Visual Search Performance in Dyslexia

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According to the magnocellular theory of dyslexia, otherwise intelligent children may fail to learn to read because of abnormalities in the magnocellular layers of the lateral geniculate nucleus (mLGN). If this were the case, one would predict that dyslexic subjects who show a deficit on low-level psychophysical tasks which tax the magnocellular system would also have deficits on higher-level visual tasks which do not rely on the properties of mLGN cells but depend upon the functioning of areas whose main inputs originate in the mLGN. In other words, magnocellular deficits should be traceable at later stages of visual processing. One area where such later processing is thought to occur is the posterior parietal cortex, damage to which impairs function on some classes of visual search. To test this hypothesis, we tested two groups of dyslexic subjects and a group of non-dyslexic controls on a range of visual search tasks. One group of dyslexic subjects had elevated motion coherence thresholds, a sign of deficits at the early levels (e.g. mLGN) of visual processing, and the other group had normal motion coherence thresholds. If the magnocellular deficits extended to the parietal cortex, it follows that the subjects with elevated motion coherence thresholds should have deficit in visual search, whereas those with normal motion coherence thresholds should not. The dyslexics with a motion coherence deficit were also impaired on serial visual search tasks but not on a parallel search. The dyslexics with normal motion coherence performance were unimpaired on visual search. The deficit was expressed as an elevation in reaction times, but there was no difference between the groups either in error rates or in the way the tasks were ranked according to difficulty. The results suggest that those dyslexics who have visual problems related to magnocellular functions also have visual–attentional problems related to the functions of areas such as the parietal cortex, which are dominated by inputs originating in the magnocellular LGN.

Keywords: visual search; magnocellular hypothesis; parietal cortex

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INTRODUCTION

Dyslexia is a developmental disorder affecting between 3% and 10% of the population (Yule *et al.*, 1974). The disorder comprises a range of language-related deficits, including poor reading, writing and speech production skills, and many of these deficits can be accounted for within phonological accounts of dyslexia (e.g. Vellutino, 1977; Ellis and Miles, 1978; Llundberg, Olofsson and Wall, 1980; Bradley and Bryant, 1983; Liberman, 1989; Snowling and Rack, 1991; Frith *et al.*, 1995). Some dyslexics also possess deficits in their low-level visual abilities (e.g. Lovegrove, Heddle and Slaghuis, 1980; Badcock and Lovegrove, 1981; Slaghuis and Lovegrove, 1985; Martin and Lovegrove, 1987; Williams *et al.*, 1989, 1990; Livingstone *et al.*, 1991; Stein and Walsh, 1997), but a hypothesis to account for the role of visual problems is not well established. Indeed, visual deficits are sometimes not considered relevant to a dyslexic's poor performance in reading tasks (e.g. Frith and Frith, 1996; Shaywitz, 1996). A recurring error in the reasoning behind this dismissal of vision is to consider visual and phonological accounts of dyslexia as mutually exclusive. As argued elsewhere, they may be just two faces of a very complex disorder which demands a more sophisticated, supramodal explanation (Stein and Walsh, 1997).

The Evidence

Impairment on visual tasks which require rapid processing of visual information has been found consistently in dyslexics, implying that the magnocellular cells in the lateral geniculate nucleus (mLGN), important in detecting fast, low-contrast information, may be impaired in dyslexia (Livingstone *et al.*, 1991; see also Breitmeyer *et al.*, 1981; Lovegrove, Gazia and Nicholson, 1990; Stein, 1993, 1994; Cornelissen *et al.*, 1995; Borsting *et al.*, 1996; Edwards *et al.*, 1996). Three recent studies are of particular relevance to this paper. Cornelissen *et al.* (1995) tested the ability of dyslexic and non-dyslexic children and adults to detect motion coherence in random-dot kinematograms. On average, motion coherence thresholds in dyslexics were 3%–4% higher than in normal readers of the same age. A subset of dyslexics had a significant motion coherence impairment, a fact we have exploited in the experiment reported here. The most direct demonstration to date, however, is a recent functional magnetic resonance imaging (fMRI) study in which moving stimuli failed to activate V5 (an important motion area) in dyslexic subjects (Eden *et al.*, 1996). Other studies suggest that there is no such visual impairment in dyslexia (Victor *et al.*, 1993; Gross-Glenn *et al.*, 1995; Johannes *et al.*, 1996). The most parsimonious way of interpreting the disparities between different reports would be to accept that some dyslexics do and some dyslexics do not have low-level visual deficits. Accordingly, we have used two such populations in this study.

The Rationale

Relating an LGN deficit to reading may appear to present problems, because cells in this area do not possess the characteristics of a 'reading area'; for example, they have small receptive fields, respond to simple stimuli and are insensitive to form. Magnocellular LGN outputs serve areas other than the cortical motion area (V5), and

one would therefore expect visual deficits to reflect this. A major output area of the magnocellular system is the parietal cortex, which receives mLGN afferents via areas V3 and V5 in extrastriate cortex. Functions associated with parietal visual areas such as attention and visuospatial integration could both be regarded as important in reading (La Berge and Samuels, 1974; Riddoch *et al.*, 1990; Atkinson, 1991). The parietal cortex has been associated with dyslexia for some time (see Stein and Walsh, 1997). Eden, Stein and Wood (1991), for example, showed that the tendency to draw clocks showing signs of left-sided distortion is much more common in reading-disabled children than in normal readers. This is similar in character to the spatial hemineglect seen after right parietal lesions. Children with poor vergence control show evidence of impaired right posterior parietal function (Stein, Riddell and Fowler, 1989), and patients with a damaged right posterior parietal cortex (PPC) show markedly impaired vergence control (Fowler *et al.*, 1989). Valdois *et al.* (1995) also report attentional deficits in a developmental dyslexic which mirror those seen in acquired dyslexia associated with parietal cortex damage (Kinsbourne and Warrington, 1962; Behrman *et al.*, 1990).

The rationale for this study, then, rests on three observations: first, an established abnormality in mLGN of some dyslexics; second, the similarity between the effects of PPC damage and some visuospatial deficits in developmental dyslexics; and third, a well-established, strong anatomical link between mLGN and PPC.

Attention and Search

A function which appears to be dependent upon the parietal cortex is visuospatial attention (Posner, 1980; Lawler and Cowey, 1987; Arguin, Joanette and Cavanaugh, 1993). Attention is demanded in and therefore often tested using spatial cueing tasks or visual search tasks. Dyslexics perform abnormally on spatial cueing tasks (Brannan and Williams, 1987) and have also been found to be impaired on visual search tasks (Williams, Brannan and Lartigue, 1987; Ruddock, 1991). Some search tasks incorporate components of reading: target identification may correspond to letter or word identification; inhibition of distractor items can resemble inhibition or grouping of unattended letters or words—and rapid integration across space is required in both tasks. Williams, Brannan and Lartigue (1987) required subjects to search through lists of characters for a target character. Subjects were instructed to scan each line of the array from top to bottom as quickly as possible for the previously specified target letter. Search time was found to increase with the target's position in the array. Although the poor readers were much slower on all arrays compared with good readers and adults, it is possible that the arrays and instructions may have encouraged reading-like eye movements. A visual search task which does not induce reading-like strategies would be more useful in determining some of the basic visual processing differences between good and poor readers.

A consistent finding in the visual search literature is that different strategies are used when the nature of the detection task changes (Treisman and Gelade, 1980). Parallel search or 'pop-out' occurs when the target appears to leap from the display with little effort required from the observer, search time not being affected by the number of items in the display. Serial search is an attentive process and is used, for example, when a conjunction of two features needs to be found; search time then increases with an increasing number of distractors present in the array.

Table 1. Details of the handedness, sex, IQ and reading abilities of the subjects used in this study

	MDs	NMDs	Controls	*P*
Handedness	7R, 1L	7R, 1L	7R, 1L	
IQ	117 (8.86)	117 (8.69)	115 (9.48)	>0.05
Reading words				
Time	102 (9.67)	109 (23.39)	54 (2.82)	<0.001
Errors	12 (5.52)	9 (6.10)	0 (0)	<0.001
Non-words				
Time	48 (7.24)	44 (16.18)	23 (3.60)	<0.001
Errors	7 (1.8)	7 (2.87)	1 (0.50)	<0.001
Motion coherence threshold[a]	15.93 (3.75)	8.02 (1.16)	9.98 (3.36)	

[a]Motion coherence thresholds were measured by Cornelissen *et al.* (1995).

For this study we hypothesized that mLGN abnormalities would manifest as a parietal cortex deficit in dyslexic subjects, which could thus contribute to the reading disability. Visual search tasks were given to three groups: (i) a control group, (ii) a group of dyslexics who had normal motion coherence thresholds and (iii) a group of dyslexics who had elevated motion coherence thresholds. If visual search is dominated by the integrity of the parietal cortex, and visual dyslexia is partly a consequence of parietal cortex dysfunction (Stein, 1993), then deficits on visual search task performance by dyslexics are to be expected. In addition, we predicted that dyslexics with clear deficits in the perception of motion coherence would be more impaired than dyslexics without motion coherence deficits.

METHOD

Subjects

Twenty-four subjects took part in the experiment: (i) eight controls, (ii) eight dyslexics with normal motion coherence thresholds (NMDs) and (iii) eight dyslexics with elevated motion coherence thresholds (MDs). Subjects were matched for age (controls, mean 25, range 20–43; NMDs, mean 27, range 15–47; MDs, mean 32, range 17–46; one-way ANOVA $P>0.05$) and sex (controls, six male, two female; NMDs, seven male, one female; MDs, six male, two female). The dyslexics were taken from the population studied by Cornelissen *et al.* (1995). All had been previously diagnosed as dyslexic by educational psychologists, and we confirmed this with measures of reading real and nonsense words (Table 1). The motion deficit (MD) group had motion coherence thresholds at least 1.5 standard deviations above those of the normal population; the non-motion deficit group (NMD) did not differ from the control population. Details of reading ability, motion coherence thresholds and IQ are shown in Table 1.

Stimuli

Eight visual search tasks were used, as represented schematically in Figure 1. A simple motor reaction time task was also given to all subjects. The subjects were required to

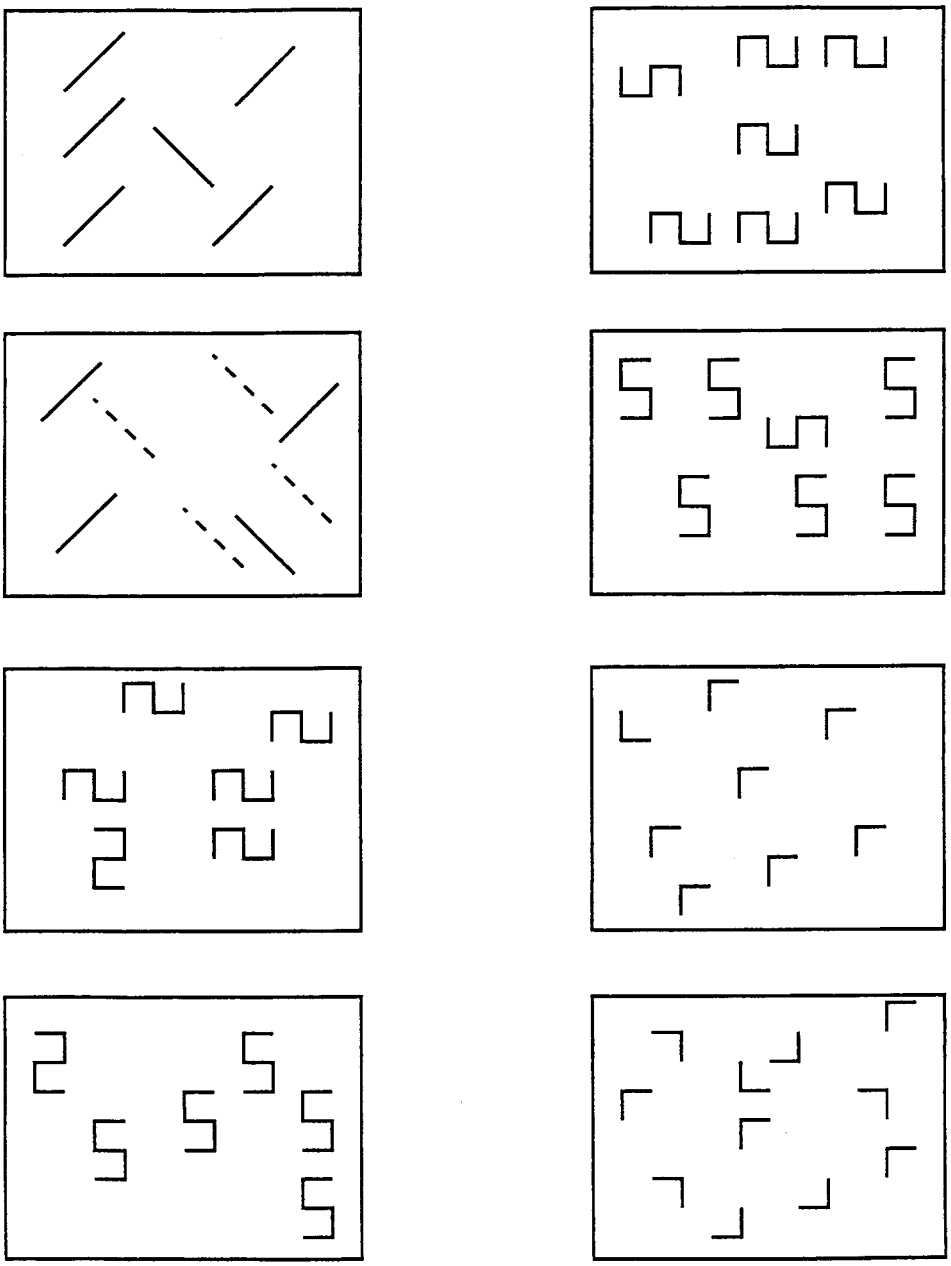

Figure 1. Schematics of the eight visual search tasks used in this study. The top left shows an orientation pop-out array. The succeeding arrays in the left-hand column show an example of an orientation 'colour' conjunction, familiar target/unfamiliar distractors and familiar target/familiar distractors. The right-hand column shows an unfamiliar/unfamiliar array, unfamiliar/familiar, homogeneous distractors and heterogeneous distractors.

respond with a mouse press to the appearance of any visual stimulus on the monitor. The same arrays were used for this as for the search testing.

Stimuli were generated on a 280 mm × 200 mm monitor driven by a 486DXZ 66 MHz PC. In each condition the target and distractors appeared randomly at one of 48 possible sites. With the exception of task 2 (colour/orientation conjunction), white stimuli were presented on a black background. The line thickness of the stimuli was 3 mm, and the stimuli were drawn to fill virtual boxes of size 35 mm × 33 mm, allowing for a random jitter of 3 mm in any direction to prevent alignment of stimuli. On each trial the target was either present or absent; the number of distractor items was randomly selected from 1, 2, 4, 8 or 16. The target was present on 50% of the trials in each block.

Procedure

Subjects sat 95 cm away from the screen. Eight blocks of 100 trials were presented to each subject; in each block only one pair of stimuli was tested. A typical testing session took 1 h 15 min. At the beginning of each trial a white fixation point appeared in the centre of the screen and a tone was sounded for 500 ms to alert the subject. The stimuli appeared immediately after the tone and remained visible until the subject responded. The inter-trial interval was 3000 ms and no feedback was given. The subjects placed their dominant hand over a computer mouse and were instructed to press the left button if the target was present and the right button if it was absent. The mouse was fixed at 90° to the subject such that the response was top button for present and bottom button for absent.

Speed and accuracy of response were stressed. Before the eight test blocks, the subject was given 160 practice trials, or until the error was less than 5%. In general, only one practice session was required for each subject.

RESULTS

There were no differences between the groups on the simple motor reaction time (RT) task: one-way ANOVA $P>0.05$ (mean RTs ± 1SD) for the control, NMD and MD groups were 435 ± 56, 480 ± 80 and 405 ± 52 ms respectively. The search time data for all eight conditions were analysed by a two-factor mixed-measured ANOVA (subject group × distractors). Figure 2 shows the RTs as a function of number of distractors for all tasks. Only present and correct responses were used for analysis.

Distractor Main Effect

In condition 1, the pop-out condition, the main effect of distractors was not significant ($P>0.05$). In all conditions 2–8 the main effect of distractors was significant ($P<0.0001$).

Subject Group Main Effect

The main effect of subject group was not significant in condition 1. In condition 2, a conjunction of colour and orientation, the main effect of subject group was significant

($F=4.46$, $P=0.0242$) and there was also an interaction between group and number of distractors ($F=2.34$, $P=0.0254$). In condition 3, when the target was familiar and distractors were unfamiliar, the main effect of subject group was significant ($F=6.80$, $P=0.0053$). The group × distractors interaction was not significant. In condition 4, when target and distractors were both familiar, the main effect of subject group was significant ($F=3.61$, $P=0.0449$). Again, the interaction was not significant. In condition 5, when target and distractors were both unfamiliar, the main effect of subject group was significant ($F=11.38$, $P=0.0004$) and the interaction between distractors and subject group was significant ($F=2.29$, $P=0.0286$). In condition 6, when the target was unfamiliar and distractors were familiar, the main effect of subject group was significant ($F=7.57$, $P=0.0033$). The interaction between distractors and subject group was not significant. In condition 7, when distractors were homogeneous, the main effect of subject group was significant ($F=10.90$, $P=0.0012$). The interaction was not significant. In condition 8, when distractors were heterogeneous, the subject group effect was significant ($F=5.90$, $P=0.0129$). The interaction was not significant.

Post hoc Tests

Planned comparisons between the groups showed that the MD group was significantly slower than the control group on tasks 2–8. In order of tasks the effects were: (task 2) $F=8.73$, $P<0.01$; (task 3) $F=12.52$, $P<0.01$; (task 4) $F=7.11$, $P<0.05$; (task 5) $F=22.37$, $P<0.01$; (task 6) $F=11.69$, $P<0.01$; (task 7) $F=20.60$, $P<0.01$; and (task 8) $F=11.65$, $P<0.01$. However, in only one condition was there any significant difference between the control group and the NMD group. This was condition 5 (unfamiliar target and unfamiliar distractors, $F=3.36$, $P<0.05$).

Task Difficulty

To illustrate the relative difficulty of all the tasks, linear regressions of the slopes were compared. These are displayed in Figure 3, which shows clearly that all groups performed the colour pop-out task as a parallel, pre-attentive search (i.e. a slope of less than 10 ms per item). In all cases the groups appeared to rank the tasks in the same order of difficulty. The only qualitative differences between the groups is on task 7 (homogeneous distractors), which the control group performed as a parallel search but the two dyslexic groups performed as a serial search (16 and 24 ms per item for the NMD and MD groups respectively).

In terms of errors there are no differences between any of the groups (Figure 4) (Kruskall–Wallis, $P>0.05$ in all cases).

DISCUSSION

The results show that dyslexics with elevated motion coherence thresholds are impaired on visual search tasks which require serial search, but are unimpaired on a simple pop-out task (task 1). Dyslexics with normal motion coherence thresholds are impaired on only one of the visual search tasks (task 5). Both groups of dyslexic subjects ranked the tasks in the same order of difficulty as the non-dyslexic controls;

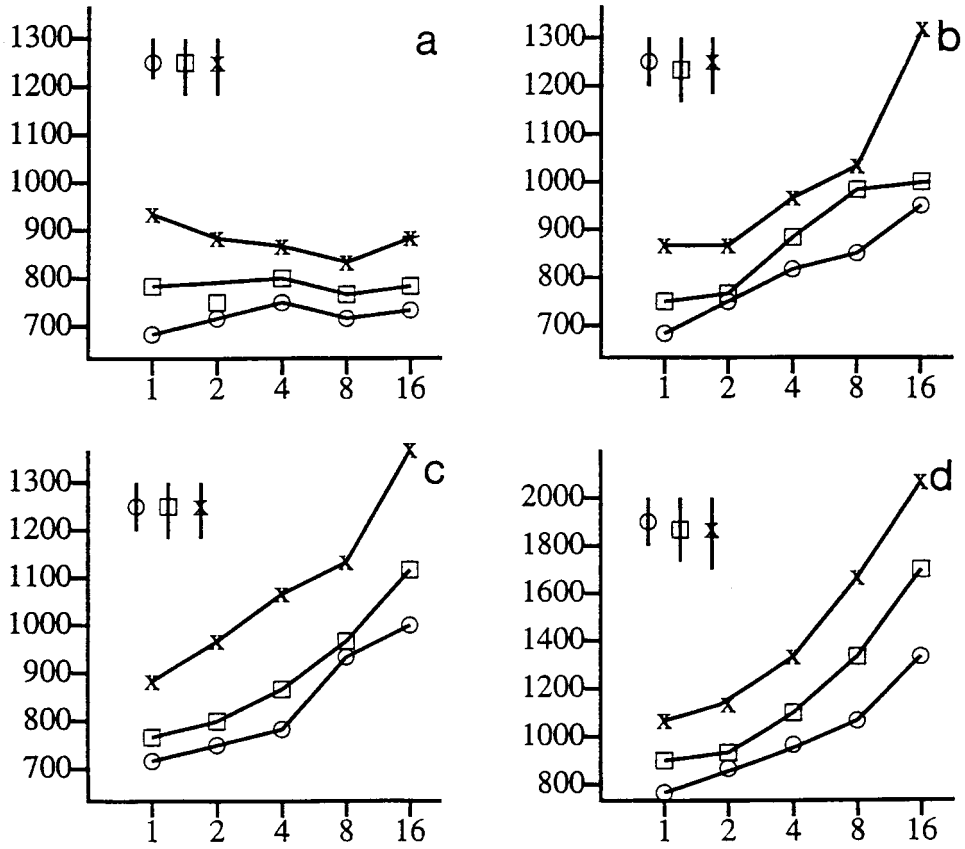

Figure 2. (a)–(d).

that is, the deficits observed represent a quantitative rather than a qualitative difference between the groups. The only exception to this is on task 7 (homogeneous distractors), which the control group performed in parallel, whereas the dyslexic groups performed the task with a shallow serial slope. The two dyslexic groups differed significantly from each other on four tasks (tasks 3, 5, 6 and 7), which were all classified as non-reading-like. However, on tasks 4 and 8, which we classified as reading-like, there were no significant differences between the two groups of dyslexics. The results cannot be explained an an effect of simple RT deficits, otherwise the impairment would have penetrated task 1. Further, simple RT effects cannot explain the interaction effect between set size and group (task 5).

Conjunction search requires focal attention and feature binding (Treisman and Gelade, 1980) which are associated with normal parietal cortex functioning, and patients with parietal lesions are impaired at conjunction search (Arguin, Joanette and Cavanaugh, 1993). We suggest, therefore, that mLGN abnormalities, as indexed by deficits in motion coherence tasks, can extend beyond V5 and produce deficits akin to those seen following damage to the parietal cortex in man. The result reported here adds to a catalogue of visuospatial deficits seen in dyslexics: left neglect (Eden, Stein and Wood, 1991; Stein, 1993), eye movement abnormalities (Eden *et al.*, 1994), visual attention (Brannan and Williams, 1987; Valdois *et al.*, 1995), crowding (Atkinson,

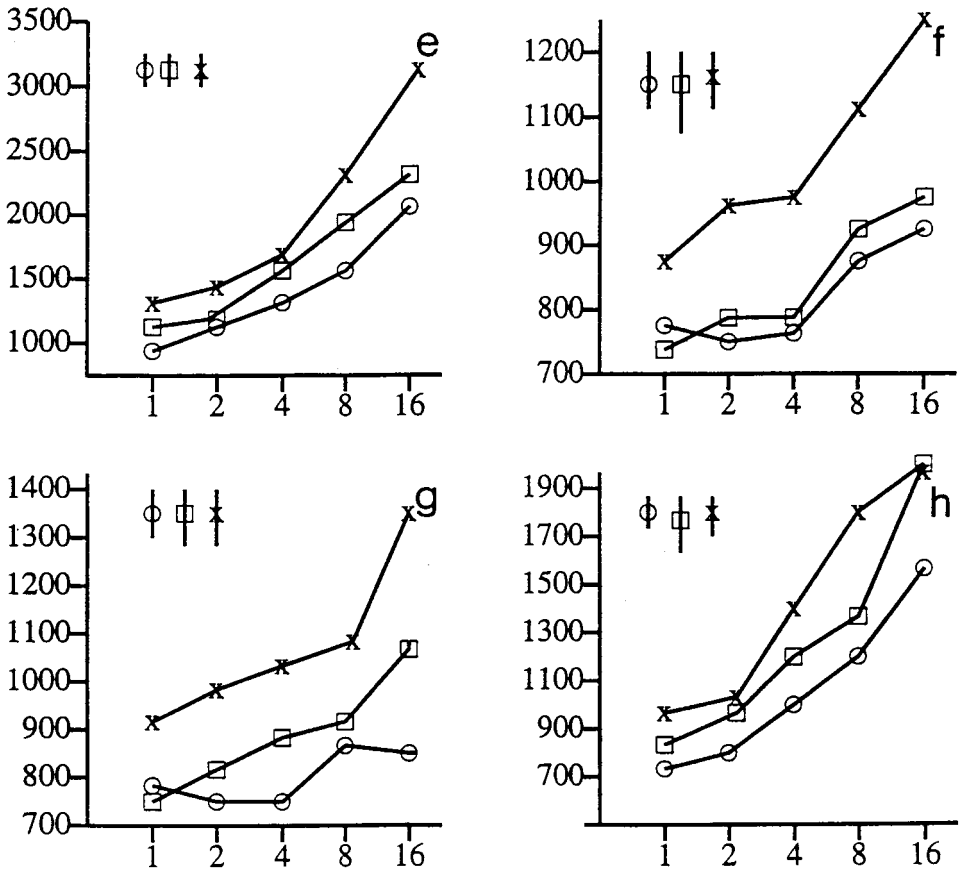

Figure 2. (e)–(h).

Figure 2. Set size × reaction time functions for all eight tasks. In all cases the control group is represented by open circles, the non-motion deficit dyslexic group by open squares and the motion deficit dyslexic group by crosses. (a) Orientation pop-out task; (b) colour/orientation conjunction; (c) familiar target/unfamiliar distractors; (d) familiar target/familiar distractors; (e) unfamiliar target/unfamiliar distractors; (f) unfamiliar target/familiar distractors; (g) homogeneous distractors; (h) heterogeneous distractors.

1991), peripheral vision (Geiger and Lettvin, 1987) and visual search (Williams, Brannan and Lartigue, 1987; Ruddock, 1991).

Comparison with Other Studies of Search and Dyslexia

The results concur with the findings of Williams, Brannan and Lartigue (1987) and Ruddock (1991). However, there are some differences between the tasks used and between the underlying rationales of those studies and our experiment. As noted in the Introduction, it is not at all clear that the results of Williams, Brannan and Lartigue (1987) can be regarded as a visual search effect owing to some of the reading-like qualities of their task. Our results therefore extend their conclusions to visual search in non-reading-like arrays and with non-lexical stimuli. Ruddock (1991) demonstrated

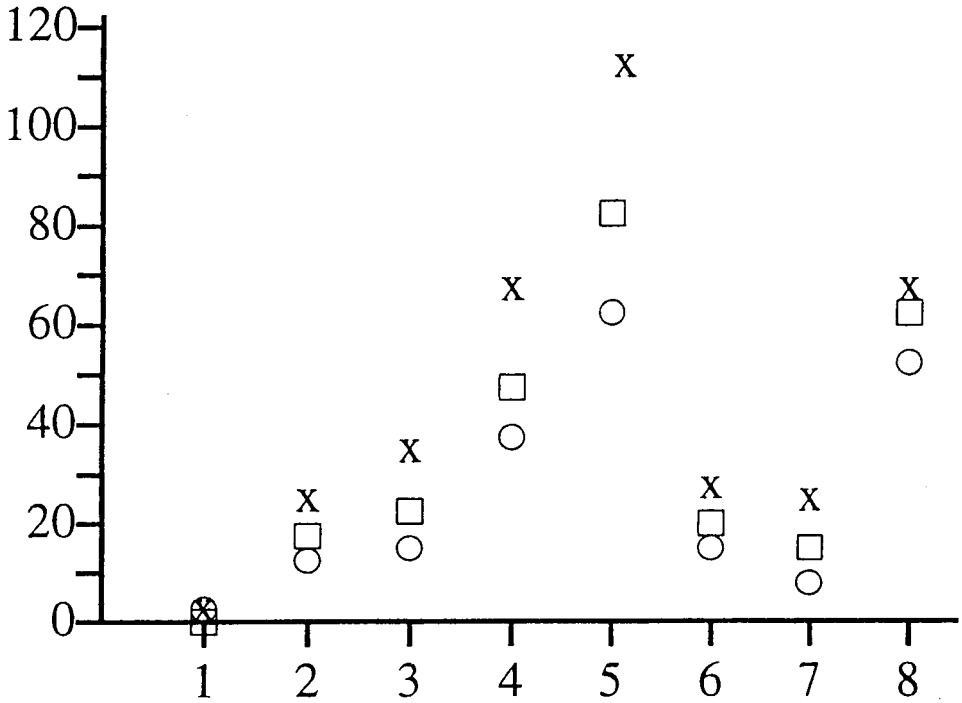

Figure 3. Slopes of the RT × distractor functions for the eight tasks. The abscissa contains the tasks (1–8) and the ordinate the slope calculated in milliseconds per item. Symbols as for Figure 2.

search effects in two dyslexic subjects (M.H. and A.B.). One of these (M.H.) performed an orientation pop-out task in parallel, but failed to perform in parallel another pop-out task which required detection of an equilateral triangle from distractors which were rotated versions of the target. Two other subjects (A.W. and K.B.) were tested on a difficult search task in which a square or a rotated square had to be detected from other rotated squares. One of the subjects performed this task with a similar slope to the control subject, but both subjects made 10–20 times more errors than the control. Ruddock's dyslexic subjects were clearly different from ours, who did not make any more errors than the controls (Figure 4). Despite differences between the studies, however, the collective conclusion is that some dyslexics have visual search deficits.

Visual Deficits Unrelated to Language Difficulties

Several functions of parietal cortex are good candidates for being able to cause deficits in visual search: eye movement abnormalities, focal attention problems, crowding and failure to inhibit irrelevant stimuli. It is our contention that deficits in any one of these functions can be accounted for without invoking language problems. Further, all of these functions may be considered as necessary contributors to reading.

Visual crowding is prevalent in young children (3–5 years) and in dyslexic children when compared with reading-age-matched or chronological-age-matched controls (Atkinson, 1991). It is also a good example of a visual deficit which cannot be accommodated within a purely phonological framework of reading impairments. We

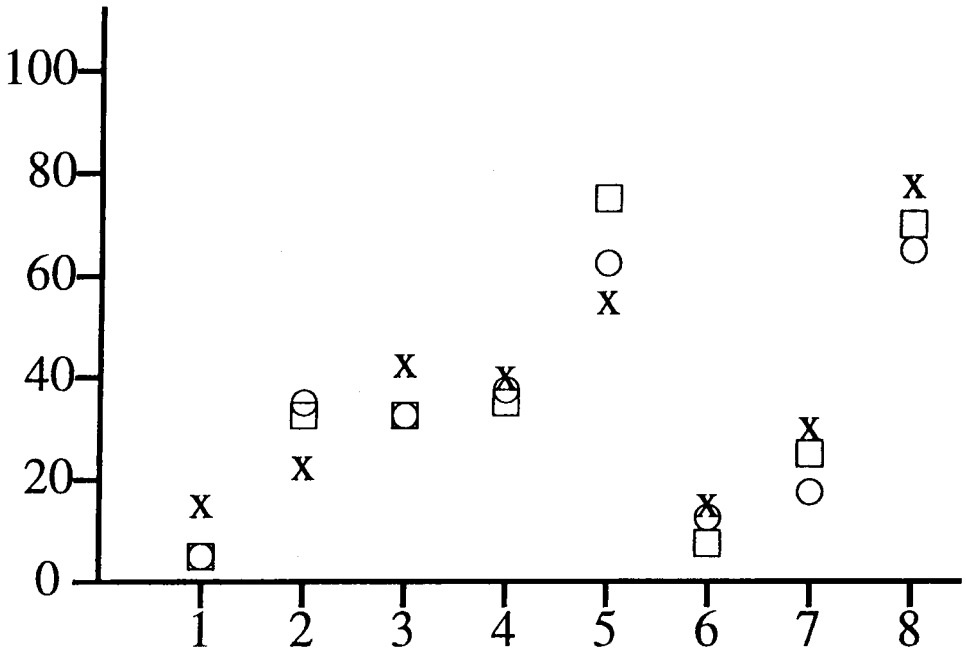

Figure 4. Total number of errors made by each of the three groups on the eight search tasks. Symbols as for Figure 2.

cannot exclude the possibility that crowding contributed to the deficits we observed in visual search, but we think it unlikely. If crowding were a major factor, then one would have predicted a set size × group interaction effect on all tasks, since an increase in the number of distractors would have increased the probability of distractors being close to the target.

Visual attention problems constitute another class of deficits related to parietal cortex damage. Dyslexics have been shown to be impaired on a range of attention tasks: the Posner task (Brannan and Williams, 1987), perceptual grouping (Williams and Bologna, 1984) and visual search. Attention also requires inhibition of the stimuli which are not the focus of attention, and there is also evidence that dyslexics may fail on this aspect of attention (Rayner *et al.*, 1989). Parietal cortex is also associated with visual processing of stimuli in the periphery of the visual field, yet another aspect of visual processing which is abnormal in some dyslexics.

A referee of this paper commented that perhaps the deficits we observed could be accounted for by general attentional and motivational factors. This may be the case, but it would broaden the hypothesis from one concerning the magnocellular system to one which proposed that any component of the attention system could be deficient. This is not at odds with our thinking, but even if the hypothesis were broadened, it would doubtless have recourse to the role of the parietal visual cortex which is magno-dominated.

mLGN Hypothesis and Reading

In extending the mLGN hypothesis to parietal cortex, we are able to challenge the view that while dyslexics may have visual deficits, these are either a consequence of

phonological deficits or are irrelevant to the reading process (Frith and Frith, 1996). The counter argument has three components. First, the anatomical continuity between mLGN and parietal cortex is well established. Parietal cortex receives a strong input from V3 and V5, which in turn have received a strong input from the magno-recipient laminae of V1. We of course recognize the intermingling of parvocellular and magnocellular pathways; nevertheless, there is still a meaningful pattern of relative segregation through the cortex (Ungerleider and Mishkin, 1982; Maunsell and Merigan, 1993). Second, in principle and in practice it has been established that deficits on psychophysical tasks can be associated reliably with diminished activity in magno-recipient cortical regions (Eden *et al*., 1996). Third, as we have argued above, some deficits on visual tasks are (a) unrelated to phonological processing and (b) reflect processes involved in reading.

Visual deficits are not, of course, the only source of dyslexia. Dyslexics have deficits in auditory processing which share some of the features of the deficits in visual processing. An important similarity between visual and auditory deficits is that they are most apparent when dyslexics are required to process rapid temporal information in either domain (Stein, 1994; Tallal, Miller and Fitch, 1993). Further, just as there is anatomical evidence for the visual magnocellular deficit (Livingstone *et al*., 1991), so too there is anatomical evidence for abnormalities in the medial geniculate nucleus (MGN) of dyslexics (Galaburda, Menard and Rosen, 1994); MGN neurons are smaller and organized differently in dyslexics than in the brains of normal readers. Indeed, the notion that dyslexics have impairments in processing fast temporal information may be extended to the motor system (Nicolson and Fawcett, 1995), where a range of deficits have also been found in populations of dyslexic subjects.

CONCLUSIONS

The visual search deficits we observed in a subpopulation of dyslexics extend the magnocellular LGN hypothesis of dyslexia. It therefore seems clear that an understanding of dyslexia will not be achieved without an account of the role of the visual system in reading. To this extent, our study supports those of Lovegrove, Stein, Eden, Breitmeyer and others. However, given the wealth of data on visual and auditory performance in dyslexics, it also seems clear that neither a visual nor a phonological explanation of dyslexia will be sufficient to capture the richness and heterogeneity of the performance profiles of dyslexics on a wide range of tasks.

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