

# More to dyslexia than deficits in visual attention?

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## *Summary*

The aim of the experiment reported in this paper was to establish whether dyslectics have specific attentional problems. To this end the performance between a group of 12 adult dyslectics and 14 adult controls on a Posner task with endogenous attentional cuing was compared. In addition participants also made a number of clinically used pen and paper tasks. During the Posner task, EEG was measured. For the cue target interval lateralized event related potentials associated with attentional shifting were compared. One recent theory states that dyslexia is caused by abnormal functioning of the magnocellular pathways. The magnocellular system is responsible for handling fast sensory information and projects, in the case of visual information, onto the posterior parietal cortex. Therefore it is reasoned that dyslectics would suffer in tasks involving the posterior parietal cortex, like directing visual attention. Our results do not confirm the attentional hypothesis. No behavioral differences between dyslectics and controls were found for both the psychological tests and the Posner task. On the Posner task both groups benefited equally from attentional cues and subsequent attentional shifts. Analysis of EEG data in the cue target interval revealed the early directing attention component (EDAN) to be 40 ms longer in dyslectics. Reasoned is that this reflects different functioning of the posterior parietal cortex in dyslectics, unrelated to specific attentional problems but instead related to task-related aspects of the cue.

## *Introduction*

Developmental dyslexia is a disorder that is found in ten to fifteen percent of school aged children, and has been defined as a specific reading disorder that exists despite normal intelligence and schooling, in the absence of impaired vision or hearing (American psychological association 1994; Willows 1998; Willows and Terepocki 1993). The aim of the experiment in this paper was to establish if dyslectics have specific attentional problems. Attentional problems in dyslexia would be expected on the basis of the organization of the human visual system and previous research that found deviant functioning of this system in dyslectics. In this introduction, we'll turn to the physiology of the human visual system first. Second, the problems with low-level visual processing in dyslectics are looked at. Third, explained is how deviant functioning of such a system could lead to higher level problems like attentional deficits. Fourth, we pose several hypotheses and explain the experimental paradigm used to test them.

### *The human visual system*

The human visual system starts in the retina and on a sub-cortical level consists of two distinct systems (Ungerleider and Mishkin 1982). Ten percent of the retina consists of Magno cells and the other ninety percent of Parvo cells. Parvo cells are small, respond slowly, are sensitive to colors and have small receptive fields that contribute to high spatial resolution in vision. The bigger Magno cells are sensitive to fast changing stimuli and moving objects and are insensitive to colors (Palmer, 1999). From the retina visual information is relayed to the lateral geniculate nucleus (LGN). In the LGN the division between Magno and Parvo cells is maintained. These two systems are called the magnocellular or M-stream and the parvocellular or P-stream. Through the LGN these two streams reach their own layers in the extra striate cortex (V1).

Having reached a cortical level there are two pathways projecting from V1: A dorsal pathway that projects to the posterior parietal cortex (PPC), and a ventral pathway that projects to the inferior-temporal cortex (ITC). In a neuroimaging study using positron emission tomography Ungerleider and Haxby (1994) demonstrated this division to be present in humans. According to these authors the dorsal system deals with spatial aspects or the 'where' of visual information and the ventral system deals with the identification or the "what" of visual input.

The PCC is known to be involved in directing visual attention (Lynch, Mountcastle, Talbot, Yin 1977) and from clinical practice it is also known that damage to the PCC affects the ability to disengage visual attention (Posner et al., 1984). Research has also shown the PCC to be involved in guiding limbs and eye movements (Glickstein 2000; Glimcher, 1999). Neuroimaging studies have implicated the PCC in covert spatial orienting (Corbetta, Akbudak, Conturo, Snyder, Ollinger, Drury, 1998; Rosen, Rao, Caffarra, Scaglioni, Bobholz, Woodley, 1999). In covert spatial orienting, visual attention is directed towards a location without movement of the eyes. In the case of overt spatial orienting the eyes together with attention are directed to be attended location. According to Milner and Goodale (Milner and Goodale 1995) the dorsal route of processing extracts from the visual stream that information which serves to execute visually guided actions.

### *The magnocellular pathway and dyslexia*

Problems with low level visual processing in dyslectics was first demonstrated in a study by Lovegrove (1980). He found that dyslectics had a reduced sensitivity for high temporal and low spatial frequencies. Since then others have also found problems related to the magnocellular system. Problems with motion perception, flicker sensitivity and timing (see Farmer & Klein, 1995; Klein, 2002, for reviews). Motion sensitivity in dyslectics has been measured by presenting two panels with randomly moving dots. In one panel a certain percentage of the dots move together so they look like a cloud. This percentage is reduced until subjects begin to error when asked in which of the panels the dots are moving together. This yields a motion coherence threshold and differences between the thresholds of controls and dyslectics have been found (Cornelissen, Bradley, Fowler, Stein, 1994; Cornelissen, Richardson, Mason, Fowler, Stein, 1994b; Talcott et al., 1998, 2000b). Physiological evidence for an impaired magnocellular system in dyslectics came

from studies by Livingstone et al., (1991) and Galaburda and Livingstone (1993). They examined the brains of dyslectics post mortem and found that the neurons in the magnocellular layers of the LGC of the thalamus were some 30% smaller than those of controls.

In light of this research an M-stream theory of dyslexia has been proposed (Stein and Walsh 1997; Stein et al. 2000, 2001) According to this theory, impairments along the magnocellular pathway would cause problems with reading. Reading proficiency has been found to correlate with performance on an m-stream mediated task like coherent motion detection (Cornelissen, Bradley, Fowler, & Stein, 1994; Cornelissen, Hansen, Hutton, Evangelinou, Stein, 1998) and velocity discrimination (Demb, Boynton, Heeger, 1997). Reading achievement also correlates with fMRI activation produced by moving stimuli in the medial temporal area of the cortex and V1 (Demb et al., 1997; Eden, VanMeter, Rumsey, Maisog, Woods, Zeffiro 1996). Although it seems magnocellular function is disrupted the exact nature of these deficits and how they exactly relate to reading problems is unknown. The general idea behind the role of the magnocellular system in reading is that the symptoms of dyslexia would be caused by the visual demands of reading that draw on the magnocellular system. It is reasoned that any weakness in such a system can cause visual confusion of letter order and poor visual memory for written word. Problems with vergence control, ocular stability and saccadic movement would be able to cause difficulty perceiving letters. For a more detailed description of the proposed role the m-stream plays in reading see Boden and Giachi (2007).

Some researches dispute the magnocellular theory (Skottun, 2002). Some studies have failed to find any contrast sensitivity deficiencies in dyslectics (Ben-Yehudah Sackett, Malchi-Ginzberg, Ahissar, 2001; Williams, Stuart, Castles, McAnally, 2003). Some researchers have argued that magnocellular deficits are a separate disorder existing alongside dyslexia. Another possibility would be that magnocellular deficits are only present in a certain subtypes of dyslexia. Other researchers like Nicolson, Fawcett and Dean (2001) propose a purely cerebellar theory of dyslexia. Because it is known that cerebellar dysfunction impairs linguistic processing, abstract thinking, balance and acquisition and automatisisation of new cognitive procedures (Ito 1984, 1990; Krupa, Thompson, J.K, Thompson, J.F., 1993; Molinari et al. 1997). This cerebellar theory can explain a great number of the problems also associated with a deficient magnocellular system except those of low level visual processing. However it is also possible that, because the magnocellular system projects onto the cerebellum, deviant functioning of the magnocellular pathway could be responsible for cerebellar dysfunctions found in dyslectics (Nicolson, Fawcett 1999). For now an overview of such a discussion is beyond the scope of this paper and we limit ourselves to the link between PCC functioning, visual attention and dyslexia.

### *Attention and the PCC in dyslexia*

Considering the evidence about impaired functioning of the magnocellular system in dyslectics and the projection of this system on the PPC, the M-stream theory would predict that dyslectics would show signs of impaired PPC functioning. The idea of impaired PPC functioning because of inadequate M-stream input is not new (Vidyasagar 2004; see Jaskowski and Rusiak 2005 for reviews). Impaired PPC functioning could lead to symptoms of neglect, problems with directing visual attention, word recognition, and difficulty programming saccadic eye movements.

Recent studies have shown covert orienting deficits in individuals with dyslexia (Buchholz & Davies, 2005; Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000). In a recent study Facoetti and coauthors (2003a) found that dyslectic children did not gain any benefit from exogenous attentional cuing. During an exogenously cued shift of attention the attention of a participant is directed by using a cue at the spot of the to be presented stimulus. This in contrast to an endogenously cued shift in which the cue indicates the to be attended area. Experiments with another paradigm for measuring visual attention, the visual search task, have found abnormalities in dyslectics (Facoetti, Paganoni, & Lorusso, 2000). In such a task participants have to search for a stimulus in an array of other stimuli. Discrimination is made on the basis of one or multiple features. Iles, Walsh, and Richardson (2000) found that individuals with dyslexia who had elevated motion coherence thresholds showed impaired serial visual search, whereas those with normal thresholds were not impaired.

The link between PCC functioning and reading was demonstrated in a study using transcranial magnetic stimulation (TMS) (Braet & Humphreys, 2006). When adult proficient readers were required to name single and mixed case words as fast and accurately as they could, reading of both lowercase and mixed case (e.g. MiXeD) words was perturbed by TMS. The effect was greater on mixed case words. The authors proposed that the right parietal lobe mediates the recognition of words in unfamiliar formats.

Studies by Facoetti and Molteni (2001) found a mild form of neglect in dyslectic adults termed "mini-neglect". In a Posner task with peripheral and central cues which validly or invalidly cued the target location subjects had to react to the target side. Abrupt onset of the peripheral presented cues would present stimulus driven attention. Performance in normal readers benefited more from validly cued targets compared to invalidly cued targets. They also showed no asymmetry in stimulus preference. Dyslectics on the other hand showed no cue effect when the target was presented in the right visual field. Surprisingly, when the target was presented in the left visual field the cue effect was even greater in dyslexics than in normal readers. A smaller but similar asymmetry of cue effect was found when central cues were used. This last finding indicates that this attentional asymmetry also concerns the goal-driven attention. Asymmetry was also found in a study by Hari, Renvall, Tanskanen (2001) on a temporal order judgment task. In such a task two visual stimuli are presented to the left and the right of the fixation point. When asked to indicate the order of appearance it was found that dyslectics performed worse when the left stimulus was displayed first.

Hypothesized was that dyslectics would show problems directing their attentional spotlight, exhibit a mild form of unilateral neglect, both indicative of deviant PPC functioning, and would show a decreased sensitivity towards low spatial frequencies which is indicative of reduced effectiveness of the magnocellular pathway. In order to establish these we employed neuropsychological tests and an experiment based on a variant of the Posner task similar to the one used by Van der Lubbe, Neggers, Verleger, Kenemans (2006). EEG was used to record brain activity. For the cue target interval we expected that dyslexics would show differences in lateralized event related potentials (ERP) associated with attention.

During the Posner task used in this experiment controls and dyslexics had to respond, using either their left or right hand, to horizontal and vertical lines. Visuo-spatial frequency was varied by placing the lines closer together filling the circles with more or less lines. These lines were presented in two circles one at the left and one at the right side of the visual field. After trial onset a cue presented in the middle of the screen, at the fixation point, was used to indicate the to be filled circle. At the onset of this cue the participants had to covertly shift their attention to the indicated circle. Thus the Posner task was constructed in such a way that it measured the effects of attentional cuing by using a cue indicating stimulus location. The Posner task is sensitive to the Simon effect which presents itself as slower reaction times when visual stimuli are presented contra lateral to the side of response. Previous studies have found endogenously cuing in a Posner task to modulate the Simon effect (Abrahamse, Van der Lubbe 2007) which resulted in a decreased effect when the location was validly cued. If dyslectics would suffer from attentional deficits it's hypothesized that their performance would benefit less from an attentional cue and the cue itself would be less effective in modulating the Simon effect. This would be an indication that dyslectics have a problem shifting their attention towards the relevant location, a problem translating the attentional cue into an attention shift or a problem gaining the same performance related benefits from attention itself.

It was expected that dyslectics would show a mild form of unilateral neglect. Expected was that dyslectics would show slower reaction times towards stimuli presented on the left side of the screen. It was expected that dyslectics would have longer reaction times when the circles were filled with lines opposed to less. This would be a sign of insensitivity towards low spatial (LSF) frequencies.

The battery of neuropsychological tests consisted of a Bourdon, Trailmaking and Balloons test (Harcourt assessment, 1998). These tests are measures for visual attention, visual search, neglect and task switching and have a widespread clinical use. The trail making test consists of a piece of paper with randomly placed letters and numbers. During the trail making test participants had to use a pen to string letters and numbers together. Participants alternated between searching for letters and numbers stringing them together producing a sequence like 1A2B3C4D.. This test measured visual search and the capability to distribute attentional processes by switching between searching for letters and numbers. It was expected that dyslectics would perform worse on this test.

The Bourdon Test consisted of a sheet of paper with groupings of three, four and five dots.

Participants were instructed to look for groupings for and indicate them with pencil. This test is used to test attention, low level visual processing and concentration (Bourdon, B., Wiersma, E.D., 1910). Expected was that dyslectics would make more mistakes.

The Balloons test consisted of two A3 sized sheets of paper filled with balloons among circles in one condition (subtest A) and with circles among balloons in the other (subtest B). Participants are required to locate and put a line through balloons and circles. The Balloons test is based on the phenomenon of pop-out (Treisman and Gelade, 1980; Treisman and Gormican, 1988). Detection of balloons among the circles (as in subtest A), has been shown to be a relatively parallel process. The time taken to detect targets of this kind does not increase significantly as the number of distractors increases (Eglin, Robertson, Knight, 1989). In subtest B targets do not pop-out but instead have to be searched for in a serial fashion. Several studies show that serial search performance is much more impaired than parallel search in unilateral visual neglect (Humphreys, Riddoch, Quinlan 1989; Eglin et al., 1989). Expected was that dyslectics would be slower in the serial search task (subtest B), make more mistakes on the left side of both tests and would generally be slower completing the tests than controls. These would be signs of neglect and attentional problems respectively.

The EEG data was used to study three attention related potentials. These were first identified by Harter, Miller, Price, LaLonde and Keyes (1989) in a visual cuing paradigm: An anterior directing attention negativity (ADAN) which presents itself over the frontal lobes and peaks at 400 ms (Hopf & Mangun, 2000; Nobre, Sebestyen, Miniussi, 2000), an early directing attention negativity (EDAN) (e.g. Hopf and Mangun, 2000; Van der Lubbe, Jaskowski, Verleger, 2005; Yamaguchi, Tsuchiya, Kobayashi, 1994, 1995) that peaks roughly 300 ms after cue onset, and a late directing attention positivity (LDAP) which peaks at roughly 600 ms (Nobre et al., 2000; Yamaguchi, Tsuchiya, & Kobayashi, 1994). Both of them are found mainly above the occipito-parietal part of the cortex.

The ADAN has been thought to reflect processes involved in the control of the allocation of attention in anticipation of an expected task-relevant stimulus. This is in line with work by Posner and Peterson (1990) in which the frontal lobes have been shown to contribute to the attentional control of voluntary focusing and the maintenance of attention. The LDAP is assumed to reflect the modulation of the cortical excitability in regions involved in receiving the upcoming visual information. The EDAN is thought to reflect brain activity related to the processing of the cue and the resulting shifting of spatial attention. However another function for the EDAN has been proposed by Van Velzen and Eimer (2003). They placed a cue on the opposite side of where they indicated the target. So for example, a cue on the left side would indicate an attentional shift towards a target on the right side. They found the EDAN to be present contra lateral to the cue side in case of this example the right hemisphere. Since this is the case they reasoned the EDAN to reflect the selection of a task-relevant aspect of a cue. If the EDAN was related to the shifting of attention then activity would have been found contra lateral of the target side.

Considering there is still debate about what the attention related ERP's mean there are

several hypotheses making different predictions for what we might find in dyslectics. First It's hypothesized that problems with the shifting of visual attention would become evident in absent or deviant EDAN or ADAN. Secondly if the problem would lie in gaining benefit from visual attention and not the shifting attention itself this become visible in a deviant LDAP for dyslectics. Thirdly in line with Van Velzen and Eimer(2003) If dyslectics would have trouble recognizing the task relevant aspects of the cue this would manifest itself in differences at the EDAN component.

## Methods

### *Participants*

The experimental group consisted of 13 (7 male and 6 female) dyslectics. Twelve of them were confirmed to be right handed by using the Annet handedness inventory (Annet,1970). The control group consisted of 14 (9 males and 5 females) subjects. All of them except one were right handed. Both groups consisted of students recruited from the university population. All 27 participants gave informed consent prior to the experiment. One male participant from the dyslectic population was excluded from the EEG analysis because of difficulty completing the experimental procedure. Twenty four participants had normal or corrected to normal vision and intact color vision. Days for their participation the study was approved by the local ethics committee of the social sciences faculty of the University of Twente. Both groups had no history of neuronal disorder except for dyslexia in the experimental group.

### *Neuropsychological measures.*

The controls and dyslectics made a DST<sup>NL</sup> dyslexia screening test (NIP, 2005). The test consisted of subtests 3, 6, 7, 8a,8b, 9 measuring writing speed, spelling ability, reading of normal words, reading of nonsense words and working memory. Time to complete was measured using a stopwatch. Mistakes were scored afterwards in the case of spelling and scored directly in the case of reading.

The neuropsychological tests consisted of a Bourdon , Trailmaking and Balloons test. All instructions were given verbally and all tests were administered by the same individual. The trail-making test consisted of an A and a B each printed on an a4 sized piece of paper. The A part contained circles with numbers in them randomly placed on the paper . The B part contained circles with numbers and circles with letters also randomly placed on the paper. During the trail making test participants were instructed to use a pen to string numbers (Trailmaking A) and letters and numbers (Trailmaking B) together. In the A variant Participants had to search for numbers. In the B variant of the test participants had to make a trail between letters producing a string like 1,A,2,B,3,C .. and so forth until no numbers and letters remained.

The Bourdon Test consisted of a sheet of paper with groupings of three, four and five dots arranged in a series of 50 lines. Participants were instructed to inspect the paper line by line moving from left to right and from top to bottom indicating with pencil groupings of four. They were



instructed to do this as fast and accurately as they could. A stopwatch was used to register the time taken for each line. Afterwards tests were scored for omissions and errors.

The Balloons test is made up of two a3 sized sheets of paper containing randomly placed circles and balloons. Participants were instructed to search for the balloons in a pop out task (subtest A) and search for circles in a serial search task (subtest B) both conditions participants were instructed to be as fast and accurate as they could. In clinical settings the test would have a time limit of three minutes. Pilot testing revealed this to be long resulting in perfect test performance. Therefore time was introduced as an extra measure. All subjects were instructed to indicate when they were finished. After indicating this all subjects were asked if they were really sure. Time to complete, tracked with a stopwatch, was the interval from the start of the search to the second time subjects said they were finished

### *Stimulus Posner Task*

For the Posner task all stimuli were presented on a black computer screen. During each trial, a light-gray fixation dot ( $0.164^\circ \times 0.164^\circ$ ) was continuously presented in the center of the screen, accompanied by two light-gray open circles ( $r = 0.614^\circ$ ) located  $12.06^\circ$  to the left and right of the fixation dot default-display. Start of a trial was indicated when a sound was presented and the fixation dot increased in size and luminance (see fig1.). The enlarged fixation dot lasted for 400 ms, after which the default-display was presented for 600 ms. Next a centrally placed cue replaced the fixation dot for a duration of 400 ms. The cue was a diamond (height  $1.31^\circ$ , width  $2.62^\circ$ ) constructed of a green and a red triangle, each pointing to one of the circles. After the cue, the default-display was presented again for 600 ms. Thus, the preparatory interval from cue onset to target onset amounted to 1,000 ms. Next the target was displayed within one of the circles for a duration of 300 ms, consisting of either horizontal or vertical lines. Two lines (width  $0.25^\circ$ ) were used as a stimulus of low spatial frequency and 6 lines (width  $0.082^\circ$ ) were used as a stimulus with high spatial frequency. Participants had to react as fast as they could and after target offset the default-display was presented for another 1,100 ms. Participants were seated in a comfortable armchair in a silenced and darkened chamber, in front of a 17 screen monitor (DELL) at a distance of 70 cm. Presentation software (version 0.43 developed by Neurobehavioral Systems) was used for stimulus presentation and the production of external triggers.

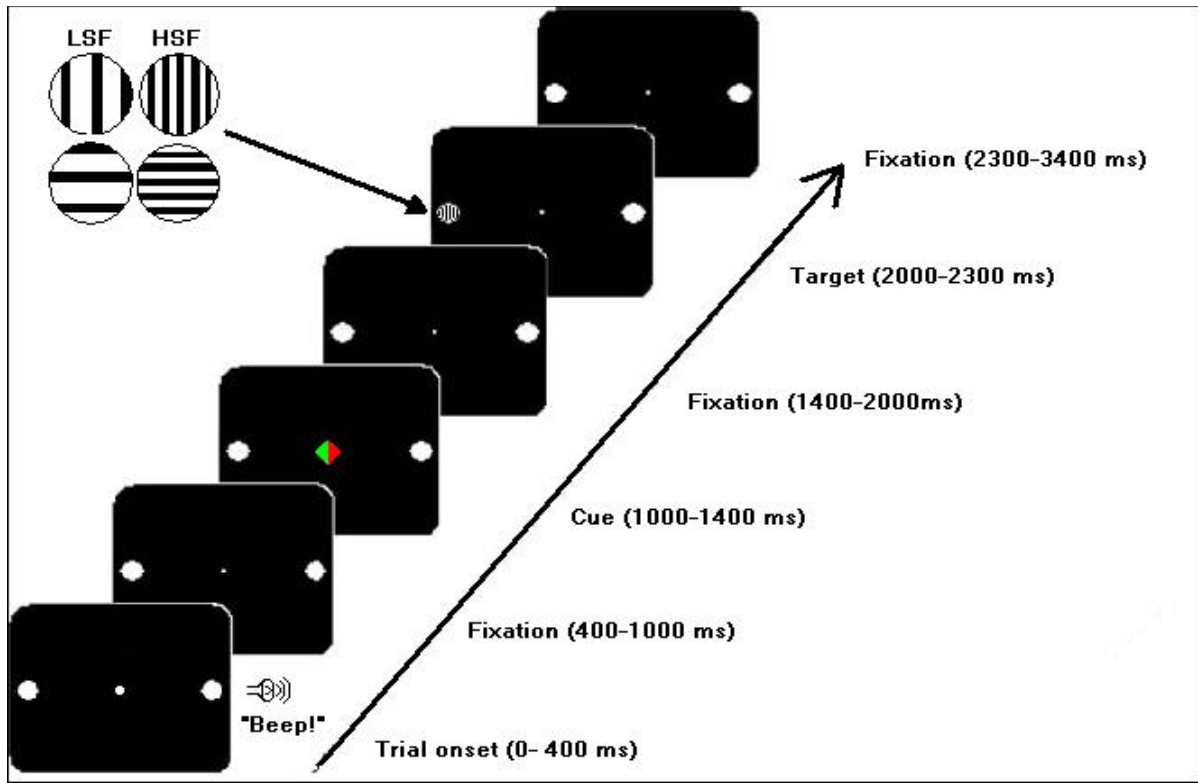


Fig. 1 An example of the stimuli and their temporal order as employed in the Experiment

#### Task and procedure Poser task

Participants were instructed to keep their eyes on the fixation dot during the cue-target interval, and to press the left or right button when one of the circles was filled with horizontal or vertical lines, respectively. These vertical and horizontal came in two varieties one with two lines, low spatial frequency (LSF), and one with six lines, high spatial frequency (HSF). Button presses had to be as fast and accurate as possible. As target position and the required button press varied independently, target position and response side could correspond or not (corresponding vs. non-corresponding trials). The Poser task consisted of 672 trials, lasting for approximately 70 min. Each task was divided into two parts. In the first part, half of the participants were informed that the circle indicated by the green side of the cue was the most probable target location (on 80% of the trials, i.e. the valid trials). On 15% of the trials, the target occurred in the other circle (invalid trials), whereas on 5% of the trials, no target occurred (catch trials). In the second part, they were informed that the red side of the cue indicated the most probable target location. For the other half of the participants, this order was reversed. The trials were divided into four blocks of 168 trials, which were each preceded by 20 practice trials.

### *Recording*

EEG was recorded continuously from Ag/AgCl ring electrodes placed at the following 61 standard scalp sites: Fpz, Fp1, Fp2, AFz, AF3, AF4, AF7, AF8, Fz, F1, F2, F3, F4, F5, F6, F7, F8, FCz, FC1, FC2, FC3, FC4, FC5, FC6, FT7, FT8, Cz, C1, C2, C3, C4, C5, C6, T7, T8, TP7, TP8, CPz, CP1, CP2, CP3, CP4, CP5, CP6, Pz, P1, P2, P3, P4, P5, P6, P7, P8, POz, PO3, PO4, PO7, PO8, Oz, O1, O2, mounted in an elastic cap (Braincap, Brainproducts GmbH). The external triggers were received by Vision Recorder (version 1.0 b BrainProducts GmbH) which measured participants' electrooculography (EOG), EEG and button presses. A normal computer keyboard was used to record button presses. Participants had to press left or right ctrl-keys on the keyboard. EOG was measured above and below the left eye and horizontally from the outer canthi of both eyes to determine the vEOG and hEOG. Online, all electrodes were referenced to the grand average of all electrodes. EEG and EOG were amplified by a BrainAmp amplifier (Brainproducts GmbH) and were recorded at 500 Hz and digitally filtered (TC = 5.0 s, low-pass filter of 100 Hz, notch filter of 50 Hz) by Vision Recorder. Electrode resistance was kept below 5 k $\Omega$ . Trials with detectable sideward eye movements (exceeding 60  $\mu$  in the hEOG recording) from cue onset until target onset were removed from all analyses. This procedure left on average (82%) of the trials on the control group and (a 81%) on the experimental group. For the statistical analyses, Huynh-Feldt epsilon correction was applied to adjust the degrees of freedom whenever appropriate.

### *Behavioral Measures*

.For each participant the proportion of correct responses (PC) was calculated for each condition. Reaction time (RT) was measured relative to target onset. Responses faster than 100 ms (premature responses) or slower than 1500 ms (misses) and erroneous responses were excluded from the RT analyses. The RT's and the PC's were evaluated statistically by multivariate analysis (MANOVA) with the factors Cor(corresponding and non-corresponding) , Side (side of stimulus presentation), Freq ( High or low spatial frequency) and Val (validly vs. invalidly cued intervals).

### *EEG parameters.*

EEG was on line referenced to the average reference and was analyzed for the interval from -100 to 1000 ms after cue onset. Trials with eye movements were marked and removed using a level trigger ( $\pm 60$ , for hEOG and  $\pm 120$  for vHEOG). Trials with other types of artifacts were removed (max-min:  $\pm 200$ , 150, and 100  $\mu$ V for frontal, central, and parietal electrodes, respectively; gradient criterion 120  $\mu$ V per sample point, zero lines for 50 ms). The baseline was determined from -100 to 0 ms before cue onset. For small eye movements an ocular correction (Gratton & Coles 1983) was performed. This left two subjects with too little trials (<60%). Inspection of their EEG data revealed that single electrodes were responsible for a great proportion of the artifacts. The activity of these electrodes was replaced by the average of surrounding electrodes. Cue-locked contraipsilateral difference potentials, relative to relevant cue direction, were determined by

calculating the differences between the two electrodes in a pair. For example: for the cues pointing to the left, the difference was computed as PO8 - PO7. For cues indicating the right, the difference was computed as PO7-PO8. Averaging these differences yields the general absolute difference contralateral minus ipsilateral relative to cue direction (e.g. PO8/7). For statistical analysis Average amplitudes were computed per participant in 20 ms intervals from cue onset until 840ms. Relevant time windows consisting of multiple 20 ms intervals associated with relevant LERP were analyzed using the factors time, lateral-medial axis (2), and anterior-posterior axis (7), containing the electrode pairs AF4/3, AF8/7, F4/3, F8/7, FC4/3, FC6/5, C4/3, C6/5, CP4/3, CP6/5, P4/3, P6/5, PO4/3, PO8/7. Intervals were considered to be of interest, either when the signal differed from baseline (Intercept), or when activity differed among electrodes ( $P < 0.01$ ).

## Results

The dyslectics (mean age = 23.15) were 2.2 years older than the control group (mean age = 20.43)  $t(25) = -2.1$   $p = 0.042$ . To control for the possibility that observed effects could have been caused by age we introduced it as a covariate in the analyses of EEG, behavioral and neuropsychological test data. Results revealed that age differences were unaccountable for observed effects.. Therefore age was left out as a covariate in the analyses used to obtain the results treated in the upcoming section.

The scores on the dyslexia screening test were analyzed using a multivariate test yielding a significant difference between groups  $F(6,20) = 4.8$   $p = 0.003$  with the dyslectics scoring especially poor on the nonsense words subtest  $F(1,27) = 27.6$   $p < 0.001$ . Mean score dyslectics = 75.93 mean score controls = 127.00. An age related difference was found on the one minute writing subtest  $F(1,27) = 4.3$   $p = 0.049$ . The test also provided norms for scoring. All the norm scores of the subtest combined formed the sum of the norm scores. Due to time restrictions not all subtest were used making the norm scores less reliable. Scoring lower than 38 indicated a high risk of dyslexia whereas scoring higher indicated normal psycholinguistic functioning. On the basis of norm scores a difference between groups was found  $t(24) = 4.2$   $p < 0.001$  The mean of the control group = 57.9 and the means of the dyslectic group = 46.8. Using a one-sampled t-test  $t(13) = 12.9$   $p < 0.001$  the controls where found to be scoring higher than 38. The dyslectics where also found to score higher than the norm of 38  $t(12) = -3.8$   $p = 0.002$ .

Differences between means of the Bourdon, Trail making and Balloons tests were analyzed using an ANOVA (see *table1* and *table2*). One female dyslectic participant was excluded from analysis of neuropsychological data due to previous experience with the tests used. Further investigation of the data yielded no significant differences between controls and dyslectics.

Group	Bourdon	Bourdon (omissions)	Trailmaking A	TrailmakingB	Balloons A	Bal-A (Fault)	Balloon s B	Bal-B (Fault)
Control	12.63 (2.02)	15.35 (12.07)	26.54 (7.92)	48.26(13.00)	33.21 (20.55)	.14(0.54)	97,89 (36,60)	,93 (1,82)
Dyslectic	11.60 (1.67)	16.25 (12.10)	24.92 (5.44)	58.13(26.87)	30.48 (11.80)	.08(0.30)	78,20 (28,56)	2,17 (3,27)

*Table 1. Mean scores for Bourdon, Trail making and Balloons test.*

	F	Sig.
Trailmaking A	,301	,588
TrailmakingB	1,330	,260
Bourdon	1,953	,175
Bourdon(omissions)	,035	,853
Balloons A	,166	,688
Bal-A (Fault)	,119	,734
Balloons B	2,274	,145
Bal-B (Fault)	1,480	,236

Table 2. Separate ANOVA's comparing differences between groups for each neuropsychological test.

Analysis of behavioral measures revealed that the mean PC on high spatial frequencies was lower than 60% for 14 subjects. The data was therefore considered to be unreliable due to chance effects and an indication that the high spatial frequencies were too difficult a task for many subjects. Therefore these 14 subjects were taken out of the analysis of the high spatial frequencies leaving 3 dyslectics and 9 controls. Low and high spatial frequencies were analyzed separate. For the lower spatial frequencies all subjects were used as the performance on these spatial frequencies was above the threshold of 60% correct. Analysis of the data with repeated measures involving correspondence, side and validity as factors and percentage correct and reaction time as measures revealed the following (see table 3): For the low spatial conditions dyslectics and controls showed an effect of validity over invalidly cued intervals  $F(2,23)= 28.074$   $p < 0.000$ . Univariate tests revealed this effect to be present for RT and PC with  $F(1,24)= 40.3$   $p = < 0.001$  and  $F(1,24)=16.1$   $p = 0.001$  respectively. An interaction on correspondence\*validity  $F(2,23)=3.881$   $p = 0.035$  was also found. Further univariate tests  $F(1,24)=6.0$   $p = 0.022$  revealed this effect only to be present for RT

For the HSF condition effects for validity were found for both RT  $F(1,11)=25.7$   $p=0.000$  and PC  $F(1,11)=8.5$   $p=0.016$ . For RT a cor\*side interaction was found for  $F(1,10)=29.3$   $p < 0.001$ , also for RT a cor\*side\*val\*group interaction was found  $F(1,10)=7.2$   $p=0.023$ .

Cue	Reaction time(RT)	Proportion correct (PC)
Valid	708.08 (19.33)	90.13(1.44)
Invalid	750.91 (18.67)	85.93 (1.94)

Table3. At the side Means for reaction time and proportion correct for factor validity. Below means for the factor validity\*correspondence. Both for conditions involving stimuli with low spatial frequency.

Stimulus	Cue	Reaction time(RT)	Proportion correct (PC)
Corresponding	Valid	711.95 (20.41)	90.12 (1.70)
	Invalid	742.95 (19.43)	86.82 (2.03)

Stimulus	Cue	Reaction time(RT)	Proportion correct (PC)
Non-corresponding	Valid	704.20 (18.86)	90.27 (1.56)
	Invalid	758.88 (19.65)	85.05 (2.30)

Separate analyses for left and right sides with factors correspondence(2) and validity(2) revealed the effects for correspondence to be reversed for reaction times to stimuli presented on the right with  $F(1,11)=12.0$   $p=0.006$ . No significant effects for correspondence were found for stimuli presented on the left side. For mean reaction times for both left and right sides on corresponding and non corresponding conditions see table 4.

Stimulus	Left	Right
Corresponding	757,00 (44,20)	830,92 (38,13)
Non corresponding	823,10 (27,85)	745,83 (38,10)

*Table 4 Mean Reaction times for HSF stimuli presented on the left and the right side for corresponding and non corresponding conditions. As can be seen the effect of correspondence is reversed for stimuli presented on the right side of the screen.*

EEG data was visualized by interpolating electric field strength between lateralized electrode pairs. Spline maps by Vision analyzer at positive and negative maximums for both groups at crucial time intervals are displayed in Fig.2. Investigation of these maps reveals a negative peak at 280 ms and a positive peak at 600 ms largest over posterior sides. This corresponds with EDAN and LDAP components respectively. Indications of ADAN are less pronounced but still visible as activity over anterior sites at 420 ms.

Analysis of the factors time (9) anterior-posterior axis(7) and lateral-medial axis(2) for 20 ms time windows in the interval (260-440) related to EDAN revealed an interaction for anterior-posterior\*lateral-medial  $F(6,19)=4.077$   $\epsilon=0.812$   $p=0.002$ , indicating that the activity was centered at a specific electrode. Investigation of the mean differences from baseline revealed that the largest activity came from the most posterior and lateral electrode pair PO8-PO7. Furthermore an interaction for time\*group  $F(8,17)=2.6$   $\epsilon=0.55$   $p=0.035$  was found. Investigations of the maximum differences at each time interval revealed that the intervals 400-420 and 420-440 were the most likely source for this time\*group interaction. Considering these findings an analysis of the lateralized ERP's from electrode pair PO8-PO7 was conducted. This revealed significant between group effects for time intervals 400-420 and 420 and 440 ms giving  $F(1,25)=4.353$   $p=0.048$  and  $F(1,25)=8.321$   $p=0.008$  respectively.

Analysis for the factors time (17) anterior-posterior axis(7) and lateral-medial axis(2) for 20 ms time windows in the interval 500-840 revealed Interactions for time  $F(9,16)=7.6$   $\epsilon=0.381$   $p<0.001$  indicating the mean of activity differed as a function of time. Investigation of the mean activity revealed it to be highest in interval 580-600. And Interaction for anterior-posterior\*lateral-medial\*time was found  $F(96,2304)=2.7$   $\epsilon=0.292$ ,  $p<0.001$  indicating the activity could be located at a specific electrode pair at a specific time. Inspections of the mean activity at each time

interval revealed that the intervals 580-600 contained the maximum positivity coming from electrode pair PO8/7 the most likely source of an LDAP. Elektrode pair P6/5 also reached maximum positivity at the same time interval but it wasn't as high as PO8/7. Further analysis using ANOVA of both elektrode pairs did not reveal any between group effects.

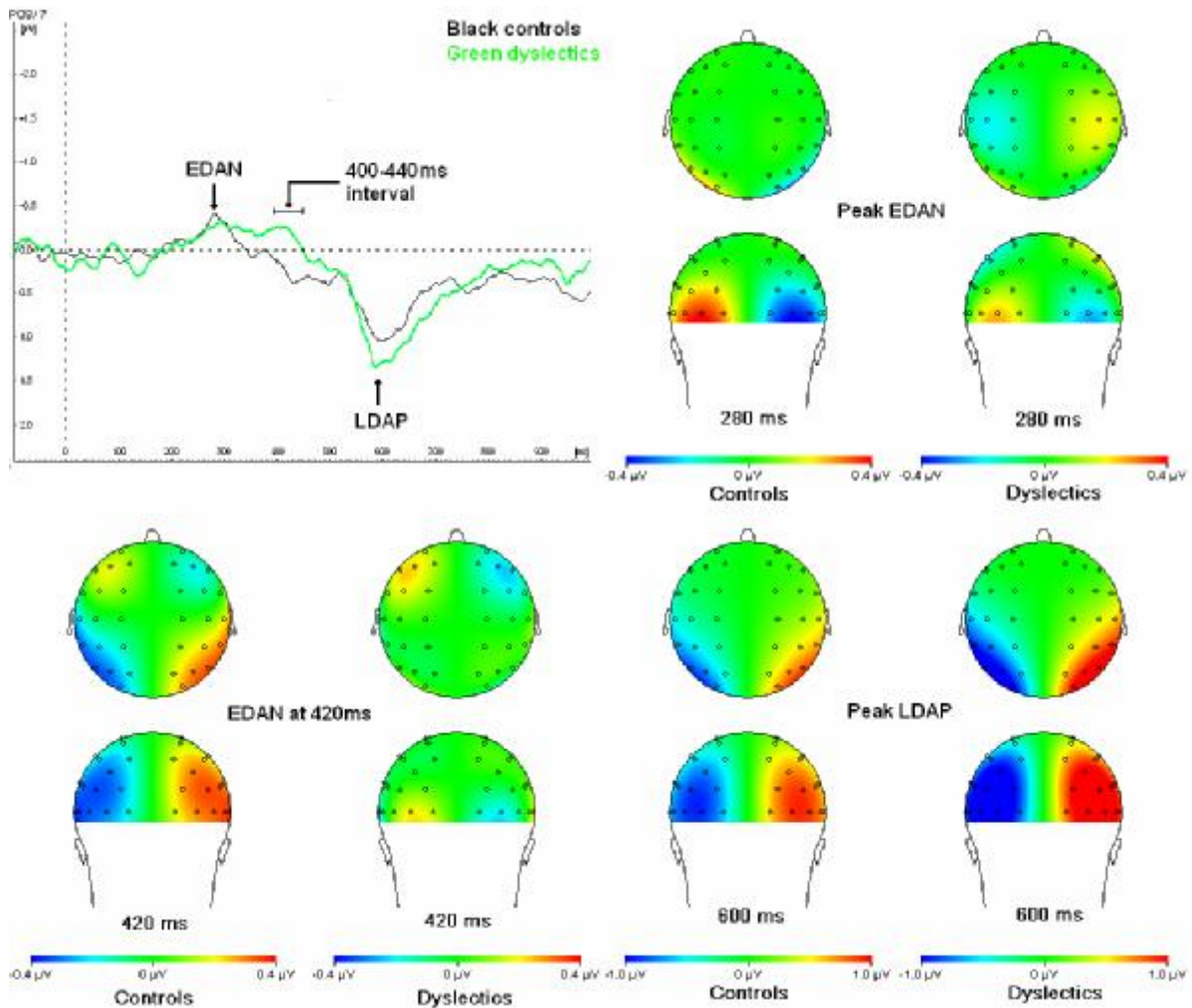


Fig.2 (Above) Lateralised event related potential for PO8/7 accompanied with the spline map at 420 ms illustrating the difference between posterior activation in controls and dyslectics. Also displayed are spline maps for the peak of the EDAN at 280 ms and the peak of the LDAP at roughly 600 ms. The spline maps have all been mirrored over the left hemisphere.

An analysis of lateralized ERP's of electrode pair F8/7, the most likely source of an ADAN, was conducted. Analysis of factors time(42) revealed activity to differ from baseline in the cue target interval  $F(1,24)=6.9$   $p=0.015$ . Further investigation of mean activity revealed interval 540-820 to be the most probable source of this interaction. By using a standard multivariate test intercepts for each time interval were obtained giving  $F(1,24)<1.3$   $p<0.05$  for the intervals 540-780. The greatest mean difference, peak of the ERP, from baseline was reached at time interval 600-620.

## Discussion

The aim of the experiment in this paper was to establish if dyslectics have specific attentional problems. Analysis of EEG data revealed an EDAN that lasted about 40 ms longer in dyslectics from time interval 400 to 440 ms. Behavioral data from the Posner Task and the data from the neuropsychological test did not reveal any group differences. Implications of these results for the link between dyslexia and attentional deficits as well as the magnocellular theory of dyslexia will be discussed.

Regarding the EEG data this experiment found EDAN and LDAP components at electrode pair PO8/7. The ADAN, at electrode pair F8/7 found in this experiment came about at 540 ms after cue onset. The literature reports typical ADAN activity at 400 ms after cue onset but there have been reports of slightly earlier and later occurrences of ADAN (Eimer, 1995; Eimer et al., 2002; Nobre et al., 2000b; Yamaguchi et al., 1994, 1995).

It was hypothesized that a deviant or absent EDAN would reflect a difference in the effectiveness of shifting visual attention. Behavioral results from the Posner task do not show corresponding results therefore it is reasoned that the extra 40ms are not needed to shift attention but in line with Van Velzen and Eimer (2003) could be taken to select the meaning of the cue. The meaning in this case can be interpreted as the task relevant aspects of the cue. Namely shifting the attention over to the relevant location. In the present experiment the cue did not present a clear cut rule but a probability. In this case the longer EDAN could mean that dyslectics need more time to resolve the task relevant aspects when the task indicated by the cue is more complex or of greater ambiguity. However, if such ambiguity would exist it would be reasonable to assume for it to be also present in the control group. In the current experimental setup it was impossible to see whether the modulating the action specific nature of the cue, simple vs. complex, would be able to modulate the EDAN component in both groups. Other studies need to be conducted in order to clarify this.

Behavioral measures of the Posner task revealed an cue effect, thus a general effect of attention, on RT and PC and an interaction between correspondence and cue validity in line with earlier results by Abrahamse & van der Lubbe (2007) demonstrating that responses were faster for validly cued intervals and that endogenous cuing modulates the Simon effect. Interactions between correspondence and side were found on conditions involving HSF stimuli. In this case the effect for correspondence on reaction time was normal for stimuli presented on the left side but the effect was reversed for stimuli presented on the right side. Because of the difficulty of the HSF condition and the absence of such an effect on LSF conditions it is reasoned that responses were biased towards the left hand whenever participants had a hard time discerning stimulus orientation. The absence of any between group differences on the behavioral data suggests that the ability to direct or benefit from visual attention is no different from the control population.

Two alternate explanations for these findings, next to the absence of specific attentional deficits in dyslexia, exist. First it is possible that problems with visual attention only exist in a subset of the dyslectic population. Results by Amitay et al. (2002) and the fact that deficiencies in



magnocellular function have been replicated with varying results seem to confirm this. There is the possibility that developmental dyslexia is not a heterogeneous disorder at all and that the same overt problems in reading and spelling can have multiple underlying causes. Some variants of dyslexia could be caused by a magnocellular deficit and others only be accompanied by them.

The second explanation is that a different experimental setup and procedure which could be making differences between dyslectics and controls with respect to attentional shifting fragile. For example: Wijers et al (2005) found differences between dyslectics and controls on the hemispherical distribution of the activity relating to ADAN. Controls showed the ADAN over predominantly one hemisphere whereas dyslectics showed it more distributed over both hemispheres. In their experimental setup the cue was presented for 750 ms instead of 400 ms, the target consisted of a letter and was presented for 50 ms instead of 300 ms in a rectangular area. A shorter stimulus presentation could have taxed the magnocellular system to a greater extent thus causing differences between groups to become visible. Also the fact that Wijers used letters as a target could explain a lot of the behavioral effects and possibly mis-attributing them to attention. The nature of the cue, whether it is presented as a rule, as in the study by Wijers, or a probability could have an effect on attentional resources. In the case of a rule attentional resources would be allocated quicker without any reserve. This could increase the effects of attention on performance and could therefore bring out differences in dyslectics and controls.

The behavioral data from the Posner task, Bourdon test, trailmaking test and Balloons test in this study does not support the view that dyslectics have specific attentional problems. On the Posner task the effect of the cue and the effect of attention related to it was the same for both groups. Furthermore there are no indications of neglect because of the absence of an effect for side. Magnocellular deficiencies as a result of impaired processing of stimuli of LSF could not be adequately determined. Dyslectics had greater difficulty with the HSF conditions, which doesn't plead for the occurrence of magnocellular deficiencies. Although the attrition in the dyslectic sample was bigger no statistical differences between the PC of controls and dyslectics could have been found.

The case for specific attentional problems is weakened by the results of this study. Interestingly enough this study did found a slightly longer EDAN in dyslectics during cue target interval. This is an indication for a difference between PPC functioning in controls and dyslectics. Furthermore because of the absence of behavioral data confirming attentional problems the EDAN is thought to reflect the selection of task-relevant aspects of the cue instead of directing attention.

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