

**VISUAL MAGNOCELLULAR IMPAIRMENT IN
ADULT DEVELOPMENTAL DYSLEXICS**

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Short Title: Visual deficits in developmental dyslexia

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Neuro-Ophthalmology 20, 187-201 (1998).

ABSTRACT

Previous research has suggested that visual magnocellular impairment may be characteristic of up to 75% of developmental dyslexics. In this study we compared 18 adult dyslexics and 18 controls on two tasks of putative visual magnocellular function. We examined whether these tasks could discriminate dyslexics from controls and also the relationship between these measures and nonword reading, a sensitive measure of phonological awareness. Our results showed that dyslexics were significantly less sensitive than controls for detection of coherent motion in random dot kinematograms (RDK) and also the highest frequency at which temporal modulation at full contrast was detectable, the critical flicker fusion frequency (CFF). Across the two groups and within each group examined separately, motion and flicker sensitivity correlated strongly with nonword reading ability. Together, the temporal perception measures were able to discriminate 72.2% of the dyslexics from controls, so this type of visual deficit may be an important feature of dyslexia. Our results support the hypothesis that dyslexics' reading problems are not entirely caused by a specific deficit in language processing. These visual deficits are also found in younger subjects; hence visual temporal perception measures may be used to identify children at risk for dyslexia prior to actual reading failure.

Key words: dyslexia, magnocellular, flicker fusion, motion

INTRODUCTION

Background

A significant proportion of school age children (3 - 9%) and adults have a substantial and unexpected discrepancy between their reading ability and their cognitive aptitude.¹ For most, the deficit is specific to reading and spelling and does not result from any other identifiable factors (e.g., differences in educational opportunity, motivation, attention deficits, or brain damage). Hence these children may be classified as having developmental dyslexia or specific reading disability.

The predominant view is that developmental dyslexia results from a failure to learn the proper relationships between letter combinations and speech sounds. It is argued that such impaired phonological awareness should be considered the primary causal factor in the aetiology of specific reading difficulties.^{2,3} Tests that require phonological skill, such as the accurate naming of phonologically consistent nonwords (e.g., 'drile') and Pig Latin and Spoonerisms tasks (which require word-sound deletion, transposition and addition), can discriminate good from poor readers at early ages.^{4,5} Poor phonological awareness has also been shown to persist in adult dyslexics.⁶

While it is accepted that dyslexics' extreme difficulties on tasks such as nonword naming result primarily from deficient phonological skills,^{7,8} other studies have shown that many dyslexics have other problems that are not easily explained by a phonological deficit.^{9,41} For example, some dyslexics have no apparent deficits for decoding phonetically regular words (e.g., chicken, context) but substantial difficulties when naming and spelling phonetically irregular words (e.g., island, colonel).⁴¹ These persons often make 'phonological regularisation errors', mispronouncing or misspelling irregular words because they try to apply the usual phonetic rules (e.g., iland for island), so it appears that their phonological skills are quite intact. These reading and spelling errors for irregular words might be better explained by deficits in visual coding, representation, and memory rather than phonological deficits;¹⁰⁻¹¹ a hypothesis that is supported by recent experimental evidence from Cornelissen and colleagues.¹² They showed that unselected normal readers who had high thresholds (i.e., low sensitivity) for detecting coherent visual motion were more likely than those with low detection thresholds to make false positive responses to visual anagrams in a word/anagram (e.g., *bowl* versus *bolw*) discrimination task.

It is therefore unlikely that poor phonological skills alone can account for the array of problems that dyslexics manifest while reading; it is also probable that dysfunction of early visual processes contributes to their reading problems. One result of this early

visual dysfunction might be to disrupt accurate letter position encoding which is necessary for efficient word decoding and lexical access.¹² Also, because reading is inherently a visual behaviour, any visual deficit would necessarily interact with a language processing deficit to compound the dyslexics' reading difficulties.

It is now clear that many dyslexics consistently differ from controls on a number of sensory processing tasks for both auditory and visual stimuli.¹³⁻²¹ However, these deficits are usually identified only when the paradigm specifically measures temporal perception. Perception can be said to be temporal when stimulus detection depends upon the detection of the temporal properties of a stimulus (i.e., processing of rate) rather than the perception of stimuli with short durations or short interstimulus intervals (i.e., rate of processing).²² Specific deficits for the detection of temporal stimuli suggests that anomalies in particular neural pathways contribute to dyslexics' reading difficulties.

In the visual system, fast temporal perception is mediated primarily by cells within a retinocortical projection referred to as the M-pathway or the transient system.^{23,24}

Differentiation of the M and P visual pathways

Visual analysis is achieved by parsing the information that impinges on the retina into two primary information processing streams that can, to some extent, be differentiated by anatomical, physiological and psychophysical methods.²⁵⁻²⁹ Figure 1 depicts the principal anatomical projections of these two primary retinocortical streams.

<INSERT FIGURE 1 ABOUT HERE>

The anatomical segregation of the two retinocortical projections is first distinguishable in the retinal ganglion cell layer of the retina and continues through the lateral geniculate nucleus of the thalamus (LGN) to primary visual cortex (V1).²⁶⁻²⁹ M-retinal ganglion cell efferents project selectively to the more ventral layers (layers 1-2) of the LGN, whereas P-retinal ganglion cell efferents project specifically to parvocellular LGN layers (layers 4-6) more dorsally. Because these streams are clearly segregated subcortically, their efferent fibres are often referred to as the magnocellular ('M') and

Although this anatomical segregation continues through to the input layers of V1, where M-pathway axons terminate in layers 4C α and 4B and P-pathway axons terminate in layer 4C β , the functional and structural separation of the M and P pathways beyond this stage is not as complete as at lower levels. In most of the cortex there is a significant functional and structural intermixing between the M and P streams.^{26,28} Nevertheless,

M-cells provide a predominant input to a dorsal stream projecting to parietal lobe structures, whereas both M- and P-cells input to a ventral stream terminating in the temporal lobe.²⁸ Information about colour and form are processed primarily in the interconnected ventral stream areas, with information about object location and motion processed primarily by dorsal stream structures.³⁰

Of particular importance to this study is extrastriate area MT (V5), an area within the dorsal stream that receives inputs primarily from M-cells via area 4B and from the superior colliculus.^{26,28} Newsome and Pare showed that cells within primate MT were extremely sensitive to motion in random dot kinematogram (RDK) stimuli.³¹ This finding has since been replicated in the human homologue of area MT by a variety of methods including functional magnetic resonance imaging (fMRI).^{30,32} Furthermore, damage to this area can selectively affect motion perception in humans without compromising other visual functions such as colour perception and visual acuity.³³

The M- and P-pathways can also be distinguished on the basis of their receptive field properties for various aspects of the visual array. Ganglion cells in primate retina can be separated into two broad classes on the basis of their different physiological responses for spatial, temporal, and chromatic information in the visual world.³⁴ Some of these properties that provide the basis for a structural and functional separation between the two visual channels are outlined in Figure 2.

<INSERT FIGURE 2 ABOUT HERE>

M-cell receptive fields differ from those of P-cells in at least two major ways. Firstly, M-cell receptive fields are larger than those of P-cells. This receptive field difference reflects the spatial summation over which photoreceptor responses are pooled. Therefore M-cells have high sensitivity to coarse visual features (i.e., low spatial frequencies), to low luminance contrasts and to overall changes in space averaged luminance, but not to colour differences. Thus they respond at low contrasts and luminances to which P-cells are insensitive. P-cells have smaller receptive fields that result from much more specific and fewer cone inputs to both receptive field centre and surround. As a result, they respond optimally to fine visual details (i.e., high spatial frequencies), and are colour opponent.

Secondly, M- and P-cells respond to visual stimuli with different temporal responses. The axons of M-cells are larger and more heavily myelinated than P-cells. As a result M-cells have faster conduction velocities. M-cells also respond more transiently, firing primarily at stimulus onsets and offsets. P-cells have slower conduction velocities and

fire with a more sustained response throughout the entire duration an appropriate stimulus is present. On the basis of these temporal response differences, the M- and P-cell streams are often referred to as the transient and sustained subsystems respectively.^{23,34}

In summary as a result of their different spatial and temporal receptive field properties, M-cells are more sensitive to lower spatial and higher temporal frequencies whereas P-cells are more sensitive to higher spatial and lower temporal frequencies.^{27,29} The sensitivity of both of these pathways is important for reading because visual word recognition depends upon accurate processing of both high and low spatial frequencies.³⁴

M-pathway visual processing deficits are currently the focus of an effort to specify dyslexics' visual problems because the majority of the experimental evidence has shown that their visual perception deficits are limited to stimuli with low spatial frequency, low contrast, low luminance and high temporal frequency characteristics.^{20,21} It is not suggested that dyslexics' lowered contrast sensitivity at low luminances and low spatial but high temporal frequencies *directly* interferes with their reading, but rather *indirectly*, perhaps via effects on visual persistence, visual stability or eye movements.^{24,50}

Because M-pathway functions may be selectively affected in dyslexia we sought to examine dyslexics' performance on psychophysical tasks of putative magnocellular function. Specifically we wanted to ascertain whether or not such tasks could discriminate dyslexics from controls. We also wanted to examine the inter-relationship between phonological skills, as assessed by nonword naming, and our M-pathway measures for both dyslexics and normal readers. Strong relationships between higher level language skill and our visual transient measures would provide further evidence for a link between basic perceptual functions and reading skill in both good and poor readers. To this aim we employed the following visual detection measures.

Visual detection tasks

We chose random dot kinematograms (RDK) similar to those employed by Newsome and colleagues^{31,35} and critical flicker fusion (CFF). These tests have the advantage that they measure sensitivity to rapidly changing stimuli which predominately stimulate the magnocellular system, and, unlike for contrast sensitivity measurements, these can be performed at high luminance. Again however, it should be noted that impaired

performance in these tests probably affects reading only indirectly. Nevertheless Cornelissen *et al.* showed that RDK motion detection thresholds were higher in dyslexic adults and in children than in chronological age matched controls.¹³ Eden *et al.* also showed, using functional magnetic resonance imaging, that dyslexics had lower activation of area MT when presented with RDK stimuli.³⁶ (But see Vanni *et al.* for a different view.³⁷) Also as mentioned earlier, Cornelissen *et al.*¹² showed that poor motion detectors were more likely to make letter position errors in their word/anagram discrimination task.

Because high temporal frequencies are selectively detected by cellular mechanisms within the M-pathway,^{27,29,38} we also measured dyslexics' CFF. CFF is the highest frequency at which temporal modulation (i.e., flicker) can be detected at 100% luminance contrast. Previous research indicated that selective lesions to M-pathway layers of LGN selectively decreased sensitivity for high temporal frequency stimuli in non-human primates.³⁸ Several samples of dyslexics have also been shown to be less sensitive than controls for flickering stimuli, particularly for higher temporal frequencies.^{15,16,18}

MATERIALS AND METHODS

Subjects

All of our methods were performed in accordance with the guidelines set forth in the Declaration of Helsinki and had approval from the local ethics committee. The subjects were 18 right-handed adult dyslexics (mean age = 27.6, range = 18-41) and 18 (mean age = 24.5, range = 19-34) right-handed controls. Of these 12 of the dyslexics and 12 of the controls were male. All were native English speakers. Each of the dyslexics had been previously diagnosed as reading disabled by educational or clinical psychologists. We also confirmed their reading disability at the time of testing by measuring their full-scale WAIS-R intelligence³⁹ and WRAT reading and spelling ability.⁴⁰ Full-scale WAIS-IQ was pro-rated for each dyslexic and control on the basis of scores on the Block Design, Picture Arrangement, Similarities and Vocabulary subscales. Raw scores obtained from the WAIS-R and WRAT were then converted into age-adjusted standard scores. Each dyslexic had a reading and spelling deficit of at least 1.5 standard deviations from their pro-rated, full-scale WAIS-R intelligence. None of the controls had such a discrepancy.

In addition to the reading and intelligence measures, each subject completed a battery of nonword naming measures including the Castles and Coltheart nonwords,⁴¹ and the Snowling nonwords.⁴² These measures were combined into a total nonword naming

metric. Hand preference was ascertained by both questionnaire⁴³ and timed performance on a pegboard task. The psychometric statistics for the dyslexic and control subjects are shown in Figure 3.

<INSERT FIGURE 3 ABOUT HERE>

Stimuli and Procedures

Critical flicker fusion (CFF) paradigm

The flicker stimulus was a 2.8 degree circular aperture of a twin panel 567 nm (yellow) LED display (L.E.D. Technologies) viewed binocularly from a fixed distance of 57 cm in a dark room (average luminance = 1.8 cd/m²). All luminance measurements were recorded with the OptiCal digital photometer (Cambridge Research Systems). The circular aperture was centred over the border of the two equiluminant, unpatterned (luminance = 49 cd/m²) panels so that each half-circle contained inputs from only one of them. The flicker rate and position (i.e., left or right side of the aperture) of the target stimulus was controlled by toggle switches under the experimenter's control with the position of the flicker randomised between trials.

CFF measures the highest temporal frequency that can be detected at 100% luminance contrast. Therefore the contrast of the flicker targets were always set at this maximum value. The non-target panel was set at the maximum flicker frequency of 208 Hz. At this temporal frequency, flicker cannot be perceived and the panel appears homogeneously illuminated. The flicker frequency of the target patch was manipulated by the experimenter using a 2-alternative forced-choice, one-up, one-down staircase with a step size of 2 Hz. A supra-threshold flicker (usually 44 Hz) was selected as the starting point for the staircase. Subjects were required to report the side of the aperture ('left' or 'right') that appeared to flicker, and guessed when necessary. The subject's CFF threshold was defined as the arithmetic average of the eight reversal points following the first reversal.

Coherent motion paradigm

The RDK stimuli comprised a patch of 150 high luminance (average = 96.9 cd/m²), white dots (1 pixel), presented on the nominal black background (average = 5.2 cd/m²) of a 17" CRT monitor display (Gateway 2000, Vivitron 1776). At a constant viewing distance of 57 cm the patch subtended 7 x 7 retinal degrees, centred on the fovea. Michelson contrast $[(L_{max}-L_{min})/(L_{max}+L_{min})]$ between the stimulus patch and the background

was held at a constant 89.8% with viewing conducted in a dark room under mesopic luminance conditions (space averaged luminance = 1.8 cd/m²). The overall space averaged luminance of the kinematogram patches was 5.4 cd/m². The percentage of coherently moving dots (angular velocity = 8.8 deg/sec) within a given software animation frame (duration = 50.1 ms) was controlled and varied to the subject's detection threshold by custom software designed for personal computers. Coherent motion percentage was defined as the total number of dots moving together in a single direction in either of the primary horizontal axes between consecutive frames. The non-coherent dots moved randomly between frames in a Brownian manner. In order to eliminate the possibility of detecting the direction of coherent motion by following a single dot, each dot had a fixed lifetime of 2 animation frames (101.4 msec) after which it would disappear before being regenerated at a random place within the stimulus patch. The total stimulus duration was 18 animation frames or 901.8 ms.

The subjects were asked to fixate a cross that preceded the appearance of the stimulus patch and remained in the centre of the RDK for the entire stimulus duration. After termination of the stimulus, the subjects reported the direction of perceived coherent motion by pressing an appropriate key and were told to guess when necessary. Coherent motion was varied to the subject's motion detection threshold by a 3dB-up, 1-dB-down, two alternative forced choice, staircase procedure.⁵⁶ Thresholds for detection were computed by taking the geometric average of the last 8 of 10 reversal points within a given series of trials. Each series was repeated at least three times with the mean of these series comprising the subject's overall motion detection threshold.

RESULTS

Critical flicker fusion (CFF) thresholds

Figure 4 plots both the dyslexics' and controls' thresholds on the CFF task. By independent samples t-test, the dyslexics were found to have significantly lower flicker fusion frequencies than the controls [$t(34)=2.898$, $p=.007$, two-tailed]. The mean effect size was approximately 4 Hz [(dyslexic mean (SEM) = 52.8 (.873); control mean (SEM) = 57.1 (1.178)].

<INSERT FIGURE 4 ABOUT HERE>

Coherent motion thresholds

There was a substantial variance inhomogeneity between the groups on the motion coherence task; the control group showed little variability in coherent motion thresholds, whereas the dyslexics were substantially more variable. We therefore used an independent t-test with degrees of freedom corrected for variance inhomogeneity to assess the significance of the group difference in motion coherence thresholds. The coherent motion thresholds were found to be significantly different between groups [$t(20.5)=3.06$, $p=.006$, two-tailed]. The dyslexics required on average approximately 8% greater motion coherence at threshold [(dyslexic mean (SEM) = 18.1 (2.471); control mean (SEM) = 10.3 (.783)]. For the 150 dot display, this corresponds to an additional 10 target dots at the detection threshold. Compared to the average thresholds of the controls this represents a motion coherence deficit of approximately 2dB. Figure 5 shows the dyslexics' and controls' detection thresholds for this task.

<INSERT FIGURE 5 ABOUT HERE>

Discriminant analysis

A major impetus of this study was to determine whether individual dyslexic and control subjects could be correctly classified into their respective groups on the basis of our M-pathway measures. As can be seen from Figures 4 and 5, there was significant overlap between the groups on each of the M-pathway tasks considered alone. Therefore, we performed a discriminant function analysis using SPSS for Windows (SPSS, Inc.) to ascertain whether or not the two M-pathway tasks could be used together to predict group membership (i.e., dyslexic or control). Like multiple regression, discriminant function analysis can assess the relationship between a group of predictor variables (e.g., CFF and coherent motion detection thresholds) and a grouping variable (e.g., dyslexic or control).⁵⁷ Discriminant function scores are calculated for each subject from the set of chosen predictors, with each predictor weighted by a coefficient that maximises the difference between groups relative to the difference within groups. A mean discriminant score for each group, the group centroid is calculated and each individual's discriminant score is then compared to the respective group centroids. Group membership for each individual case is then predicted. The ability of the set of variables to correctly predict group membership can then be ascertained by comparing the ratio of correctly classified cases to incorrectly classified cases for the entire population studied and also for each separate group.

Both motion detection and CFF thresholds were entered simultaneously into the discriminant analysis. As there were only two predictor variables assessed, only one discriminant function was calculated. This function was significant [$\chi^2(2)=10.583$, $p=.005$], and showed that there was a strong association between groups and predictors. Overall, this function accounted for 52% of the between group variability and correctly classified 77.8% of the individuals in the study. Thirteen of 18 dyslexics (72.2%) and 15 of 18 (83.3%) controls were correctly classified by their combined CFF and motion detection thresholds. Stated another way this means that the two visual measures provide a sensitivity (i.e., the probability of correctly identifying a dyslexic) of 72.2% and a selectivity (i.e., the probability of *not* cross-classifying a control) of 83.3%.

Correlational analyses

Because mean data can obscure important relationships among continuous variables, it is important to examine the inter-relationship between these variables. We therefore calculated Pearson product moment correlations to ascertain the nature of the relationship between the experimental measures employed in this study.

The relationship between age and each of the experimental measures was negligible for both groups with correlations ranging from -.082 (RDK) to -.233 (CFF). This indicates that, although the dyslexic group was slightly older than the controls, although not significant statistically [$t(34)=1.577$, $p=.124$, two-tailed], this age difference was not responsible for any of the group differences found.

There were also no significant relationships between the handedness measures and any of the nonword naming or M-pathway measures. This may have resulted from our decision to equate the groups on handedness (only right-handed subjects were included).

The two measures of putative M-pathway sensitivity were found to be moderately correlated with one another for the sample as a whole ($r = -.494$, $p<.05$) and also for both groups when examined separately (dyslexics, $r = -.445$; controls, $r = -.529$). These negative correlations show that increases in individual's flicker thresholds (i.e., higher sensitivity), were associated with decreases in the number of coherently moving dots needed for detection of coherent motion (i.e., higher sensitivity).

<INSERT FIGURES 6 AND 7 ABOUT HERE>

Nonword naming accuracy was related to sensitivity on each of the M-pathway tasks for both groups. Figures 6 and 7 show the relationship between nonword naming performance and visual detection thresholds for the CFF and coherent motion detection tasks respectively. We have removed one statistically significant outlier, a dyslexic, from each figure. This outlier was the same individual in each of the analyses. The correlations between nonword naming accuracy and CFF were .562 and .461 for the dyslexic and control groups respectively (Figure 6). This shows that as CFF thresholds decreased, the number of nonwords named correctly decreased correspondingly. A similar pattern of result was found for the motion coherence task (Figure 8). For both groups, persons who were less sensitive to coherent motion (i.e., with higher motion detection thresholds) made more nonword naming errors (dyslexics, $r = -.401$; controls, $r = -.356$).

<INSERT FIGURE 8 ABOUT HERE>

Most importantly, subjects' nonword reading was strongly correlated with a combined M-pathway measure that incorporated thresholds from both of our temporal perception tasks. Figure 8 shows dyslexics' and controls' nonword reading performance plotted as a function of a combined measure of M-pathway sensitivity. This combined measure is given by an optimal scaling procedure (linear principal components analysis (PCA)) which equally weighted and combined the thresholds from the two visual measures into a single value. Thus, distance along the PCA axis represents an individual's combined M-pathway sensitivity relative to the other subjects' sensitivities. In this case a higher combined M-pathway sensitivity is plotted as a higher score on the PCA axis. Nonword naming performance was found to be strongly related to magnocellular pathway sensitivity ($r = .691$, $p \leq .01$). In our sample nearly 48% of the variance in nonword naming could be predicted from subjects' overall M-pathway sensitivity on our two temporal perception tasks.

DISCUSSION

Our results showed that dyslexics were less sensitive than controls for both coherent motion in RDK stimuli and also for detecting high frequency flicker (CFF). Discriminant analysis demonstrated that these deficits were identifiable in a majority of our dyslexic sample but not in our controls. Over 72% of dyslexics, but only 17% of controls could be classified as having a visual deficit on the basis of their combined performance on the two M-pathway measures. This suggests that magnocellular sensitivity, as assessed by coherent motion perception and critical flicker fusion, is deficient in a majority of adult dyslexics. This result supports the findings of Slaghuys and Lovegrove who showed that

as many as 75% of dyslexic children could be classified as having a visual deficit within the transient subsystem.⁴⁴

The finding that most, but not all dyslexics have a temporal visual deficit is particularly interesting in light of recent research which has shown that visual deficits to flickering stimuli might be found only in particular subtypes of dyslexia.^{45,58} We did not classify our sample according to dyslexic subtypes, and we did not preselect our dyslexic subjects on any basis other than the presence of a significant reading discrepancy. Therefore, if our dyslexic sample contained the usual cross-section of the putative subtypes found in the dyslexic population, our results suggest that visual deficits can be identified in the majority, but not all of the individual cases. It is possible that the five dyslexic subjects in our sample who did not demonstrate M-pathway deficits were of a particular dyslexia subtype that might not have visual deficits.

We also showed that M-pathway sensitivity and nonword naming ability co-varied for both dyslexics and controls. This finding concurs with that of Cornelissen *et al.* who showed that, even in normal populations who are not expected to demonstrate M-pathway deficits, visual reading skills were positively correlated with M-pathway sensitivity.¹² These findings highlight the importance of sensitive M-pathway function in the reading process. What remains to be answered is the exact mechanism(s) by which M-pathway dysfunction disrupts visual analysis, representation and recall.

Dyslexics' deficits for the detection of temporal stimuli may exist in the auditory^{17,46-48} and sensorimotor systems⁴⁹ as well. The magnocellular deficit hypothesis proposes that the temporal perception deficit results from a dysfunction of the pathways that are specialised for the rapid conduction and processing of information in all sensory and motor systems.^{50,51} Galaburda and Livingstone have presented evidence for anatomical loci of this temporal processing deficit.⁵² They showed that a sample of dyslexic brains had cellular anomalies in auditory and visual nuclei of the thalamus (medial geniculate and lateral geniculate respectively) that were specific to magnocellular lamina. Another potential locus was suggested by Eden *et al.*³⁶ They showed that some dyslexics either lacked altogether or had an altered pattern of response to RDK stimuli in extrastriate visual area MT (V5).

Although our psychophysical tasks cannot isolate a single locus within the visual system, detection of our stimuli is likely to be dependent on the sensitivity of dorsal stream structures. (See Figure 1.) Detection of coherent motion in the RDK stimuli should be mediated primarily by cells within MT because this type of motion detection requires spatial summation over a relatively large extent of the retina. Also magnocellular

neurons at lower levels of the visual system in the retina and LGN are differentially sensitive to flickering stimuli.⁵³⁻⁵⁵

Dyslexics have been shown to have temporal processing deficits for the perception of auditory stimuli similar to the ones described for visual processing.^{17,46-48} It has been argued that one consequence of such an auditory temporal processing deficit would be difficulties in discriminating between speech sounds necessary for adequate phoneme awareness.²¹ In support of this pan-sensory deficit, we showed that thresholds for our M-pathway tasks were correlated strongly with nonword naming when the subjects were not separated into groups. If accurate nonword naming requires temporally acute auditory perception, then, to the extent that the magnocellular deficit hypothesis is correct, we would expect these deficits to correlate strongly with temporal deficits in other sensory domains as well.

In summary, despite the strong evidence for the prominence of phonological processing deficits in the aetiology of dyslexia, our results show that dyslexics also differ reliably from controls on low level visual tasks, specifically ones that measure temporal perception (temporal acuity). These lower level sensory deficits, that have also been found in early childhood^{10,15} could provide an important nonlinguistic metric to identify children at risk of reading problems prior to the occurrence of actual reading failure. Thus our visual tests may be useful for identifying those at risk of dyslexic problems before their reading begins to fail.

ACKNOWLEDGEMENTS

Supported by the Wellcome Trust and the McDonnell-Pew Centre for Cognitive Neuroscience. The authors wish to thank Jonathan Winter for his help with the figures, Anna Corrie and Liz Westwood for their time spent running subjects, and Piers Cornelissen and Sioux France for their helpful critique at several stages of this work. We would also like to acknowledge the helpful comments provided by two anonymous reviewers. Portions of this work were presented at the 3rd meeting of the European Neuro-Ophthalmology Society; London, England: May, 1997 and the 4th World Congress on Dyslexia; Macedonia, Greece: September, 1997.

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Figure Legends

FIGURE 1

Figure 1 is a simplified schematic representation of the two primary visual processing streams emanating from the retina (bottom) and projecting to higher levels of the visual cortex (top). LGN: Lateral Geniculate Nucleus. MT: Middle Temporal Cortex. MST: Middle Superior Temporal Cortex. VIP: Ventral Intraparietal Sulcus. LIP: Lateral Intraparietal Sulcus. PIT: Posterior Inferotemporal Cortex. CIT: Central Inferotemporal Cortex. AIT: Anterior Inferotemporal Cortex (Adapted from reference 28.)

FIGURE 2

Figure 2 delineates some of the major psychophysical, physiological and anatomical properties that distinguish the M and P retinocortical pathways.

FIGURE 3

Figure 3 summarises the dyslexics' and controls' performance on the experimental measures used in the present study. Significance evaluated by independent groups t-test (n.s = $p > .05$, two-tailed).

FIGURE 4

Figure 4 plots the dyslexics' (closed circles) and controls' (open circles) thresholds for Critical Flicker Fusion (CFF). Each point represents one individual's average detection threshold.

FIGURE 5

Figure 5 plots the dyslexics' (closed circles) and controls' (open circles) coherent motion thresholds for the Random Dot Kinematogram (RDK) stimuli. Each point represents an individual's average detection threshold.

FIGURE 6

Figure 6 shows the relationship between nonword naming performance and CFF for both the dyslexics (closed circles) and controls (open circles). One significant outlier, a dyslexic, has been removed.

FIGURE 7

Figure 7 depicts the relationship between nonword naming and coherent motion detection for the dyslexics (closed circles) and controls (open circles). The same outlier as for Figure 6 has been removed.

FIGURE 8

Figure 8 shows the relationship between nonword naming and M-pathway sensitivity as defined by an optimal scaling procedure (nonlinear principal components analysis) that combined individual thresholds from the coherent motion detection and CFF paradigms. (See text for details.)