Adults with Dyslexia Show Deficits on Spatial Frequency Doubling and Visual Attention Tasks

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We examine the visual processing of high-functioning adults with developmental dyslexia (mean Performance IQ=126.5) and current phonological problems. In comparison to an age- and IQ-matched control group, the group with dyslexia showed deficits in two tasks associated with magnocellular/dorsal pathway function. For the 'frequency doubling' stimulus (grating of 0.25 cpd modulated at 25 Hz counterphase flicker), contrast thresholds for detection were raised in the dyslexic group. In conjunction visual search, a display time sufficient for controls to achieve ceiling accuracy at all set sizes (30 ms per item) was inadequate to allow shifts of attention around the display for the group with dyslexia. In contrast, normal performance was found on 'popout' visual search and on a ventral stream acuity task. Correlational analysis revealed a significant relationship between degree of deficit in conjunction search and phonological difficulty. The deficits revealed were specific to functions that rely on magnocellular input. They cannot be attributed to concentration lapses, eye movement problems or slow reaction times in the dyslexic group. Copyright © 2004 John Wiley & Sons, Ltd.

Keywords: magnocellular; dyslexia; visual attention; dorsal stream; visual search; spatial frequency doubling illusion

INTRODUCTION

In childhood dyslexia, one of the most commonly identified deficits involves phonological skill (e.g. Brady & Shankweiler, 1991; Frith, 1995). By adulthood, dyslexics with phonological problems may have developed a

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large sight vocabulary, but reading difficulties usually remain. These include slow reading and spelling speeds, impaired novel or nonsense word reading (i.e. poor grapheme to phoneme conversion), and poor verbal fluency (Bruck, 1990, 1992; Felton, Naylor, & Wood, 1990; Nicolson & Fawcett, 1995).

As will be reviewed shortly, deficits in visual processing have also been extensively reported in subjects with dyslexia. Deficits have been observed particularly in tasks associated with magnocellular/dorsal pathway functioning. The idea that a magnocellular deficit is involved in dyslexia is not universally supported, however. Some researchers suggest that magnocellular deficits occur only in dyslexics who have phonological difficulties and not in other subtypes (e.g. Borsting *et al*., 1996; Ridder, Borsting, Cooper, McNeel, & Huang, 1997; Slaghuis & Ryan, 1999: although see Ridder, Borsting, & Banton, 2001). It has also been suggested that the poor performance of dyslexics could be due to general factors such as lapses in concentration rather than an underlying deficit in magnocellular/dorsal pathway function (Davis, Castles, McAnally, & Gray, 2001; Stuart, McAnally, & Castles, 2001). A final difficulty for a 'magnocellular' interpretation of dyslexia is that it remains somewhat unclear how the deficits observed on specific visual tasks are related, first, to phonological problems and, second, to actual reading performance (Frith & Frith, 1996; Shaywitz, 1996).

The present study extends current knowledge of visual processing in adult developmental dyslexics with phonological problems. The dyslexics tested were high-IQ individuals with demonstrated ability to perform well on concentration-demanding tasks. Performance was examined on three visual tasks, chosen to specifically tap either magno-cell, dorsal pathway or ventral pathway function.

Visual Processing and Dyslexia

Initial processing of visual information is primarily segregated into two major pathways, magnocellular and parvocellular, beginning at the retina and projecting to different layers of the lateral geniculate nucleus (LGN) (see Calloway, 1998). Magnocellular (*M*) cells are more sensitive to lower spatial frequencies and higher temporal frequencies than Parvocellular (*P*) cells. The cell types also differ in that *M* cells have faster transmission times, respond in a transient fashion at stimulus onset/offset rather than throughout stimulus presentation, and respond more strongly than *P* cells at low contrasts (Schiller, Logothetis, & Charles, 1990). Beyond V1 at the cortical level, input from magnocellular layers dominates the dorsal processing stream, while parvocellular input dominates the ventral stream (Merigan & Maunsell, 1993).

Tests of magnocellular function

Many studies have reported that both children and adults with dyslexia show deficits for visual stimuli optimized for magnocellular processing (e.g. Chase & Jenner, 1993; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Martin & Lovegrove, 1987; Mason, Cornelissen, Fowler, & Stein, 1993; Talcott *et al*., 1998).

The present study used a particular test of magnocellular function, based on a 'frequency doubling' (FD) stimulus (this is a low spatial frequency grating (0.1–4 c/deg) whose contrast is reversed at high temporal frequencies (>15 Hz), which when detected at all, is perceived with twice the actual spatial frequency;

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Bernadete, Kaplan, & Knight, 1992). Physiological studies have determined the presence of two sub-groups of magnocellular cells; $M(x)$ and the larger $M(y)$. Both exhibit fast transmission times but there are clear functional differences between them (Bernadete, Kaplan, & Knight, 1992) for review see Merigan & Maunsell, 1993). Of particular interest to this study is the evidence that when presented with an FD stimulus, $M(y)$ cell responses at very low contrasts ($<2\%$ contrast) are much stronger than $M(x)$ and P cell responses (Bedford, Maddess, Rose, & James, 1997; James, Maddess, Rouhan, Bedford, & Snowball, 1995; Maddess, Hemmi, & James, 1992; Maddess, Bedford, James, & Rose, 1997). Results from glaucoma studies indicate that contrast thresholds to detect the frequency doubling stimulus (measured in terms of the minimum contrast necessary for detection) can be used to assess the integrity of $M(y)$-driven function. Glaucoma leads to retinal ganglion cell death, and, since $M(y)$ cells are more sparsely distributed on the retina than either $M(x)$ and P cells (see Maddess *et al*., 1999), early stage glaucoma results in a selective deficit of $M(y)$ output. Reduced sensitivity to the frequency doubling stimulus has then been shown to provide a reliable behavioural indicator of the beginnings of glaucoma (Maddess *et al*., 1999; Johnson & Samuels, 1997).

Turning to dyslexia, there is no reason to suspect the same type of cell death that occurs in glaucoma. However, if $M(y)$-driven processing has failed to properly develop in some manner (e.g. fewer $M(y)$ cells, slower transmission times, smaller LGN receptive field sizes), detection of the frequency doubling stimulus might be impaired in dyslexia. Pammer and Wheatley (2001) reported such a result in children with dyslexia. In the present study, we examine whether the deficit is also present in adults with dyslexia.

Tests of dorsal stream function

Beyond the LGN segregation of magnocellular and parvocellular pathways is less clear, however input from the magnocellular layers dominates the dorsal stream of cortical processing. Early stages of this stream support functions of spatial localization and motion processing (involving V5/MT cells in extrastriate cortex). Consistent with a dorsal stream deficit, decreased sensitivity to motion stimuli has often been reported in subjects with dyslexia (e.g. Cornelissen *et al*., 1995, 1998; Demb, Boynton, Best, & Heeger, 1998a; Demb, Boynton, & Heeger, 1998b; Eden *et al*., 1996; Hansen, Stein, Orde, Winter, & Talcott, 2001; Pammer & Wheatley, 2001; Ridder *et al*., 2001; Slaghuis & Ryan, 1999; Talcott *et al*., 1998; Talcott, Hansen, Assoku, & Stein, 2000; Witton *et al*., 1998), although there have been reports of normal motion sensitivity in some individuals (e.g. Everett, Bradshaw, & Hibbard, 1999; Victor, Conte, Burton, & Nass, 1993).

A later processing stage in the dorsal stream involves the parietal cortex. A major function of this region is in the deployment and control of visual attention. Electro-physiological investigations in macaques, for example, have found significant attentional modulation in the activity of neurones in the posterior parietal cortex (PPC) (Colby, Duhamel, & Goldberg, 1996; Luck, Chelazzi, Hillyard, & Desimone, 1997; Motter, 1993; Steinmetz & Constantinidis, 1995). Moreover, in humans, both lesion and brain imaging studies have indicated a specific role for the parietal cortex in shifting attention to different regions of the visual field (e.g. Arguin, Joanette, & Cavanaugh, 1993; Corbetta, Miezin,

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Shulman, & Petersen, 1993, 1995; Friedman-Hill, Robertson, & Treisman, 1995; Nobre *et al*., 1997).

The attentional shift function of the parietal cortex can be tested by a particular type of visual search task. There are two common types of visual search (e.g. Treisman & Gelade, 1980). In a *feature search*, the target differs from the distractors on the basis of a single feature (e.g. a red target amongst blue distractors). Under these conditions, popout occurs, and the reaction time to determine the presence of the target is independent of the number of distractors (the 'set size'). Feature search is assumed to represent a spatially parallel process and to *not* require any shifts of attention around the display. In a *conjunction search*, however, the target differs from the distractors by a combination of two features (e.g. a red vertical target amongst blue vertical and red horizontal distractors). Here, reaction times increase linearly with increasing set size, and search slopes for target-absent trials (approx. 40–60 ms/item) are about twice those for target-present trials (approx. 20–30 ms/item). These results are taken to indicate that conjunction searches require sequential shifts of attention around the display (e.g. to serially examine each item in turn). In support of the claim that conjunction search assesses parietal function, while feature search does not, only conjunction search is affected when parietal cortex is disrupted via transcranial magnetic stimulation (TMS) in normal observers (e.g. Ashbridge, Walsh, & Cowey, 1997), or by lesions (e.g. Friedman-Hill *et al*., 1995).

Several studies have examined visual search performance in developmental dyslexia. A deficit in conjunction search has been reported in children (Casco & Prunetti, 1996; Vidyasagar & Pammer, 1999) and in one study in adults (Iles, Walsh, & Richardson, 2000). A potential difficulty with these studies, however, is that all have employed the standard version of the visual search task, in which stimulus display time is unlimited and reaction time (to respond present–absent) is the measure. Given that poor motor control appears to be common in those with dyslexia (e.g. Fawcett & Nicolson, 1995; Felmingham & Jakobson, 1995; Velay, Daffaure, Giraud, & Habib, 2002; Wolff, Michel, Ovrut, & Drake, 1990), reaction times to press a button may not provide a fair measure of dyslexic performance. Thus, in the present study, the stimulus display was presented for a limited time only, and accuracy of present–absent decisions was used as the measure. Larger sets were displayed for longer periods, with the display time (equal to the set size multiplied by 30 ms) chosen to be sufficient for normal readers to search all items in the display for a target-present conjunction search. (Increasing set size should then lead to a *decrease* in accuracy in dyslexia, if dyslexics require more than the normal 20–30 ms/item to shift their attention around the display.) Also note in order to minimize the potential contribution of dyslexic deficits in controlling eye movements (Eden, Stein, Wood, & Wood, 1994; Griffen, Christenson, Wesson, & Erickson, 1988; Stein, Riddell, & Fowler, 1988) to search performance, subjects were required to fixate at the centre of the presentation at all times. Further, if any subject failed to fixate, our longest display time (for 11-item displays) of 330 ms allowed at most one or two eye movements.

Tests of ventral stream function

Parvocellular layers of the LGN and V1 provide inputs mainly to the ventral temporal stream, post the primary visual cortex. This stream mediates processing

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of form information (Logothetis & Sheinberg, 1996; Merigan & Maunsell, 1993; Schiller *et al*., 1990), and its functioning is standardly examined using tasks that measure the ability to see fine spatial detail (i.e. test of visual acuity). Several studies have reported that dyslexics show normal levels of performance on such tasks (e.g. Pammer & Wheatley, 2001). The specific test employed here was the Landolt-C (ring-gap) test (Riggs, 1965).

Visual Deficits and Dyslexic Sub-Types

Some studies have failed to find a deficit in magno-dominated visual functioning of dyslexics (e.g. Gross-Glenn *et al*., 1995; Hayduk, Bruck, & Cavanagh, 1996; Johannes, Kussmaul, Munte, & Mangun, 1996; Victor *et al*., 1993). This could potentially be due to over representation in the subject samples of dyslexics without phonological difficulties. Several findings suggest that dyslexics who have phonological difficulties might show more severe magnocellular pathway deficits than those who do not. Normal contrast sensitivity for stimuli processed by the magnocellular pathway has been reported in the latter (Borsting *et al*., 1996; Ridder *et al*., 1997; Slaghuis & Ryan, 1999). Cestnick and Coltheart (1999) have also reported subtype differences in perception of apparent motion (Ternus task), although Ridder *et al*. (2001) observed motion coherence deficits in all subtypes. Thus, in the present study, we focussed on participants with a clear phonological component to their dyslexia.

Visual Deficits and Lapses of Concentration

In a recent critique of magnocellular/dorsal pathway involvement in dyslexia, Stuart *et al*. (2001) suggested that the poor performance of dyslexics on many magnocellular/dorsal tasks could simply be due to occasional lapses of concentration. Such lapses might affect results obtained from the adaptive staircase procedures commonly used to measure threshold performance (Davis *et al*., 2001; Stuart *et al*., 2001). Stuart *et al*. suggested that concentration lapses were more likely to occur in dyslexic than in non-dyslexic children, partly because of the high rate of comorbidity of dyslexia with Attention Deficit Hyperactivity Disorder in children (Lambert & Sandoval, 1980; see also Willcutt & Pennington, 2000). In the present study, we selected a sample for which greater-than-normal rates of concentration lapses were considered unlikely. Our participants were high-IQ adults who had performed successfully in tertiary education and had no indications of ADHD.

In addition, the concentration lapse hypothesis can be assessed indirectly by comparisons across different tasks. Even in the previous literature, the studies that have used staircase procedures in parvocellular/ventral stream tasks have failed to reveal any differences between the control and dyslexia groups (Pammer & Wheatley, 2001). In the present study, the claim that visual processing deficits are specific to magnocellular mediated processes would predict that performance of those with dyslexia on the 'frequency doubling' task (involving a staircase measure) and/or the conjunction search task (no staircase) should be poor compared to controls. At the same time, however, no deficits on the Landolt-C visual acuity test (staircase) or the feature search task (no staircase) would be predicted.

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METHOD

Participants

All participants were recruited from the Australian National University Campus through flyer advertising and had successfully completed at least one unit at University. In some cases a PhD had been attained. The experimental group consisted of 10 adults who had received a diagnosis of dyslexia as children, and currently met the criteria of dyslexia as determined on the Dyslexic Adult Screening Test (DAST: Fawcett & Nicolson, 1998). The DAST provides a dyslexia indicator score relative to age norms ranging from −3 to +1 (where: −3=1st–4th percentile; −2=5th–11th percentile; −1=12th–22nd percentile; 0=23rd–77th percentile; +1=78th–100th percentile). A measure of phonological ability (PA) was determined by averaging scores on the phonemic segmentation and nonsense passage reading components of the DAST. Similarly, an overall literacy measure was obtained by averaging one minute reading and two minute spelling scores of the DAST. Untimed single word reading and spelling abilities were evaluated using the Wide Range Achievement Test (WRAT: Jastak & Wilkinson, 1984). Intellectual functioning was assessed using the Wechsler Abbreviated Scale of Intelligence (WASI®).

The control group consisted of 10 adults without any past history of reading difficulties. The groups were matched (at group level) for age, gender, Wechsler Performance IQ and educational status. All participants reported that they did not suffer an attention deficit disorder nor mood disorder (substantiated directly through questions based on DSM-IV criteria; American Psychiatric Association, 2000, and indirectly through observation by a trained clinician) and had completed an optometric assessment within the last two years.

Group characteristics are presented in Table 1. Verbal but not Performance IQ was lower in the dyslexic group (although still at the upper end of the average range with respect to population norms). Highly significant group differences were documented in measures of phonological ability and literacy from the DAST, as well as both sub-tests of the WRAT. All dyslexic subject scores fell

Table 1. Participant group characteristics. Values are mean (S.D.) unless otherwise noted

	Control n=10	Dyslexic n=10
Mean Age (Years)	33.7 (7.5)	34.4 (8.4)
WASI (Standard: M=100, S.D.=15)		
Full-scale IQ	128 (8.3)	121 (4.9)
Verbal IQ*	124.4 (8)	113.5 (6)
Performance IQ	125.2 (8.3)	126.5 (10.1)
DAST(Range: −3 to +1, negative		
Phonological Ability (PA)**	0.55 (0.61)	−1.85 (0.75)
Literacy measure (LM)**	0.55 (0.56)	−1.90 (0.66)
WRAT (Standard:		
Reading**	111.2 (4.1)	92.8 (9.7)
Spelling**	113.8 (7.5)	91.4 (11.9)
Highest level of education	Tertiary	Tertiary

*$p < 0.01$.
**$p < 0.001$.

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outside the 95% confidence intervals of the control sample on each significant measure, with the exception of two dyslexic VIQ scores (scores=120 and 123), and a single dyslexic score in spelling on the WRAT (score=110). Furthermore all dyslexic subjects demonstrated phonological reading deficits.

Materials and Procedure

All participants were tested individually and when necessary wore their normal optical correction. Testing was carried out over two sessions for a total of 3 h. The first session consisted of literacy tasks (DAST and WRAT, reading and spelling), ring-gap detection and frequency doubling perimetry. The second session consisted of the WASI tasks and visual search testing. Within each session tasks were counterbalanced across participants in each group such that half received the psychophysical tests before the screening tasks.

Frequency doubling task

Each stimulus consisted of a 0.25 pd sinusoidal grating, modulated at a 25 Hz counterphase flicker; the perception at low contrasts is of a grating with twice the spatial frequency of the actual stimulus (hence the name frequency doubling). Multiple retinal locations were tested using an Humphrey® Instruments FDT Visual Field Instrument® (Zeiss Humphrey Systems, 1971). This instrument contains age normed data which allows calculation of overall deviation scores of each participant. A small black fixation square remained on throughout the session. An example test stimulus pattern is presented in Figure 1(a). Stimulus duration was 720 ms, consisting of 160 ms ramped onset and offset, and to avoid anticipatory responses and visual persistence a variable inter-stimulus interval between 300 and 500 ms was employed.

Each eye was tested separately beginning with the right eye. Following a practice session the testing sequence lasted approximately 4 min. Participants could pause the display at any time and all were given a 5 min break between testing of each eye.

Participants were required to press a response button each time the pattern was detected. The dependent variable was the contrast threshold for pattern detection. To determine this threshold, a modified binary search (MOBS, Johnson & Shapiro, 1989) threshold strategy was used to manipulate the contrast of the

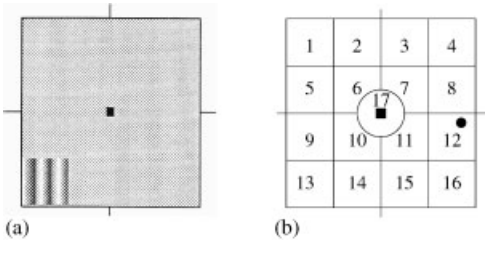

Figure 1. (a) A representation of the display produced by the FDT. The test stimulus pattern is presented in retinal location 13. (b) Retinal location configuration used with the FDT. Fixation point in location 17. Blind spot in location 12, for right eye and location 9 for left eye (not to scale).

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stimulus at each retinal location. At least four staircase reversals, plus upper and lower staircase boundaries within 0.3 log units of each other, make up staircase completion. The mean of the last upper and lower presentations satisfying the staircase completion criteria represented the MOBS threshold, which could range between 0 dB (~100%, maximum contrast and lowest sensitivity) and 56 dB (~0%, minimum contrast and highest sensitivity). The mean background illumination of the display was 100 cd/m^2.

Testing was carried out at 17 retinal locations throughout the central 20° radius of the visual field (see Figure 1(b)). The central location tested was a 5° diameter circle, all other locations were 10° diameter squares.

Targets were occasionally presented in the blind spot to monitor fixation errors (6 trials/eye). False positive errors were also measured by the number of times a participant responded during a pause in testing (6 trials/eye), and false negative errors were indicated by a response to a pattern at maximum contrast (3 trials/eye).

Visual search

Participants were seated approximately 90 cm from the computer monitor. The tasks were generated using an IBM PC (386) with a 17.8 ms refresh rate.

Stimulus items consisted of small (1.5 × 3 mm) and large (3 × 6 mm) rectangles, in either horizontal or vertical orientation. Items were white against a black background. The number of items in the display (set size) varied across 3 levels (5, 7, and 11 items); items were positioned at randomly chosen locations on an invisible grid consisting of 24 locations in a 4 × 6 array (approx. 2.2° × 3.2° visual angle). The target appeared in the stimulus array in a random 50% of the trials, and to maintain set size an extra distractor item was substituted when it was absent. Trials were organised into blocks consisting of 72 trials each. In each block, the target item was a small horizontal rectangle in 50% of trials and a small vertical rectangle in the other 50% of trials. Participants were informed that they could take a break at any time and were given a 3 min break at the end of each block regardless of whether any other breaks had been taken.

The design of a single feature search trial (orientation) and a single conjunction search trial are illustrated in Figure 2. The first two blocks constituted the feature search conditions (e.g. in orientation search if the target was small and horizontal, then all the distractors were small and vertical), and the conjunction search condition comprised four blocks (e.g. if the target was small and vertical, distractors were small and horizontal plus large and vertical).

On each trial, the participant was shown the target to be detected by presenting it in the centre of the screen for 1000 ms. After a delay of 500 ms, the stimulus array was presented. The delay minimized retinal visual persistence of the target. Each stimulus array remained on the screen such that the total display time was 30 ms/item (i.e. 150 ms for 5-item displays, 210 ms for 7-item displays, 330 ms for 11-item displays). At these display times, controls were expected to show close to perfect levels of accuracy at all display sizes (see Introduction). After a delay of 100 ms mask, consisting of a random line pattern, was presented for 75 ms, thus terminating stimulus processing. Participants responded either present or absent via the keyboard. The dependent measure was accuracy of responses.

[A possible methodological criticism of our visual search paradigm is the claim (Sagi, 1988) that a conjunction search on the particular features of orientation and

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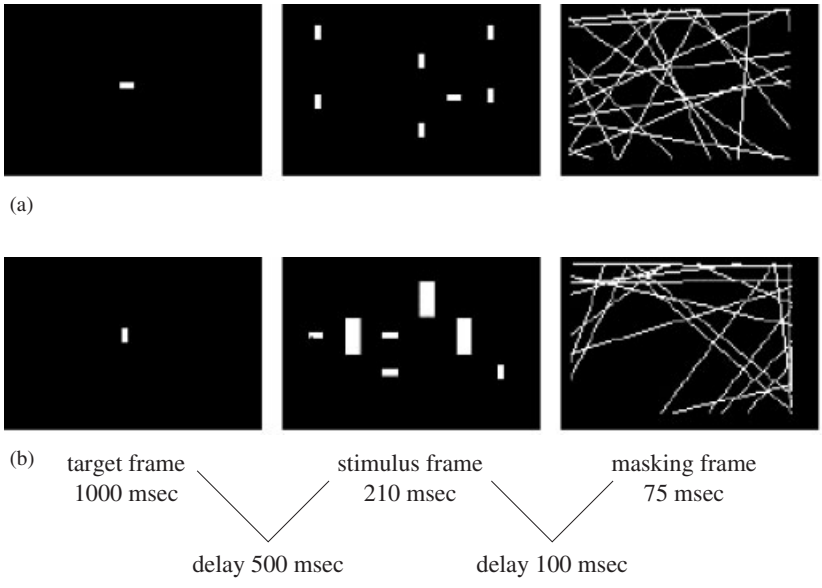

Figure 2. Example of a target present trial (among seven items) in (a) a feature and (b) a conjunction search. Target frames were followed by a stimulus frame (30 msec × number of items) which were followed by a masking frame.

size do not constitute a serial search. Thus, a standard version of conjunction visual search was run for the control group. In this task the stimulus array remained visible until the participant responded and response time was measured. Slopes (ms/item) were calculated using the line-of-best-fit for each participant for target present and target absent conditions. A paired sample *t*-test showed a significant difference between the slopes, $t(9)=2.99$, $p<0.02$, with the rate of increase in response time approximately doubled when the target was absent (23.6 ms/item) compared to when present (13.1 ms/item). The slope for target present trials was also significantly greater than zero slope, $t(9)=4.39$, $p<0.01$. These findings are consistent with the task requiring serial search of the items in the field.]

Landolt ring-gap detection

This task examined ability to resolve fine spatial information, providing a measure of ventral pathway sensitivity. The Landolt ring-gap detection display stimulus consisted of a broken circle (*C*) oriented randomly such that the gap occurred in one of four positions; to the right, left, up or down (cf. Riggs, 1965). The circle subtended a visual angle of 0.57° situated within a square subtending a 2.8° angle. This square was centrally positioned on an otherwise black screen. The *C* was presented for 72 ms, and a fixation dot in the middle of the screen remained visible throughout the trials. Example test displays are shown in Figure 3.

Participants were required to indicate the orientation of the gap by pressing a corresponding key. The dependent measure was the minimum gap size required for accurate detection. This threshold was determined by the PEST staircase procedure (Taylor & Creelman, 1967) beginning with a gap of 20° arc (0.11° visual

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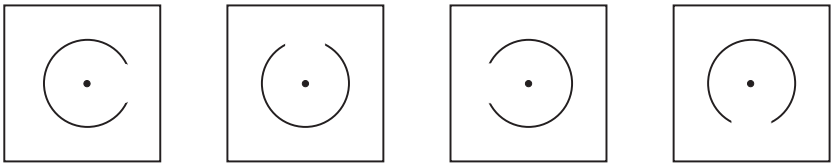

Figure 3. The four possible display configurations of the stimulus C (not to scale).

angle) on the first trial. A total of 14 practice trials with a constant gap size (20°) were given, followed by three blocks of test trials. No zero gap trials were included.

RESULTS

FDT Responses

There were no significant differences between the two reading groups on the reliability indices, such as fixation errors (Control: M=0.3, $S.D.$=0.48, Dyslexic: M=0.4, $S.D.$=0.69; $t<1$) and false positive errors (Control: M=0.2, $S.D.$=0.42, Dyslexic: M=0.3, $S.D.$=0.48; $t<1$). No false negative errors were made by either group.

Figure 4 shows mean threshold contrast levels for each of the 17 visual field locations for each eye. Lower scores (dB) indicate poorer performance; that is, greater contrasts are needed in order to detect the presence of the grating.

A 3-way ANOVA (reading group × eye × retinal location) was performed to determine whether thresholds differed between the two reading groups. The results indicated significant main effects of reading group, $F(1,36)=4.88$. $MSE=92.6$, $p<0.05$, and retinal location, $F(1,11)=2.29$, $MSE=111.9$, $p<0.05$ (corrected for sphericity violation using Greenhouse–Geisser adjustment), but no main effect of eye, $F<1$, $MSE=103.66$. There were no 2-way or 3-way interactions between the variables (largest $F=1.42$, smallest $p>0.2$). These results indicate that the dyslexic group (M=30.45 dB, $S.D.$=1.87) was less sensitive overall than the age and IQ matched control group (M=32.11 dB, $S.D.$=2.72) in detecting the grating.

Age adjusted field loss was also determined for which the FDT instrument provided a mean deviation index (MDI) score for each participant. Positive values indicate above average sensitivity compared to the subjects age group, while negative scores indicate below average sensitivity. Of the individual dyslexics MDI scores (−4.04, −3.08, −2.44, −2.28, −1.80, −1.11, −1.02, −0.73, **1.60**, **2.14**), only two fell within the 95% confidence interval of the control slope scores (95% CI=−0.64 to 1.54).

Visual Search

Feature search

Visual search accuracy is shown in Table 2. Two feature searches were conducted: in one the target feature was orientation and in the other the target feature was size. Note that with the accuracy measure, any effect of number of distractors

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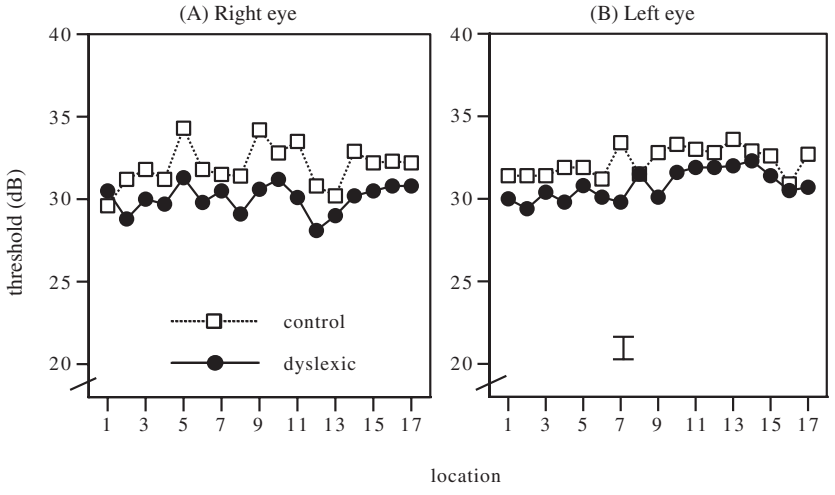

Figure 4. FDT thresholds for dyslexic and control groups over each of the 17 retinal locations for the right eye (A), and the left eye (B). Error bar is ± 1 s.e.m., appropriate for between-subjects comparison of dyslexics versus control group.

Table 2. Mean accuracy scores (standard deviation) for target detection across three set sizes in each feature search, for each reading group: when target present and when absent.

		Feature			
		Orientation		Size	
		Reading Group			
		Control	Dyslexic	Control	Dyslexic
Target present					
	5	91.3 (8.4)	92.5 (8.7)	97.5 (5.3)	96.3 (6.0)
	Set size	93.8 (8.8)	91.3 (10.3)	97.5 (5.3)	95.0 (6.5)
	11	92.5 (8.7)	90.0 (7.9)	98.7 (3.9)	97.5 (5.3)
Target absent					
	5	82.5 (20.6)	85.0 (17.5)	91.3 (10.3)	95.0 (6.5)
	Set size	86.3 (12.4)	86.3 (14.9)	95.0 (6.5)	95.0 (10.5)
	11	87.5 (13.2)	82.5 (16.8)	97.5 (5.3)	95.0 (8.7)

would be demonstrated as decreasing accuracy with increasing set size (5, 7,11 items).

A 4-way ANOVA (reading group × set size × target feature × target presence/absence) indicated no main effects of reading group, $F<1$, MSE=338.4, or set size, $F<1$, MSE=132.5. Significant main effects of target feature, $F(1,18)$=29.8, MSE=113.3, $p<0.001$, and target presence/absence. $F(1,18)$=6.57, MSE=191.9, $p<0.05$, were found. Performance was poorer in the orientation feature search than in the size feature search, and determining the absence of a target in the stimulus set was more difficult than determining its presence. No 2-way, 3-way nor 4-way interactions amongst the factors were indicated, largest F=3.23,

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smallest $p>0.08$. Table 2 confirms that accuracy was not affected by increasing the stimulus set size for either reading group. Furthermore, there was no effect of set size on accuracy even when performance was below ceiling levels (i.e. orientation searches). These results confirm that the ability of the group with dyslexia to perform popout single feature searches is normal.

Conjunction search

Means and standard errors of accuracy scores, determined for each reading group, are shown in Figure 5. A 3-way ANOVA revealed interactions involving target presence/absence, so target present and target absent responses are analysed separately.

For target present responses see Figure 5(a). A 2-way ANOVA (reading group $\times$ set size) indicated a significant main effect of reading group, $F(1,18)=9.23$, $MSE=365.8$, $p<0.01$, indicating that overall accuracy of the group with dyslexia ($M=80.32$, $S.E.=4.82$) was lower than that of the control group ($M=95.31$, $S.E.=1.07$). The main effect of set size was significant, $F(2,17)=11.46$, $MSE=35.6$, $p<0.01$), but this was modified by a significant interaction with reading group, $F(2, 17)=9.69$, $p<0.01$. For the dyslexic group, a follow-up 1-way ANOVA revealed a significant effect of set size, $F(2,8)=14.56$, $MSE=20.5$, $p<0.001$, reflecting a significant linear decrease in accuracy with more distractors, $F(1,9)=48.99$. $MSE=12.2$, $p<0.001$, and no quadratic component. For the control group, a follow-up 1 -way ANOVA revealed no effect of set size, $F<1$, $MSE=15.1$. Thus, as predicted, when the stimulus duration time is set such that normal participants should have sufficient time to move attention across items (30 ms/item) to find a target, controls are highly accurate regardless of set size. The

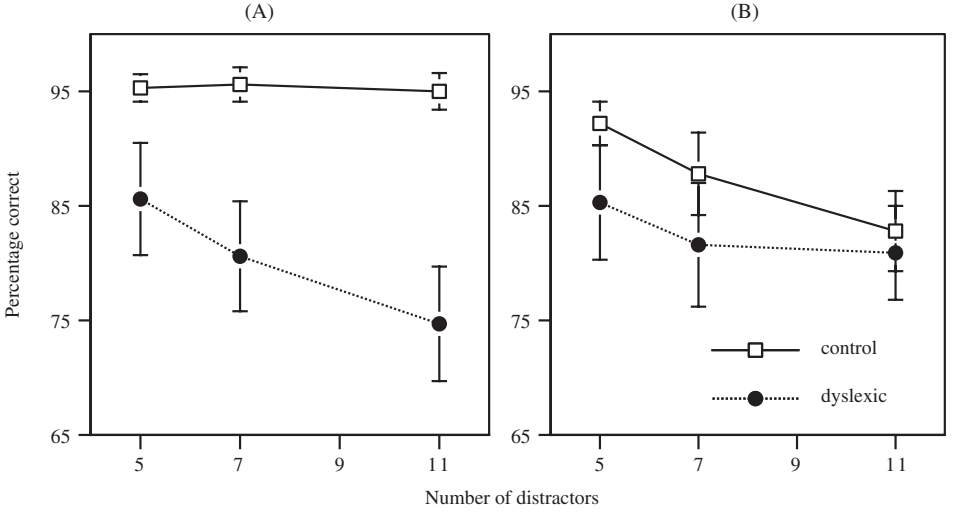

Figure 5. Mean percentage correct responses for each reading group as set size increases, in a conjunction search (orientation and size) when (A) target present and (B) target absent. There was a significant difference between groups when the target was present with differences increasing significantly as the number of distractors increased. There was no significant difference between groups when the target was absent. Error bars represent $\pm$ 1 s.e.m.

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group with dyslexia, however, show a decrease in accuracy as set size increases, indicating a deficit in conjunction search that becomes more severe as more shifts in attention are required. Of the individual dyslexics mean slope scores (−3.7, −2.1, −2.1, −2.1, −2.1, −1.6, −1.6, −1.0, −1.0, −**0.52**), only one fell within the 95% confidence interval of the control slope scores (95% CI=−0.76 to 0.66).

For target absent responses see Figure 5(b). A 2-way ANOVA (reading group × set size) indicated no significant main effect of reading group, $F<1$, MSE=427.6, although the overall accuracy of the dyslexic group (M=82.62, $S.E.$=4.63) was somewhat lower than that of the control group (M=87.61, $S.E.$=2.67). A significant effect of set size, $F(2,17)=7.36$, MSE=36.9, $p<0.01$) with a non-significant two-way interaction was also indicated, $F<1$. A follow-up trend analysis was carried out on the combined data of the two groups and found to be significantly linear, $F(1,18)=15.39$, MSE=30.8, $p<0.001$, with no quadratic component. Thus, accuracy of both groups declines as more items are included in the stimulus set. For both groups, this indicates that target absent search requires more time than that allocated per item.

Ring-Gap Detection

The thresholds of ring gap detection, measured in degrees of visual arc, were determined. Repeated measures ANOVA indicated no main effect of reading group, $F<1$, MSE=3.8; in fact the group with dyslexia showed slightly better performance (M=0.040, $S.D.$=0.007) than controls (M=0.042, $S.D.$=0.005). Thus, performance of the dyslexia group on a ventral pathway acuity function was normal.

Correlational Analysis between Different Tasks for the Dyslexic Group Alone

So far, the group with dyslexia has been shown to perform more poorly than controls on two measures associated with magnocellular-dominated visual processing (FDT and slope of conjunction search with target present). To address the question of whether the *severity* of dyslexia within the dyslexic group is related to the degree of visual deficit, correlational analyses (Pearson's product moment) were performed between the literacy measures (DAST and WRAT) and the two visual processing measures (FDT, as assessed by the mean deviation index, and conjunction search slope). Results are presented in Table 3. Significant correlations were obtained between the search slope and both phonological ability and the literacy measures of the DAST. The direction of the correlations

Table 3. Correlations (Pearson's product moment) between performance on visual tasks and literacy abilities of dyslexic participants (N = 10).

	Literacy tasks			
Visual tasks	PA	LM	Readine	Spelling
MDI	0.28	0.23	0.52	0.36
Search slope	0.62[a]	0.56[a]	0.29	0.28

[a] Correlation significant at the 0.05 level (2 -tailed).
N. B.: The direction of scores have been coded so that, in all cases, positive correlations indicate that more severe deficits on one task are related to more severe deficits on the correlated task.

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were such that, when the visuo-spatial deficit was more severe so too were deficits on the DAST measures.

Correlation between the two visual tasks was also calculated, $r(9)=0.011$. Thus there does not appear to be a relationship between performance on the FDT and that on conjunction search within the dyslexic group.

DISCUSSION

The results of this study have demonstrated that adults with dyslexia are less sensitive than non-dyslexic adults to a stimulus primarily processed by $M(y)$ cells in the magnocellular pathway, namely the frequency doubling grating (i.e. a low-spatial, high temporal frequency, and very low contrast stimulus). Impairment was also observed on the conjunction visual search task, with which parietal cortex (dorsal stream) attentional shift function is associated. These deficits on magnocellular/dorsal stream dominated tasks were observed in the context of normal performance on a ventral stream task (acuity) and also on feature search. These results are consistent with the many previous findings of a magnocellular/dorsal stream deficit in developmental dyslexia.

In terms of severity of the deficits, on the FDT task all but two dyslexics performed outside the 95% CI for control performance. [The mean deficit for the group with dyslexia relative to controls on this task (1.72 dB MDI) was smaller than that found by Pammer and Wheatley (2001), in children with dyslexia (4.54 dB MDI). A possible contribution of attention lapses in the children with dyslexia cannot be ruled out, since Pammer and Wheatley did not ''address the possibility of ADHD comorbidity.''] On the conjunction visual search task all but one dyslexic's performance were outside the 95% CI for control performance.

Within the dyslexic group, the two magno/dorsal visual measures did not correlate with each other. However, a relationship was found between conjunction visual search deficit and phonological ability, and between conjunction visual search deficit and timed reading and spelling measures (from the DAST). These findings add to the results of previous studies which have observed a correlation between magnocellular dominated visual processes and phonological ability (e.g. Cestnick & Coltheart, 1999; Olson & Datta, 2002; Pammer & Wheatley, 2001; Witton *et al*., 1998).

Can Concentration Lapses Account for Dyslexic Deficits?

Our results argue that deficits of those with dyslexia on magnocellular-related tasks cannot be attributed simply to concentration lapses (Davis *et al*., 2001; Stuart *et al*., 2001). First, the FDT instrument used in this study provided inbuilt control for factors such as inattention and level of motivation through measures of false positive, false negative and fixation errors. Second, if inattention and/or motivation were contributing factors, then the group with dyslexia should have demonstrated deficits on all tasks with an adaptive threshold, specifically including the Landolt Ring-gap task. Finally, while a general sample of children with dyslexia might well show poor concentration, resulting in poor performance on psychophysical tasks, this study examined high functioning adults without a history of ADHD who performed well on other demanding tasks requiring good

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concentration. (e.g. performance tests of the WASI). [An anonymous reviewer suggested that another way of directly assessing the concentration lapse idea would be to compare the variance of the staircase reversal points across dyslexics and controls. Unfortunately, the apparatus used in this study did not allow access to this measure.]

Specific Type of Parietal Deficit

The results of this study support previous findings of a conjunction visual search deficit in dyslexia (e.g. Buchholz *et al*., in prep; Casco & Prunetti, 1996; Iles *et al*., 2000; Vidyasagar & Pammer, 1999). There are several functions of the parietal cortex in which dyslexics have shown deficits and which could be the origin of poor visual search performance. In principle, these include abnormal eye movements (e.g. Eden *et al*., 1994; Griffen *et al*., 1998; Stein, Riddell & Fowler, 1988), spatial crowding (Atkinson, 1991), and peripheral stimulus processing abnormalities (Valdois, Gerard, Vanault, & Dugar, 1995), as well as visual attention *per se* (Brannan & Williams, 1987). We believe, however, that it is unlikely that eye movement or peripheral processing difficulties were major contributors to the deficits observed here. Firstly, in this study, eye movements were minimised by requiring subjects to fixate at the centre of each presentation, with the maximum time of presentation being 330 ms and secondly, visual search performance has been shown to be independent of eye movements (Corbetta *et al*., 1995). Crowding is also unlikely to be a major factor since set size$^{\infty}$ group interaction effects were not seen on feature search tasks (see Iles *et al*., 2000). Thus the deficits shown appear primarily related to the visual attention function of the parietal cortex, specifically that of locating an object requiring a binding of two features.

Theoretical Relationship between Visual Deficits and Reading Performance

Phonological decoding skills are a primary component of developing reading performance in children (Fowler, 1991; Share, 1995). Thus, in theoretical terms, the correlation (or otherwise) between our magnocellular-driven visual tasks and our phonological awareness measure is of particular interest. Performance on the FDT task—designed to tap early-stage magnocellular processing—did not correlate significantly (r=0.28) with phonological awareness. In contrast, performance on the conjunction visual search task—designed to tap later-stage dorsal processing—correlated strongly (r=0.62) with phonological awareness. This pattern is consistent with the idea that visual processing associated with dorsal stream functioning constrains reading ability, rather than low level magnocellular function *per se*.

How could a deficit in parietal cortex function relate to problems with reading? One possibility (Vidyasagar, 1999, 2001) is that a deficit in the parietal function of directing spatial attention plays a direct causal role in dyslexia, since reading text requires controlled shifts of attention to different locations in space. Vidyasagar proposes that these shifts do not occur naturally (Horowitz & Wolfe, 1998), and that perhaps dyslexics are poor at learning to guide visual attention. A similar idea has been proposed by Cestnick and Coltheart (1999) specifically with respect

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to the grapheme-to-phoneme conversion process required in nonword reading, in that, 'nonword reading requires successive allocation of attention to successive positions in the letter string' (p. 250). Keeping in mind that, for a child, all real words are at first nonwords, a deficit in allocation of attention to successive graphemes could well produce developmental problems in learning to read.

Finally, while we suspect that visual deficits do play some causal role in dyslexia, we note that dyslexic perceptual difficulties are clearly not limited to vision. Many studies have reported problems on a number of auditory tasks, such as temporal order judgement (Tallal, 1980; Nagarajan *et al*., 1999) and frequency discrimination (McAnally & Stein, 1996, Ahissar, Protopapas, Reid, & Merzenich, 2000) (for reviews see Farmer and Klein, 1995; McArthur & Bishop, 2001). These could well be the immediate cause of dyslexic problems with phonological tasks such as auditory nonword repetition and phoneme deletion (Cestnick & Jerger, 2000). Moreover, poor representation of phoneme order is likely to lead to problems in learning to read novel words as much as is poor representation of letter order (grapheme-to-phoneme conversion would fail in either case). Thus, phonological dyslexia could result, in individual subjects, from visual deficits, or from auditory deficits, or perhaps from a combination of both caused by a general problem with the development of magnocellular paths in both perceptual systems (see Cestnick, 2001).

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