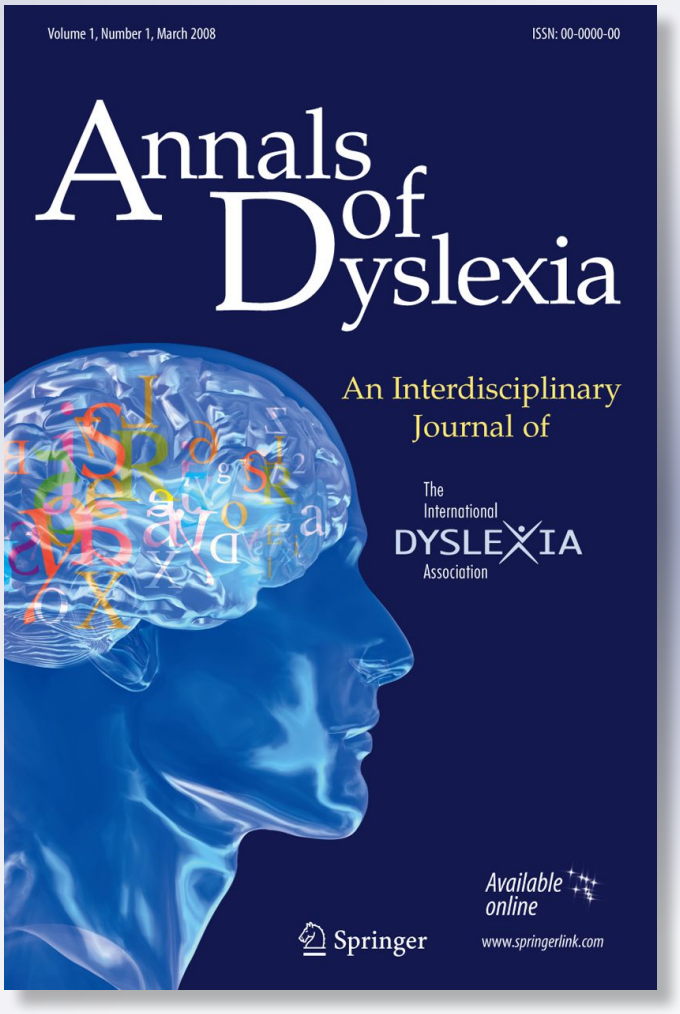
Visual search deficits are independent of magnocellular deficits in dyslexia

Craig M. Wright, Elizabeth G. Conlon & Murray Dyck

Annals of Dyslexia
An Interdisciplinary Journal of The International Dyslexia Association

ISSN 0736-9387

Ann. of Dyslexia
DOI 10.1007/s11881-011-0061-1



Springer

Ann. of Dyslexia
DOI 10.1007/s11881-011-0061-1

Visual search deficits are independent of magnocellular deficits in dyslexia

Craig M. Wright · Elizabeth G. Conlon · Murray Dyck

Received: 23 August 2010 / Accepted: 6 October 2011

Abstract The aim of this study was to investigate the theory that visual magnocellular deficits seen in groups with dyslexia are linked to reading via the mechanisms of visual attention. Visual attention was measured with a serial search task and magnocellular function with a coherent motion task. A large group of children with dyslexia (n=70) had slower serial search times than a control group of typical readers. However, the effect size was small (η_p^2=0.05) indicating considerable overlap between the groups. When the dyslexia sample was split into those with or without a magnocellular deficit, there was no difference in visual search reaction time between either group and controls. The data suggest that magnocellular sensitivity and visual spatial attention weaknesses are independent of one another. They also provide more evidence of heterogeneity in response to psychophysical tasks in groups with dyslexia. Alternative explanations for poor performance on visual attention tasks are proposed along with avenues for future research.

Keywords Dyslexia · Visual attention · Visual search

Dyslexia is a word-reading problem that affects 5–10% of the population (Yule, Rutter, Berger, & Thompson, 1973). Although the notion of dyslexia being a modular deficit in phonology remains (Bonifacci & Snowling, 2008), there is increasing recognition that dyslexia is a heterogeneous disorder associated with multiple cognitive and perceptual weaknesses (Shanahan et al., 2006) that have been explained with a range of theories (see Vellutino, Fletcher, Snowling, & Scanlon, 2004, for review).

One explanation of dyslexia is the magnocellular deficit theory. Reduced sensitivity to stimuli that are processed in the magnocellular and/or dorsal visual stream has been found in groups or individuals with dyslexia compared to typical readers (e.g. Conlon, Sanders, & Wright, 2009; Conlon, Sanders, & Zapart, 2004; Conlon, Wright, Norris, & Chekaluk,

C. M. Wright (✉) · E. G. Conlon · M. Dyck
Behavioural Basis of Health Research Group, Griffith Health Institute, Griffith University, Gold Coast, QLD, Australia
e-mail: c.wright@griffith.edu.au

C. M. Wright
Understanding Minds, Mermaid Beach, QLD, Australia

Published online: 17 November 2011

2011; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Demb, Boynton, Best, & Heeger, 1998; Felmingham & Jakobson, 1995; Kubova, Kuba, Peregrin, & Novakova, 1996; Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Lovegrove, Martin, & Slaghuis, 1986; Slaghuis & Ryan, 2006; Wilmer, Richardson, Chen, & Stein, 2004; Wright & Conlon, 2009). Regardless of the visual location at which this deficit is found, it is commonly described as "magnocellular" (M-deficit; Hansen, Stein, Orde, Winter, & Talcott, 2001; Stein & Walsh, 1997; Vidyasagar, 2004; Wright & Conlon, 2009). A coherent theory linking these physiological weaknesses to cognition and reading behaviour is yet to be established. Explanations including poor capacity of the magnocellular system to inhibit the parvocellular system (Breitmeyer, 1980; Breitmeyer & Ganz, 1976) and poor eye movement control (Stein & Fowler, 1980, 1981, 1985; Stein, Fowler, & Richardson, 2000; Stein, Riddell, & Fowler, 1988) have been proposed. However, these have not proven satisfactory (Goulandris, McIntyre, Snowling, Bethel, & Lee, 1998; Skottun, 2000).

One theory that bears investigation has observed that the M-pathway primarily projects to a ventral visual stream (of which MT/V5 is a part), which in turn projects to the right posterior parietal cortex (PPC), an area that modulates visuospatial attention (Vidyasagar & Pammer, 2010). The theory proposes that impoverished M-inputs to the PPC adversely affect an individual's ability to deploy spatial visual attention. Because visual attention is necessary for visual–orthographic analysis of single words (Friedmann, Kerbel, & Shvimer, 2010), a visual attention deficit could contribute to the single-word reading weaknesses seen in dyslexia (Hari, Valta, & Uutela, 1999; Iles, Walsh, & Richardson, 2000; Steinman, Steinman, & Garzia, 1998; Vidyasagar, 1999, 2004; Vidyasagar & Pammer, 1999).

There is evidence for weaknesses in visual attention in dyslexia. Studies have shown that groups with dyslexia have prolonged attentional dwell time (Buckholz & Aimola Davies, 2007; Hari et al., 1999; Visser, Boden, & Giaschi, 2004), difficulties maintaining and focusing attention (Facoetti et al. 2003; Facoetti, Paganoni, & Lurosso, 2000), in orienting attention on spatial cueing tasks (Buckholz & Aimola Davies, 2005; Facoetti, Turatto, Lorusso, & Mascetti, 2001; Facoetti et al., 2006; Roach & Hogben, 2004, 2007; Sireteanu, Goertz, Bachert, & Wandert, 2005; Steinman et al., 1998; Valdois, Gerard, Vanault, & Dugas, 1995) and in inhibiting irrelevant information from the periphery of the visual field (Bednarek, Saldana, Quintero-Gallego, Garcia, Grabowska et al., 2004; Facoetti et al., 2000). Poorer performance has also been found in groups with dyslexia when compared to controls on visual search tasks (Buchholz & McKone, 2004; Casco, Tessoldi, & Dellantonio, 1998; Iles et al., 2000; Sireteanu et al., 2008; Vidyasagar & Pammer, 1999; Williams, Brannan, & Lartigue, 1987). Furthermore, some studies have found associations between word-level reading skills and performance on visual attention tasks (Facoetti et al., 2006; Valdois, Bosse, & Tainturier, 2004). However, it is unclear whether the visual attention weaknesses are due to M-deficits.

One way of measuring the ability to deploy visual–spatial attention is with serial search tasks. When a target differs from distractors by a conjunction of features, or when the unique feature of the target is not salient, for example when a circle with a small gap in its circumference is presented among many circles, target search requires focused attention to each of the items presented (Triesman, 1988). In serial search tasks, search times are a function of the number of distractors in the display (Triesman & Gelade, 1980). It has been proposed that at any given time when performing serial search, a spotlight of attention focuses on a specific object in the visual field. This spotlight has two basic functions. First, it highlights an object in the visual field, prior to overt eye movements being made to the location of that object. Second, by focusing attention on a single object, it allows the unambiguous binding of features from that object without interference from other objects

(Triesman & Gelade, 1980; Triesman & Sato, 1990). As already noted, the visual attention theory of dyslexia proposes that this attention mechanism is adversely affected by impoverished M-inputs to the right PPC (Vidyasagar, 1999, 2004). Visual search in dyslexia has been investigated. Studies pertinent to the current investigation are reviewed below.

In their study using a serial search task, Vidyasagar and Pammer (1999) had groups of children with (n=11) or without dyslexia (n=9) search for a target defined by a conjunction of two features (a yellow triangle among yellow circles and purple triangles). There were four search conditions involving 10, 24, 36 or 70 items. The group with dyslexia only had slower serial search times than the control group when 70 items were in the display. This finding was interpreted as being the result of impoverished M-inputs to visual attention areas in the PPC. However, as no test of magnocellular function was reported, this conclusion was based on the assumption that the children with dyslexia had an M-deficit. It is therefore unclear whether the slower search time observed in the group with dyslexia can be attributed to an M-deficit, to a general visual attention deficit or to some other cognitive variable associated with task performance.

A second study has tested the hypothesis more systematically in adults (Iles et al., 2000). One group with dyslexia had previously been shown to be impaired on a coherent motion task (to have a motion detection deficit (MD)) and so was reported to have an M-deficit. The other group with dyslexia had normal coherent motion detection thresholds (no motion detection deficit, NMD). The MD group had significantly slower response times than the control group on a number of serial search tasks. In contrast, the NMD group performed significantly more slowly than controls on only one serial search task. The NMD group also had significantly faster response times on all but two tasks compared to the MD group. These results were interpreted to support the hypothesis that an M-deficit can affect an individual's ability to efficiently allocate spatial attention during serial search. However, interpretation of the results is complicated by the observation that error rates for at least four of the eight tasks approached or exceeded chance levels (50% errors) in both groups. Furthermore, as the NMD group had significantly slower response times than the control group on at least one serial search task and because the two dyslexia groups had equivalent search times on two serial search tasks, it is difficult to conclude that poor capacity to allocate spatial visual attention occurs only in the presence of an M-deficit.

Two other studies have also demonstrated groups with dyslexia are less efficient than control groups on serial search tasks (Casco et al., 1998; Williams et al., 1987). However, both studies used alphabet characters as search stimuli. Therefore, interpretation of slower visual search was confounded by poorer reading ability and familiarity with letters in the group with dyslexia.

Using a cue target paradigm, Roach and Hogben (2007) demonstrated that a group of adults with dyslexia had a significantly slower average reaction time than controls in the valid cue condition but not when no cue was presented. These findings indicate that the group with dyslexia was less able than the control group when making a covert shift of attention in response to the cue. The same group with dyslexia was found to perform in a similar fashion to the control group on a flicker contrast sensitivity task (using low spatial and high temporal frequency stimuli known to maximally stimulate the M-pathway) but had significantly poorer sensitivity than the control group on a coherent motion task. These results were interpreted as evidence of poorer capacity of the dyslexia group in attentional processing areas in the PPC and in frontal areas and not as a consequence of a sensory processing deficit in the M-system (Roach & Hogben, 2007).

To summarize, some previous data indicate that groups with dyslexia have an M-deficit and that they are slow to shift visual attention in visual search tasks. However, the hypothesis that poor visual attention is due to an M-deficit (Vidyasagar, 1999) is yet to be fully tested. The current study aims to investigate the premise that visuospatial attention deficits in dyslexia are caused by M-pathway dysfunction by comparing the performance of a group of children with dyslexia who also had a coherent motion detection deficit to a group with dyslexia who had typical coherent motion detection and to normally reading controls. It was hypothesized that poor visual attention, indexed by slow visual search times, would only be seen in the group with dyslexia and an M-deficit.

Method

Participants

One hundred thirty participants with English as a first language were recruited from eight primary schools. There were 75 children with dyslexia (M=8.5 years; SD=1.4 years; 45 male) and 55 children who were normal readers (M=8.5 years; SD=1.25 years; 34 male). Children were included in the sample if they had a standardized score of 90 or above on the Colored Progressive Matrices (Raven, Court, & Raven, 1995), a standardized measure of intellectual ability. Twelve of the children with dyslexia were found to have a consistent weakness on a global motion coherence task. These children were used to form a motion deficit group that was carefully matched with 12 control children and 12 children from the dyslexia group who did not have a motion processing weakness. The characteristics of these groups are shown in Table 1.

Reading skills were assessed using the Basic Reading Cluster (BRC) of the Woodcock Diagnostic Reading Battery (WDRB; Woodcock, 1997). The BRC is derived from the participant's scores on the Word Identification and Word Attack subtests. The rationale behind using single word-reading and word-decoding measures rather than text reading accuracy was that these measures provided a context-free measure of the word-level skills that are accepted to be the most basic and ubiquitous cause of dyslexia (Vellutino et al., 2004). Given that the word-reading deficits in dyslexia are dimensional rather than categorical (Shaywitz, Escobar, Shaywitz, Fletcher, & Makugh, 1992), it was necessary to adopt criteria to define dyslexia. Where to place the cutoff point is a methodological problem for all studies. In the current study, a participant scoring at or below the 15th percentile (more than 1 standard deviation below the population mean) on the BRC was included in the group with dyslexia. Participants included in the control group had to score at or above the 40th percentile on the BRC. The use of these criteria has been suggested by Snowling (2000) who regards word-level skills at or below the 15th percentile on the BRC in otherwise typical children as a good indicator of the anomalous deficits in word-decoding that are the hallmark of dyslexia. These criteria have been adopted in a number of studies (Vellutino et al., 1996; Wright & Conlon, 2009).

No child had a history of (a) recurrent ear infections, (b) severe hearing problems and uncorrected vision problems, (c) severe emotional problems, (d) a diagnosis of attention deficit hyperactivity disorder or (e) a diagnosed developmental disorder (e.g. autism spectrum disorder). Evidence of the presence of each of these disorders was obtained from the students' school records. All had normal or corrected to normal vision.

The data from five children with dyslexia and three controls were subsequently removed from the sample because of excessive motor activity and inattention observed during

Table 1 Cognitive and reading scores including means and 95% confidence intervals for dyslexia and control groups and for the motion deficit, non-motion deficit and matched control groups

	Overall sample		Sub-groups (n=12 per group)		
	Control (n=52)	Dyslexia (n=70)	Dyslexia: MD	Dyslexia: NMD	Control
Age (years)	8.58	8.6	7.3	7.3	7.6
Range	6.1–10.2	6.0–11.1	6.0–10.1	6.0–10.5	6.1–10.2
IQ[a]	106.93 (104–109)	104.54 (103–106)	100.2 (96–104)	99.3 (95–103)	105.2 (101–110)
WI[b]	106 (103.5–107.3)	80.3 (78.3–81.3)	64.6 (54.9–74.3)	66.3 (55.2–77.3)	106.2 (93.7–118.7)
NW[c]	110.6 (108–114)	83 (81.7–83.9)	76.9 (69.7–84)	80.2 (74.5–85.9)	100 (94.4–105.5)
BRC[d]	108.4 (106.2–110)	81.8 (81.2–82.3)	74.3 (67.6–80.9)	75.1 (69.7–80.3)	100 (95.6–104.3)
PA[e]	31.5 (30.3–32.7)	25.3 (24.1–26.5)	17.1 (15.3–18.8)	18.2 (17–19.4)	19.8 (18.8–20.8)
Ortho[f]	60.4 (57.3–63.5)	50.7 (48.6–52.7)	44.5 (40.2–48.9)	44.8 (41.6–48)	53.5 (45.9–61)
NVPS[g]	31.9 (22.9–33.9)	28.6 (26.8–30.3)	23.3 (19.4–27.2)	22.8 (17.9–27.7)	28.4 (23.7–33.1)
RAN[h]	28.8 (25.5–30.6)	42.6 (38.4–46.8)	46.3 (38.1–54.5)	53.3 (44.6–66.0)	30.6 (24.4–36.9)

The range of scores is included for age

[a] Standard score on Coloured Progressive Matrices

[b] WDRB word identification standard score

[c] WDRB nonword standard score

[d] WDRB Basic Reading Cluster standard score

[e] Phonological awareness composite measure raw score

[f] Word–pseudohomophone task raw score

[g] Nonverbal processing speed raw score in seconds

[h] DST rapid automatic naming raw score in seconds

testing. The final sample consisted of 70 children with dyslexia and 52 controls. The study had approval from the University Human Ethics Committee with parents of all selected children providing written informed consent for their child to participate in the study.

Materials and stimuli

Psychometric and reading tests

The Colored Progressive Matrices (Raven et al., 1995) were used as a measure of intellectual ability. Word and nonword reading skills were measured using the WDRB (Woodcock, 1997).

Orthographic skill

The word–pseudohomophone task (Olson, Fosberg, Wise, & Rack, 1994) was used to measure orthographic skill. Stimuli were generated by the V-Scope software package (Enns & Rensink, 1992) on a Power Macintosh with a standard monitor. Two words were presented side by side in 28-point Arial font. One was a high-frequency word (e.g. take), and the other was a nonsense word with identical phonological output (e.g. taik). A block of ten practice trials was conducted prior to presentation of 80 test items that were presented in four blocks of 20 trials. Feedback was given after each practice trial. On each trial, participants were instructed to point to the word from each pair that was correctly spelled. Split-half reliability for this task is 0.93 (Olson et al., 1994).

Phonological awareness

The Phoneme Segmentation subtest from the Dyslexia Screening Test (Fawcett & Nicolson, 1996) was used as a measure of phoneme elision ability (maximum score=15; r=0.88 for test–retest). Syllable blending, phoneme blending, rhyme oddity awareness and phoneme segmentation abilities were assessed using selected subtests of the Sound Linkage Test of Phonological Awareness (Hatcher, 2000; maximum=24; r=0.94 for internal consistency). Scores from the two tests were summed to form a phonological awareness composite measure.

Processing speed

Rapid automatic naming The rapid automatic naming (RAN) subtest from the Dyslexia Screening Test (Fawcett & Nicolson, 1996) required participants to produce the names of 20 familiar objects presented as two-dimensional drawings on a card. The test provides the opportunity for the participant to make him/herself familiar with an untimed practice trial using identical stimuli to the test stimuli. Participants were asked to name the test stimuli as soon as possible following presentation without making mistakes. Performance was taken as the time in seconds to correctly name the entire test stimuli (r=0.85 for test–retest reliability).

Visual matching The visual matching task from the WDRB (Woodcock, 1997) was selected as a contrast to RAN as it involved fewer linguistic demands. Participants were required to circle two identical numbers in a line of five numbers. It is similar in psychomotor, attention and decision demands to other processing tasks such as coding and symbol search.

Performance was taken as the number of items answered correctly in 120 s. This task will be referred to as non-verbal processing speed.

Visual–motor response time

This task measured simple motor reaction time in response to the presence of a visual symbol on a computer screen. This task was included because, although research has shown that groups with dyslexia are unimpaired on simple reaction time (Nicolson & Fawcett, 1994), the serial search task requires a motor response once a search target has been acquired, and it is important to rule out psychomotor response speed as a factor.

Visual symbols were black circles subtending a visual angle of 2° at 57 cm. A single circle could appear at any of eight random points on the screen. Stimuli were generated and randomized by the V-Scope software package (Enns & Rensink, 1992) and were administered on a Macintosh Power Mac with standard computer monitor. Stimuli were presented on a grey background with space-averaged luminance held constant at 15 cd/m^2.

Participants were instructed to respond as soon as possible to the appearance of any visual symbol appearing on the screen by pressing the space bar. A block of five practice trials was given prior to the test trials to make participants familiar with the task. A block of ten experimental trials was then administered. Simple motor reaction time was defined as the mean of the response times for the ten trials.

Serial search

Targets were black circles and the distractors were circles with a gap located at randomly designated points on each circle's circumference. At a viewing distance of 57 cm, each circle subtended a visual angle of 0.5° with the gap for distractors subtending a visual angle of 0.15°. The target, present for half the trials, appeared randomly within the array. In any one trial, 4, 8, 16, or 32 items were presented. All displays subtended a visual angle of 12° vertically and 14° horizontally. Stimuli were generated by the V-Scope software package (Enns & Rensink, 1992) and were administered on a Macintosh Power Mac with standard computer monitor. Stimuli were presented on a grey background with space-averaged luminance held constant at 15 cd/m^2.

Participants were instructed to respond as quickly as possible to the presence or absence of the target without error. If the target was present, participants were instructed to press a key marked "P" or a key marked "A" if the target was absent. Following a response, the stimulus was removed from the screen and response accuracy feedback in the form of a plus (correct) or a minus (incorrect) sign was displayed. A block of 20 practice trials was administered and experimental trials consisted of four blocks of 16 trials with eight trials per condition. Mean correct response times and accuracy data were obtained.

Global motion coherence

Thresholds for detecting coherent motion were measured using the double panel task developed by Hansen et al. (2001). Stimuli were displayed on a laptop PC with a 15-in. LCD screen. The frame rate of the monitor was 13.3 ms (screen refresh 75 Hz). In the motion task two panels, each with 300 high luminance (130 cd/m^2), white dots (one pixel) were presented on a low luminance (0.98 cd/m^2) background. From a viewing distance of 57 cm, each of the panels subtended 10×14° of visual angle. These were separated by a

dark stripe subtending 5° of visual angle (see Fig. 1). One panel contained a variable percentage of signal dots that moved coherently with an angular velocity of 7.0°/s. During a single trial, the direction of motion of the signal dots, left or right, was reversed every 572 ms. The second panel contained noise elements only. A single animation frame was 26.6 ms with each signal dot having a lifetime of three animation frames (79.8 ms), after which the signal dots disappeared and were then regenerated at a randomly selected location within the same stimulus panel. Motion coherence threshold percentage was corrected for this finite dot lifetime. Noise dots randomly changed direction in a Brownian manner with each screen refresh. Each trial contained 25 frames with a total stimulus duration of 2.7 s.

Percentage of coherent motion was corrected for the finite lifetime of the dots. The percentage of target dots within a given software animation frame was varied to each participant's detection threshold from an initial starting value of 75% coherence using a weighted one-up, one-down adaptive staircase technique. This produced threshold estimates at the 75% correct level (Kaernbach, 1991). For correct responses, the motion coherence of the target stimulus was decreased by 1 dB (a factor of 1.122). For incorrect responses, the proportion of signal dots was increased by 3 dB (a factor of 1.412). The staircase procedure was terminated after ten reversals and detection threshold was defined as the geometric mean of the final eight reversals. Catch trials in which coherent motion depth was the same as the starting coherency (75%) were included at random (at least once every five trials) during each block to evaluate participant vigilance. Two blocks of test trials were conducted. The threshold of greatest sensitivity was taken as the participant's motion detection threshold.

Binocular viewing of the random dot kinematogram patches was conducted in a darkened room where lighting was held constant at ~5 cd/m^2. All participants were light adapted prior to presentation at a viewing distance of 57 cm held constant by a chinrest. Participants were instructed to inspect the two stimulus patches and report which patch contained coherent motion ("which had the dots moving side to side"). Feedback was given by means of a high (correct) or low (incorrect) tone after each response. Each participant completed practice trials before testing began. The coherence for the practice trials was fixed at a value well above the average threshold for all participants. Practice testing continued until participants reached the criterion of five consecutively correct trials.

Motion detection thresholds were obtained in two separate testing sessions. The proportion of children with dyslexia and coherent motion detection deficits was determined

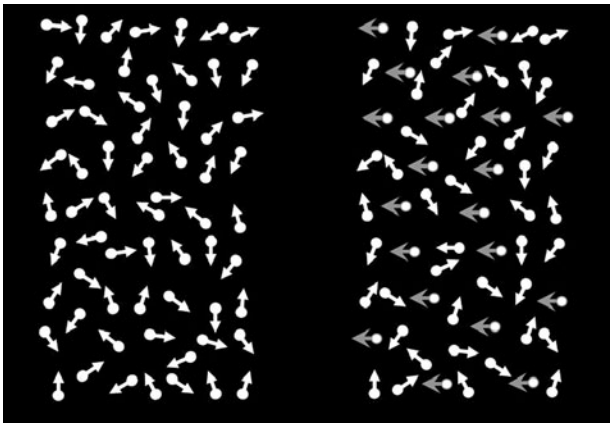


Fig. 1 Motion coherence task

using deviance analysis (Ramus et al., 2003). The threshold sensitivity used to determine the presence of a sensory deficit was 1.65 standard deviations (one tail 95% confidence interval) above the control group mean, after children from the control group with extreme scores were removed from the sample. See Wright and Conlon (2009) for a more complete description of the method and group data.

Procedure

The psychometric and reading tests were administered in a quiet room at the participant's school, free from visual and acoustic distractions. The visual search data were collected in a separate session. All testing was conducted by a trained examiner.

Results

Psychometric and reading variables

The group with dyslexia was significantly less accurate than controls on the measures of phonological awareness, $t(120)=7.3$, $p<0.001$; rapid automatic naming, $t(120)=-5.4$, $p=<0.001$; non-verbal processing speed, $t(120)=2.47$, $p=0.01$; orthographic skill, $t(120)=5.5$, $p<0.001$; word identification, $t(120)=9.35$, $p<0.001$; nonword decoding, $t(120)=11.3$, $p<0.001$ and WDRB Basic Reading Cluster, $t(120)=10.3$, $p<0.001$ (see Table 1).

Visual–motor response time

No significant differences between the mean reaction time to visual stimuli in the group with dyslexia ($M=311.5$ ms, SD=45.4) and control group ($M=311.4$ ms, SD=41.2) were found, $t(120)=0.014$, $p=0.989$, Cohen's $d=0.002$. Therefore, any significant between group differences in terms of response times for serial search that are observed in the following analyses cannot be attributed simply to motor reaction slowness. These data are consistent with previous that has shown groups with dyslexia are unimpaired on simple reaction time (Nicolson & Fawcett, 1994). No further analyses were conducted on visual–motor response time data.

Serial visual search

Response time There was a significant main effect of the number of distractors, $F(3, 360)=429.0$, $p<0.001$, $\eta_p^2=0.78$. Figure 2 shows an increase in response time as the number of distractors increased. There was no significant interaction between distractors and group, $F(3, 360)=1.38$, $p=0.25$, $\eta_p^2=0.01$. The main effect of reader group was significant, $F(1, 120)=6.2$, $p=0.014$, $\eta_p^2=0.05$. The group with dyslexia performed more slowly than the control group regardless of the number of distractors in the display. Cohen's d effect sizes for the group effects for the 4, 8, 16 and 32 item conditions were $d=0.60$, 0.46, 0.25 and 0.38, respectively.

Accuracy There was a significant main effect for number of the number of distractors presented, $F(3, 360)=53.2$, $p<0.001$, $\eta_p^2=0.31$. In general, error rates increased as the number of distractors increased. There was no statistically significant interaction effect involving group, $F(3, 360)=0.63$, $p=0.59$, $\eta_p^2=0.001$. Nor was there a significant main

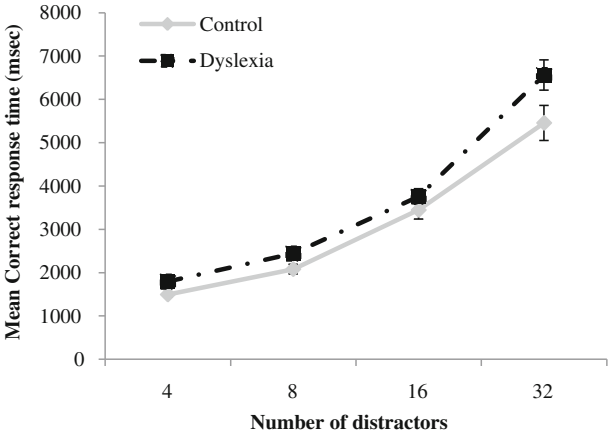


Fig. 2 Mean correct response time for the group with dyslexia (n=70) and the control group (n=52). *Error bars* represent±1 standard errors

effect of reader group, $F(1, 120)=0.16$, $p=0.69$, $\eta_p^2=0.001$. Response accuracy for both groups was above 85% in all conditions. The high accuracy rate demonstrates that participants were not trading speed for accuracy. Therefore, no further analyses were conducted on these data.

Comparison between controls and dyslexia sub-groups with and without M-deficits

On the coherent motion task, reported in Wright and Conlon (2009), 36.2% (26) of the children with dyslexia had a deficit at the first testing session and 27% (19) at the second testing time. There were 17.1% (12) children with an MD at both testing phases. Fifty-three percent (37) of the children with dyslexia did not have an MD deficit at either testing session.

For the purposes of this study, only those 12 children who showed a reliable weakness in motion detection across both testing sessions were included in the MD group. The NMD group showed no evidence of a motion detection deficit at either testing session. These individuals were matched to the MD group for age, IQ and reading ability. Both groups were compared to a matched group of skilled readers selected from the primary sample. The characteristics of these groups have been presented in Table 1.

There were no significant group differences for age, $F(2, 33)=0.17$, $p=0.83$, or IQ, $F(2, 33)=2.7$, $p=0.07$. There was a significant main effect of group for basic reading skills, $F(2, 33)=13.7$; $p<0.001$. Post hoc analyses showed that the skilled reader group had significantly higher word-reading skills compared to both groups with dyslexia. The two groups with dyslexia did not differ ($p=0.95$). The two groups with dyslexia did not differ significantly from the overall sample of children with dyslexia on IQ or other reading measures. However, these children were significantly younger than those in the overall sample, $F(2, 67)=19.0$, $p<0.001$. In a similar way, the only difference between the control group from the main sample and children selected was that the sub-group used were significantly younger than the overall control group, $t(50)=3.15$, $p=0.003$ (see Table 1).

Visual–motor response time

The main effect of group for visual–motor response time was not significant, $F(2, 33)=1.28$, $p=0.29$. These data show that the visual search times to be presented below were not

affected by more basic deficits in visual–motor response time in any of the groups. No further analyses were conducted on these data.

Serial visual search

Response time There was a significant main effect of number of distractors, $F(3, 99)=91.36$, $p<0.001$, $\eta_p^2=0.73$, with response time increasing as the number of distractors increased (see Fig. 3). The main effect of reader group was not significant, $F(2, 33)=1.02$, $p=0.37$, $\eta_p^2=0.06$. There was a significant interaction between distractors and group, $F(3, 99)=2.75$, $p=0.016$, $\eta_p^2=0.14$. The significant interaction occurred with the presentation of 32 distractors. The two groups with dyslexia did not differ in response time, $t(67)=0.51$, $p=0.62$. When combined, these groups had a significantly longer correct response time than the control group, $t(67)=6.14$, $p<0.001$.

Accuracy There was a significant main effect of number of distractors, $F(3, 99)=15.0$, $p<0.001$, $\eta_p^2=0.31$. In general, accuracy of responding decreased as the number of distractors increased. The group by distractors interaction was not significant, $F(6, 99)=1.01$, $p=0.42$, $\eta_p^2=0.06$. The main effect of group was significant, $F(2, 33)=3.37$, $p=0.047$, $\eta_p^2=0.17$. Regardless of the number of distractors presented, the NMD group ($M_{correct}=90.3\%$, SD=4.9%) and control group ($M_{correct}=92.7\%$, SD=3.8%) did not differ on the proportion or errors made, $t(33)=1.13$, $p=0.26$. When combined, these groups made significantly more errors than the MD group ($M_{correct}=95.1\%$, SD=2.81%).

Discussion

Visual magnocellular weaknesses have been found in groups with dyslexia (Conlon et al., 2011; Talcott, Hansen, Assoku, & Stein, 2000; Wright & Conlon, 2009); however, a satisfactory theory linking these weaknesses to reading development has yet to be found.

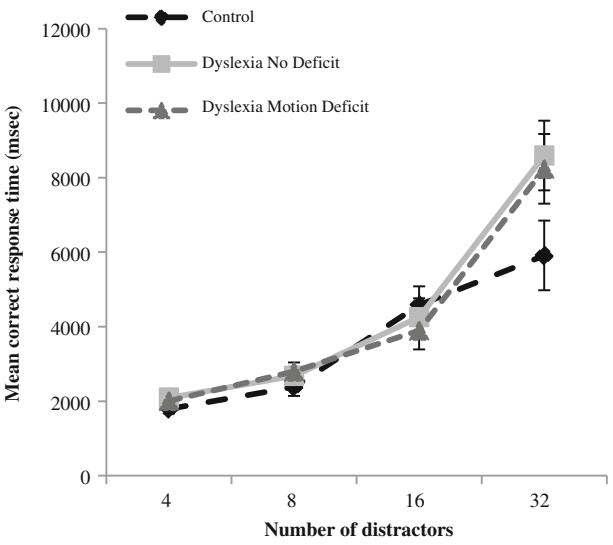


Fig. 3 Mean correct response to for the sub-groups with dyslexia with or without a motion deficit and for the control group. Each group had 12 participants. The standard *error bars* represent±1 standard errors

This study tested the proposal that the visual magnocellular weaknesses seen in dyslexia are linked to reading via the mechanism of visual attention (Vidyasagar, 1999). Results showed that a large group of children with dyslexia had slower visual search times than a control group regardless of the number of items in any condition. However, the effect size was small (η_p^2=0.05) which indicates there was considerable overlap in the distributions of groups with and without dyslexia. Overlap in the psychophysical thresholds of groups with dyslexia and control groups is common within the literature for both visual (Ramus et al., 2003; Wright & Conlon, 2009) and auditory stimuli (Witton, Stein, Stoodley, Rosner, & Talcott, 2002; Wright & Conlon, 2009). The current data are consistent with the conclusion that slower performance on visual search tasks is characteristic of groups with dyslexia, but it is not a core characteristic for each child with dyslexia.

The current findings could not be accounted for by simple motor reaction time. These data are important because some previous studies of visual attention have been criticized for failing to take response speed of this type into account (Roach & Hogben, 2007). This study has shown that the speed at which individuals with dyslexia can make psychomotor responses to visual stimuli does not explain slow search times.

Are visual attention deficits linked to poor M-processing?

The proposal that slow visual search in dyslexia results from poor magnocellular/dorsal stream functioning (Iles et al., 2000; Steinman et al., 1998; Vidyasagar, 1999, 2004; Vidyasagar & Pammer, 1999) was not supported by the current findings. There was no significant difference between the visual search times of two groups with dyslexia, one with coherent motion detection deficits and one without, and carefully matched controls.

These data conflict with the results of a similar study, which reported that a group of adults with dyslexia who had coherent motion detection deficits had significantly slower search performance than control readers or a group with dyslexia and no coherent motion detection deficit (Iles et al., 2000). Two possible explanations exist for the conflicting results. First, the conclusion reached in the Iles et al. study may be based on findings from only some of their serial visual search tasks. While their dyslexia group with a motion deficit was generally slower than controls, the group without a motion deficit was also slower than controls on one serial search task. Furthermore, the data from the two groups with dyslexia were indistinguishable on two serial search tasks. Second, error rates in some tasks approached chance levels suggesting that either the tasks were more difficult than that used in the current study or that some participants were trading accuracy for speed.

The current data are more consistent with a study that showed a group with dyslexia to be less responsive to spatial cues on a single fixation visual search task (Roach & Hogben, 2007). Although the group with dyslexia had significantly poorer sensitivity on a coherent motion task, no group differences in sensitivity were found on a measure of flicker contrast sensitivity. Furthermore, the discriminative accuracy of the spatial cueing task in discriminating individuals with and without dyslexia was high and coherent global motion low (Roach & Hogben, 2007). These findings suggest that the presence of a coherent motion deficit does not explain poorer capacity to use spatial attention on the cueing task. The current data are consistent with these findings and indicate that reduced sensitivity to coherent motion in dyslexia, indicated by reduced activity at MT/V5 in the dorsal stream (Demb, Boynton, & Heeger, 1998; Eden et al., 1996), is independent of deficits on tasks that require top-down focused visual attention. On this basis, the hypothesis that visual attention deficits in dyslexia occur because of an M-deficit are not supported.

Heterogeneity in visual processing in dyslexia

A notable result from the current study was that the original sample with dyslexia performed significantly more slowly than the control group on the visual search task. However, when the dyslexia sample was divided into sub-groups of individuals with and without global motion coherence deficits in order to test the primary hypothesis, no differences in search speed were found between the MD and NMD groups. The control group responded significantly faster than either group with dyslexia only in the 32-distracter condition. This suggests that the original group effect was partially based on the characteristics of at least some of the individuals who were not included in the MD versus NMD versus controls analyses. It is common to find that visual and/or sensory processing deficits in dyslexia groups are based on a small sub-group (15–30%; e.g. Ramus et al., 2003; Wright & Conlon, 2009). However, perhaps the most important point is that some of the children who were not included in the MD versus NMD versus controls analyses were not included because of variable responses to the coherent motion task (see Wright & Conlon, 2009). These data seem to highlight the need for careful measurement of psychophysical responses in children. It is suggested that future studies conduct repeated measurements of psychophysical tasks as we did here with the motion coherence task to avoid false conclusions based on inter- and intra-subject variability. These data also suggest that there may be something about the cognitive characteristics of some children with dyslexia that leads to considerable variation in response to psychophysical/sensory tasks and that group differences on some tasks may be based on these characteristics rather than to sensory processing weaknesses.

Cognitive factors which may influence visual attention performance

Two cognitive factors that may lead to inter- and intra-subject variability are processing speed and working memory. Both have been associated with the dyslexia phenotype (de Jong, 1998; Smith-Spark & Fisk, 2006; Shanahan et al., 2006; Willcutt, Pennington, Olson, Chhabilda, & Huslander, 2005; Wolf, 1991).

Weaknesses in groups with dyslexia have been found on verbal and non-verbal processing speed tasks (Denckla & Rudel, 1972; Fawcett & Nicolson, 1994; Wolf et al., 2002; Nicolson & Fawcett, 1994; Shanahan et al., 2006; Willcutt et al., 2005; see Wolf & Bowers, 1999 and Savage, 2004, for reviews). This study found that the group with dyslexia was significantly slower than controls on two processing speed tasks (rapid automatic naming and non-verbal processing speed). Serial search tasks are timed and therefore vulnerable to processing speed effects that may be independent of visual attention. Future research will need to investigate the visual search performance of groups with dyslexia with and without processing speed deficits to determine the influence of this cognitive variable on visual attention tasks.

Working memory refers to a cognitive function that permits the simultaneous storage and processing of information (Baddeley, 1986). There is growing evidence for both verbal and visuospatial working memory weaknesses in dyslexia (e.g. de Jong, 1998; Willcutt et al., 2005). In the case of visual search, some individuals with dyslexia may have difficulty using working memory to keep a representation of the target stimulus active while performing the search task. This may affect search strategies and slow search times.

Possible impact of co-existing conditions

In the current study, care was taken to exclude children with formal diagnoses of developmental disorders other than dyslexia. However, exclusion was based on existing

school records, and clinical diagnostic assessment was not performed. Therefore, it is possible that the slow visual search times seen in some individuals in the current study may have been due to a co-existing disorder that escaped detection. A likely candidate is attention deficit hyperactivity disorder, a condition that frequently co-exists with dyslexia (Semrud-Clikeman, Biederman, Sprich-Buckminster, Lehman, & Faraone, 1992; Willcutt, Pennington, & DeFries, 2000), as lapses in attention could explain slow search speed.

Finally, the identification of dyslexia itself poses a problem that is rarely, if ever, discussed in the literature. While a discrepancy method, average IQ in the presence of poor word-level reading skills, is scientifically defensible and allows replication, it does not guarantee exclusion of children who have reading problems that are at least partly environmental rather than being due to the neurological weaknesses presumed to underlie dyslexia (Lyon, Shaywitz, & Shaywitz, 2003). Studies that have investigated response to reading intervention in young children typically show that 60–70% exhibit good growth in reading skills and return to a level considered within normal limits (e.g. above the 30th or 40th percentile; Torgesen et al., 1999; Vellutino et al., 1996). Conversely, 30–40% of children remain poor readers at the end of treatment. The proportion of children with refractory reading problems is similar to the number of children (~30%) identified by studies that have evaluated individual children as having sensory processing deficits (Wright & Conlon, 2009). Therefore, it is possible that only the children with complex, refractory reading problems also exhibit sensory processing weaknesses. Whether or not these sensory processing weaknesses are part of the aetiology of dyslexia in these individuals or an artefact of some co-existing disorder or characteristics remains to be discovered. However, it seems reasonable to suggest that future studies adopt a response-to-intervention (RTI) approach to participant selection (Fletcher, Francis, Morris, & Lyon, 2005). The RTI approach ensures that all individuals within the sample have access to evidence-based teaching and therefore will mostly ensure that children whose reading difficulties have an environmental rather than a neurological origin do not become part of the sample with dyslexia. Only those children who remain poor readers post-intervention should be included in future studies. We are not aware of any study that has attempted this to date. Nevertheless, it should become standard practice to help tease out who has and who does not have sensory deficits and how they may or may not be related to reading.

References

Baddeley, A. (1986). *Working memory*. Oxford: Oxford University Press.

Bednarek, D. B., Saldana, D., Quintero-Gallego, E., Garcia, I., Grabowska, A., & Gomez, C. M. (2004). Attentional deficit in dyslexia: a general or specific impairment? *NeuroReport, 15*, 1787–1790.

Bonifacci, B., & Snowling, M. J. (2008). Speed of processing and reading disability: a cross-linguistic investigation of dyslexia and borderline intellectual functioning. *Cognition, 107*, 999–1017.

Breitmeyer, B. G. (1980). Unmasking visual masking: a look at the "why" behind the veil of "how". *Psychological Review, 87*, 52–69.

Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual patter making, saccadic suppression and information processing. *Psychological Review, 83*, 1–36.

Buchholz, J., & Aimola Davies, A. M. (2005). Adults with dyslexia demonstrate space-based and object-based covert attention deficits. *Brain and Cognition, 57*, 30–34.

Buchholz, J., & Aimola Davies, A. (2007). Attentional blink deficits observed in dyslexia depend upon task demands. *Vision Research, 47*, 1292–1302.

Buchholz, J., & McKone, E. (2004). Adults with dyslexia show deficits on spatial frequency doubling and visual attention tasks. *Dyslexia, 10*, 24–43.

Casco, C., Tessoldi, P. E., & Dellantonio, A. (1998). Visual selective attention and reading efficiency are related in children. *Cortex, 34*, 531–546.

Conlon, E. G., Sanders, M. A., & Wright, C. M. (2009). Relationships between global motion and global form processing, practice, cognitive and visual processing in adults with dyslexia or visual discomfort. *Neuropsychologia, 47*, 907–915.

Conlon, E., Sanders, M., & Zapart, S. (2004). Temporal processing in poor adult readers. *Neuropsychologia, 42*, 142–157.

Conlon, E. G., Wright, C. M., Norris, K., & Chekaluk, E. (2011). Does a sensory processing deficit explain counting accuracy on rapid visual sequencing tasks in adults with and without dyslexia? *Brain and Cognition, 76*, 197–205.

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*, 1483–1493.

de Jong, P. F. (1998). Working memory deficits of reading disabled children. *Journal of Experimental Child Psychology, 70*, 75–96.

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

Demb, J. B., Boynton, G. M., & Heeger, D. J. (1998). Functional magnetic imaging of early visual pathways in dyslexia. *The Journal of Neuroscience, 18*, 6939–6951.

Denckla, M. B., & Rudel, R. G. (1972). Color-naming in dyslexic boys. *Cortex, 8*, 164–176.

Eden, G., VanMeter, J., Rumsey, J., Maisog, J., Woods, R., & Zeffiro, T. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature, 382*, 66–69.

Enns, J. T., & Rensink, R. A. (1992). *V Scope: general purpose tachistoscope for Macintosh*. Vancouver: Micropsych Software.

Facoetti, A., Lorusso, M. L., Paganoni, P., Cattaneo, C., Galli, R., & Mascetti, G. G. (2003). The time course of attentional focusing in dyslexic and normally reading children. *Brain and Cognition, 53*, 181–184.

Facoetti, A., Paganoni, P., & Lorusso, M. L. (2000). The spatial distribution of visual attention in developmental dyslexia. *Experimental Brain Research, 132*, 531–538.

Facoetti, A., Turatto, M., Lorusso, M. L., & Mascetti, G. G. (2001). Orienting of visual attention in dyslexia: evidence for asymmetrical hemispheric control of attention. *Experimental Brain Research, 138*, 46–53.

Facoetti, A., Zorzi, M., Cestnick, L., Lurusso, M. L., Molteni, M., Paganoni, P., et al. (2006). The relationship between visuo-spatial attention and nonword reading in developmental dyslexia. *Cognitive Neuropsychology, 23*, 841–855.

Fawcett, A. J., & Nicolson, R. I. (1994). Naming speed in children with dyslexia. *Journal of Learning Disabilities, 27*, 641–646.

Fawcett, A. J., & Nicolson, R. I. (1996). *The Dyslexia Screening Test*. London: The Psychological Corporation.

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

Fletcher, J. M., Francis, D. J., Morris, R. D., & Lyon, G. R. (2005). Evidence-based assessment of learning disabilities in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*, 506–522.

Friedmann, N., Kerbel, N., & Shvimer, L. (2010). Developmental attentional dyslexia. *Cortex, 46*, 1216–1237.

Goulandris, N., McIntyre, A., Snowling, M. J., Bethel, J. M., & Lee, J. P. (1998). A comparison of dyslexic and normal readers using orthoptic assessment procedures. *Dyslexia, 4*, 30–48.

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.

Hari, R., Valta, M., & Uutela, K. (1999). Prolonged attentional dwell time in dyslexic adults. *Neuroscience Letters, 271*, 202–204.

Hatcher, P. (2000). *Sound linkage: an integrated program for overcoming reading difficulties*. London: Whurr.

Iles, J., Walsh, V., & Richardson, A. (2000). Visual search performance in dyslexia. *Dyslexia, 6*, 163–177.

Kaernbach, C. (1991). Simple adaptive testing with the weighted up-down method. *Perception & Psychophysics, 49*, 227–229.

Kubova, Z., Kuba, M., Peregrin, J., & Novakova, V. (1996). Visually evoked potential evidence for magnocellular system deficit in dyslexia. *Physiological Research, 45*, 87–89.

Lehmkuhle, S. M., Garzia, R. P., Turner, L., Hash, T., & Baro, J. A. (1993). A defective visual pathway in children with reading disability. *The New England Journal of Medicine, 328*, 989–996.

Lovegrove, W. J., Martin, F., & Slaghuis, W. (1986). A theoretical and experimental case for a visual deficit in specific reading disability. *Cognitive Neuropsychology, 3*, 225–267.

Lyon, G. R., Shaywitz, S. E., & Shaywitz, B. A. (2003). A definition of dyslexia. *Annals of Dyslexia, 53*, 1–14.

Nicolson, R. I., & Fawcett, A. J. (1994). Reaction times and dyslexia. *Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology, 47A*, 29–48.

Olson, R., Forsberg, H., Wise, B., & Rack, J. (1994). Measurement of word recognition, orthographic and phonological skills. In G. Reid Lyon (Ed.), *Frames of reference for the assessment of learning disabilities: new views on measurement issues* (pp. 243–278). Baltimore: Brookes.

Ramus, F., Rosen, S., Dakin, S. C., Day, B. L., Catelotte, J. M., White, S., et al. (2003). Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults. *Brain, 126*, 841–865.

Raven, J. C., Court, J. H., & Raven, J. (1995). *Coloured progressive matrices*. Oxford: Oxford Psychologists Press.

Roach, N. W., & Hogben, J. H. (2004). Attentional modulation of visual processing in adult dyslexia: a spatial cueing deficit. *Psychological Science, 15*, 650–654.

Roach, N. W., & Hogben, J. H. (2007). Impaired filtering of behaviorally irrelevant visual information in dyslexia. *Brain, 130*, 771–785.

Savage, R. (2004). Motor skills, automaticity and developmental dyslexia. *Reading and Writing, 17*, 301–324.

Semrud-Clikeman, M., Biederman, J., Sprich-Buckminster, S., Lehman, B. K., Faraone, S. V., & Norman, D. (1992). Comorbidity between ADDH and LD: a review and report in a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 439–448.

Shanahan, M. A., Pennington, B. F., Yerys, B. E., Scott, A., Boada, R., Willcutt, E. G., et al. (2006). Processing speed deficits in attention deficit/hyperactivity disorder and reading disability. *Journal of Abnormal Child Psychology, 34*, 584–602.

Shaywitz, S., Escobar, M., Shaywitz, B., Fletcher, J., & Makugh, R. (1992). Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. *The New England Journal of Medicine, 326*, 145–150.

Sireteanu, R., Goebel, C., Goertz, R., & Wandert, T. (2005). Do children with developmental dyslexia show a selective visual attention deficit? *Strabismus, 14*, 85–93.

Sireteanu, R., Goebel, C., Goertz, R., Werner, I., Nalewajko, M., & Thiel, A. (2008). Impaired visual search in children with developmental dyslexia. *Annals of the New York Academy of Sciences, 1145*, 199–211.

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

Slaghuis, W. L., & Ryan, J. F. (2006). Directional motion contrast sensitivity in developmental dyslexia. *Vision Research, 46*, 3291–3303.

Smith-Spark, J. H., & Fisk, J. E. (2006). Working memory functioning in developmental dyslexia. *Memory, 15*, 34–56.

Snowling, M. J. (2000). *Dyslexia*. Oxford: Blackwell.

Stein, J. F., & Fowler, M. S. (1980). Visual dyslexia. *British Orthopedic Journal, 37*, 11.

Stein, J. F., & Fowler, M. S. (1981). Visual dyslexia. *Trends in Neuroscience, 4*, 77–80.

Stein, J. F., & Fowler, M. S. (1985). Effect of monocular occlusion on visuomotor perception and reading in dyslexic children. *Lancet, 2*(8446), 69–73.

Stein, J. F., Fowler, M. S., & Richardson, A. (2000). Monocular occlusion can improve binocular control and reading in dyslexics. *Brain, 123*, 164–170.

Stein, J. F., Riddell, P. M., & Fowler, M. S. (1988). Disordered vergence control in dyslexic children. *British Journal of Ophthalmology, 72*, 162–166.

Stein, J., & Walsh, V. (1997). To see but not to read: the magnocellular theory of dyslexia. *Trends in Neurosciences, 20*, 147–152.

Steinman, S. B., Steinman, B. A., & Garzia, R. P. (1998). Vision and attention. II: is visual attention a mechanism through which a deficient magnocellular pathway might cause reading disability? *Optometry and Vision Science, 75*, 674–681.

Talcott, J. B., Hansen, P. C., Assoku, E. L., & Stein, J. F. (2000). Visual motion sensitivity in dyslexia: evidence for temporal and energy integration deficits. *Neuropsychologia, 38*, 935–943.

Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Lindamood, P., Rose, E., Conway, T., et al. (1999). Preventing reading failure in young children with phonological processing disabilities: group and individual differences to instruction. *Journal of Educational Psychology, 91*, 579–593.

Triesman, A. M. (1988). Features and objects: the fourteenth Bartlett memorial lecture. *Quarterly Journal of Experimental Psychology, 40A*, 201–237.

Triesman, A. M., & Gelade, G. (1980). A feature integration theory of attention. *Cognitive Psychology, 12*, 97–136.

Triesman, A., & Sato, S. (1990). Conjunction search revisited. *Journal of Experimental Psychology. Human Perception and Performance, 16*, 459–478.

Valdois, S., Bosse, M. L., & Tainturier, M. J. (2004). Cognitive correlates of developmental dyslexia: review of evidence for a selective visual attentional deficit. *Dyslexia, 10*, 1–25.

Valdois, S., Gérard, C., Vanauld, P., & Dugas, M. (1995). Developmental dyslexia: a visual attentional account? *Cognitive Neuropsychology, 12*, 31–67.

Vellutino, F. R., Fletcher, J. M., Snowling, M. J., & Scanlon, D. M. (2004). Specific reading disability (dyslexia): what have we learned in the past four decades? *Journal of Child Psychology and Psychiatry, 45*, 2–40.

Vellutino, F. R., Scanlon, D. M., Sipay, E. R., Small, S. G., Pratt, A., Chen, R., et al. (1996). Cognitive profiles of difficult-to-remediate and readily remediated poor readers: early intervention as a vehicle for distinguishing between cognitive and experiential deficits as basic causes of specific reading disability. *Journal of Educational Psychology, 88*, 601–638.

Vidyasagar, T. R. (1999). A neuronal model of attentional spotlight: parietal guiding the temporal. *Brain Research Reviews, 30*, 6–76.

Vidyasagar, T. R. (2004). Neural underpinnings of dyslexia as a disorder of visuo-spatial attention. *Clinical & Experimental Optometry, 87*, 4–10.

Vidyasagar, T. R., & Pammer, K. (1999). Impaired visual search in dyslexia relates to the role of the magnocellular pathway in attention. *NeuroReport, 10*, 1283–1287.

Vidyasagar, T., & Pammer, K. (2010). Dyslexia: a deficit in visuospatial attention, not in phonological processing. *Trends in Cognitive Sciences, 14*(2), 57–63.

Visser, T. A. W., Boden, C., & Giaschi, D. E. (2004). Children with dyslexia: evidence for visual attention deficits in perception of rapid sequences of objects. *Vision Research, 44*, 2521–2535.

Willcutt, E. G., Pennington, B. F., & DeFries, J. C. (2000). Twin study of the etiology of comorbidity between reading disability and attention-deficit/hyperactivity disorder. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 96*, 293–301.

Willcutt, E. G., Pennington, B. F., Olson, R. K., Chhabildas, N., & Huslander, J. (2005). Neuropsychological analyses of comorbidity between reading disability and attention-deficit/hyperactivity disorder: in search of the common deficit. *Developmental Neuropsychology, 27*, 35–78.

Williams, M. C., Brannan, J. R., & Lartigue, E. K. (1987). Visual search in good and poor readers. *Clinical Vision Sciences, 1*, 367–371.

Wilmer, J. B., Richardson, A. J., Chen, Y., & Stein, J. F. (2004). Two visual motion processing deficits in developmental dyslexia associated with different reading skills deficits. *Journal of Cognitive Neuroscience, 16*, 1–13.

Witton, C., Stein, J. F., Stoodley, C. J., Rosner, B. S., & Talcott, J. B. (2002). Separate influences of acoustic AM and FM sensitivity on the phonological decoding skills if impaired and normal readers. *Journal of Cognitive Neuroscience, 14*, 866–874.

Wolf, M. (1991). Naming speed and reading: the contribution of the cognitive neuro-sciences. *Reading Research Quarterly, 26*, 123–141.

Wolf, M., & Bowers, P. G. (1999). The double-deficit hypothesis for the developmental dyslexias. *Journal of Educational Psychology, 91*, 415–438.

Wolf, M., O'Rourke, G. A., Gidney, C., Lovett, M., Cirino, P., & Morris, R. (2002). The second deficit: an investigation of the independence of phonological and naming-speed deficits in developmental dyslexia. *Reading and Writing: An Interdisciplinary Journal, 15*, 43–72.

Woodcock, R. W. (1997). *Woodcock Diagnostic Reading Battery.* Itasca: Riverside.

Wright, C. M., & Conlon, E. G. (2009). Auditory and visual processing in children with dyslexia. *Developmental Neuropsychology, 34*, 330–355.

Yule, W., Rutter, M., Berger, M., & Thompson, B. (1973). Over and under achievement in reading. *British Journal of Educational Psychology, 44*, 1–12.