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Contrast Sensitivity and Coherent Motion Detection Measured at Photopic Luminance Levels in Dyslexics and Controls

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Developmental dyslexics perform differently from controls on a number of low level visual tasks. We carried out three experiments to explore some of these differences. Dyslexics have been found to have reduced luminance contrast sensitivity at mesopic luminance levels. We failed to replicate this finding at photopic luminance levels. We also compared the (photopic) coherent motion detection thresholds of groups of child and adult dyslexics with those of age matched controls. Dyslexics were significantly less sensitive to motion. The results are discussed in relation to a recent suggestion that developmental dyslexia may be associated with a magnocellular visual deficit.

Dyslexia Photopic Contrast sensitivity Coherent motion Magnocellular

INTRODUCTION

General background

Developmental dyslexia or specific reading disability affects 3–10% of school children (Rutter & Yule, 1975). For practical purposes, dyslexia is most often defined in terms of discrepancy: despite adequate educational opportunity, dyslexics' reading ability is significantly lower than that predicted from their age and IQ. Most of the research into developmental dyslexia has focused on the impaired phonological skills of disabled readers; e.g. they have poor phoneme awareness—i.e. the ability to detect that "fan" is the odd item in the sequence "hat", "mat", "fan" and "cat". Phoneme awareness is thought by many researchers to be a pre-requisite for mastering the phonological rules relating spelling to sound during reading development (Bradley & Bryant, 1983; Lundberg, Olofsson & Wall, 1980; Wagner & Torgeson, 1987). Consequently, dyslexics find it particularly difficult to read nonwords, a skill which depends on application of spelling-sound rules, even when they are compared to younger children of the same reading age (Snowling & Rack, 1991).

Contrary to the views originally expressed by Vellutino and others (Benton, 1975; Vellutino, 1979), research in the last decade has also shown convincingly that dyslexics perform differently from controls at low level visual tasks (Willows, Kruk & Corcos, 1993). Moreover, there have been recent suggestions that developmental dyslexia may be associated with abnormalities in the magnocellular pathways of both the visual and auditory systems (Tallal, Galaburda, Llinas & von Euler, 1993). In this paper we consider the question of whether dyslexia may be associated with a visual magnocellular (M pathway) deficit.

M pathway dysfunction and dyslexia

Two lines of evidence have been adduced in support of an M pathway deficit in dyslexia. The most direct approach was described by Livingstone Rosen, Drislane and Galaburda (1991) who carried out anatomical and visually evoked potential (VEP) studies. In a histological comparison of five normal vs five dyslexic brains, these authors found that the ventral, magnocellular layers of the lateral geniculate nucleus (LGN) in dyslexic brains contained fewer, smaller cells than the comparable layers in normals. By contrast, no group differences were found in the cell sizes of the parvocellular layers of the LGN. The VEP data showed that dyslexics had reduced amplitudes in their responses to high temporal frequency, low luminance, low contrast stimuli—conditions which are thought to stimulate the M pathway optimally (see also Riddell & Hainline, 1993; Lehmkuhle, Garzia, Turner, Hash & Baro, 1992). A failure to replicate the VEP results was reported by Victor, Conte, Burton and Nass (1993). But, as these authors point out themselves, it is not clear that their selection of subjects was comparable with other studies. For example, Victor *et al.* used a wide age range of subjects (6–46 yr) and they relied primarily on a

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previous history of unexpected reading failure instead of directly measuring the reading deficit.

The second line of evidence for an M pathway deficit in developmental dyslexia is less direct. It is one of several possible interpretations of the differences in luminance contrast sensitivity which have been found when groups of dyslexic subjects were compared with controls. Moderate reductions in dyslexics' contrast sensitivity for static gratings (*c.* 0.1 log units) under mesopic luminance conditions have been shown at low spatial frequencies (<6 c/deg) and slightly increased sensitivity (<0.1 log units) for higher spatial frequencies (>6 c/deg) (Lovegrove, Martin, Bowling, Blackwood, Badcock & Paxton, 1982). Larger differences, up to *c.* 0.3 log units have been found in the temporal domain (Martin & Lovegrove, 1987; Mason, Cornelissen, Fowler & Stein, 1993). Thus dyslexics have been shown to have reduced sensitivity to flickering gratings over a wide range of spatial frequencies (0.5–12 c/deg), an effect which is most prominent at temporal frequencies of 20 Hz or more.

One way to interpret these results is to compare them with recent behavioural studies in which contrast sensitivity responses have been obtained from macaques with specific lesions in the M or P pathways. Unlike P pathway lesions, those in the M pathway cause a large decrease in luminance contrast sensitivity for stimuli of higher temporal frequency and lower spatial frequency (Merigan & Maunsell, 1993). The reduction of sensitivity to low spatial and high temporal frequencies results in reduced visibility of rapidly moving or rapidly flickering stimuli. In the light of lesion experiments in macaques, it is feasible that the modest reductions in luminance contrast sensitivity shown by dyslexics could be due to M pathway dysfunction.

The present study

It is significant that in the above experiments in which dyslexics' luminance contrast sensitivity was measured, the mean screen luminance of the stimuli used was in the mesopic range (i.e. 3–10 cd/m^2). By comparison, most of our waking activity, including reading, is conducted in a world which is brightly illuminated (i.e. in the photopic luminance range of 30–40 cd/m^2 or more) and filled with colour as well as luminance contrasts. Therefore, while the contrast sensitivity findings can be interpreted as an M pathway correlate of dyslexia, they cannot tell us whether abnormal luminance contrast detection may affect children's reading, because the measurements have not been carried out under the same luminance conditions as reading. Indeed, Legge, Rubin and Luebker (1987) showed that the contrast between text and background had to be reduced by almost 1 log unit before normal adults' reading rate was halved. So it is difficult in principle to see how the smaller reductions in dyslexics' contrast sensitivity (*c.* one-third of the size) might affect their reading.

One aim of the present study was to test whether a "contrast detection deficit" could plausibly affect children's reading. Therefore we investigated whether dyslexics' contrast sensitivity loss persisted at the luminance levels usually experienced during reading—i.e. in the photopic range. Martin and Lovegrove (1984) provided some evidence of this kind by comparing the luminance contrast sensitivities of dyslexics and controls at 10 and 102 cd/m^2. Their stimuli were static gratings which subtended 4 deg at the retina. At 10 cd/m^2, in agreement with the majority of their previous findings, dyslexics showed a reduction in sensitivity for low spatial frequencies (approx. 0.2 log units over 1–4 c/deg), relative to controls, coupled with a moderate increase (0.3 log units) at 12 c/deg. However, at 102 cd/m^2 there was negligible difference between the groups, although Martin and Lovegrove did not explicitly state whether the small difference between dyslexics and controls remained statistically significant. Hence, in our first experiment, we attempted to replicate Martin and Lovegrove's findings by measuring static contrast sensitivity in dyslexic and control children using stimuli which were well within the photopic range (112 cd/m^2). Then, because mesopic flicker contrast sensitivity has proven to distinguish between dyslexics and controls better than static contrast sensitivity, we also measured photopic flicker contrast sensitivity in age matched dyslexics and controls.

Our second aim was to test the hypothesis that dyslexics have an impaired M pathway more directly. To do this, we compared the abilities of dyslexics and controls to detect coherent motion in random-dot kinematograms. Such stimuli consist of a series of patterns of random dots presented in rapid succession. Between frames, some proportion of the dots is systematically shifted to new positions thereby presenting the visual system with a temporal correspondence problem (Braddick, 1974). Correspondences are solved and the perception of coherent motion extracted by spatial and temporal integration of local motion signals (Baker & Braddick, 1982; McKee & Welch, 1985; Snowden & Braddick, 1989a,b). Thus the figural contours in the stimuli are defined not by luminance contrast, but only by the motion signal.

There is evidence suggesting that information carried by the M pathway plays a key part in detecting motion in random-dot displays. For example, Schiller, Logothetis and Charles (1990) injected ibotenic acid into the LGN of rhesus monkeys to induce scotomata in restricted parts of their visual fields. These authors found that magnocellular, but not parvocellular injections induced scotomata in which sensitivity to random-dot motion was dramatically reduced. Furthermore, area MT in the macaque contains cells whose receptive fields are exquisitely sensitive to the direction of motion (Newsome & Pare, 1988), and whose patterns of neuronal firing are highly correlated with the behavioural responses made by monkeys during motion discrimination tasks (Newsome, Britten & Movshon, 1989; Snowden, Treue, Erickson & Andersen, 1991). Simultaneous inactivation of either parvo- or magnocellular LGN combined with single unit recordings in MT showed that magno- rather than parvocellular blockade was most detrimental to the motion sensitivity of MT neurones (Maunsell, Nealey &

DePriest, 1990). In the light of these electrophysiological data, a motion detection paradigm using random-dot stimuli seemed the most appropriate method to test for impaired M pathway function in dyslexics.

In summary, in the first experiment, we aimed to test whether dyslexics' reduced luminance contrast sensitivity persisted under photopic luminance conditions. In the second experiment we measured the ability of dyslexic children and controls to detect coherent motion. In the third experiment we compared motion coherence thresholds of reading disabled adults with those of controls.

EXPERIMENT 1

Objective

If poor luminance contrast detection contributes in some way to dyslexics' reading difficulties, then we should still observe differences in sensitivity between disabled readers and controls when their luminance contrast sensitivity is measured at photopic luminance levels.

Methods

Subjects. We selected 14 reading disabled subjects from a population of children who had been referred to the Royal Berkshire Hospital, Reading, for orthoptic assessment because of their reading difficulties. We used a standard definition of reading disability which is based on the regression of reading age on IQ (cf. Rutter & Yule, 1975). Children were defined as dyslexic if their reading age measured on the British Ability Scales (BAS) reading test fell 2 or more SDs behind that predicted from their age and BAS IQ. The BAS is a standardized IQ test battery which includes an assessment of reading accuracy (Elliott, Murray & Pearson, 1979). We pre-tested 21 children as potential controls from a local primary school. We then selected as our control subjects the 14 children whose chronological ages and IQs best matched the dyslexic children. The characteristics of the 14 dyslexics and 14 controls are summarized in Table 1.

Orthoptic and psychological assessment. Every child was examined to exclude orthoptic and gross ophthalmological pathology. Assessment included separate measurements of the Snellen acuities of the two eyes. In this test subjects view a standardized high contrast letter chart in which the size of the letter targets is systematically reduced. A subject's acuity is recorded as a fraction. The numerator represents the distance the letter chart was viewed from (6 m). The denominator refers to the distance (in m) at which the smallest letter that the subject can resolve would have to be viewed to appear the same size as a reference. Hence the larger the denominator, the poorer the subject's acuity—typically values vary between 6/4 and 6/18 with the reference value being 6/6. In addition we measured each child's near point for convergence and accommodation using the "Royal Air Force" rule. In this test, a letter target (for accommodation) or small dot (for convergence) is moved smoothly in the sagittal plane towards the subject who states when the target becomes blurry (*c.* 6–8 cm for near point of accommodation) or diplopic (*c.* 6–8 cm for near point of convergence). Summary data for these four measures are also shown in Table 1.

Children's reading ages were measured with the BAS reading accuracy test. Their IQs were estimated with the shortened form of the BAS IQ battery. They were calculated from the mean of the Matrices (Test F) and Similarities (Test A) subtest *t*-scores. The Matrices test assesses nonverbal reasoning and involves completion of a series of picture puzzles. The Similarities test is a measure of verbal reasoning in which children are asked to describe the connection between sets of three items (e.g. red, blue and brown are all colours). An important point to note is that this IQ measure is crude. In particular, children who score near the upper or lower extremes on the Matrices (Test F) may subsequently score a relatively high or low IQ respectively. However, this mostly reflects the fact that the step size between consecutive *t*-scores

TABLE 1. Characteristics of dyslexic and control subjects in Expt 1

		Dyslexics (n = 14)	Controls (n = 14)
Chronological age (months)	Mean (SD)	118.6 (15.6)	116.1 (9.6)
	Range	97–147	99–133
IQ (BAS)	Mean (SD)	103.9 (11.1)	102.1 (19.6)
	Range	88–126	57–123
Reading age (months)	Mean (SD)	87.4 (12.8)	128.4 (20.2)
	Range	63–112	87–153
Left eye acuity [denominator of Snellen acuity (m)]	Mean (SD)	4.4 (0.6)	4.7 (1.4)
	Range	4–5.5	4–9
Right eye acuity [denominator of Snellen acuity (m)]	Mean (SD)	4.3 (0.5)	5.0 (1.8)
	Range	4–5.5	4–9
Smallest accommodation distance (cm)	Mean (SD)	7.4 (1.6)	6.9 (1.0)
	Range	6–12	6–8
Critical convergence distance (cm)	Mean (SD)	6.1 (0.5)	6.0 (0.0)
	Range	6–8	6–6

t-Test comparisons between dyslexics and controls failed to show significant differences for all of these measures except for reading age.

becomes increasingly coarse toward the tails of the distribution. Therefore, unlike a full-scale WISC IQ, for example, it should not be especially alarming that the lowest BAS IQ recorded in Table 1 was 57.

Apparatus. With both eyes open, children viewed a Joyce Electronics CRT display from a distance of 1.5 m, with natural pupils and without a fixation target. The mean screen luminance was set to 112 cd/m^2 measured with a Tektronix J6503 photometer. The mean room illuminance was 2.8 lx. The edges of the CRT were masked with dark card, so that only a central, circular portion of the screen, subtending 8 deg, was visible. The CRT's screen refresh rate was set at 200 Hz.

A signal generator was used to present sinusoidal grating patterns of 0.5, 1.5, 3 and 6 c/deg. Stimuli were sinusoidally modulated in counterphase at 20 Hz for the flicker contrast measurements. For each trial, the tester triggered a timer which allowed the grating patterns to appear for exactly 1000 msec. Therefore, children saw a grey field, then a grating pattern with the same mean luminance, and then a grey field again. Grating onset and offset had a square-wave profile which meant that our "static" patterns were contaminated with high temporal frequency transients. Stimulus contrast was modulated manually by means of a signal attenuation unit. The smallest attenuation step which this unit could make was 1 dB.

We used the Michelson fraction to define stimulus contrast, i.e.

$$\text{contrast} = (L_{\min} - L_{\max})/(L_{\max} + L_{\min})$$

where $L_{\min}$ is the luminance of the darkest part of the stimulus (in cd/m^2) and $L_{\max}$ is the luminance of the brightest part of the stimulus (in cd/m^2).

Threshold measurements. To identify childrens' thresholds, contrast was systematically reduced by means of a modified 1-up/1-down staircase procedure. At the start of testing, children were shown a high contrast grating which was vertically oriented. They were then shown that the lines could be oriented in one of four ways: horizontal; vertical; 45 deg upwards to the left; or 45 deg upwards to the right. It was explained that all the child had to do was identify the direction of the lines, and they were encouraged to do this by drawing the direction in the air with a finger.

For each spatial frequency (0.5, 1.5, 3.0 and 6.0 c/deg) children were shown a high contrast grating. The contrast was then reduced in 5 dB steps without changing the orientation, until children claimed that they could no longer see it. The grating contrast was then raised to 15 or 20 dB above this level (typically to about 30% contrast) and the staircase using randomized orientations was started from there. Contrast was then reduced in 5 dB steps until children said they could no longer see the grating, or until an error was made. Contrast was then increased in 5 dB steps until the orientation was correctly identified again. After this contrast was changed in 3 dB steps and the staircase continued for two more reversals. Once the 3 dB steps were being used, only the vertical and horizontal gratings were in fact presented, although the children still believed that the gratings might appear in all four orientations. This was necessary to ensure that the contrast thresholds we measured were not elevated by the oblique effect (Appelle, 1972). We defined threshold as the average contrast of the last two correct responses.

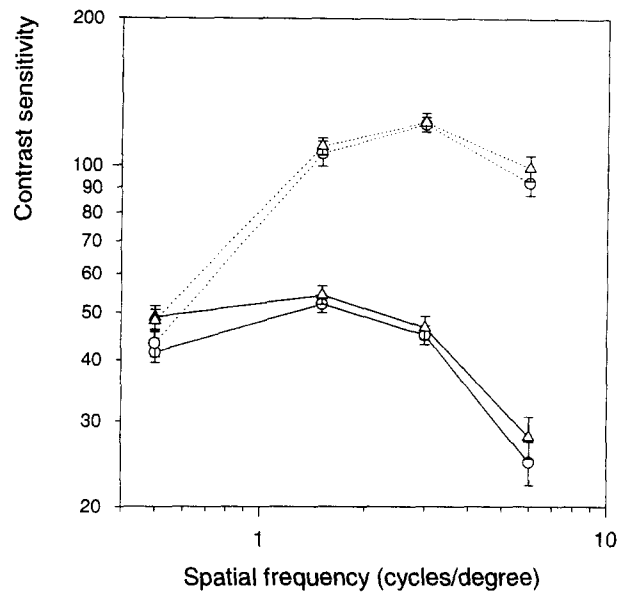


FIGURE 1. Plot of contrast sensitivity against spatial frequency for the static (dotted lines) and flickering (solid lines) grating stimuli. Both x and y axes are log scaled. ○ Dyslexics; □controls. Error bars represent ±1 SE for each mean.

This nonstandard technique was probably not as rigorous in estimating threshold as some which might have been employed. However, its advantage was that it was fast and engaging enough to prevent children getting bored. Also it was flexible as it was controlled mechanically by the tester, so the exceptional behaviour of some children could be accommodated, e.g. if a child was failing to pay attention to the stimulus his response was ignored and he was asked to repeat the measure.

Results

Figure 1 shows a plot of mean contrast sensitivity (i.e. 1/Michelson contrast at threshold) against spatial frequency for both the static (dotted lines) and flickering (solid lines) stimuli. The circles represent dyslexics and the triangles the controls. It is clear from Fig. 1 that, for both static and flickering stimuli, there was negligible difference in threshold at any spatial frequency between dyslexic and control children at 112 cd/m^2. The only exception to this was for the flickering gratings at 0.5 c/deg.

Correlations between IQ, age, Snellen acuity, accommodation, convergence and the two contrast sensitivity measurements never exceeded 0.1 and were all nonsignificant ($P > 0.2$). Therefore we excluded all these measures from further statistical analysis of the contrast data. We used a repeated measures analysis of variance to look for effects of spatial frequency and group (i.e. dyslexic vs control) on contrast sensitivity. For the static grating stimuli, the main effect of spatial frequency was

TABLE 2. Characteristics of control and dyslexic subjects in Expt 2

		Dyslexics ($n = 29$)	Controls ($n = 29$)
Chronological age (months)	Mean (SD)	116.7 (14.2)	118.7 (8.4)
	Range	90–148	100–131
IQ (BAS)	Mean (SD)	109.9 (13.5)	110.4 (14.8)
	Range	80–136	83–131
Reading age (months)	Mean (SD)	92.7 (12.9)	138.5 (23.6)
	Range	74–122	96–173
Left eye acuity [denominator of Snellen acuity (m)]	Mean (SD)	5.3 (2.2)	4.7 (1.6)
	Range	4–12	4–12
Right eye acuity [denominator of Snellen acuity (m)]	Mean (SD)	4.8 (1.2)	4.7 (1.4)
	Range	4–9	4–9
Smallest accommodation distance (cm)	Mean (SD)	7.0 (0.9)	6.7 (0.6)
	Range	6–12	6–8
Critical convergence distance (cm)	Mean (SD)	6.9 (2.3)	6.7 (1.1)
	Range	6–18	6–10

t-Test comparisons between dyslexics and controls failed to show significant differences for all of these measures except for reading age.

significant ($F_{3,78} = 133.06$, $P < 0.001$*), while the effects of group and the interaction between group and spatial frequency were not ($F_{1,26} = 0.85$, $P = 0.36$ and $F_{3,78} = 0.16$, $P = 0.92$ respectively). Thus we replicated Martin *et al.*'s finding of a negligible difference in contrast sensitivity between dyslexic children and controls for static gratings viewed at photopic luminance levels.

Figure 1 shows that the temporally modulated contrast sensitivity curves were flatter than those for the static gratings which is to be expected (Robson, 1966). We performed a second analysis of variance for the flicker contrast sensitivity data. Again, the main effect of spatial frequency was significant ($F_{3,78} = 82.87$, $P < 0.001$), while the effects of group and the interaction between group and spatial frequency were not ($F_{1,26} = 2.00$, $P = 0.17$ and $F_{3,78} = 1.09$, $P = 0.36$ respectively). However, it should be noted that by doubling the sample size, the small difference at 0.5 c/deg would have tended to produce a small but significant group difference.

EXPERIMENT 2

Objective

We aimed to test more directly whether dyslexics have an M pathway deficit. We used random-dot kinematograms to compare the ability of dyslexic and control children to detect coherent motion. Dyslexics and controls had the same mean chronological age and IQ.

Methods

Subjects. We selected 29 reading disabled subjects (6 females and 23 males) from a population of children who had been referred to the same orthoptic department as in Expt 1. The criteria for defining children as dyslexic were the same as in Expt 1. We compared the dyslexics with 29 normally reading controls (16 females and 13 males) selected from a local primary school. Dyslexic children were matched as closely as possible with controls for chronological age and IQ, as summarized in Table 2. In order to do this, we obtained a complete set of psychological and psychophysical measurements from 38 primary school children. Attending only to the psychological variables, we then made *a posteriori* matches with the dyslexic children.

Orthoptic and psychological assessment. The same orthoptic and psychological tests as in Expt 1 were performed on each child. Summary data for all these measures are shown in Table 2.

Apparatus. Subjects sat 60 cm from a VGA monitor, watching the stimuli which were generated by an Acorn A5000 computer. The room was illuminated by strip lights alone, producing a room illuminance of approx. 700 lx. The stimulus patterns comprised two rectangular patches of 1056 randomly arranged, square white dots on a darker background. Each patch had the dimensions on screen of 93 × 120 mm. The stimuli are illustrated in Fig. 2.

Each dot was 2 × 2 mm, subtending 0.024 deg at the viewing distance of 60 cm. The luminance of each white dot was 168.9 cd/m^2 while the luminance of the darker background was set to 0.51 cd/m^2 (measured with a Tektronix J6503 photometer). The space average luminance of each stimulus patch (taking account of any overlap between dots) was 58.7 cd/m^2. The luminance contrast between the dots and background, calculated from the Michelson fraction, was 99%.

The perception of motion coherence (i.e. the percentage of dots that moved coherently) in the two stimulus patches was generated in the following way (Wattam-Bell, 1992). Each patch contained 1056 dots, written to screen at every refresh (20 msec). At 0% coherence, because there was no correlation between the positions of dots in successive frames, each patch looked like a pattern of scintillations. With increasing coherence, an increasing proportion of the 1056 dots in each patch moved in a coherent way. In Expt 2, coherent dots existed on screen for 1.5 sec, i.e. 75 frames. One of the two patches was segregated into three alternating horizontal strips in

*An F ratio is a variance ratio > 0. In the context of an analysis of variance it is equal to the mean squares between the test populations divided by the mean squares within the test populations. The subscripted numbers denote the appropriate degrees of freedom to allow the significance of an F ratio to be calculated.

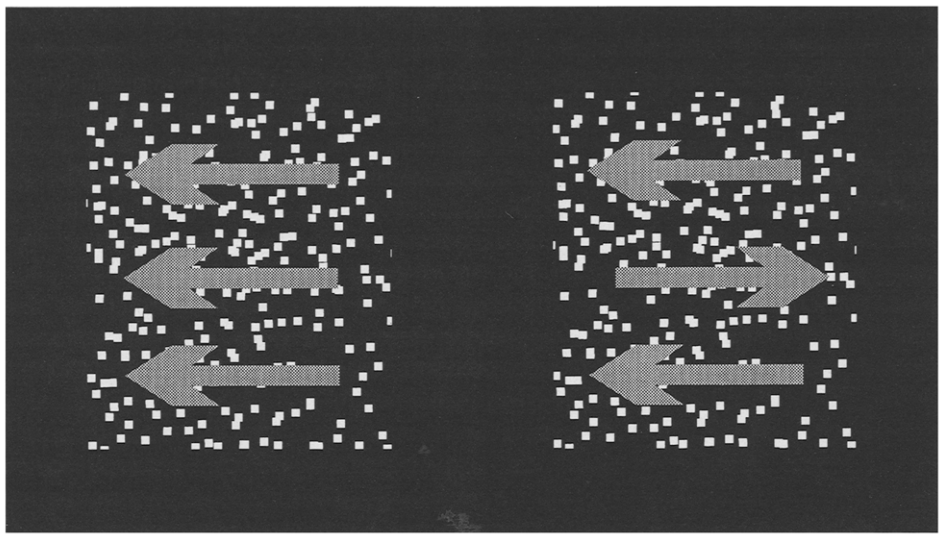


FIGURE 2. Diagram showing both patches of the motion stimulus. See text for detailed description. In this example the segregated stimulus is shown on the right side with the direction of movement of each segment indicated by the arrows.

which the coherent dots moved in opposite directions (see Fig. 2). The uniform pattern, on the opposite side of the display, was not segregated; all the coherent dots moved in the same direction. The velocity of the coherently moving dots was 2.5 deg/sec. Finally, the direction of coherent motion was reversed every 240 msec, producing oscillating movement with a triangular wave velocity profile.

Threshold measurements. We used a two-alternative force-choice method (2AFC) to identify children's coherence thresholds. The experimenter initiated each trial which lasted for 1.5 sec. Children were asked to indicate the segregated panel (as in Fig. 2) either by pointing or by naming the side (labelled 1 or 2) on which it appeared. For each trial, the Acorn computer randomized which panel would be segregated. Once the experimenter keyed in each child's response the next trial started automatically 1 sec later. We felt it would be unrealistic to ask 9–12 yr olds to stare at a fixation spot; instead children were encouraged to freely fixate both panels.

Motion coherence was varied according to a modified 2-up/1-down staircase method (Wetherill & Levitt, 1965). Coherence started at 100% and decreased by 1.5 dB between each trial until the first incorrect response. The coherence level at which this occurred formed the starting point for two reversals of the 2-up/1-down procedure with a spacing between coherence levels of 3 dB. This was followed by a further six reversals with a spacing of 1.5 dB (Wattam-Bell, 1992). Threshold was defined as the mean of the coherence levels at which the last six reversals occurred. Children performed the entire procedure four times, so that we obtained a total of four thresholds for each subject. Henceforth, each of the four repetitions is referred to as a trial block.

Results

Figure 3 is a plot of percentage coherence at threshold against trial blocks 1, 2, 3 and 4. All 232 thresholds from each of the dyslexic and control groups are presented. Two points are clear from this graph. First there is significant overlap between groups for each of the four blocks of trials. Second, the thresholds from blocks 2, 3 and 4 are on average higher for dyslexics than controls. This is made clearer by the table to the side of the graph in Fig. 3. It shows the means and SDs for the threshold values for each block of trials. In addition the grand average across all four trial blocks is shown, as well as the average for blocks 2, 3 and 4.

We correlated left and right eye Snellen acuities, convergence and accommodation with motion detection thresholds. No correlation coefficient exceeded 0.15 and all were nonsignificant ($P > 0.1$). Therefore we felt it was justified to exclude the orthoptic and ophthalmological measures from further analysis of motion detection thresholds.

We used linear multiple regression analysis to test whether the difference in threshold between dyslexic and control children was significant. The regression model took the form:

$$y = a + b_1x_1 + b_2x_2 + b_3x_3 + b_4x_4 + e$$

where y is the coherence threshold, a is a constant, e is a random error term, b_1x_1 is the effect of age, b_2x_2 is the effect of sex, b_3x_3 is the effect of IQ and b_4x_4 is the effect of group (i.e. dyslexic vs normal).

To take account of the fact that we made repeated measures, which would introduce an additional error term for within subject variance, we averaged thresholds across trial blocks. In the first analysis we entered the average threshold across all four trial blocks as the dependent variable in the model. In the second analysis, we used the average across trials 2, 3 and 4.

In the first analysis the effect of group (i.e. dyslexic children vs controls) was significant ($F_{1,54} = 6.27$, $P = 0.01$) while the effects of IQ, sex and age were not

($F_{1,54} = 0.63$, $P > 0.1$; $F_{1,54} = 1.95$, $P > 0.1$ and $F_{1,54} = 3.03$, $P > 0.05$ respectively). These findings suggests that, on average, dyslexic children were worse at detecting coherent motion in random-dot displays than controls of the same age, despite statistical controls for IQ, sex and age.

It is clear from Fig. 3 that all subjects performed better (i.e. had lower thresholds) in blocks 2, 3 and 4 than block 1. Therefore, we felt that it was justifiable to consider the first block of trials as a learning period. So we carried out a second analysis, ignoring the first trial block, and entering the average threshold for blocks 2, 3 and 4 as the dependent variable into the above regression model. This time the effect of group was more significant ($F_{1,54} = 12.15$, $P = 0.001$) while IQ, sex and age were still not significant ($F_{1,54} = 0.94$, $P > 0.1$; $F_{1,54} = 1.70$, $P > 0.1$ and $F_{1,54} = 2.55$, $P > 0.1$ respectively).

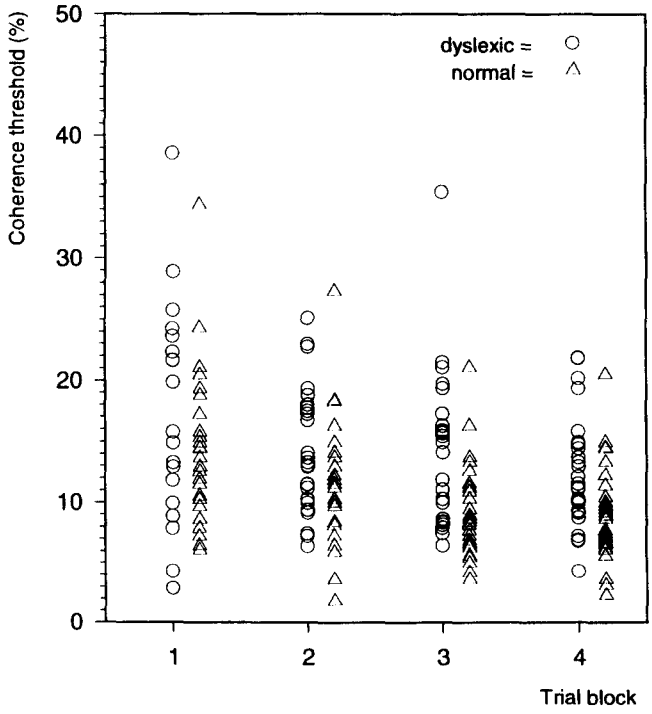
Finally, Fig. 3 shows that some degree of learning occurred over the course of four trials. Therefore it was feasible that the dyslexics might have performed less well than controls merely because they were slower at learning the motion detection task. To test this possibility, we carried out a comparison t-test of the mean coherence thresholds for the asymptotic performance at trial 4. The difference between dyslexics and controls remained significant ($t_{56} = 2.61$, $P = 0.01$) thereby discounting the possibility of differential learning between groups as a major factor explaining the group difference in coherence thresholds.

EXPERIMENT 3

Objective

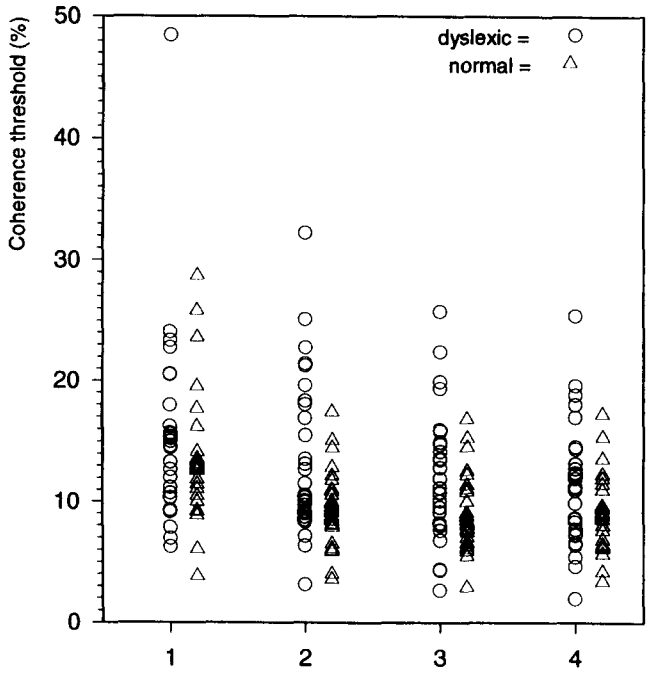
Bryant and Bradley (1985) argued persuasively that, in order to show that a processing deficit of some kind affects reading, it is necessary to compare older dyslexic subjects with younger children who are reading at the same level—a reading age match design. This approach controls for the possibility that the processing deficit observed arises simply from the different reading experiences of the two groups. However, there are at least two problems with applying this approach to measuring visual deficits in groups of dyslexic and control children. Firstly, it is often difficult to get 6 and 7 yr old children to apply as much concentration to a psychophysical task as 10–12 yr olds. Secondly, there is the possibility that normal developmental changes in visual performance would confound a reading age match design (e.g. Stein, Riddell & Fowler, 1986; Wattam-Bell, 1994). Fortunately we were also able to measure coherence thresholds in adult dyslexics. Therefore we could make a stringent test of the hypothesis that reading experience was unlikely to account for dyslexics' poor motion perception by comparing dyslexic adults with normally reading children.

In Expt 3 we measured motion coherence thresholds in a group of dyslexic adults and compared them with a group of adult controls. We assessed whether the dyslexic/normal difference we had found in Expt 2 persisted in adults. Then we made the "reading age match" type of comparison between dyslexic adults and normally reading children.



Dyslexic n=29 mean (sd)	Normal n=29 mean (sd)	Trial block
16.57 (8.4)	14.26 (6.14)	1
14.46 (4.94)	11.83 (4.88)	2
14.06 (6.19)	9.07 (3.91)	3
11.96 (4.51)	9.04 (4.01)	4
13.71 (4.43)	11.05 (3.61)	1,2,3,4
13.27 (3.92)	9.98 (3.36)	2,3,4

FIGURE 3. Scatter plot of percentage coherence at threshold against trial block for the dyslexic children and their controls. The offset between the data points for each group is included to allow easier comparison between groups. The table shows the means and SDs for the thresholds for each block as well as the means across blocks 2, 3 and 4, as well as across all four blocks.



Dyslexic n=29 mean (sd)	Normal n=29 mean (sd)	Trial block
15.34 (7.90)	13.48 (6.36)	1
13.44 (6.57)	9.39 (3.09)	2
11.91 (5.31)	8.99 (3.08)	3
12.67 (9.33)	8.96 (3.14)	4
13.34 (6.07)	10.21 (3.14)	1,2,3,4
12.67 (6.02)	9.12 (2.74)	2,3,4

FIGURE 4. Scatter plot of percentage coherence at threshold against trial block for the dyslexic adults and their controls. The offset between the data points for each group is included to allow easier comparison between groups. The table shows the means and SDs for the thresholds for each block as well as the means across blocks 2, 3 and 4, as well as across all four blocks.

Methods

Subjects. Twenty-nine dyslexic adults (13 females and 16 males) and 29 controls (13 females and 16 males) took part in Expt 2. The two groups of subjects were matched as closely as possible for chronological age (dyslexics' mean age 29 yr 0 months, SD 9 yr 10 months; controls mean age 28 yr 10 months, SD 7 yr 7 months). To do this, 42 controls were tested initially, *a posteriori* age matches with the dyslexics were then made with the aim of keeping the male/female subject ratios as close as possible between groups. The adult dyslexics had all previously been diagnosed by educational psychologists on the basis of a significant discrepancy between general ability and written language skills. Unfortunately we were unable to obtain either the orthoptic measures, IQs or reading abilities on our adult subjects. However, the educational psychologists' reports on the adult subjects showed that their reading accuracy was in excess of the 10–12 yr old level. Those who required spectacle correction wore their glasses.

Apparatus and threshold measurements. We used the same setup as in Expt 2 with the same luminance levels and the same staircase procedure for obtaining thresholds. As in Expt 2, each adult subject completed four blocks of trials to produce four thresholds. The only difference between Expts 2 and 3 was that the stimuli were presented on screen for 1 sec instead of the 1.5 sec we used for children.* Therefore coherent dot lifetime was 50 frames in Expt 3 compared with 75 frames in Expt 2.

In fact, as Wattam-Bell (1994) has recently shown, the difference in stimulus presentation time between Expts 2 and 3 should not have influenced our results. He used exactly the same stimuli to measure coherence thresholds in adults and children, systematically examining the effects of increasing the interval between direction reversals as well as the number of segregated regions in the stimulus. Wattam-Bell presented stimuli for 1.9 sec and obtained the same thresholds as we did for normal adults, using the same reversal interval and segregation parameters.

Results

Figure 4 is another plot of percentage coherence at threshold against trial blocks 1, 2, 3 and 4. As in Fig. 3, there is significant overlap between groups for each of the four blocks of trials. Nevertheless the thresholds from blocks 2, 3 and 4 are on average higher for dyslexics than controls. Summary statistics are again given in the table to the side of the graph in Fig. 4.

We used the following multiple regression model to test whether the mean difference in threshold between dyslexics and controls was significant:

$$y = a + b_1x_1 + b_2x_2 + b_3x_3 + e$$

where y is the coherence threshold, a is a constant, e is a random error term, b_1x_1 is the effect of age, b_2x_2 is the effect of sex and b_3x_3 is the effect of group (i.e. dyslexic vs normal).

For the first analysis we entered the average threshold across all four trials as the dependent variable. As before,

*The default setting for stimulus presentation time for the stimulus software (Wattam-Bell, 1992) is 1 sec, and we used this for the adult experiment. However, in our pilot study we found that children seemed to need slightly longer stimulus presentation times to make reliable judgements, hence we used 1.5 sec in Expt 2.

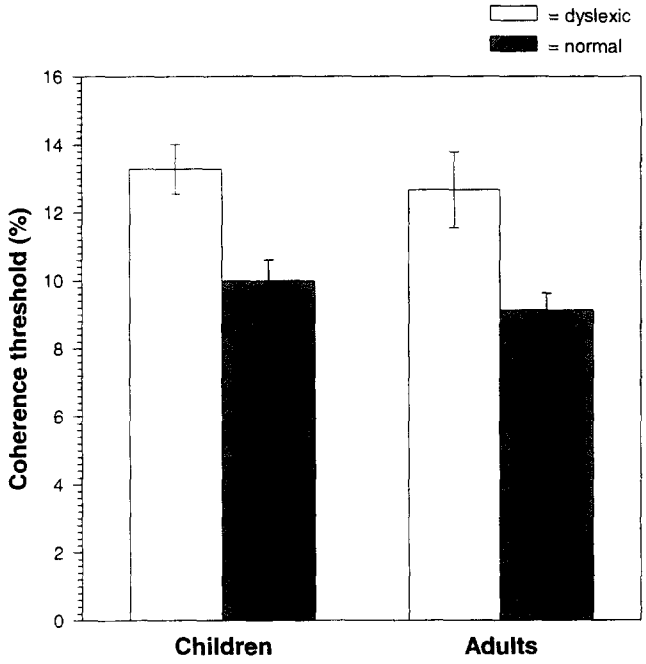


FIGURE 5. Bar chart of mean percentage coherence at threshold calculated from trial blocks 2, 3 and 4 only. Dyslexics are shown as the open bars and controls by the shaded bars. Error bars represent ± 1 SE for each mean.

the main effect of group was significant ($F_{1,54} = 6.92$, $P = 0.01$). However the effect of age was also significant ($F_{1,54} = 7.04$, $P < 0.05$); older subjects had higher thresholds than younger subjects. This age effect was equally strong for dyslexics and controls. However there was no effect of sex ($F_{1,54} = 2.54$, $P > 0.1$). Next we used the average threshold across trials 2, 3 and 4 as the dependent variable. The main effect of group ($F_{1,54} = 9.27$, $P < 0.005$) was more significant. The effect of age was less significant ($F_{1,54} = 5.32$, $P < 0.05$). There was no effect of sex ($F_{1,54} = 2.38$, $P > 0.1$). Thus dyslexic adults found it harder on average than controls to detect coherent motion even when the effects of age and sex were statistically controlled for. A t-test comparison between dyslexics and controls on trial block 4 was also significant ($t_{56} = 2.03$, $P < 0.05$).

Figure 5 shows a bar chart for mean coherence thresholds comparing dyslexics and controls for both child and adult groups of subjects. In all cases, the means were calculated from the thresholds for trial blocks 2, 3 and 4.

Finally, we carried out t-test comparisons between dyslexic adults and control children of mean thresholds (blocks 2, 3, and 4) as well as the thresholds for block 4 alone. Both tests were significant (means 12.67% vs 9.98%, $t_{56} = 2.09$, $P < 0.05$; and 12.67% vs 9.04%, $t_{56} = 1.92$, $P = 0.05$ respectively).

DISCUSSION

Experiment 1: photopic luminance contrast sensitivity

We measured luminance contrast sensitivity under the conditions that children usually experience when they read, i.e. bright light. We reasoned that if poor luminance contrast detection has some direct influence on dyslexics' reading, then differences in contrast sensitivity between dyslexic children and controls ought to be observed at photopic luminance levels.

Like Martin and Lovegrove (1984), we found negligible difference in contrast sensitivity between dyslexic children and age matched controls when they viewed static gratings in the photopic luminance range. We also failed to find a significant difference between disabled readers and controls when they viewed photopic counterphase modulated stimuli. However, by doubling our sample size, the residual difference at 0.5 c/deg for flickering gratings in particular would have tended to produce a statistically significant group effect at the 1% level. Nevertheless, the fact remains that by viewing flickering grating stimuli under photopic instead of mesopic conditions, luminance contrast sensitivity provides poorer discrimination between dyslexics and controls.

Given that this is a negative result, it would have been reassuring to have been able to measure the same children's static and flicker contrast sensitivities at low luminance, as well as at high luminance, in order to confirm our previous findings (Mason *et al.*, 1993). Unfortunately, both outpatient time constraints as well as the number of tasks children were already being asked to undertake, prevented us from obtaining all four measures. However we feel there is little reason to suspect that the children studied in this experiment would have been systematically different from those who took part in the previous study. Indeed, the mean ages of the two samples (108 months vs 116 months) were well within 1 SD of each other, as were their IQs. Therefore, we fully expect that we would have found low luminance differences in contrast sensitivity, had we been able to look for them.

Our results suggest that threshold contrast sensitivity measures differentiate dyslexic from normal children at low luminance levels (Mason *et al.*, 1993; Martin & Lovegrove, 1987), but very poorly at high luminance levels (this paper; Martin & Lovegrove, 1984). Together these findings support the idea that low luminance contrast measures can provide a psychophysical marker to discriminate between groups of disabled readers and controls. However, because there were no reliable differences between dyslexics and controls at photopic luminance levels, defective luminance contrast detection is unlikely to contribute directly to children's reading problems.

Experiment 2: motion coherence detection in dyslexic children and controls. Each coherently moving dot in our stimuli lasted on screen for as long as the entire stimulus appeared, i.e. 1 or 1.5 sec. In this respect our stimuli differed from the kind of random-dot kinematograms in which all dots have a limited lifespan (e.g. 2 video frames), irrespective of whether their motion is correlated between frames or not (Williams & Sekuler, 1984). Nevertheless, both kinds of stimulus are likely to satisfy the criteria for motion detection of the kind envisaged by Reichardt and

others (Reichardt, 1987; Borst & Egelhaaf, 1989), i.e. motion coherence can only be signalled if global velocity information is extracted from the stimulus by spatio-temporal integration. Therefore, we feel that our stimuli provided an adequate test of coherent motion detection.

We found that dyslexic children's motion coherence thresholds were, on average, some 3–4% higher than those of normally reading children who were the same age. This finding remained significant despite statistical controls for IQ, chronological age and sex. Therefore, like the mesopic contrast sensitivity findings, coherent motion detection provided a statistical discriminator between dyslexics and controls. However, the considerable overlap between the distributions of the thresholds from each group (see Fig. 3) suggests that either the visual deficit we have measured was very subtle, or that our measuring technique was noisy, or that there was considerable heterogeneity in the two groups of children studied. The latter suggestion may fit with the results of the serial case studies carried out by Seymour (1986). In a detailed study of 21 dyslexics and 13 controls, Seymour showed that individual dyslexics show markedly heterogenous patterns of visual and phonological difficulties in their reading strategies; hence it may be unreasonable to expect a low level visual task alone to provide perfect discrimination between groups of dyslexics and controls.

Experiment 3: coherent motion detection in dyslexic adults and controls. Like the children in Expt 2, dyslexic adults produced detection thresholds which were 3–4% higher on average than adult controls of the same age. Furthermore, the normal children were better at detecting coherent motion than the adult dyslexics. According to the argument presented by Bradley and Bryant which was presented earlier, this result strongly suggests that the reduction in motion sensitivity we have found in dylexics cannot be attributed simply to lack of reading experience. It therefore provides support for the idea that poor motion detection could be causally related to poor reading in dyslexics.

A potential problem with the adult data is that of subject selection because of the lack of strict statistical criteria for reading disability. It has been common practice to select adult dyslexic subjects on the basis of a previous history of reading difficulties. This is largely because there are few adequate psychometric tests for adults. However, attempts have been made to derive statistical discrepancy definitions of reading disability for adults on the basis of the regression of reading accuracy on IQ (Finucci, Whitehouse, Isaacs & Childs, 1984). Recently, Kinsbourne, Rufo, Gamzu, Palmer and Berliner (1991) studied adults defined as dyslexic by Finucci's criteria, adults defined as dyslexic on the basis of previous history and normally reading controls. They compared the performance of the three groups of adults on a large battery of neuropsychological and literacy tests. The important finding for our purposes was that Kinsbourne *et al.* found little qualitative difference between the two groups of dyslexic subjects. Thus we believe our definition of reading disability in adults to be appropriate.

Conclusion: an M pathway deficit in dyslexia? When considering the possibility of an M pathway deficit in dyslexia it is helpful to draw a distinction between sub-cortical and cortical visual pathways. In their histological comparison of five dyslexic and five normal brains, Livingstone *et al.* (1991) found sub-cortical abnormalities in the magnocellular LGN. If this anatomical feature was commonly found in dyslexics, then it might well account for both the contrast sensitivity and coherent motion data (see Introduction for references). However, if dyslexia is associated more with cortical than sub-cortical abnormalities (Galaburda, Sherman, Rosen, Aboitz & Geschwind, 1985) then it becomes difficult to make a strong case for a specific M pathway deficit in dyslexics. For example, neurons in V1 layer 4B and V2 thick stripes have high contrast sensitivity which matches that in the M pathway (Blasdel & Fitzpatrick, 1984; Hawken & Parker, 1984). But cortical responses like these could be elaborated by summing inputs from many relatively insensitive parvocellular neurons to give a response just as sensitive as any in the M pathway (Watson, 1992). Summation of this sort has been demonstrated by the existence of cortical units that have greater sensitivity than neurons in either subdivision of the LGN (Sclar, Maunsell & Lennie, 1990). Therefore dyslexics' reduced sensitivity to luminance contrast could be caused by abnormal pooling of P pathway input just as easily as by abnormal M pathway input. Similarly, our finding that dyslexics found it harder than controls to detect coherent motion could plausibly be a sign of generalized dysfunction in the parietal visual pathway, which includes areas like MT and MST, as opposed to a specific failure of an M pathway contribution. Indeed recent electroencephalographic and regional blood flow studies have revealed unusual patterns of activation which is consistent with abnormal activity in the parietal cortices of dyslexic subjects (Duffy, Denckla, Bartels & Sandini, 1980; Wood, Felton, Flowers & Naylor, 1991).

Not only do our results need to be replicated, but it is also important to discover how specific the findings are to dylexia. In addition, there is the question of whether an M pathway deficit might affect dyslexics' reading? Recently, Hill and Lovegrove (1993) showed that dyslexics made more errors when they read a whole line of text as opposed to single words presented in isolation. Their results are qualitatively similar to those of Geiger and Lettvin (1987), and suggest that abnormal interactions between peripheral and foveal vision in some dyslexics may affect their reading. A hypothesis linking these findings with an M pathway deficit involves the "shift effect" in which the detectability of a foveal target may be reduced by movement in the periphery (Breitmeyer, Valberg, Kurtenbach & Neumeyer, 1980; Mattingley & Badcock, 1991). Electrophysiologically, the

shift effect is most pronounced in cells projecting to and within magnocellular LGN (Krüger, Fischer & Barth, 1975). Therefore, an M pathway deficit might in some way disturb the balance between central and peripheral processing during reading fixations causing reduced efficiency of letter detection [note this view differs from Breitmeyer's (1980) impaired saccadic suppression model]. Clearly, further research is required to elucidate these questions.

In summary, the idea of an M pathway deficit in dyslexia is appealing largely because of its simplicity. However, although some of the available experimental evidence can be interpreted as supporting this conclusion, on the one hand it is easy to provide alternative explanations for the same data, while on the other hand it is difficult to reconcile such a simple hypothesis with the observed variability between dyslexic subjects. Nevertheless, it remains plausible that an M pathway deficit could specifically affect some dyslexics.

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