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Contrast sensitivity in subgroups of developmental dyslexia

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Abstract

It has been proposed that developmental dyslexia is associated with a deficit in the magnocellular pathway of the visual system. Other research focuses upon the heterogeneous nature of developmental dyslexia, and evidence that subgroups of dyslexia may be identified based on selective deficits in specific component reading skills. This study tested the hypothesis that visual processing deficits may be present in different subgroups of developmental dyslexia by comparing the visual contrast sensitivity of three subgroups of dyslexic children (phonological, surface and mixed) and controls. The stimulus designed to measure magnocellular visual function was a low spatial frequency Gaussian blob, flickered sinusoidally at a temporal frequency of 8.33 Hz. The control stimulus, designed to measure parvocellular visual function, was a relatively high spatial frequency Gaussian windowed grating (8 c/deg) slowly ramped on and off. There were no significant differences between the groups of dyslexic and control children in contrast sensitivity to either stimulus. The findings do not support the existence of a magnocellular system deficit in dyslexia.

Keywords: Dyslexia; Magnocell; Parvocell; Contrast sensitivity

1. Introduction

Developmental dyslexia is defined as the failure to acquire the level of reading skill expected for one's age, given normal educational opportunity, average intelligence, and the absence of sensory deficits, psychiatric or neurological disorder (Critchley, 1964). Research investigating the underlying causes of dyslexia has implicated a number of underlying neural mechanisms. An influential theory is the magnocellular deficit theory, which postulates a deficit at the level of one of the two parallel retinocortical pathways in the visual system, specifically the magnocellular pathway of the lateral geniculate nucleus (Habib, 2000; Lovegrove, 1996; Stein & Walsh, 1997).

Parallel research investigating the patterns of reading deficit in developmental dyslexics has provided considerable evidence for the existence of subgroups of developmental dyslexia (Castles & Coltheart, 1993; Castles, Datta, Gayan, & Olson, 1999; Manis, Seidenberg, Doi, McBride-Chang, & Petersen, 1996; Stanovich, Siegel, & Gottardo, 1997). There have been only a few recent studies that have investigated the relationship between the magnocellular deficit theory of dyslexia and subgroups of dyslexia. If only some subgroups exhibit visual deficits, this may explain why there has been mixed support for the magnocellular deficit theory (Skottun, 2000).

1.1. Contrast sensitivity and magnocellular function in dyslexia

The psychophysical evidence strongly supports the theory that at least two mechanisms underlie threshold contrast sensitivity. As originally demonstrated by Tolhurst (1973), the spatio-temporal contrast sensitivity function exhibits an interaction between spatial and temporal sensitivity, indicating that the mechanism sensitive to high temporal frequencies is more sensitive to lower spatial frequencies. This basic finding has been replicated a

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number of times (see Peli, Arend, Young, & Goldstein, 1993). Koenderink and van Doorn (1979) demonstrated two distinct peaks in the contrast sensitivity function by using temporal modulation rates as slow as 0.1 Hz. In addition, recent studies of individual differences in contrast sensitivity functions support the existence of at least two independent channels (see Dobkins, Gunther, & Peterzell, 2000).

The original studies investigating early visual deficits in dyslexia used spatio-temporal contrast sensitivity as an indicator of "transient" and "sustained" visual function (Lovegrove, Bowling, Badcock, & Blackwood, 1980; Martin & Lovegrove, 1984; Martin & Lovegrove, 1988). With advances in primate single-cell neurophysiology, these functions have now been associated with the magnocellular and parvocellular systems of the lateral geniculate nucleus respectively, and it has been theorised that in dyslexia there is specific damage to the magnocellular system (Livingstone, Drislane, Rosen, & Galaburda, 1991; Lovegrove, 1996; Stein & Walsh, 1997). Habib (2000) concluded that selective losses of contrast sensitivity represent critical evidence for a magnocellular deficit in dyslexia.

Two recent critical reviews of studies of contrast sensitivity in dyslexia (Skottun, 2000; Stuart, McAnally, & Castles, 2001) have raised questions about the strength of the evidence for a magnocellular deficit in dyslexia. Skottun (2000) pointed out although some studies have demonstrated significant differences in contrast sensitivity between dyslexic and control groups, these differences were not always consistent with theoretical predictions. Stuart et al. (2001) re-examined those studies that Skottun (2000) considered to be consistent with the magnocellular deficit theory, and concluded there was more evidence for reduced sensitivity across all spatial and temporal frequencies tested, rather than the specific deficits expected from magnocellular dysfunction. This pattern of results is consistent with general difficulties in completing the psychophysical task successfully, possibly due to a lack of attention or motivation. The only studies of contrast sensitivity that yielded statistically significant results consistent with a specific magnocellular deficit were those of Lovegrove et al. (1982), Martin and Lovegrove (1984, 1988) and Felmingham and Jakobson (1995).

One possible explanation of the above findings is that the magnocellular system alone is responsible for achromatic contrast sensitivity across a wide range of spatial and temporal sensitivities, thus meaning one would expect to see nonspecific reductions in contrast sensitivity. Despite the common association of sustained and transient psychophysical channels with the parvocellular and magnocellular systems respectively, there is an alternative view that the magnocellular system alone governs achromatic contrast sensitivity. Consistent with this view, Burbeck and Kelly (1980) showed that it is possible to model the contrast sensitivity function using a single type of antagonistic centre-surround receptive field with delays between centre and surround responses. Further, it has been demonstrated that individual cells in the magnocellular pathway show greater contrast sensitivity, and greater contrast gain, than parvocellular cells across a broad range of spatial and temporal frequencies (Kaplan, Lee, & Shapley, 1990).

Studies that have been cited in support of the contention that the magnocellular system has superior contrast sensitivity to that of the parvocellular system were concerned with chromatic and achromatic temporal modulation sensitivity in response to relatively large circular patches of light (Lee, Pokorny, Smith, Martin, & Valberg, 1990; Smith, Pokorny, Davis, & Yeh, 1995). This type of stimulus favours achromatic detection by the magnocellular system. In contrast, Hicks, Lee, and Vidyasagar (1983), using grating stimuli, showed that at very low temporal frequencies, magnocellular neurons failed to respond at any spatial frequency, whereas parvocellular neurons responded vigorously. This indicates that there are some achromatic stimuli that will preferentially stimulate the parvocellular system at threshold.

Perhaps the best evidence for parvocellular system involvement in achromatic contrast detection derives from studies using macaque monkeys with selective magnocellular and parvocellular lesions, and from the study of retinal diseases in humans that selectively affect contrast sensitivity. Lesions of the magnocellular layer of the lateral geniculate nucleus of monkeys resulted in a loss of sensitivity restricted to stimuli with both high temporal and low spatial frequencies (Merigan & Maunsell, 1993). In particular, monkeys with such lesions were virtually blind to a large (approximately 4 deg) Gaussian blob flickering at 10 Hz. Conversely, the visibility of relatively stationary stimuli, even at moderate spatial frequencies, is severely reduced in monkeys with parvocellular lateral geniculate nucleus lesions (Merigan, Katz, & Maunsell, 1991).

In humans, Wolf and Arden (1996) demonstrated that in melanoma-associated retinopathy there was a profound loss of sensitivity to Gaussian blobs across a range of temporal frequencies, with a preservation of sensitivity to 1 c/deg gratings flickering slowly at 0.5 Hz. Selective loss of contrast sensitivity should not have occurred if magnocellular cells alone determined threshold contrast sensitivity. Thus, both single cell physiology and studies of damage to the magnocellular and parvocellular pathways as a whole indicate that it is possible to selectively test the sensitivity of the two pathways using achromatic stimuli at threshold. However, the spatial and temporal characteristics of the stimuli must be chosen carefully. This includes the spatio-temporal envelope within which the stimuli are presented (Peli et al., 1993; Spehar & Zaidi, 1997).

1.2. Subgroups of developmental dyslexia

There is considerable evidence for subgroups of dyslexics, with divergent patterns of reading deficits, and it has also been theorised that different patterns of dyslexia may reflect different aetiologies (Castles & Coltheart, 1993; Manis et al., 1996; Stanovich et al., 1997). Therefore, as Hogben (1996) has suggested, it is possible that some of the inconsistency in the contrast sensitivity studies may stem from a failure to control for sample heterogeneity and differences in the proportion of each subgroup represented in respective samples.

The dual route model of reading aloud (e.g. Coltheart, 1978; Coltheart, Curtis, Atkins, & Haller, 1993; Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001; Morton & Patterson, 1980) has provided the theoretical framework for some recent subgrouping attempts. The model proposes that reading aloud involves two, at least partially independent, procedures. The *lexical* procedure involves retrieving from a mental dictionary (or lexicon), the phonological form appropriate to a particular orthographic stimulus. The sublexical procedure involves the application of grapheme–phoneme correspondence rules in the decoding of print. The integrity of the lexical route is typically investigated via the reading of irregular words, which deviate from grapheme–phoneme correspondence rules (e.g. *yacht*). Sublexical skill, on the other hand, can be investigated via the reading of novel nonwords (e.g. *glop*). Regular word reading can theoretically be achieved by either the lexical or sublexical routes.

In a large group study of reading patterns in developmental dyslexia, Castles and Coltheart (1993) found evidence for dissociations between irregular and nonword reading. One subgroup of children had poor nonword reading and normal range irregular word reading, and were interpreted as having specifically impaired sublexical skills or *phonological* dyslexia. Another subgroup had poor irregular word reading skills and age-appropriate nonword reading skills, and were therefore interpreted as having a specific difficulty reading via the lexical route or *surface* dyslexia. These basic subgroups have since been replicated in two further studies (Manis et al., 1996; Stanovich et al., 1997). However, it should be noted that, while children with discrepant patterns of reading deficit exist, a large population of children has difficulty reading via both lexical and sublexical procedures, and they may be characterised as a *mixed* dyslexia subgroup.

1.3. Contrast sensitivity and subgroups of dyslexia

Some recent research has explored the relationship between dyslexia subgroups and magnocellular functioning using contrast sensitivity tasks (Borsting et al., 1996; Ridder, Borsting, Cooper, McNeel, & Huang, 1997; Slaghuis & Ryan, 1999; Spinelli et al., 1997). Three of these studies (Borsting et al., 1996; Ridder et al., 1997; Slaghuis & Ryan, 1999) subgrouped their dyslexic sample using the typology originally advocated by Boder (1971). Although this typology is not based on an explicit model of reading, the subgroups obtained are likely to be similar to those obtained using the Castles and Coltheart (1993) method. The fourth study (Spinelli et al., 1997) used an Italian measure for identifying "surface" dyslexia in their Italian speaking sample (Sartori, Job, & Tressoldi, 1995).

A consistent finding across these studies is that the surface dyslexics, or dyseidetic dyslexics in the Boder typology, do not differ from controls in performance on contrast sensitivity tasks at low spatial and high temporal frequencies (Borsting et al., 1996; Ridder et al., 1997; Slaghuis & Ryan, 1999; Spinelli et al., 1997), and therefore do not have a magnocellular deficit. The relationship between the other two subgroups and magnocellular function is less clear. Slaghuis and Ryan (1999) analysed contrast sensitivity on a linear scale. When Stuart et al. (2001) replotted this data on a log scale there was little indication of any selective loss of contrast sensitivity in the dyslexia group. In Borsting et al.'s (1996) study of adults, there was some evidence of a loss of sensitivity at low spatial/high temporal frequencies among dysphoneidetic (mixed dyslexic) readers, but a direct test failed to show a significant interaction between reading group and spatial frequency at a temporal frequency of 10 Hz. No pure dysphonetic dyslexic (phonological dyslexic) readers were tested. Ridder et al. (1997) did include three subgroups in their follow-up study. The dysphoneidetic group and some participants with dysphonetic dyslexia displayed a reduction in contrast sensitivity. However, because there was no stimulus in this study designed to measure parvocellular contrast sensitivity, these results are equivocal.

The possibility of contrast sensitivity deficits being specific to phonological dyslexia is consistent with recent theories of the relationship between magnocellular functioning and reading. Stein (1993) has proposed that in dyslexia there is a generalised defect in the processing of rapidly changing sensory stimuli, which occurs across the visual, auditory, and motor domains. Auditory temporal deficits are thought to result in speech perception deficits, which are associated with difficulty manipulating speech sounds, or phonological awareness deficits. There is much evidence that poor sublexical skill is associated with poor phonological awareness (Rack, Snowling, & Olson, 1992; Wagner & Torgesen, 1987). Phonological dyslexia should therefore reflect auditory temporal deficits, and be seen concurrently in individuals with magnocellular visual deficits (Witton et al., 1998).

1.4. The present study

The present study aimed to investigate the relationship between visual magnocellular processing and different patterns of developmental dyslexia. We aimed to build on previous research in two ways. First, we selected our subgroups based on the Castles and Coltheart (1993) methodology, which arguably produces purer subtypes because it is based on an explicit model of component processes in reading. Second, bearing in mind the issues raised above, we used visual stimuli which were more carefully controlled than in some previous studies with subgroups of dyslexia and which unequivocally reflect both magnocellular and parvocellular function. Specifically, we precisely varied the spatial and temporal characteristics of achromatic stimuli presented at threshold.

A control group and three subgroups of dyslexics were selected: phonological dyslexics, surface dyslexics and a group proposed to have both lexical and sublexical deficits (mixed dyslexics). Without subscribing to the idea of strictly discrete subgroups, this sampling strategy ensured some dissociation between irregular and nonword reading skills in the dyslexia sample.

2. Methods

2.1. Participants

The participants included in the study were 20 dyslexics and 23 controls between the ages of 8 years and 12 years. Characteristics of the sample are presented in Table 1. Dyslexics were recruited from a learning difficulties clinic, and consisted of 12 males and 8 females. Twenty-one dyslexics were initially included in the study, however, one of these participants was identified as an influential outlier on both visual tasks and therefore not included in the final sample. All of these children had completed full neuropsychological assessments at the clinic. Results of the Wechsler Intelligence Scale for Children-Third Edition (WISC-III) (Wechsler, 1991) were available on file. Reading age was reviewed at the time of the study experiment using the Word Identification subtest of the Woodcock Reading Mastery Test-Revised (WRMT-R) (Woodcock, 1987).

The dyslexia sample were selected based on the following criteria: (1) a history of reading difficulties, (2) reading age delay of at least 18 months on the WRMT-R (Woodcock, 1987), (3) Full Scale Intelligence Quotient score of at least 85 on the WISC-III (Wechsler, 1991), (4) no known neurological deficit, (5) no known visual acuity or auditory acuity impairment, (6) no known psychiatric disorder. [1] Two of the dyslexics had a reading lag of between 12 and 18 months, but were included in the sample as their lag on initial neuropsychological assessment had been greater than 18 months, they had a strong history of reading difficulties, and they met the criteria for a specific subtype of dyslexia.

Classification of the children as meeting general criteria for dyslexia was further confirmed by the calculation of discrepancy scores between reading achievement and intelligence. These scores were calculated by subtracting the Word Identification subtest standard score (Mean = 100, SD = 15), from the Full Scale Intelligence Quotient score as measured by the WISC-III. Seventeen of the 20 dyslexics had a discrepancy of greater than one standard deviation (15 points), the standard cut-off point for diagnosis. Three dyslexics had discrepancies less than 15 points. Two of these children met the criteria for phonological dyslexia, while the other met the criteria for surface dyslexia. Pure subtypes such as these may be expected to perform somewhat better than mixed dyslexics on general reading measures, due to the preserved integrity of one reading route, and thus may not show significant discrepancies between broad reading and intelligence measures. These children were included in the final sample as they met the subgrouping criteria described below.

The control sample was recruited from a private primary school in an outer eastern suburb of Melbourne, Australia, and consisted of 17 males and 6 females. The sample had good or corrected vision, as indicated by visual acuity tests (Random Es) for near vision and also at 6 m. Most of the dyslexia sample had undergone full optometric testing, and wore their spectacles if prescribed. Full Scale Intelligence was prorated based on the Vocabulary and Block Design subtests of the WISC-III for the control sample. All control children were reading within or above the expected range for their age, as measured by their reading age on the Word Identification subtest of the WRMT-R.

2.2. Subgrouping criteria

The sample was subgrouped based on norms for reading 30 regular words, 30 irregular words and 30 nonwords from Coltheart and Leahy (1996). Normal reading performance for either regular, irregular or nonword reading was determined to be a reading score greater than, or equal to, the minimum normal score for their age. An abnormal performance was indicated by a reading score at least two standard deviations below the mean for their age.

The following criteria were utilised: Phonological dyslexics had normal irregular word reading scores and a nonword reading score at least two standard deviations below the mean for their age. Conversely, the surface dyslexics had a normal nonword reading score and deficient irregular word reading scores. Mixed

[1] With the exception of Attention Deficit Hyperactivity Disorder.

Table 1
Sample characteristics

	Control ($N = 23$)	Dyslexic ($N = 20$)	t-Test (df = 41)	P
Age				
Mean	10 years, 8 months	10 years, 7 months	0.16	$p > 0.05$
Range	8 years 6 months to 12 years 8 months	8 years 6 months to 12 years 9 months		
WISC-III full scale IQ				
Mean (SD)	110 (16)	104 (11)	1.54	$p > 0.05$
WRMT-R (months)				
Mean (SD)	141 (27)	98 (10)	6.70	$p < 0.001$
Mean reading delay (months)				
Mean (SD)	−15 (22)	29 (11)	8.03	$p < 0.001$
Modified Castles list accuracy (/30) (Coltheart & Leahy, 1996)				
Regular words	29.2 (0.9)	20.7 (6.1)	6.62	$p < 0.001$
Irregular words	23.7 (2.6)	14.7 (5.3)	7.28	$p < 0.001$
Nonwords	26.0 (3.1)	12.3 (6.3)	9.28	$p < 0.001$

dyslexics had irregular word and nonword reading scores at least two standard deviations below the mean for their age. Of the 20 dyslexics, eight participants met the criteria for phonological dyslexia, four met the criteria for surface dyslexia, and eight met the criteria for mixed dyslexia. All controls had reading scores within the normal range for their age.

Sample characteristics for the controls and subgroups of dyslexics are presented in Table 2. A one-way analysis of variance revealed a significant between group difference in reading age, $F(3, 39) = 15.88$, $p < 0.001$. Tukey's HSD post hoc analysis revealed that the controls' reading age was significantly superior to that of the dyslexic groups. There were no significant differences in reading age between the subgroups.

One-way analyses of variance were carried out to examine between-group differences in regular, irregular and nonword reading scores. A significant difference in regular word reading scores was found, $F(3, 39) = 41.38$, $p < 0.001$. Tukey's HSD post hoc analysis revealed that the controls' regular word reading accuracy was significantly greater than that of the dyslexics as a group. Within the dyslexic groups, the phonological and surface dyslexics' regular word scores were higher than those of the mixed dyslexics. The poor regular word reading skill of the mixed dyslexics is likely to reflect the cumulative effect of poor lexical and sublexical skills. A significant between-group difference in irregular word reading scores was also found with the controls' scores being significantly higher than those of the dyslexic group, $F(3, 39) = 46.46$, $p < 0.001$. Within the dyslexic groups, the mixed and surface dyslexics were equally poor in their ability to read irregular words and were significantly worse than the phonological dyslexics. Finally, the expected group differences in nonword reading were also found, with the control group obtaining significantly higher scores than the dyslexic group, $F(3, 39) = 58.3051$, $p < 0.001$. Within the dyslexic

Table 2
Sample characteristics of the subgroups of dyslexics and controls

	Control ($N = 23$)	Phonological dyslexics ($N = 8$)	Surface dyslexics ($N = 4$)	Mixed dyslexics ($N = 8$)
Age				
Mean (SD)	10 years, 8 months	10 years, 7 months	10 years, 7 months	10 years, 7 months
Range	8 years 6 months to 12 years 8 months	8 years, 9 months to 11 years, 11 months	8 years, 7 months to 11 years, 10 months	9 years, 3 months to 12 years, 9 months
WISC-III full scale IQ				
Mean (SD)	110 (16)	106 (12)	104 (10)	101 (11)
WRMT-R (months)				
Mean (SD)	141 (27)	104 (8)	103 (12)	89 (4)
Mean reading delay (months)				
Mean (SD)	−15 (22)	23 (3)	24 (8)	38 (13)
Modified Castles list accuracy (/30) (Coltheart & Leahy, 1996)				
Regular words	29.2 (0.9)	24.3 (3.5)	24.0 (2.9)	15.4 (5.8)
Irregular words	23.7 (2.6)	19.5 (4.0)	13.8 (2.2)	10.4 (3.2)
Nonwords	26.0 (3.1)	13.0 (4.4)	20.0 (3.7)	7.6 (4.7)

groups, significant differences between the nonword reading accuracy of each group were seen, with the surface dyslexic scores being highest, followed by the phonological dyslexics, and the weakest performance demonstrated by the mixed dyslexic group. These results serve to validate the subgrouping of the participants.

2.3. Psychophysical materials and procedure

The visual stimuli were generated using an IBM compatible computer with a VSG 2/3 (Cambridge Research Systems) high-resolution graphics card and a Sony high-resolution monitor, with a vertical refresh rate of 100 Hz. A large white card (41.6 deg by 28.6 deg) with a central disk removed (8.6 deg) surrounded the monitor, and was front illuminated by two lamps equipped with daylight filters to 12 cd/m^2. The average luminance of the stimulus display field was also 12 cd/m^2. Luminance levels were measured using a Tektronix Lumacolor luminance meter with a J18 monitor sensor. The luminance-output relationship was calibrated regularly. Contrast was defined as Michelson contrast: $(I_{\max} - I_{\min})/(I_{\max} + I_{\min})$.

The visual stimulus presented as a measure of transient/magnocellular visual function was a Gaussian blob (SD = 1.17°), which is an unpatterned stimulus with a very low spatial frequency content. It flickered sinusoidally at 8.33 Hz for 1 s. This stimulus will be described for the remainder of this paper as the flicker sensitivity task. The stimulus used as a measure of sustained/parvocellular visual functioning was a moderately high spatial frequency (8 c/deg) vertically oriented Gabor patch the same size as the Gaussian blob. This was presented for one second with additional 500 ms on- and off-ramps with linear temporal profiles. The task will be described as the static sensitivity task.

Thresholds for the visual stimuli were determined using a modified 3 down, 1 up two-alternative forced choice staircase procedure (Badcock & Sevdalis, 1987; Wetherill & Levitt, 1965). The staircase procedure proceeded until eight reversals were obtained and the average of the last four reversals was used as the threshold. This staircase converges to the 79% correct threshold. Each trial consisted of two intervals, each of a duration of 2 s, paired with a tone. The first interval was paired with a 2500-Hz tone, which was presented for a duration of 50 ms. The second interval was paired with a 400-Hz tone presented for 50 ms. The stimulus was presented either during the first or second interval. The interval not containing the stimulus consisted of a blank field of 12 cd/m^2. The stimulus was presented for a duration of 1 s within the 2 s interval, padded by either 500 ms temporal ramps (8 c/deg Gabor patch) or blank intervals (8.33-Hz flickering Gaussian blob). The participant verbally indicated within which of two intervals the stimulus was presented by stating "one" or "two". The experimenter then entered the response by pressing one of two buttons.

Participants were seated at a distance of 1 m from the monitor in an otherwise darkened room. They rested their chin on a rest, and were instructed to focus their gaze on a small dot positioned at the centre of the monitor. The order of administration of the two tasks was counterbalanced across and within all subgroups. The two threshold tasks took approximately 10 min each to complete.

3. Results

3.1. Contrast sensitivity tasks

Contrast thresholds were converted to contrast sensitivity scores ($\log_{10}$ 1/threshold) for all statistical analyses. As previously described, one dyslexic participant who met criteria for mixed dyslexia was identified as an influential outlier and was excluded from the final sample. Order of administration made a significant contribution to variance in threshold sensitivity, as shown in Fig. 1. Mixed-effect analysis of variance showed that, overall, there was a highly significant effect of order of administration, $F(1, 82) = 12.737$, $p = <0.001$. There was also, as expected, a difference between thresholds to the flickering Gaussian and stationary Gabor stimuli, $F(1, 82) = 10.854$, $p = <0.001$. However, there was no interaction between order of administration and the type of stimulus, $F(1, 82) = 0.221$, $p > 0.05$. This meant that the thresholds could be ad-

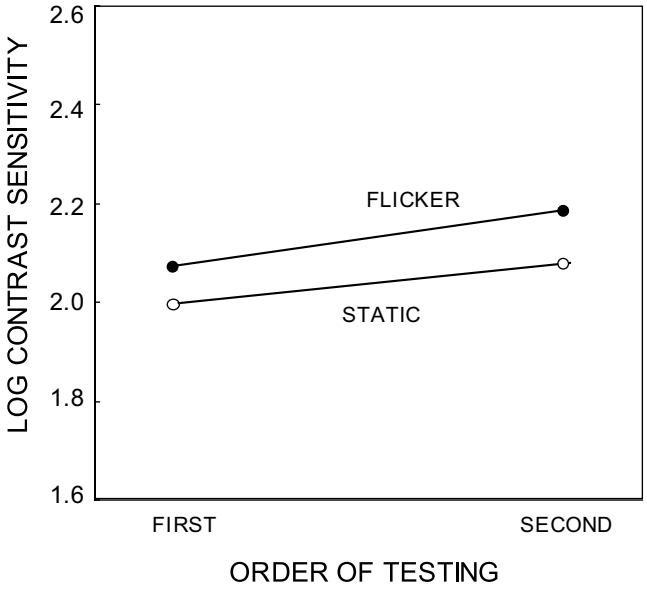

Fig. 1. Effect of order of administration on measured sensitivity to the 8.33-Hz flickering Gaussian and static 8 c/deg Gabor patch stimuli for the combined sample of children with dyslexia and controls.

justed to remove the main effect of order of administration for the purpose of subsequent analysis.

Individual order-corrected thresholds for the flickering Gaussian blob and the static Gabor stimulus are shown in Fig. 2, broken down by group (controls, surface dyslexia, phonological dyslexia and mixed dyslexia). The most striking feature of these scatterplots is that the thresholds for the dyslexic observers fall within the range of the normal controls. This is true of both sets of thresholds.

A 2×4 ANOVA was conducted, using the two thresholds for each participant as a repeated measures factor, and the four reading groups as a between-groups factor. The mean thresholds are given in Table 3. There was no significant main effect of group on contrast threshold, $F(3, 39) = 0.654$, $p > 0.05$. Although there was a significant difference in threshold sensitivity to the flickering and static stimuli, $F(1, 39) = 8.177$, $p = <0.01$, there was no interaction between this factor and sample group, $F(1, 82) = 0.221$, $p > 0.05$. This finding was not due to a lack of power to detect differences in contrast sensitivity between the control and dyslexia groups. When the dyslexia subgroups were combined, the mean log contrast flicker sensitivity of the two groups was the same to three decimal places, a value of 2.147. The standard deviation in log contrast sensitivity was 0.150, varying only slightly between the groups. This meant that it would have been possible to detect a reduction in contrast sensitivity in the dyslexic group down to 1.975 using a two-tailed test, or 1.990 using a one tailed test, at an alpha level of 0.05 with 95% statistical power. This represents a clinically insignificant difference, a reduction in threshold contrast from 0.71% to 1.02%, which is well within the normal range. The diagnostic significance of such a reduction would be very poor.

Table 3
Means and standard deviations (in brackets) of the subgroups and controls for the flicker and static contrast sensitivity tasks

	Flicker sensitivity	Static sensitivity
Control	2.15 (0.15)	2.04 (0.16)
Phonological	2.17 (0.15)	2.01 (0.18)
Surface	2.17 (0.09)	1.93 (0.19)
Mixed	2.15 (0.15)	2.07 (0.18)

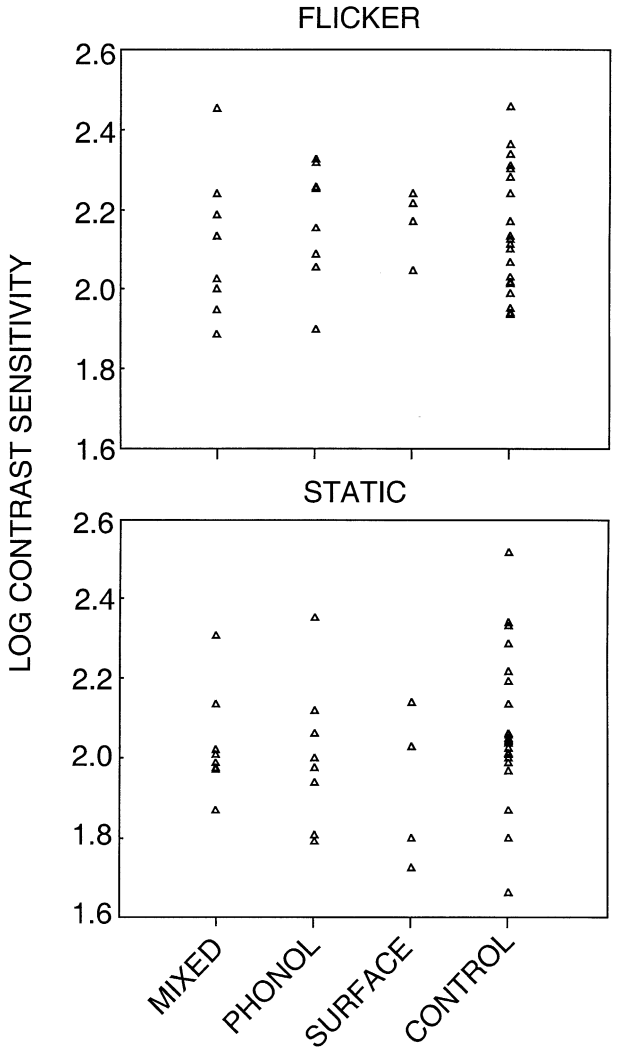

Fig. 2. Individual threshold sensitivities to an 8.33-Hz flickering Gaussian blob (top) and a static 8 c/deg Gabor patch (bottom), broken down by reading group. Thresholds are corrected for the effects of order of administration.

Further analyses were carried out by collapsing the groups of dyslexics and controls and using the word/nonword reading scores to directly investigate the relationship between component reading skills and sensitivity to the flickering Gaussian blob used to measure transient/magnocellular system sensitivity.

3.2. The relationship between component reading skills and contrast sensitivity

Multiple regression analysis was used to determine whether component reading skills could be used to predict thresholds to the flickering Gaussian blob. This allows the within-group variance in reading skills within the subgroups to be used in the analysis, as the boundaries between the groups may be somewhat arbitrary. The effect of age on the independent variables was controlled for by converting the regular word, irregular word and nonword reading scores into age adjusted scores and using these scores in the regression analyses. Raw reading scores were age adjusted using the norms of Edwards and Hogben (1999) by equating the minimum raw score (0/30), the maximum raw score (30/30) and the 10, 50, and 90th percentiles to those of the average of the 9 and 10 year olds and interpolating between these values.

A standard multiple regression analysis was performed with order-corrected flicker sensitivity as the dependent variable. Age-adjusted regular word, irregular word and nonword accuracy were used as predictor variables. Three covariates were also included in the model; order-corrected static contrast sensitivity, full scale IQ, and age in months. The multiple R^2 was 0.209, and was not significant, $F(5, 37) = 1.949$, $p > 0.05$. The adjusted R^2 was only 0.077, reflecting the limited sample size relative to the number of predictors. Inspection of the significance of individual beta coefficients within the multiple regression analysis showed that the threshold for the static stimulus was negatively related to the threshold for the flickering stimulus, beta $= -0.316$, $t = -2.028$, $p = 0.050$. This result was nearly significant in a univariate regression using the static contrast as the sole predictor, beta $= -0.280$, $t = -1.900$, df $= 41$, $p = 0.064$. However, inspection of scatterplots indicated that this result depended on a few individual observations, and so was unlikely to be generalized beyond the sample. The only other trend in the multiple regression model was a near-significant beta coefficient for regular word reading ability, beta $= 0.727$, $t = 1.935$, $p = 0.061$, but only when the other reading scores were in the model. The univariate regression between regular word reading and flicker sensitivity was not significant, beta $= 0.178$, $t = 1.155$, $p > 0.05$. Although this beta coefficient borders on significance in the multiple regression, the effect is probably an overestimate, given that no adjustment has been made for overfitting, nor for multiple inference within the overall model. No other combination of reading scores and covariates yielded significant beta values.

4. Discussion

This experiment demonstrated no significant differences between dyslexics and controls, or subgroups of dyslexics and controls, in their contrast sensitivity to flicker or static pattern. The single case data revealed that the range of scores for the dyslexics on the contrast sensitivity tasks closely paralleled that of the controls, and fell within a narrow band. The finding that dyslexics and controls did not differ in their performance on the flicker task is consistent with the results of a number of other studies of flicker contrast sensitivity in dyslexia (e.g. Cornelissen, Richardson, Mason, Fowler, & Stein, 1995, experiment 2; Demb, Boynton, Best, & Heeger, 1998; Hayduck, Bruck, & Cavanagh, 1995, experiment 1; Hill & Lovegrove, 1993, experiment 2; Walther-Muller, 1995, experiment 2). Cornelissen et al. (1995) suggested that their failure to demonstrate reduced flicker contrast sensitivity in dyslexia was due to the use of high base luminance levels. However, we failed to demonstrate an effect at lower luminance levels similar to those used in most other studies.

It has been suggested that null findings in studies of visual temporal processing can be attributed to methodological flaws (Martin, 1995) or sample heterogeneity (Hogben, 1996). The stimuli used in the present study were carefully chosen based on stimuli that show elevated thresholds following lesions of the LGN in monkeys. They were also close to the peak sensitivities of the two psychophysical mechanisms identified by Koenderink and van Doorn (1979). The tasks should therefore have had the potential to detect visual deficits if they were present. The absence of transient system deficits in dyslexia using these measures suggests that this sample of children with dyslexia did not have specific damage to the magnocellular system.

Stuart et al. (2001) have recently reviewed a number of contrast sensitivity studies reporting a magnocellular deficit in dyslexia. The review concluded that most of them did not meet the statistical criteria for an interaction between sample group (dyslexia vs control) and spatial/temporal frequency. Skottun (2000) has pointed out that some studies have produced significant interactions that are not consistent with theoretical predictions. Only Lovegrove et al. (1982), Martin and Lovegrove (1984, 1988) and Felmingham and Jakobson (1995) have produced significant findings that are definitely consistent with a magnocellular deficit. It is notable that the reading delays of the samples used in Lovegrove et al. (1982) and Martin and Lovegrove (1984, 1988) were severe (from 4 to 5 years on average). This far exceeds the level required to meet the diagnostic criteria for dyslexia. This may be why a large number of studies, including the present one, have failed to find similar visual deficits in more typical samples.

The second issue, raised by Hogben (1996), concerns sample heterogeneity. The subgrouping of our sample ensured that this source of heterogeneity was taken into account. One limitation was that, along with other studies (Borsting et al., 1996; Ridder et al., 1997; Slaghuis & Ryan, 1999) that used Boder's (1971) approach to subgrouping dyslexia, we found it difficult to find children who could be characterised as surface or dyseidetic dyslexics. Fortunately, despite the small numbers, all studies agree that this subgroup displays no contrast sensitivity deficits. However, all the studies using Boder's (1971) typology claimed that the dysphoneidetic subgroup showed a magnocellular processing deficit. This could possibly be explained by differences in the subtyping schemes. However, as outlined earlier, close inspection of the results of Borsting et al. (1996) and Ridder et al. (1997) and Slaghuis and Ryan (1999) showed that there were no statistically significant interactions between any reading group and contrast sensitivity at different spatial frequencies (see Stuart et al., 2001). Thus, no study has demonstrated specific contrast

sensitivity deficits in any subgroup of dyslexia that might indicate a magnocellular deficit.

Cestnick and Coltheart (1999) is the only other study to have examined magnocellular deficits in dyslexia using the Castles and Coltheart (1993) subtyping scheme. Here, an apparent motion task, the Ternus task, was used to indirectly measure magnocellular function. The authors found support for a magnocellular impairment in their dyslexic sample, which was then shown to be restricted to the phonological dyslexic subtype. However, we have argued elsewhere that Cestnick and Coltheart's (1999) results are more consistent with a general performance deficit in the dyslexics concerned, because although the slope of the average psychometric function for the dyslexic participants was shallow, the point of subjective equality was almost the same as that of the control group (Davis, Castles, McAnally, & Gray, 2001). In addition, the Ternus task may not be an unambiguous measure of magnocellular function, because it reflects a trade-off between spatial and temporal grouping (Kramer & Yantis, 1997).

Habib (2000) has stated that "the best demonstration of a low-level visual deficit in dyslexia is that of altered contrast sensitivity". The results of the present study, together with a critical examination of previous research, suggest that there is very limited evidence for deficits of contrast sensitivity in representative samples of dyslexics. At best, such deficits may be characteristic of only the most severely affected individuals. Our results present difficulties for those theories that assume that there is damage to the magnocellular layers of the lateral geniculate nucleus, with similar deficits in other modalities (Stein, 1993). However, there are two alternative approaches to the question of visual deficits in dyslexia that can, and have, been pursued. One possibility is that although magnocellular and parvocellular pathways are intact, there is an abnormal interaction between them, such as a failure of mutual inhibition. This theory has been invoked to explain abnormalities on tasks such as visual masking (Slaghuis & Pinkus, 1993). Another possibility is that there is dysfunction of higher visual areas that receive dominant (although not unique) projections from the magnocellular system, even though the more peripheral parts of the system may be functioning normally. In this case, visual functions such as the ability to perceive global dot motion in noise might be affected (Cornelissen et al., 1995; Slaghuis & Ryan, 1999; Talcott, Hansen, Assoku, & Stein, 2000).

A difficulty for all psychophysical research with dyslexic participants is the choice of control task, given that there are many reasons why individuals with dyslexia may under-perform compared with normal readers (Stuart et al., 2001). Recently, Hansen, Stein, Orde, Winter, and Talcott (2001) made a clever attempt to design static form perception tasks as controls for a global-dot motion perception task. They found specific impairment in the ability of dyslexic observers to detect global motion. However, this was interpreted as a deficit in one of two high level processing streams, rather than an early sensory deficit. A more suitable control task for early deficits might be to measure the relative contributions of color and luminance in a global motion task, as in the study of Edwards and Badcock (1996). This task relies not on the insensitivity of the early magnocellular pathway to colour contrast, but on the existence of a parallel projection from the colour sensitive parvocellular system to higher-level motion sensitive areas of the cortex. Until such experiments are carried out, the question of the exact nature and severity of visual deficits in dyslexia will remain open, as will the question of whether such deficits are associated with specific types of reading impairment.

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References

Badcock, D. R., & Sevdalis, E. (1987). Masking by uniform field flicker: some practical problems. *Perception, 16*, 641–647.

Boder, E. (1971). Developmental dyslexia: prevailing diagnostic concepts and a new diagnostic approach. In H. R. Mykleburst (Ed.), *Progress in learning disabilities* (Vol. II, pp. 293–321). New York: Grune and Stratton.

Borsting, E., Ridder III, W. H., Dudeck, K., Kelley, C., Matsui, L., & Motoyama, J. (1996). The presence of a magnocellular defect depends on the type of dyslexia. *Vision Research, 36*(7), 1047–1053.

Burbeck, C. A., & Kelly, D. H. (1980). Spatiotemporal characteristics of visual mechanisms: excitatory–inhibitory model. *Journal of the Optical Society of America, 70*, 1121–1126.

Castles, A., & Coltheart, M. (1993). Varieties of developmental dyslexia. *Cognition, 47*, 148–180.

Castles, A., Datta, H., Gayan, J., & Olson, R. K. (1999). Varieties of developmental reading disorder: genetic and environmental influences. *Journal of Experimental Child Psychology, 72*(2), 73–94.

Cestnick, L., & Coltheart, M. (1999). The relationship between language-processing and visual-processing deficits in developmental dyslexia. *Cognition, 71*(3), 231–255.

Coltheart, M. (1978). Lexical access in simple reading tasks. In G. Underwood (Ed.), *Strategies of information processing* (pp. 151–216). London: Academic Press.

Coltheart, M., Curtis, B., Atkins, P., & Haller, M. (1993). Models of reading aloud: dual-route and parallel-distributed processing approaches. *Psychological Review, 100*, 589–608.

Coltheart, M., & Leahy, J. (1996). Assessment of lexical and nonlexical reading abilities in children: some normative data. *Australian Journal of Psychology, 48*(3), 136–140.

Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: a dual route cascaded model of visual word recognition and reading aloud. *Psychological Review, 108*(1), 204–256.

Cornelissen, P., Richardson, A., Mason, A., Fowler, S., & Stein, J. (1995). Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Research, 10*, 1483–1494.

Critchley, M. (Ed.). (1964). *Developmental dyslexia*. Springfield: Thomas.

Davis, C., Castles, A., McAnally, K., & Gray, J. (2001). Lapses of concentration and dyslexic performance on the Ternus Task. *Cognition, 81*, B21–B31.

Demb, J., Boynton, G., Best, M., & Heeger, D. (1998). Psychophysical evidence for a magnocellular pathway deficit in dyslexia. *Vision Research, 38*(11), 1555–1559.

Dobkins, K. R., Gunther, K. L., & Peterzell, D. H. (2000). What covariance mechanisms underlie green/red isoluminance, luminance contrast sensitiviity and chormatic (green/red) sensitivity? *Vision Research, 40*, 613–628.

Edwards, M., & Badcock, D. R. (1996). Global-motion perception: Interaction of chromatic and luminance signals. *Vision Research, 36*, 2423–2431.

Edwards, V. T., & Hogben, J. H. (1999). Norms for comparing children's lexical and nonlexical reading: A further look at subtyping dyslexia. *Australian Journal of Psychology, 51*(1), 37–49.

Felmingham, K. L., & Jakobson, L. S. (1995). Visual and visuomotor performance in dyslexic children. *Experimental Brain Research, 106*, 467–474.

Habib, M. (2000). The neurological basis of developmental dyslexia: an overview and working hypothesis. *Brain, 123*, 2373–2399.

Hansen, P. C., Stein, J. F., Orde, S. R., Winter, J. L., & Talcott, J. B. (2001). Are dyslexics' visual deficits limited to measures of dorsal stream function? *NeuroReport, 12*, 1527–1530.

Hayduck, S., Bruck, M., & Cavanagh, P. (1995). Low-level visual processing skills of adults and children with dyslexia. *Cognitive Neuropsychology, 13*(7), 975–1015.

Hicks, T. P., Lee, B. B., & Vidyasagar, T. R. (1983). The responses of cells in macaque lateral geniculate nucleus to sinusoidal gratings. *Journal of Physiology, 337*, 183–200.

Hill, R., & Lovegrove, W. J. (1993). One word at a time: a solution to the visual deficit in SRDs. In S. F. Wright & R. Groner (Eds.), *Facets of dyslexia and its remediation* (pp. 65–76). Amsterdam: Elsevier Science Publishers.

Hogben, J. H. (1996). A plea for purity. *Australian Journal of Psychology, 48*(3), 172–177.

Kaplan, E., Lee, B. B., & Shapley, R. M. (1990). New views of primate retinal function. *Progress in Retinal Research, 9*, 272–336.

Koenderink, J. J., & van Doorn, A. J. (1979). Spatiotemporal contrast detection threshold surface is bimodal. *Optics Letters, 4*(1), 32–34.

Kramer, P., & Yantis, S. (1997). Perceptual grouping in space and time: evidence from the Ternus display. *Perception and Psychophysics, 59*, 87–99.

Lee, B. B., Pokorny, J., Smith, V. C., Martin, P. R., & Valberg, A. (1990). Luminance and contrast modulation sensitivity of macaque ganglion cells and human observers. *Journal of the Optical Society of America A, 7*, 2223–2236.

Livingstone, M., Drislane, F., Rosen, G., & Galaburda, A. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proceedings of the New York Academy of Sciences, 88*, 7943–7947.

Lovegrove, B. (1996). Dyslexia and a transient/magnocellular pathway deficit: the current situation and future directions. *Australian Journal of Psychology, 48*(3), 167–171.

Lovegrove, W., Bowling, A., Badcock, D., & Blackwood, M. (1980). Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. *Science, 210*, 439–440.

Lovegrove, W., Martin, F., Bowling, A., Blackwood, M., Badcock, D., & Paxton, S. (1982). Contrast sensitivity functions and specific reading disability. *Neuropsychologia, 20*(3), 309–315.

Manis, F. R., Seidenberg, M. S., Doi, L. M., McBride-Chang, C., & Petersen, A. (1996). On the bases of two subtypes of developmental dyslexia. *Cognition, 58*, 157–195.

Martin, F., & Lovegrove, W. (1984). The effects of field size and luminance on contrast sensitivity differences between specifically reading disabled and normal children. *Neuropsychologia, 22*(1), 73–77.

Martin, F., & Lovegrove, W. J. (1988). Uniform-field flicker masking in control and specifically-disabled readers. *Perception, 17*, 203–214.

Martin, R. C. (1995). Heterogeneity of deficits in developmental dyslexia and implications for methodology. *Psychonomic Bulletin and Review, 2*(4), 494–500.

Merigan, W. H., Katz, L. M., & Maunsell, J. H. R. (1991). The effects of parvocellular lateral geniculate lesions on the acuity and contrast sensitivity of macaque monkeys. *The Journal of Neuroscience, 11*(4), 994–1001.

Merigan, W. H., & Maunsell, J. H. R. (1993). How parallel are the primate visual pathways? *Annual Review of Neuroscience, 16*, 369–402.

Morton, J., & Patterson, K. E. (1980). A new attempt at an interpretation or an attempt at a new interpretation. In M. Coltheart, K. E. Patterson, & J. C. Marshall (Eds.), *Deep dyslexia* (pp. 91–118). London: Routledge & Kegan Paul.

Peli, E., Arend, L. E., Young, G. M., & Goldstein, R. B. (1993). Contrast sensitvity to patch stimuli: effects of spatial bandwidth and temporal presentation. *Spatial Vision, 7*(1), 1–14.

Rack, J. P., Snowling, M. J., & Olson, R. K. (1992). The nonword reading deficit in developmental dyslexia: a review. *Reading Research Quarterly, 27*(1), 28–53.

Ridder, W. H., Borsting, E., Cooper, M., McNeel, B., & Huang, E. (1997). Not all dyslexics are created equal. *Optometry and Vision Science, 74*(2), 99–104.

Sartori, G., Job, R., & Tressoldi, P. (1995). *Battena per la Valutazione dell Dislessia e della Disorthografia Evolutiva*. Firenze, Organizzazioni Speciali.

Skottun, B. C. (2000). The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. *Vision Research, 40*, 111–127.

Slaghuis, W. L., & Pinkus, S. Z. (1993). Visual backward masking in central and peripheral vision in late-adolescent dyslexics. *Clinical Visual Sciences, 8*, 187–199.

Slaghuis, W. L., & Ryan, J. F. (1999). Spatio-temporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. *Vision Research, 39*, 651–668.

Smith, V. C., Pokorny, J., Davis, M., & Yeh, T. (1995). Mechanisms subserving temporal modulation sensitivity in silent-cone substitution. *Journal of the Optical Society of America A, 12*, 241–249.

Spehar, B., & Zaidi, Q. (1997). Surround effects on the shape of the temporal contrast-sensitivity function. *Journal of the Optical Society of America A, 14*, 2517–2525.

Spinelli, D., Angeletti, P., De Luca, M., Di Pace, E., Judica, A., & Zoccolotti, P. (1997). Developmental surface dyslexia is not associated with deficits in the transient visual system. *NeuroReport, 8*, 1807–1812.

Stanovich, K. E., Siegel, L. S., & Gottardo, A. (1997). Converging evidence for phonological and surface subtypes of reading disability. *Journal of Educational Psychology, 89*(1), 114–127.

Stein, J. (1993). Dyslexia-impaired temporal information processing? *Annals of the New York Academy of Sciences, 682*, 83–86.

Stein, J., & Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. *TINS, 204*, 147–153.

Stuart, G. W., McAnally, K. I., & Castles, A. (2001). Can contrast sensitivity functions in dyslexics be explained by inattention rather than a magnocellular deficit? *Vision Research, 41*, 3205–3211.

Talcott, J. B., Hansen, P. C., Assoku, E. L., & Stein, J. F. (2000). Visual motion perception in dyslexia: evidence for temporal and energy integraton deficits. *Neuropsychologia, 38*, 343–935.

Tolhurst, D. J. (1973). Separate channels for the analysis of the shape and movement of a moving stimulus. *Journal of Physiology, 231*, 385–402.

Wagner, R. K., & Torgesen, J. K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin, 101*(2), 192–212.

Walther-Muller, P. U. (1995). Is there a deficit of early vision in dyslexia? *Perception, 24*, 918–936.

Wechsler, D. A. (1991). *Wechsler intelligence scale for children* (3rd Ed.). Orlando: Psychological Corporation.

Wetherill, G. B., & Levitt, H. (1965). Sequential estimation of points on a psychometric function. *British Journal of Mathematical and Statistical Psychology, 18*(1), 1–10.

Witton, C., Talcott, J., Hansen, P., Richardson, A., Griffiths, T., Rees, A., Stein, J., & Green, G. (1998). Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. *Current Biology, 8*(14), 791–797.

Wolf, J. E., & Arden, G. B. (1996). Selective magnocellular damage in melanoma-associated retionpathy: comparison with congenital stationary nightblindness. *Vision Research, 36*, 2369–2379.

Woodcock, R. W. (1987). *Woodcock Reading Mastery Tests-Revised*. Circle Pines, MN: American Guidance Service.