Visual Perception and Reading: New Clues to Patterns of Dysfunction Across Multiple Visual Channels in Developmental Dyslexia

Ana Pina Rodrigues,[1] José Rebola,[1] Helena Jorge,[1] Maria José Ribeiro,[1] Marcelino Pereira,[2] Marieke van Asselen,[1] and Miguel Castelo-Branco[1]

[1]Institute for Biomedical Imaging and Life Sciences (CNC.IBILI), Faculty of Medicine, University of Coimbra, Coimbra, Portugal
[2]Faculty of Psychology and Education Sciences, University of Coimbra, Coimbra, Portugal

Correspondence: Ana Pina Rodrigues, Azinhaga de Santa Comba, 3000-548 Coimbra, Coimbra, Portugal; ana.pina.rodrigues@gmail.com.

MvA and MC-B are joint senior authors.

APR and JR contributed equally to the work presented here and should therefore be regarded as equivalent authors.

Submitted: June 8, 2016
Accepted: December 19, 2016

Citation: Pina Rodrigues A, Rebola J, Jorge H, et al. Visual perception and reading: new clues to patterns of dysfunction across multiple visual channels in developmental dyslexia. *Invest Ophthalmol Vis Sci*. 2017;58:309–317. DOI:10.1167/iovs.16-20095

Purpose. The specificity of visual channel impairment in dyslexia has been the subject of much controversy. The purpose of this study was to determine if a differential pattern of impairment can be verified between visual channels in children with developmental dyslexia, and in particular, if the pattern of deficits is more conspicuous in tasks where the magnocellular–dorsal system recruitment prevails. Additionally, we also aimed at investigating the association between visual perception thresholds and reading.

Methods. In the present case–control study, we compared perception thresholds of 33 children diagnosed with developmental dyslexia and 34 controls in a speed discrimination task, an achromatic contrast sensitivity task, and a chromatic contrast sensitivity task. Moreover, we addressed the correlation between the different perception thresholds and reading performance, as assessed by means of a standardized reading test (accuracy and fluency). Group comparisons were performed by the Mann-Whitney *U* test, and Spearman's rho was used as a measure of correlation.

Results. Results showed that, when compared to controls, children with dyslexia were more impaired in the speed discrimination task, followed by the achromatic contrast sensitivity task, with no impairment in the chromatic contrast sensitivity task. These results are also consistent with the magnocellular theory since the impairment profile of children with dyslexia in the visual threshold tasks reflected the amount of magnocellular–dorsal stream involvement. Moreover, both speed and achromatic thresholds were significantly correlated with reading performance, in terms of accuracy and fluency. Notably, chromatic contrast sensitivity thresholds did not correlate with any of the reading measures.

Conclusions. Our evidence stands in favor of a differential visual channel deficit in children with developmental dyslexia and contributes to the debate on the pathophysiology of reading impairments.

Keywords: dyslexia, reading disorder, visual perception, magnocellular-dorsal stream, ventral stream

Developmental dyslexia is a neurodevelopmental disorder characterized by a reading impairment in spite of normal intellectual functioning and educational opportunities.[1] Although the prevalence of this condition is fairly high (7%),[2] its causes and mechanisms remain under debate and are currently a subject of intensive research. Phonological deficits are usually described as the core impairment in dyslexia and constitute the basis for the most dominant theory in the field: the phonological theory.[3] This causal hypothesis states that an inadequate correspondence between phonemes and graphemes is accountable for the reading deficits in this population. Nonetheless, in-depth study of this condition has revealed perceptual and sensory dysfunctions,[4–6] which cannot be discarded. To take these into account, a number of alternative theories have therefore been put forward. Among those, a widely discussed yet controversial sensory theory is the magnocellular account.[7,8]

The magnocellular and parvocellular retinocortical pathways carry the majority of visual information from the retina into the cortex.[9] The route from primary visual cortex (V1) projecting to V5 (MT) and to posterior parietal regions is termed dorsal stream. Magnocellular input is thought to dominate this stream, often called the magnocellular–dorsal (M-D) stream. The route from V1 projecting to V4 and on to the inferior temporal cortex is referred as the ventral stream (V).[10] The two systems have distinct characteristics, and while the M-D system is specialized in processing high temporal frequencies and low spatial frequencies, the V stream processes low temporal frequencies and high spatial frequencies.[11,12] According to the magnocellular theory, the visual perception of people with dyslexia is characterized by an abnormal functioning of the M-D stream.[13,14] Several studies have provided evidence that either favor or oppose this theory. Initially, it was supported by anatomic evidence from postmortem studies in adults with dyslexia. In their studies, Galaburda and Livingstone[15] and Livingstone et al.[16] reported anatomically abnormal magno cells in the lateral geniculate nucleus (LGN), a thalamic

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License.

Downloaded from iovs.arvojournals.org on 08/18/2023

structure that receives information from the retina and projects to V1. Electrophysiological[17,18] and fMRI[19–22] studies have also corroborated these findings by highlighting abnormal neural responses to magnocellular stimuli. Moreover, psychophysical studies have shown that both children and adults with dyslexia fail to reach a normal level of motion processing,[20,23–28] attributed to the M-D stream. Other studies focused on the differences in contrast sensitivity thresholds between dyslexic patients and controls to identify M-D differences.[5,28–34] A study by Iles et al.[24] raised an important question by adressing the upstream influence of low-level deficits on higher-level visual tasks that are mainly dependent on M-D functioning. These authors found that the adults with dyslexia who had elevated motion coherence thresholds were also impaired on visual search tasks probing the posterior parietal function, which is known to be involved in reading. Additionally, to fully understand the implications of the M-D impairment in reading deficits, a number of studies addressed its correlation with reading measures, finding significant links.[28,35–37]

Nonetheless, as already mentioned, the literature is not unanimous on the claim of particular M-D impairment in dyslexia. A number of studies report normal thresholds of motion processing[38–40] and on other M-D functioning measures,[41–45] as well as considerable performance variability in the population.[24,32,46–48] Other studies question the specificity of visual channel impairment[49–52] or its mechanistic link with reading problems.[53–55] These discrepancies are partly explained by task variability and the difficulty in isolating each visual stream.[51,56] However, by compiling multiple tasks one can build an informative test battery based on different levels of M-D/V contribution.

In the present study we tested multiple visual channels to verify if a differential pattern of impairment could be found in children with and without dyslexia and to investigate whether the pattern of results can be interpreted as a function of their relative contribution to each of the visual streams. The second goal of the study was to investigate the associations between different visual thresholds and reading performance. To achieve these goals we assessed visual function in Portuguese children with developmental dyslexia and examined the link between low-level visual processing and reading performance. We chose a battery of low-level visual tasks ranging from color to achromatic contrast sensitivity and speed discrimination. These tasks seem to differentially involve M-D and V streams. In other words, they likely lead either to a preferential M-D activation (speed discrimination task) or to V activation (chromatic contrast sensitivity task). The achromatic contrast sensitivity task using intermediate spatial frequencies probably leads to a more even pattern of activation of both streams.[57,58] Our concept of a gradient of M-D involvement was developed to overcome the known difficulty to ensure an exclusive activation of M-D stream. Our battery of tasks followed, therefore, a gradient of M-D stream contribution (from strong in local speed discrimination, to mild in the intermediate spatial frequency contrast sensitivity task and weak in the chromatic task), which allowed us to establish a profile of low-level visual deficits in terms of a gradient of M-D recruitment, instead of having to rely on the assumption of exclusive activation of the stream. Finally, scores on visual function were then confronted with reading fluency and accuracy indexes, as measured through text reading.

Methods

Ethics Statement

This study and all procedures were reviewed and approved by the Ethics Committee of the Faculty of Medicine of the University of Coimbra and were conducted in accordance with the tenets of the Declaration of Helsinki. Written informed consent was obtained from the legal representatives of the participants, after explanation of the nature of the study.

Participants

Participants included 33 children with dyslexia (mean age: 9.88 ± 1.45 years) and 34 age-matched controls (mean age: 10.06 ± 1.39 years). Both groups were assessed in terms of IQ and reading level with the Wechsler Intelligence Scale for Children (WISC-III, Portuguese version)[59] and the Fluency and Accuracy Reading Assessment Test: The King,[60] widely used in Portugal for reading assessment. This reading test has two outcomes that were further analyzed as reading measures: the Accuracy Index (AI) and the Fluency Index (FI). Accuracy Index was calculated using the formula (WCR / WR) × 100, where WCR stands for the number of words correctly read and WR for the total number of words read. Fluency Index was calculated using the formula (WRC / RT) × 60, where RT stands for the total time necessary to read the text (maximum of 180 seconds).

The recruitment period of the clinical and control samples spanned 18 months. Children with dyslexia were recruited from the diagnostic and treatment center of the Faculty of Psychology and Education Sciences of the University of Coimbra. The inclusion criteria were a 2-year lag in reading speed and/or reading accuracy on the Fluency and Accuracy Reading Assessment Test: The King,[60] and a normal level of intelligence assessed by the WISC III, Portuguese version (IQ above 90).[59] The presence of comorbid attention-deficit/hyperactivity disorder was established as exclusion criterion. The clinical sample consisted of volunteer children who fulfilled the inclusion and exclusion criteria, assessed either during the period of recruitment or previously assessed at the center. The control group comprised volunteer children recruited through distribution of flyers in local schools. These typically developing children had no history of learning, developmental, cognitive, neurologic, or neuropsychiatric problems. Groups were matched for age, education, sex, and IQ. All participants had normal or corrected-to-normal vision (visual acuity of 20/20). Characteristics of participants are summarized in Table 1.

Procedure

Three tasks were applied to assess low-level visual function: a speed discrimination task (Local Speed Discrimination), an achromatic contrast sensitivity task (Intermediate Spatial Frequency), and a chromatic contrast sensitivity task (Cambridge Colour Test). The tasks took place in a darkened room. Children executed the tasks monocularly (only the dominant eye was tested) with an opaque patch occluding the other eye. A chin and forehead rest was used to ensure a stable viewing position throughout testing.

Local Speed Discrimination (LSD). The LSD task was developed in our laboratory (adapted from Ref. 61). The task was programmed in MATLAB (MATLAB 2011a; The Mathworks, Inc., Natick, MA, USA), using the Psychophysics Toolbox (PTB-3) extension. Children were seated at a viewing distance of 50 cm. All stimuli were presented on a gamma-corrected 24-inch LCDIPS monitor (ColorEdge CG243W; Eizo, Hakusan, Japan) with a resolution of 1920 × 1200 pixels and a refresh rate of 60 Hz. Spectral and luminance measurements were made using a spectroradiometer (PR-650 SpectraScan Colorimeter; Photo Research, Inc., Chatsworth, PA, USA). The background luminance was ~0 cd/m^2.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

Table 1. Summary Statistics for the Two Groups of Participants

	Children With Dyslexia, $n = 33$			Children Without Dyslexia, $n = 34$			
Demographics and Clinical Variables	**Mean**	**Range**	**SD**	**Mean**	**Range**	**SD**	***P* Value**
Age, y	9.88	7–13	1.45	10.06	7–12	1.39	0.383
Education, y	4.36	2–7	1.22	4.76	2–7	1.39	0.082
IQ	104.70	90–127	8.45	107.50	92–132	9.14	0.251
Reading Accuracy Index	89.76	35.7–98.2	12.59	98.90	95–100	1.61	<0.001
Reading Fluency Index	58.94	14–127	28.14	138.07	93–179	20.74	<0.001
Sex, m/f	20/13			13/21			0.089

Probability values for group comparisons using Mann-Whitney *U* tests (except for sex, for which the χ^2 test was used) are reported ($P < 0.05$ values are considered significant).

The LSD is a psychophysical task that requires the discrimination of motion speed between two separated moving single dots (a reference dot and a target dot) (see Fig. 1a). In each trial the reference and target dots (two white dots moving at different velocities) were simultaneously presented for 400 ms. Stimuli consisted of squared dots measuring 0.3° × 0.3°. The reference dot velocity was always 5°/s (visual degrees per second), while the target dot velocity started at 24°/s and was then adjusted by the logarithmic staircase procedure (maximum step size of 1 decibel [dB] and minimum of 0.05 dB). Children were asked to fixate a black central cross (size of 1°) during the test. After each trial, participants were asked to press a button on a keyboard indicating which dot was moving faster ("Left/Right" for the horizontal and oblique meridians or "Up/Down" for the vertical meridian). The motion was then adjusted in the following trial, driven by a correct or incorrect response, by using a logarithmic staircase procedure. The tests ended after six reversals, and a discrimination threshold was calculated using the arithmetic mean of the last four reversals. This threshold represents the discriminated difference, in °/s, between test and reference stimulus. The test was repeated four times, corresponding to four different meridian/eccentricity pairs (the horizontal meridian, 0°, tested at 7.5° of eccentricity; the vertical meridian, 90°, at 10°; and the oblique meridians, 45° and 135°, at 15°). The four thresholds obtained from the four different meridians were averaged into a grand average in order to obtain a measure of the global motion perception of these children.

Intermediate Spatial Frequency (ISF). The ISF contrast sensitivity task, developed in our laboratory,[62,63] uses static achromatic vertical gratings with an intermediate profile. Stimuli were static vertical gratings, with a spatial frequency of 3.5 cyc/deg (mean background luminance of 51 cd/m^2, constant throughout the experiment) displayed on a 21-inch monitor (Trinitron GDM-F520 monitor; Sony, Tokyo, Japan).

The width of each stimulus was 10° of visual angle (35 grating cycles) (see Fig. 1b). Stimulus duration was 200 ms, and the interstimulus interval varied randomly between 2300 and 2800 ms. The stimuli were presented within nine locations of the visual field. Children were seated at a viewing distance of 36 cm and were instructed to fixate the black square in the center of the screen and report the presence of the targets by pressing a button. Participants' reliability was evaluated by the inclusion of false-positive (0% contrast stimuli) and false-negative (100% contrast in the central location) "catch trials." Experiments with a false-positive or false-negative rate above 33% were aborted. The task was then repeated after a small rest period. If the participant still responded with a high number of false positives or false negatives, the data were not used in the analysis.

Luminance contrast of the stimulus was expressed according to Michelson. Contrast sensitivity results were expressed in terms of decibel units, dB = 20 × log (1/c), with contrast c measured as a percentage. To obtain the psychophysical thresholds, the test uses nine randomly interleaved logarithmic staircases, one for each location tested. The contrast value used for a given trial was calculated using the previous trial value

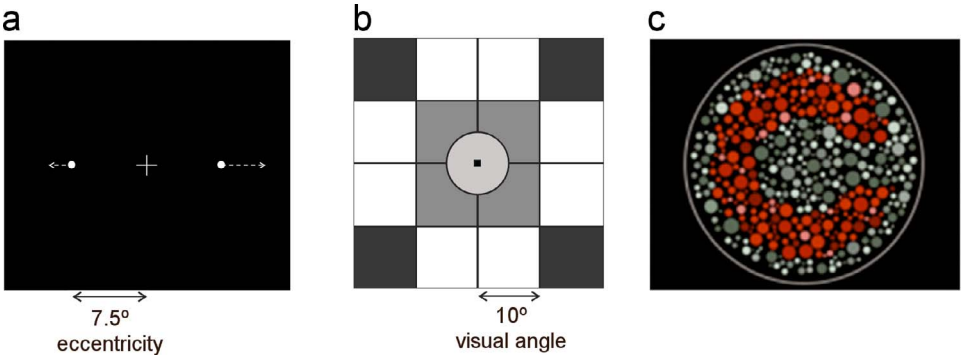

Figure 1. Schematic representation of the visual tests. (**a**) Representation of the location, at the horizontal meridian 0°, where the moving dots were presented in the speed discrimination task. Three additional meridians were tested (vertical 90°, tested at 10° of eccentricity; and oblique 45° and 135°, at 15° of eccentricity). The central cross represents the fixation cross. (**b**) Representation of the sizes and shapes of the nine locations (represented in different shades of *gray* and *black*) within the visual field where the gratings with intermediate spatial frequency were presented. Note that in the actual experiment the *shaded areas* and the *separating lines* were not present. The stimuli were shown at these locations against an overall gray background. The *black square* in the *middle of the figure* represents the fixation square. (**c**) Illustration of the stimuli used in the chromatic contrast sensitivity task (Cambridge Colour Test) representing a luminance noise stimulus with superimposed chromatic target (Landolt C shape, colored in *red*).

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

Table 2. Low-Level Visual Perception Thresholds of Children With and Without Dyslexia

	Children With Dyslexia		Children Without Dyslexia			
Psychophysical Measures	**Median**	**Q1–Q3**	**Median**	**Q1–Q3**	***U***	***P* Value**
LSD, °/s	3.59	2.27–4.50	1.42	0.96–2.64	222.50	<0.0001
ISF, dB	12.58	9.61–14.80	9.67	7.58–10.36	283.00	0.001
CCT-PD, u'v'*10^{-4}	56.25	49.37–68.75	56.00	47.38–67.13	508.00	0.644

Probability values for group comparisons using Mann-Whitney *U* tests are reported ($P < 0.05$ values are considered significant).

plus or minus the step size in dB. The step size used was 3 dB. Staircases were run for a total of four reversals. The contrast at the final two reversals was averaged to estimate the contrast threshold. For this task, data could not be collected from one of the control children.

Cambridge Colour Test (CCT). Finally, to test chromatic contrast sensitivity we used a task that establishes a threshold of color discrimination, the Cambridge Colour Test (CCT; Cambridge Research Systems, Rochester, UK). Stimuli were displayed on a 21-inch monitor (GDM-F520; Sony) and consisted of static patterns of circles of various sizes and luminances with superimposed chromatic contrast defining the letter C (gap size: 1.6°; outer diameter: 7.6°; inner diameter: 3.81°) (see Fig. 1c). Participants were positioned at a viewing distance of 1.8 m and were instructed to indicate the position of the C's gap by pressing one of four buttons (up, down, left, or right). We used a color version of the test (Trivector; CCT), where the targets differ from the background along one of the three color confusion lines, each activating one type of cone receptor: protan, deutan, or tritan. We took as the threshold for the red–green (parvocellular) chromatic channel the average of the thresholds along protan and deutan lines (CCT-PD). The test uses three randomly interleaved staircases to dynamically adjust the chromaticity of the target according to the participant's performance to establish the chromaticity difference between target and background needed for reliable report of the orientation of the C. Occasional control trials, with a target presented at maximal chromatic saturation, were introduced to ensure that the participant was alert. Testing on any one staircase was terminated after 11 reversals, and the mean of the last 6 reversals was taken as the threshold estimate for the direction being tested, as has been previously established.[61,62] Psychophysical thresholds were expressed in CIE 1976 u'v' color space units.

Statistical Analysis. All statistical analyses were performed using the IBM SPSS statistical software package, version 20.0 (SPSS, Inc., Chicago, IL, USA). Since data significantly deviated from normal distributions (verified using the Kolmogorov-Smirnov normality check and Levene homogeneity tests), we applied nonparametric statistical methods. Group comparisons were performed by Mann-Whitney *U* test. Participants scoring more than 3 SD away from the group mean were considered outliers and therefore not included in the between-group analyses. This resulted in the exclusion of one participant with dyslexia from the CCT-PD task comparison. Correlational analyses were performed using Spearman rank correlation coefficient (ρ). As in other studies,[36,64] correlations were assessed for the population as a whole. Once again, participants scoring more than 3 SD away from the overall population mean were considered outliers and therefore not included in the analyses. This was the case for five children with dyslexia: two in the ISF task, one in the CCT-PD task, and two in the AI measure.

Results

Low-Level Visual Perception in Dyslexia

The low-level visual function was assessed through a battery of tasks: the LSD, the ISF, and the CCT. Results are summarized in Table 2. Mann-Whitney *U* test analyses showed that cases and controls had similar CCT-PD thresholds ($P = 0.644$), indicating a preserved color discrimination in the dyslexic group (see Fig. 2, top). In contrast, the ISF task already revealed a significant difference between groups ($P = 0.001$; effect size $r = 0.410$), with poorer perception thresholds for children with dyslexia (see Fig. 2, middle). This performance difference between groups was further increased in the LSD task ($P < 0.0001$; effect size $r = 0.519$) (see Fig. 2, bottom).

Taken together, results showed that children with dyslexia were more impaired in the LSD task, followed by the ISF task, with no impairment in the CCT-PD task and, therefore, argue against a generalized visual perception deficit.

Correlations Between Low-Level Visual Functions and Reading

In order to address the link between visual perception and reading, Spearman correlations were computed between the low-level visual thresholds (LSD, ISF, and CCT-PD) and the reading measures (AI and FI).

No significant correlations were found between the CCT-PD thresholds and the reading measures (AI: $\rho = -0.054$ [$P = 0.670$]; FI: $\rho = -0.148$ [$P = 0.236$]). Thus, we did not find evidence of an association between chromatic sensitivity and reading. On the contrary, achromatic contrast sensitivity and speed discrimination were correlated with reading performance, in terms of both accuracy (AI) and fluency (FI). In the case of the ISF thresholds, the correlation coefficients were $\rho = -0.413$ ($P = 0.0009$) for the AI and $\rho = -0.412$ ($P = 0.0007$) for the FI. For the LSD task, correlation analysis identified significant correlations with the AI ($\rho = -0.440$; $P = 0.0003$) and with FI ($\rho = -0.520$; $P < 0.0001$) (Fig. 3). Therefore, we found that the lower the achromatic contrast sensitivity and speed discrimination thresholds, the better the reading performance, in terms of both accuracy and fluency.

Discussion

The present work compared children with and without developmental dyslexia (mean age: 9.88 and 10.06, respectively, which means that our results may not be generalizable to other age cohorts) on a battery of visuoperceptual tasks assessing chromatic and achromatic contrast sensitivity and speed discrimination. The fundamental aim of this work was to probe multiple visual channels in children diagnosed with developmental dyslexia in order to verify if a differential pattern of impairment is present in these children.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

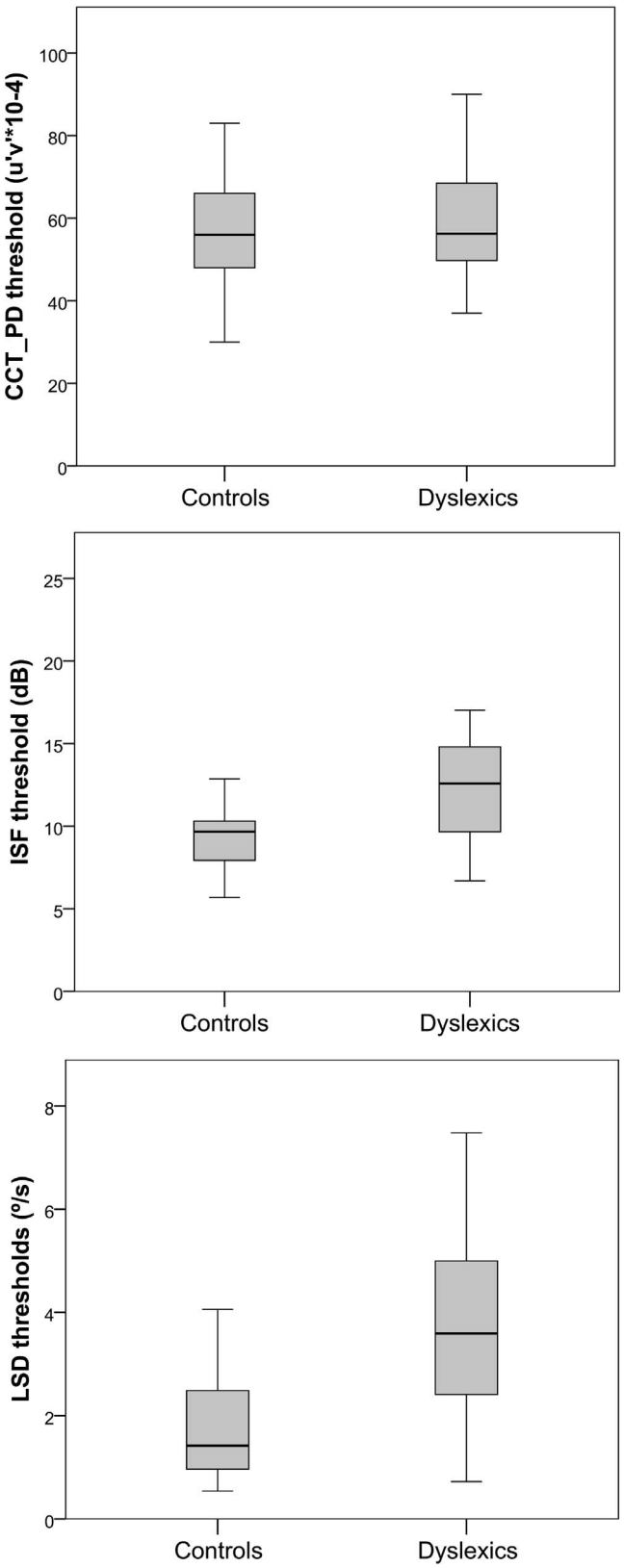

FIGURE 2. Performance of controls and dyslexics in the CCT-PD (*top*), ISF (*middle*), and LSD (*bottom*). Moving *from top to bottom*, note that dyslexics show normal chromatic contrast sensitivity, mildly impaired achromatic contrast sensitivity, and considerable speed discrimination impairment. (*Box boundaries* correspond to upper and lower 25th percentiles, *outer bars* to the 10th percentiles, and *middle bar* to the median).

We demonstrate that Portuguese children with dyslexia are substantially impaired when asked to discriminate speed, corroborating previous studies that addressed motion processing.[20,26,28,49] It should be noted that motion perception has traditionally been assessed by coherent motion detection thresholds instead of speed discrimination. However, the output of this particular type of task across studies is contradictory (see Refs. 23–25, 27, 65, but also 38 and 39). Moreover, deficits in motion coherence are present in several neurodevelopmental disorders, such as autism[66,67] and Williams syndrome.[68,69] On the contrary, speed discrimination impairments seem to be preserved in, at least, some of these disorders.[70,71] Thus, it seems that speed discrimination deficits may represent more specific motion deficits in dyslexia than coherent motion deficits. Additionally, recent genetic studies showed that some motion deficits, including coherent motion but not speed discrimination, are particularly strong in dyslexic individuals with a deletion in intron 2 of the *DCDC2* gene rather than in the whole dyslexic population.[47,72]

The second main finding of our study is that children with dyslexia have preserved chromatic contrast sensitivity. Chromatic vision in these patients, contrary to other visual functions, has not been comprehensively studied. To our knowledge there are few studies on this subject,[52,64,73–75] and only Ahmadi et al.[52] reported abnormal chromatic contrast thresholds in children with dyslexia. However, the task used in their study to assess chromatic contrast sensitivity consisted of chromatic natural scenes, which suggests that neural responses and performance may be distinct for natural chromatic scenes. Moreover, we must acknowledge that our chromatic contrast sensitivity testing was limited to the red–green chromatic channel, since it also aimed at probing V stream functioning. Nonetheless, the present study endorses the majority of the previous literature by adding supportive evidence of a preserved chromatic channel in a population where other visual channels were concomitantly studied. Finally, we show, for the first time, that children with dyslexia were mildly affected when an ISF channel was tested. Taken together, our results indicate differential low-level visual deficits in children with developmental dyslexia, arguing against the notion of a generalized visuoperceptual impairment.

The study of visual function in dyslexia has been mainly related to the debate on the magnocellular theory.[7,8] According to this theory, dyslexics suffer from specific M-D stream difficulties. Magnocellular-dorsal stream is known to activate preferentially to stimuli with low spatial frequencies and high temporal frequencies, and many studies have used these properties to vouch for or contradict a M-D deficit in dyslexia.[23,33,34,42,44,76–78] Nonetheless, there is not a definite consensus because for most frequency ranges, activation is not exclusive to this stream. In fact, the range of spatial frequencies used as a hallmark of M-D functioning has been a target for criticism.[51,56] Another marker that has been used to claim M-D deficits in dyslexia is abnormal visual coherent motion (e.g., Refs. 22–24, 26, 33, 78, 79). However, the reliability of this measure to assess M-D sensitivity has also been challenged.[80] Thus, the validation of the magnocellular theory is undermined by a fundamental issue: the difficulty, or even the impossibility, of exclusively activating M-D stream. In order to overcome this difficulty, here we designed a battery of visuoperceptual tasks with different levels of M-D contribution. Therefore, having a battery of three tasks, we can establish a profile of low-level visual deficits in terms of a "gradient" of M-D recruitment, instead of relying on the assumption of exclusive activation of this stream. In this manner, we can think of our tasks as ranging from strong (speed discrimination) to weak (chromatic

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

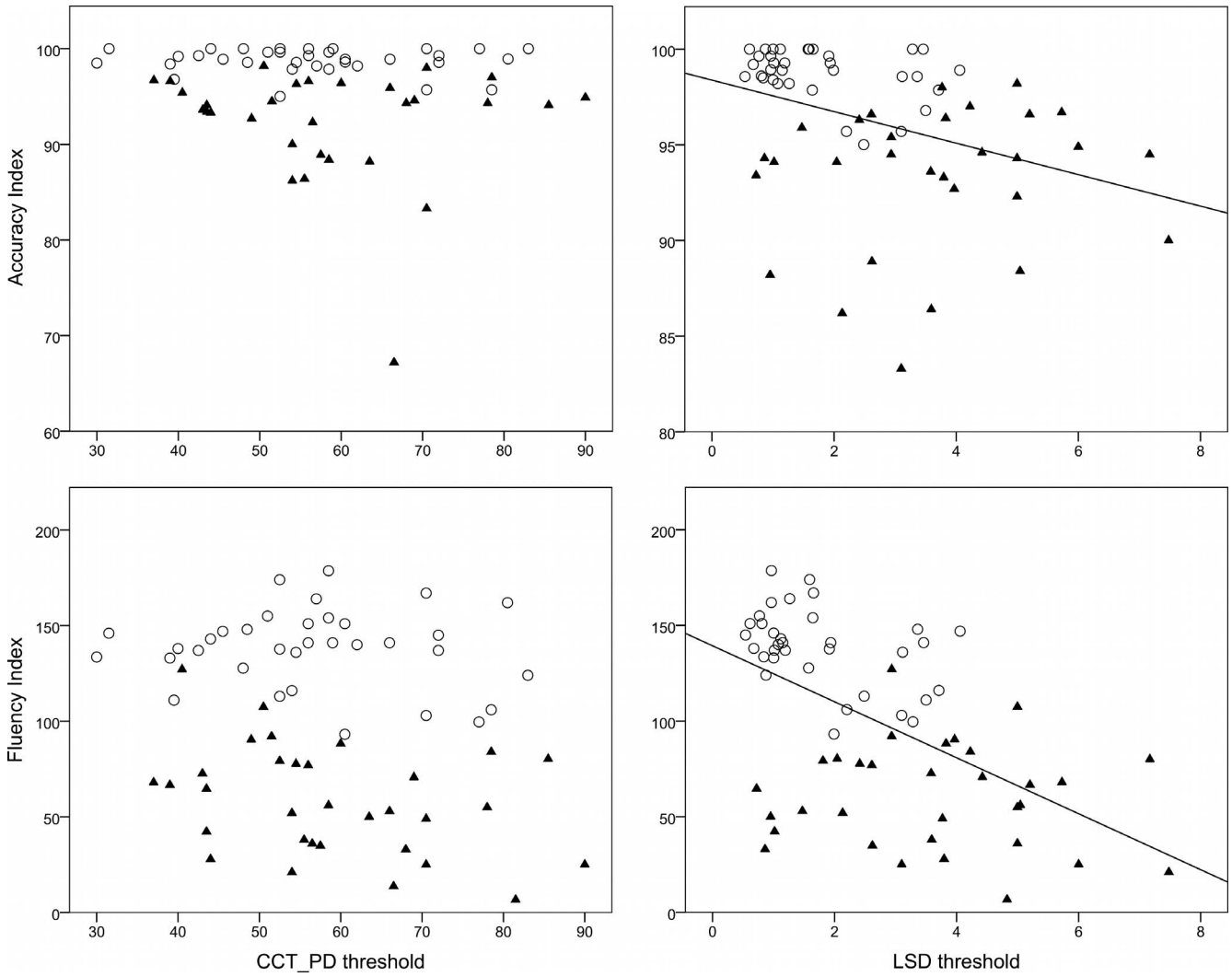

FIGURE 3. Scatter plots illustrating the correlations between the Accuracy and Fluency Indexes from the reading test with chromatic sensitivity (CCT-PD) and speed discrimination (LSD) perception thresholds. Controls (*empty dots*). Dyslexics (*filled triangles*).

sensitivity) M-D involvement. According to the magnocellular theory, we found that the higher the M-D involvement, the higher the differences between groups. Therefore, using this procedure, we report compelling evidence for a preferential M-D deficit in children with dyslexia.

To conclude, we demonstrate that speed discrimination thresholds are the ones that show the strongest correlation with reading, followed by the ISF thresholds. Notably, chromatic contrast sensitivity thresholds did not correlate with any of the reading measures. These results highlight the notion that the link between low-level visual function and reading is not generalized across different visual systems. This result is in agreement with studies both in individuals with dyslexia and in controls.[20,28,36,37] Following the M-D gradient mentioned above, we found that the higher the involvement of M-D mechanisms in the administered tasks, the stronger the correlation to both accuracy and fluency indices. This result is also in accordance with the roles that the M-D system may play in reading-related tasks. These include accurate letter position encoding through precise shifts in visual attention,[35,81,82] the ability to process information that changes rapidly over the course of time,[64] or the rapid delivery of a low-pass representation of words to guide further processing.[83] Actually, a very recent study[84] showed that M-D stream training significantly improved reading fluency and reading comprehension in individuals with dyslexia, supporting the hypothesis of a causal link between M-D processing deficits and dyslexia.

Finally, the link between low-level visual performance and reading is perhaps as important as the establishment of low-level visual deficits in dyslexia. In future studies, attention should be devoted to the understanding of how these particular visuoperceptual deficits underlie reading impairment. Only a clear unfolding of this issue can unequivocally establish those deficits as contributing to reading difficulties in dyslexia. Studies of indirect[19,34,83,85] and direct interference (TMS)[86] are already paving the way on this matter.

Acknowledgments

Supported by the Foundation for Science and Technology Portugal under Grants PTDC/SAU-NSC/113471/2009, SFRH/BD/75083/2010, and UID/NEU/04539/2013–COMPETE, POCI-01-0145-FEDER-007440.

Downloaded from iovs.arvojournals.org on 08/18/2023

Disclosure: **A. Pina Rodrigues**, None; **J. Rebola**, None; **H. Jorge**, None; **M.J. Ribeiro**, None; **M. Pereira**, None; **M. van Asselen**, None; **M. Castelo-Branco**, None

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*. 4th ed., text revision. Washington, DC: American Psychiatric Association; 2000.
2. Shaywitz SE, Shaywitz BA, Fletcher JM, Escobar MD. Prevalence of reading disability in boys and girls. Results of the Connecticut Longitudinal Study. *JAMA*. 1990;264:998–1002.
3. Snowling M. Phonemic deficits in developmental dyslexia. *Psychol Res*. 1981;43:219–234.
4. Badcock NA, Hogben JH, Fletcher JF. Dyslexia and practice in the attentional blink: evidence of slower task learning in dyslexia. *Cortex*. 2011;47:494–500.
5. Buchholz J, McKone E. Adults with dyslexia show deficits on spatial frequency doubling and visual attention tasks. *Dyslexia*. 2004;10:24–43.
6. Goswami U. Sensory theories of developmental dyslexia: three challenges for research. *Nat Rev Neurosci*. 2015;16:43–54.
7. Stein J. The magnocellular theory of developmental dyslexia. *Dyslexia*. 2001;7:12–36.
8. Stein J, Walsh V. To see but not to read; the magnocellular theory of dyslexia. *Trends Neurosci*. 1997;20:147–152.
9. Ungerleider LG, Mishkin M. Two cortical visual systems. In: Ingle D, Goodale M, Mansfield R, eds. *The Analysis of Visual Behavior*. Cambridge, MA: MIT Press; 1982:549–586.
10. Beaton A. *Dyslexia, Reading and the Brain: A Sourcebook of Psychological and Biological Research*. Hove, UK: Psychology Press; 2004.
11. Livingstone MS, Hubel DH. Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science*. 1988;6:740–749.
12. Kaplan E, Shapley RM. The primate retina contains two types of ganglion cells, with high and low contrast sensitivity. *Proc Natl Acad Sci U S A*. 1986;83:2755–2757.
13. Farmer ME, Klein RM. The evidence for a temporal processing deficit linked to dyslexia: a review. *Psychon Bull Rev*. 1995;24:460–493.
14. Boden C, Giaschi D. M-stream deficits and reading-related visual processes in developmental dyslexia. *Psychol Bull*. 2007;133:346–366.
15. Galaburda A, Livingstone M. Evidence for a magnocellular defect in developmental dyslexia. *Ann N Y Acad Sci*. 1993;682:70–82.
16. Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci U S A*. 1991;88:7943–7947.
17. Scheuerpflug P, Plume E, Vetter V, et al. Visual information processing in dyslexic children. *Clin Neurophysiol*. 2004;115:90–96.
18. Jednoróg K, Marchewka A, Tacikowski P, Heim S, Grabowska A. Electrophysiological evidence for the magnocellular-dorsal pathway deficit in dyslexia. *Dev Sci*. 2011;14:873–880.
19. Ben-Shachar M, Dougherty RF, Deutsch GK, Wandell BA. Contrast responsivity in MT+ correlates with phonological awareness and reading measures in children. *Neuroimage*. 2007;37:1396–1406.
20. Demb JB, Boynton GM, Heeger DJ. Functional magnetic resonance imaging of early visual pathways in dyslexia. *J Neurosci*. 1998;18:6939–6951.
21. Dhar M, Been PH, Minderaa RB, Althaus M. Reduced interhemispheric coherence in dyslexic adults. *Cortex*. 2010;46:794–798.
22. Demb JB, Boynton GM, Heeger DJ. Brain activity in visual cortex predicts individual differences in reading performance. *Proc Natl Acad Sci U S A*. 1997;94:13363–13366.
23. Cornelissen P, Richardson A, Mason A. Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Res*. 1995;35:1483–1494.
24. Iles J, Walsh V, Richardson A. Visual search performance in dyslexia. *Dyslexia*. 2000;6:163–177.
25. Talcott JB, Witton C, Hebb GS, et al. On the relationship between dynamic visual and auditory processing and literacy skills; results from a large primary-school study. *Dyslexia*. 2002;8:204–225.
26. Eden G, VanMeter J, Rumsey J, Maisog JM, Woods RP, Zeffiro TA. Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature*. 1996;382:66–69.
27. Everatt J, Bradshaw MF, Hibbard PB. Visual processing and dyslexia. *Perception*. 1999;28:243–254.
28. Demb JB, Boynton GM, Best M, Heeger DJ. Psychophysical evidence for a magnocellular pathway deficit in dyslexia. *Vision Res*. 1998;38:1555–1559.
29. Pellicano E, Gibson LY. Investigating the functional integrity of the dorsal visual pathway in autism and dyslexia. *Neuropsychologia*. 2008;46:2593–2596.
30. Evans BJ, Drasdo N, Richards IL. An investigation of some sensory and refractive visual factors in dyslexia. *Vision Res*. 1994;34:1913–1926.
31. Williams MJ, Stuart GW, Castles A, McAnally KI. Contrast sensitivity in subgroups of developmental dyslexia. *Vision Res*. 2003;43:467–477.
32. Borsting E, Ridder WH, Dudeck K, Kelley C, Matsui L, Motoyama J. The presence of a magnocellular defect depends on the type of dyslexia. *Vision Res*. 1996;36:1047–1053.
33. Martin F, Lovegrove W. The effects of field size and luminance on contrast sensitivity differences between specifically reading disabled and normal children. *Neuropsychologia*. 1984;22:73–77.
34. Kevan A, Pammer K. Predicting early reading skills from pre-reading measures of dorsal stream functioning. *Neuropsychologia*. 2009;47:3174–3181.
35. Cornelissen PL, Hansen PC, Gilchrist I, Cormack F, Essex J, Frankish C. Coherent motion detection and letter position encoding. *Vision Res*. 1998;38:2181–2191.
36. Conlon E, Sanders M, Zapart S. Temporal processing in poor adult readers. *Neuropsychologia*. 2004;42:142–157.
37. Levy T, Walsh V, Lavidor M. Dorsal stream modulation of visual word recognition in skilled readers. *Vision Res*. 2010;50:883–888.
38. Kronbichler M, Hutzler F, Wimmer H. Dyslexia: verbal impairments in the absence of magnocellular impairments. *Neuroreport*. 2002;13:617–620.
39. Vanni S, Uusitalo MA, Kiesilä P, Hari R. Visual motion activates V5 in dyslexics. *Neuroreport*. 1997;8:1939–1942.
40. Sperling AJ, Lu Z-L, Manis FR, Seidenberg MS. Motion-perception deficits and reading impairment: it's the noise, not the motion. *Psychol Sci*. 2006;17:1047–1053.
41. Victor JD, Conte MM, Burton L, Nass RD. Visual evoked potentials in dyslexics and normals: failure to find a difference in transient or steady-state responses. *Vis Neurosci*. 1993;10:939–946.
42. Sperling AJ, Lu Z-L, Manis FR, Seidenberg MS. Deficits in perceptual noise exclusion in developmental dyslexia. *Nat Neurosci*. 2005;8:862–863.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

43. Johannes S, Kussmaul CL, Munte TF, Mangun GR. Developmental dyslexia: passive visual stimulation provides no evidence for a magnocellular processing defect. *Neuropsychologia*. 1996;34:1123–1127.
44. Gross-Glenn K, Skottun BC, Glenn W, et al. Contrast sensitivity in dyslexia. *Vis Neurosci*. 1995;12:153–163.
45. Roach NW, Hogben JH. Attentional modulation of visual processing in adult dyslexia. *Psychol Sci*. 2004;15:650–654.
46. Kubová Z, Kuba M, Kremláček J, et al. Comparison of visual information processing in school-age dyslexics and normal readers via motion-onset visual evoked potentials. *Vision Res*. 2015;111:97–104.
47. Cicchini GM, Marino C, Mascheretti S, Perani D, Morrone MC. Strong motion deficits in dyslexia associated with DCDC2 gene alteration. *J Neurosci*. 2015;35:8059–8064.
48. Ramus F. Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults. *Brain*. 2003;126:841–865.
49. Amitay S, Ben-Yehudah G, Banai K, Ahissar M. Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. *Brain*. 2002;125(pt 10):2272–2285.
50. Farrag AF, Khedr EM, Abel-Naser W. Impaired parvocellular pathway in dyslexic children. *Eur J Neurol*. 2002;9:359–363.
51. Skottun BC. The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. *Vision Res*. 2000;40:111–127.
52. Ahmadi K, Pouretemad H, Esfandiari J, Yoonessi A, Yoonessi A. Psychophysical evidence for impaired Magno, Parvo, and Konio-cellular pathways in dyslexic children. *J Ophthalmic Vis Res*. 2015;10:433–440.
53. Heim S, Grande M, Pape-Neumann J, et al. Interaction of phonological awareness and "magnocellular" processing during normal and dyslexic reading: behavioural and fMRI investigations. *Dyslexia*. 2010;16:258–282.
54. White S, Milne E, Rosen S, Hansen P, Swettenham J, Frith U. The role of sensorimotor impairment in dyslexia: a multiple case study of dyslexic children. *Dev Sci*. 2006;9:237–255.
55. Olulade OA, Napoliello EM, Eden GF. Abnormal visual motion processing is not a cause of dyslexia. *Neuron*. 2013;79:180–190.
56. Skottun BC. On the use of spatial frequency to isolate contributions from the magnocellular and parvocellular systems and the dorsal and ventral cortical streams. *Neurosci Biobehav Rev*. 2015;56:266–275.
57. Kulikowski JJ, Robson AG. Spatial, temporal and chromatic channels: electrophysiological foundations. *J Opt Technol*. 1999;66:797.
58. Murav'eva SV, Deshkovich AA, Shelepin YE. The human magno and parvo systems and selective impairments of their functions. *Neurosci Behav Physiol*. 2009;39:535–543.
59. Menezes Rocha A. Manual da Escala de Inteligencia de Wechsler para crianças, de David Wechsler, adaptado e aferido para a população portuguesa por António Menezes Rocha. Lisbon: CEGOC; 2003.
60. da Cruz Carvalho AOD, Pereira MAM. O Rei – Um Teste para Avaliação da Fluência e Precisão da Leitura no 1° e 2° ciclos do Ensino Básico. *Psychologica*. 2009;51:283–305.
61. Mateus C, Lemos R, Silva MF, et al. Aging of low and high level vision: from chromatic and achromatic contrast sensitivity to local and 3D object motion perception. *PLoS One*. 2013;8:e55348.
62. Ribeiro MJ, Violante IR, Bernardino I, et al. Abnormal achromatic and chromatic contrast sensitivity in neurofibromatosis type 1. *Invest Ophthalmol Vis Sci*. 2012;53:287–293.
63. Silva MF, Maia-Lopes S, Mateus C, et al. Retinal and cortical patterns of spatial anisotropy in contrast sensitivity tasks. *Vision Res*. 2008;48:127–135.
64. Sperling AJ, Lu ZL, Manis FR, Seidenberg MS. Selective magnocellular deficits in dyslexia: a "phantom contour" study. *Neuropsychologia*. 2003;41:1422–1429.
65. Ridder WH, Borsting E, Banton T. All developmental dyslexic subtypes display an elevated motion coherence threshold. *Optom Vis Sci*. 2001;78:510–517.
66. Pellicano E, Gibson L, Maybery M, Durkin K, Badcock DR. Abnormal global processing along the dorsal visual pathway in autism: a possible mechanism for weak visuo-spatial coherence? *Neuropsychologia*. 2005;437:1044–1053.
67. Spencer JV, O'Brien JMD. Visual form-processing deficits in autism. *Perception*. 2006;358:1047–1055.
68. Mendes M, Silva F, Simoes L, Jorge M, Saraiva J, Castelo-Branco M. Visual magnocellular and structure from motion perceptual deficits in a neurodevelopmental model of dorsal stream function. *Cogn Brain Res*. 2005;253:788–798.
69. Atkinson J, Braddick O, Rose FE, Searcy YM, Wattam-Bell J, Bellugi U. Dorsal-stream motion processing deficits persist into adulthood in Williams syndrome. *Neuropsychologia*. 2006;44:828–833.
70. Chen Y, Norton DJ, McBain R, Gold J, Frazier JA, Coyle JT. Enhanced local processing of dynamic visual information in autism: evidence from speed discrimination. *Neuropsychologia*. 2012;50733–50739.
71. Manning C, Neil L, Karaminis T, Pellicano E. The effects of grouping on speed discrimination thresholds in adults, typically developing children, and children with autism. *J Vis*. 2015;15(11):17.
72. Gori S, Mascheretti S, Giora E, et al. The DCDC2 intron 2 deletion impairs illusory motion perception unveiling the selective role of magnocellular-dorsal stream in reading (Dis)ability. *Cereb Cortex*. 2015;25:1685–1695.
73. Pammer K, Wheatley C. Isolating the M(y)-cell response in dyslexia using the spatial frequency doubling illusion. *Vision Res*. 2001;4116:2139–2147.
74. Bednarek DB, Grabowska A. Luminance and chromatic contrast sensitivity in dyslexia: the magnocellular deficit hypothesis revisited. *Neuroreport*. 2002;13:2521–2525.
75. Dain SJ, Floyd RA, Elliot RT. Color and luminance increment thresholds in poor readers. *Vis Neurosci*. 2008;25:481–486.
76. Lovegrove W, Martin F, Slaghuis W. A theoretical and experimental case for a visual deficit in specific reading disability. *Cogn Neuropsychol*. 1986;3:225–267.
77. Lovegrove WJ, Bowling A, Badcock D, Blackwood M. Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. *Science*. 1980;210:439–440.
78. Martin F, Lovegrove W. Flicker contrast sensitivity in normal and specifically disabled readers. *Perception*. 1987;16:215–221.
79. Hansen PC, Stein JF, Orde SR, Winter JL, Talcott JB. Are dyslexics' visual deficits limited to measures of dorsal stream function? *Neuroreport*. 2001;12:1527–1530.
80. Skottun BC, Skoyles JR. Coherent motion, magnocellular sensitivity and the causation of dyslexia. *Int J Neurosci*. 2008;118:185–190.
81. Vidyasagar TR, Pammer K. Dyslexia: a deficit in visuo-spatial attention, not in phonological processing. *Trends Cogn Sci*. 2010;14:57–63.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023

82. Stein J. Dyslexia: the role of vision and visual attention. *Curr Dev Disord Rep*. 2014;1:267–280.
83. Chouake T, Levy T, Javitt DC, Lavidor M. Magnocellular training improves visual word recognition. *Front Hum Neurosci*. 2012;6:14.
84. Lawton T. Improving dorsal stream function in dyslexics by training figure/ground motion discrimination improves attention, reading fluency, and working memory. *Front Hum Neurosci*. 2016;10:397.
85. Kevan A, Pammer K. Making the link between dorsal stream sensitivity and reading. *Neuroreport*. 2008;19:467–470.
86. Laycock R, Crewther DP, Fitzgerald PB, Crewther SG. TMS disruption of V5/MT+ indicates a role for the dorsal stream in word recognition. *Exp Brain Res*. 2009;197:69–79.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 08/18/2023