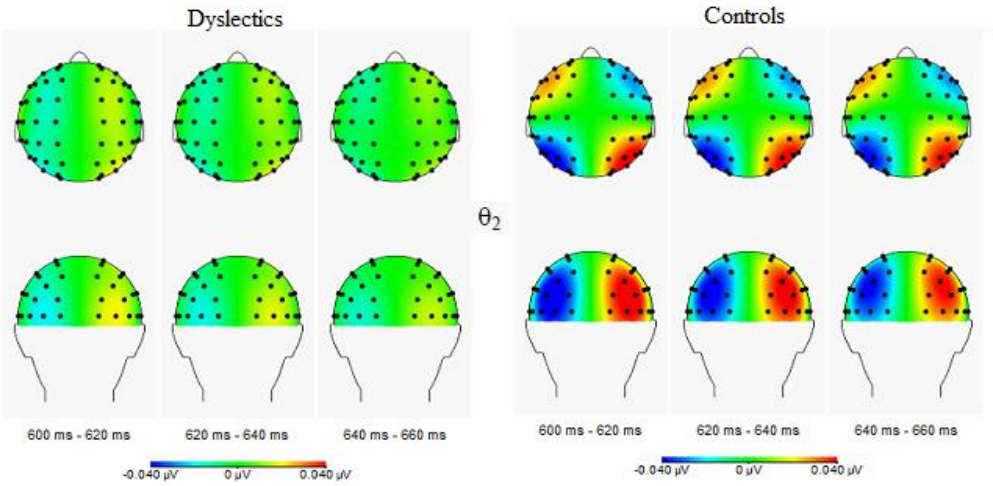

Figure 5. Topographical maps for the θ_2 band for time windows 600-660 ms after cue onset, in which a significant difference in power was observed between dyslectics and controls. The left hemisphere reflects the contra-ipsilateral power difference, whereas the right hemisphere displays the ipsi-contralateral power difference. Positive values in the right hemisphere means increased ipsilateral as compared to contralateral power.

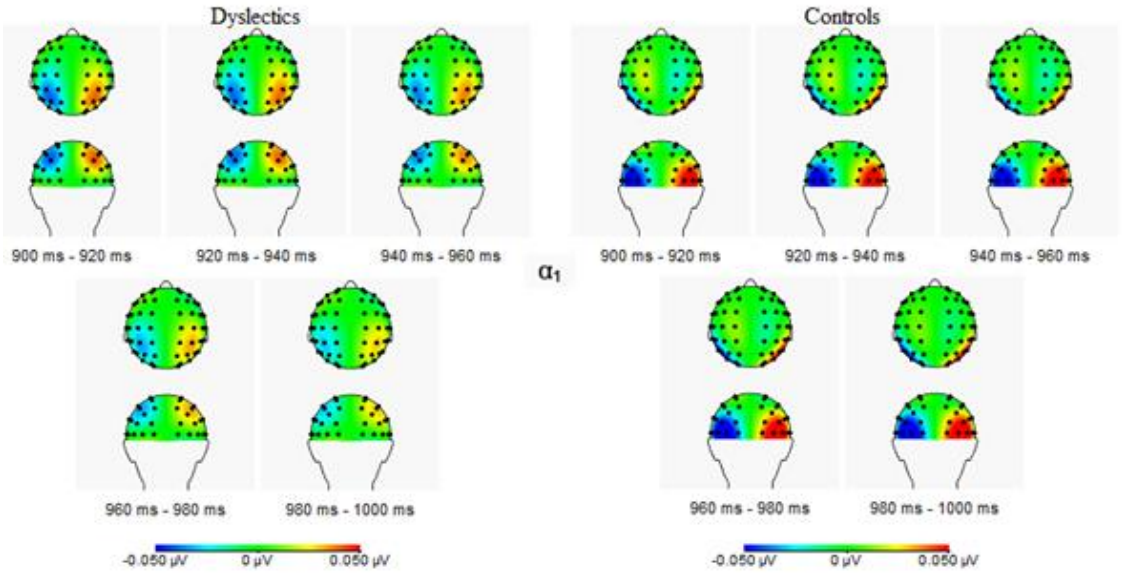

Figure 6. Topographical maps for the α_1 band for time windows 900-1000 ms after cue onset, in which a significant difference in power was observed between dyslectics and controls. The left hemisphere reflects the contra-ipsilateral power difference, whereas the right hemisphere displays the ipsi-contralateral power difference. Positive values in the right hemisphere means increased ipsilateral as compared to contralateral power.

LPS-ERP analysis

As in the LPS analysis, in the LPS-ERP analysis at least two consecutive time windows had to be significantly deviant from 0. Analysis revealed significant deviant time windows on all bands and mostly occurred in the parietal and occipital regions (see Appendix B). After determining the relevant time windows, differences between the two groups were analyzed. An effect was found for only one time window, in the electrodes P1/2 there was more ipsi-contralateral power in the control group compared to the dyslectic group on the α_1 band at 860-880 ms after cue-onset (0.036 vs. 0.303, p = .028, d = .95). A graph and topographical maps were made to illustrate this finding (see Figure 7 and Figure 8).

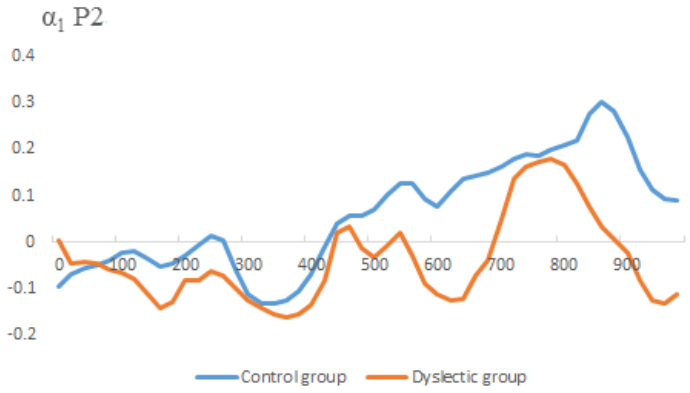

Figure 7. Grand average lateralizations of the P2 electrode on the α_1 band with group differences. There is a significant difference in ipsi-contralateral power between the groups on 860-880 ms.

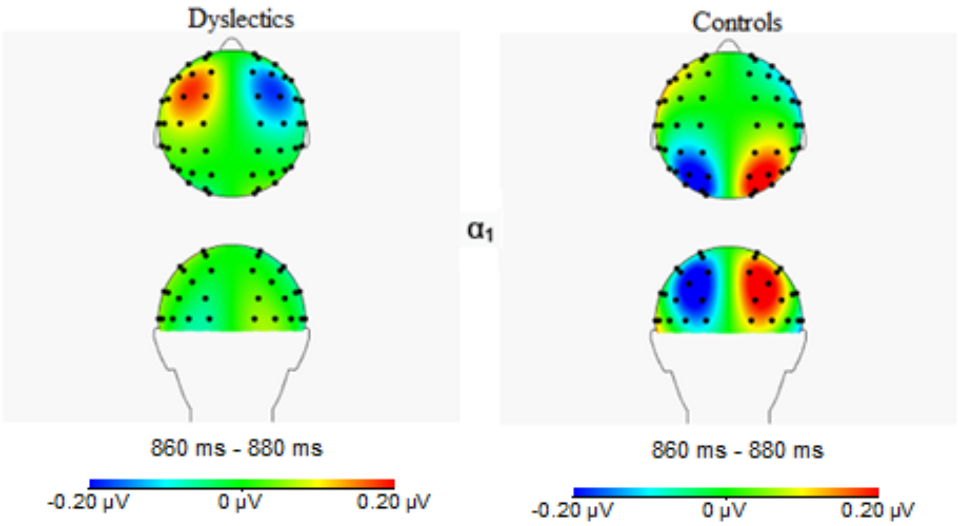

Figure 8. Topographical maps for the α_1 band for time windows 860-880 ms after cue onset, in which a significant difference in power was observed between dyslectics and controls. The left hemisphere reflects the contra-ipsilateral power difference, whereas the right hemisphere displays the ipsi-contralateral power difference. Positive values in the right hemisphere means increased ipsilateral as compared to contralateral power.

After checking both the LPS and the LPS-ERP for deviation from 0, the overlapping time windows of the belonging channels of the LPS and the LPS-ERP were checked (see Table 4). If differences in both the LPS and the LPS-ERP are present, this suggests this is due to an externally evoked event. When there is only a difference between the groups in the LPS and not the LPS-ERP, this suggests that the difference is caused by internally induced processes. None of the overlapping time windows showed a significant difference ($\alpha = 0.05$). This suggests that there are no differences in said time windows evoked by an external event and all differences are of an induced nature.

Table 4

Overview of the LPS and LPS-ERP with overlapping relevant time windows (in ms) that deviated significantly from 0 ($\alpha = 0.006$) with their corresponding frequency bands for at least two consecutive time windows

Band	LPS	Window (ms)	LPS-ERP	Window (ms)
θ_2	P6	440 - 660	P6	620 - 700
θ_2	PO4	460 - 680	PO4	560 - 720
θ_3	P6	480 - 1000	P6	460 - 560
θ_3	PO8	440 - 1000	PO8	520 - 580 & 720 - 760
α_1	PO8	440 - 600	PO8	540 - 580
α_1	P8	480 - 620	P8	520 - 560

Note: Effects are described in terms of ipsi-contralateral differences (therefore only the even electrodes are shown). LPS = lateralized power spectra. LPS-ERP = lateralized power spectra on event related potentials.

Discussion

The aim of this study was to replicate and extend the study of Wijers et al. (2005) that employed ERLs on EEG data. Trying to find differences in visuospatial attention for people suffering from dyslexia compared to controls and to provide evidence for the magnocellular hypothesis, using the Posner cueing task by assessing EEG data. Due to the nature of ERPs, and therefore ERLs, they may be less suited to employ on the onset of attentional orienting since this varied over trials and the chance exists that by using the standard averaging technique, the varying activity will be lost (Van der Lubbe & Utzerath, 2013). Therefore, it was hypothesized that there might be a difference in LPS when comparing dyslectics and controls,

due to controls more actively using posterior (parietal) reading systems when reading compared to dyslectics. For this reason another method was applied, namely the LPS and the LPS-ERP, which did not cancel out the trial to trial variation and may therefore be more suited to analyze the process of visuospatial attention.

Neuropsychological tests

Controls were better in completing the subtests of the DST^{NL} than the dyslectics, as expected, but dyslectics and controls both scored higher than the norm score of the DST^{NL}. An explanation of the higher scores for both the dyslectics and controls could be that the maximum age of the norm scores of the DST^{NL} were for sixteen year old children. The remainder of the neuropsychological tests (Bourdon, Trailmaking and Balloons) resulted in no differences between controls and dyslectics, which suggested that not one of the groups was better in visual search, visual attention, neglect or task switching in these measures. Since differences between the groups have been found in the EEG analyses, this potentially means that the neuropsychological tests were not sensitive enough or measured different concepts.

Posner task

Analysis of the Posner task revealed that dyslectics were slower on all types of trials compared to controls, but the amount of errors made was not different. Additionally, responses to valid targets were answered faster and more correct compared to responses on invalid targets, as was expected. It was found that when dyslectics were faced with a high spatial, non-corresponding target they needed the most time to answer. This corresponds to the literature suggesting that dyslectics show a sluggish attention shift (Krause, 2015). The delayed response of dyslectics on non-corresponding targets seem in line with the Simon effect, which suggests that when the target and the response button are on the same side, the reaction time is faster (Van der Lubbe, et al., 2014). Based on the behavioral measures there were no strong indications of attentional deficits in dyslectics.

EEG

Differences in amplitudes or power have been found in all the employed analyses (ERL, LPS and LPS-ERP). The ERL analysis only showed the LDAP component was prominently discernable, while both the EDAN and the LDAP were found in the study of Wijers et al. (2005). The ADAN and EDAN components were not observed in this study, although some difference in frontal power has been found 240 ms after cue onset which could be related to the ADAN component. Since the ADAN is thought to be of a more induced nature, it could be that it is present but not visible in the ERL analysis (Van der Lubbe & Utzerath, 2013). As hypothesized a difference in parietal and occipital lateralizations has been found and could be related to the LDAP component and was also found by Wijers et al. (2005). This could be an indicator of a difference in focusing visuospatial attention of dyslectics. Wijers et al. (2005) described that the LDAP could be related to attentional control processes but does not elaborate further. As described earlier, the PPC is linked to visuospatial attention and is thought to get its input from the dorsal/magnocellular pathway, a dysfunction in the magnocellular pathway might explain the difference in lateralizations on the parietal sites we found.

The LPS analysis showed a difference over fronto-central, parietal and parieto-occiptal sites (FC6, P2, P8 and PO8). These findings partly replicate the findings in Wijers et al. (2005), they hypothesized that a difference in frontal lateralization is present in dyslectics compared to controls, which was partly visible since dyslectics showed increased ipsilateral compared to contralateral power on a fronto-central site. The difference in the P2, P8 and PO8 electrodes were in line with expectations since a posterior ipsi-contralateral difference in power was expected in the controls compared to the dyslectics, which was similar to research supporting the magnocellular hypothesis. The difference in lateralizations (both the theta and alpha bands) might be explained by controls being able to inhibit irrelevant information while dyslectics have trouble to inhibit irrelevant information (Krause, 2015; Van der Schoot, Licht, Horsley &

Sergeant, 2002; Xu et al., 2015). Another plausible explanation for the difference in named electrodes is the influence of the PPC. The magnocellular pathway is thought to play an important function as input to the PPC and is used when a reading task is done. The PPC corresponds to our P2, P4 and P6 electrodes and might explain the difference in lateralization between dyslectics and controls in the LPS and LPS-ERP. Our findings support the idea that the input from the dorsal/magnocellular stream to the PPC dysfunctions which is mentioned earlier by a lot of studies (Gori et al. 2014; Gori et al. 2015a; Gori et al. 2015b; Jaśkowski & Rusiak, 2005; Vidyasagar & Pammer, 2010). Stein (1997) argued that monkeys with a lesion in the PPC were able to discriminate between visual stimuli but were unable to distinguish left-right reversals or rotations. It is commonly known that some dyslectics have related problems when reading and writing, they tend to change letters like the p and the q or reverse writing. In several other studies this link between the PPC and reversal and rotation errors in humans has been made too (Facoetti, Turatto, Larusso & Mascetti, 2001; Goswami, 2015; Jaśkowski & Rusiak, 2005). Other studies link dyslexia to a dysfunction in eye movements and motion processing, which is also thought to be functions of the PPC and could be closely related to the reversing and rotating of letters (Goswami, 2015). Since all the LPS findings were not visible in the LPS-ERP, it can be assumed that all findings were of an induced nature instead of externally evoked.

This study found, in both dyslectics and controls, mostly posterior differences in power which was hypothesized and could be related to the dorsal stream of the magnocellular hypothesis. The magnocellular hypothesis could be a possible explanation of what causes differences in amplitude between dyslectics and controls in this study. Gori et al. (2015b) found a causal relationship between the magnocellular pathway and dyslexia. They thought to have dealt with all critiques in the magnocellular pathway debate; they made a comparison between younger controls with the same reading ability as older dyslectics and their future reading

development. Additionally, they did two remediation studies in which the magnocellular pathway is specifically trained and reading improvement is established. They conclude that their results point strongly to a causal relationship between magnocellular dysfunction and dyslexia, this is shown in other research by them as well (Gori, Seitz, Ronconi, Franceschini, & Facoetti (2015a). Furthermore, they argued that, this will have repercussions for the diagnosis of dyslexia. The diagnosis can be done before reading and language disorders develop and early prevention programs can then drastically reduce the incidence of reading disorders. Gori et al. (2015a; 2015b) also stated that this settles the debate concerning magnocellular dysfunction not just being a consequence of impoverished reading experience.

In conclusion, dyslectics seem to focus visuospatial attention differently as was also found in Wijers et al. (2005). Dyslectics showed lower power ipsi-contralateral in the LPS on mostly (posterior) parietal and occipital sites, which have been related with the dorsal stream and the magnocellular hypothesis. The difference in lateralization of dyslectics around parietal sites could be linked to reversing and rotating of visual objects, which negatively influences the ability to read. Additionally, the capacity to inhibit irrelevant information or to disinhibit relevant information could be less developed in dyslectics.

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Appendix A

A summary of findings of LPS analysis on the raw EEG when the significance criterion was crossed for at least two successive time windows ($\alpha \leq 0.006$)

Wavelets		LPS		
Band	Window (ms)	Area	Maximum	t(25)
θ_1	200 - 560	Central-Parietal	CP2	3.0* - 4.7**
θ_2	220 - 660	Frontal-Central	FC6	3.0* - 3.5*
	420 - 680	Parietal-Occipital	PO8	3.0* - 5.3**
	440 - 660	Parietal	P6	3.2* - 4.4**
	540 - 660	Frontal-Central	FC6	3.0* - 3.5*
θ_3	220 - 260	Central	C4	3.0* - 3.1*
	440 - 1000	Parietal-Occipital	PO8	3.0* - 5.5**
	460 - 1000	Parietal	P6	3.1* - 5.3**
α_1	300 -380	Central-Parietal	CP4	3.3* - 3.9*
	440 - 1000	Parietal-Occipital	PO8	3.1* - 4.4**
	680 - 740	Tempo-Parietal	TP8	3.0* - 3.2*
α_2	500 - 580	Parietal-Occiptal	PO4	3.0* - 4.0**
β_1	520 - 600	Parietal-Occiptal	PO4	3.3* - 4.6**
	560 - 600	Parietal	P6	3.2* - 3.6*
	700 -740	Occipital	O2	3.2* - 3.2*
β_2	80 - 120	Central	C4	3.5* - 4.0*
	400 - 480	Frontal-Central	FC2	3.0* - 3.6*
	540 - 600	Parietal-Occiptal	PO4	3.2* - 3.4*

Note. Effects are described in ipsi-contralateral differences. Electrodes are clustered for area, most notable electrodes are shown. LPS = lateralized power spectra. *$p \leq 0.006$. **$p < 0.001$.

Appendix B

A summary of findings of the LPS-ERP analysis when the significance criterion was crossed for at least two successive time windows ($\alpha \leq 0.006$)

Wavelets		LPS-ERP		
Band	Window (ms)	Area	Maxima	t(25)
θ_1	580 - 700	Parietal	P6	3.1* - 3.5*
	580 - 700	Parietal-Occipital	PO8	3.0* - 3.1*
θ_2	200 - 240 & 560 - 720	Parietal-Occiptal	PO4	3.0* - 3.2*
	560 - 740	Central	C4	3.1* - 3.3*
	620 - 700	Parietal	P6	3.0* - 3.2*
θ_3	460 - 560	Parietal	P6	3.5* - 4.0**
	520 - 680 & 720 - 760	Parietal-Occipital	PO8	3.3* - 4.0**
α_1	260 - 340	Frontal-Central	FC6	3.0* - 3.6*
	500 - 820	Parietal-Occipital	PO8	3.0* - 3.9*
	520 - 880	Parietal	P2	3.1* - 3.7*
	620 - 660	Anterior-Frontal	AF4	3.3* - 3.4*
	940 - 980	Frontal	Fp2	3.0* - 3.1*
α_2	720 - 980	Posterior	P6	3.0* - 4.0**
β_1	460 - 500	Anterior-Frontal	AF8	3.0* - 3.1*
	840 - 980	Frontal-Central	FC2	3.3* - 4.2**
β_2	260 - 300	Parietal-Occiptal	PO8	3.1* - 3.4*
	820 - 860	Frontal	F2	3.5* - 3.5*

Note. Effects are described in ipsi-contralateral differences. Electrodes are clustered for area, most notable electrodes are shown. LPS-ERP = lateralized power spectra on event related potentials. *$p \leq 0.006$. **$p < 0.001$.