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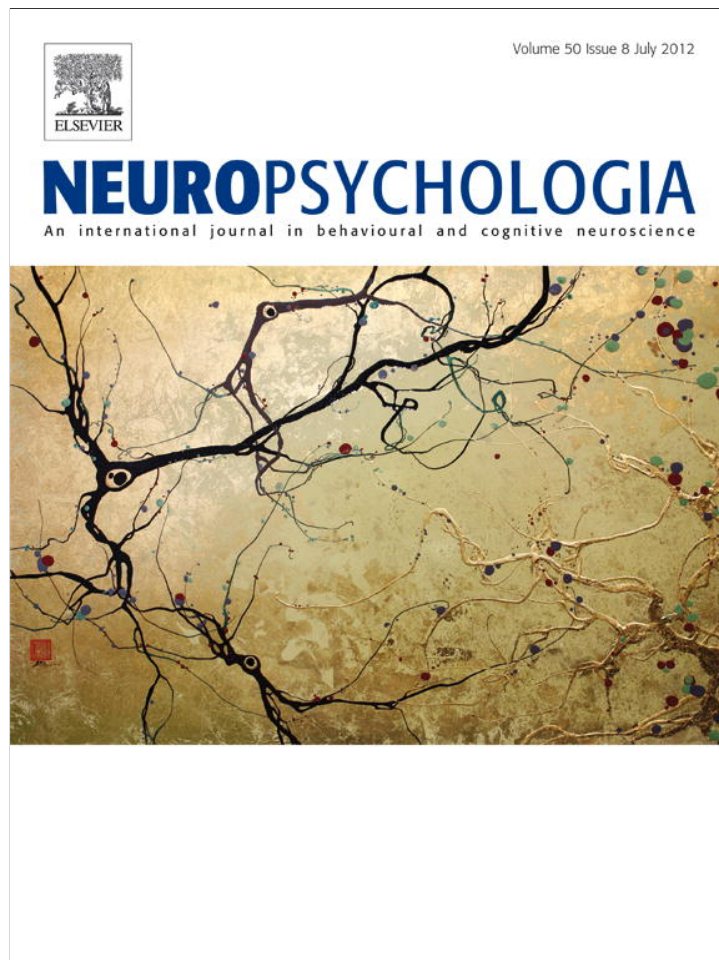
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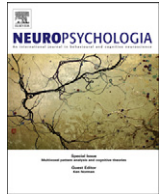
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# Abrupt and ramped flicker-defined form shows evidence for a large magnocellular impairment in dyslexia

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## ABSTRACT

Controversy still exists over whether there is a magnocellular deficit associated with developmental dyslexia. Here we utilised a magnocellular system-biased phantom contour form discrimination task defined by high temporal frequency contrast reversals to compare contrast sensitivity in a group of children with dyslexia and an age- and nonverbal intelligence-matched control group (9–14 years). Stimuli were either abruptly presented for 4 refresh frames (34 ms), or in two reduced transience conditions had contrast progressively ramped on and off over either 4 frames or 10 frames (86 ms). Children in the dyslexia group showed increased contrast thresholds compared with the control group in all three conditions, and thus strong evidence for a magnocellular deficit. Although the absolute size of the differences in threshold scores between control and dyslexic groups increased dramatically between the abrupt and the 4 and 10 frame ramped onset stimuli, the similar effect size across all tasks, and also the similar range of contrast change at the first frame of stimulus presentation across all tasks between groups suggests that a similar neural mechanism could provide the locus of the apparent magnocellular deficit in children with dyslexia for all tasks tested. These results suggest that threshold discrimination of stimuli with low contrast and high temporal frequencies designed to target the magnocellular system, and has great potential for early screening for children at risk of visually derived reading difficulties.

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## 1. Introduction

Although developmental dyslexia is still commonly associated with language or phonological impairment, a large body of evidence exists arguing for an impairment in the magnocellular visual pathway (Borsting et al., 1996; Chase & Jenner, 1993; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Demb, Boynton, & Heeger, 1998; Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Martin & Lovegrove, 1987; Rutkowski, Crewther, & Crewther, 2003; Sperling, Lu, Manis, & Seidenberg, 2003; Stein, 2001; Wang, Bi, Gao, & Wydel, 2010). The human visual system consists of two major pathways (magnocellular and parvocellular) projecting from the relatively larger magnocellular and smaller parvocellular retinal ganglion cells to the lateral geniculate nucleus (LGN) and through to primary visual cortex (V1). The subcortical magnocellular pathway has been shown in primates to possess high contrast gain and preferentially respond to higher temporal and lower spatial frequencies, whilst the parvocellular pathway responds selectively to lower temporal and higher spatial

frequencies, responds to colour and has a higher contrast threshold (reviewed in Nassi & Callaway, 2009).

This magnocellular deficit hypothesis is not without controversy (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Skottun, 2000; Sperling, Lu, Manis, & Seidenberg, 2005), though many different measures have indicated subtle abnormalities in visual processing consistent with impairment in the subcortical magnocellular system and possibly extending through the dorsal stream to parietal cortex to which the magnocellular system provides the dominant inputs. It is also possible that many of the mixed results reported may be related to the wide range of tasks used to test the functionality of the magnocellular system that may not have been optimally designed for selectively targeting both the spatial and temporal properties of subcortical magnocellular responses.

Spatial contrast sensitivity is one measure for which there is consensus that magnocellular and parvocellular responses can be effectively tested and compared (Borsting et al., 1996; Demb, Boynton, Best, & Heeger, 1998; Lovegrove et al., 1982; Skottun, 2000). In particular, high temporal frequency flicker-defined form contrast sensitivity is a paradigm that theoretically appears particularly well suited to measuring magnocellular responses (Barnard, Crewther, & Crewther, 1998; Cornelissen et al., 1995; Crewther, Crewther, Barnard, & Klistorner, 1996).

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Crewther et al. (1996) first used a flicker-defined form contrast sensitivity task to demonstrate psychophysical evidence for the development of magnocellular function until at least 8 years of age, and then Barnard et al. (1998) used the same flicker-defined form task to compare magnocellular function in good and poor readers in kindergarten, grades 3 and 6, finding insignificant differences in performance between reading groups. A similar stimulus was used by Sperling, Lu, Manis, and Seidenberg (2003) who found that children with dyslexia had lower flicker-rate thresholds for phantom-contour shape recognition in a luminant (magnocellular) but not an isoluminant (parvocellular) condition, a finding that was later replicated in an adult population (Sperling, Lu, Manis, & Seidenberg, 2006). Interestingly, Sperling et al. (2006) did not interpret their later study in terms of a magnocellular deficit, but rather on the basis of an earlier study (Sperling et al., 2005) a noise-exclusion deficit or impaired perceptual integration was proposed as a more likely explanation. More recent models emphasising the role of early, rapid magnocellular processing in driving object perception would however not see impaired perceptual integration as surprising assuming the existence of a magnocellular deficit (Bar et al., 2006; Laycock, Crewther, & Crewther, 2007).

Our earliest version of a contour flicker-defined form task (Barnard et al., 1998), although designed as a preferentially magnocellular stimulus (achromatic contrast-reversing pattern defined by lighter and darker grey dots modulating around 50% contrast that created a 'phantom-E form') in which a perceptually clear but illusory foreground/background boundary is apparent (see Fig. 1) was limited to a flicker-rate of 33 Hz. Thus the second version of the task (Crewther, Kiely, & Crewther, 2006) has now been designed for a higher screen refresh-rate (flicker rate of 58.5 Hz) to exclude the possibility of parvocellular contamination of the threshold responses (Derrington & Lennie, 1984; Hicks, Lee, & Vidyasagar, 1983). Furthermore, to better isolate the low contrast and high temporal frequency qualities preferred by the subcortical magnocellular pathway from their more dorsal stream role in fast activation of attention we have now incorporated a second ramped condition of stimulus presentation designed to reduce the stimuli's activation of transient attention mechanisms. Thus while both tasks will require subcortical magnocellular processing alongside ventral stream-related form recognition (Malach et al., 1995), the abrupt onset condition is expected to also provide a measure of more dorsal stream attention driven contrast sensitivity while the ramped onset contrast sensitivity task was designed to allow determination of the minimum contrast increase needed for perception when transient attention is minimized, and thus ventral stream processing is relied upon. These two conditions also have the advantage of retaining the same *relative signal to noise ratio* at each presentation, although

the total signal (and noise) strength is reduced when presented at low contrast. Signal strength obviously increases gradually as contrast is ramped on, but the relative signal-to-noise ratio embedded within the stimulus will remain constant during the ramped onset/offset and thus will be equivalent to the signal-to-noise ratio in the abrupt onset version.

The abrupt and ramped onset stimuli have been used previously by Kiely, Crewther, and Crewther (2007) to demonstrate that the ramped onset flicker contrast sensitivity task requires far higher levels of contrast for perception. Ramped onset required greater than 60% contrast for threshold performance for most adult participants (when ramped on over 40 frames), compared to 1–4% for the abrupt onset condition. The results demonstrated that each individual has a consistent 'contrast velocity' function (threshold divided by duration), indicating that ramped phantom flicker-defined contours, having reduced transience information, require a sufficiently rapid increase in contrast before they can be seen. In other words with a longer duration of ramped onset, stimuli become more attentionally demanding and may require a stronger or faster magnocellular response to be recognised. This is consistent with the suggestion that the magnocellular system provides the strongest input to the dorsal visual stream and parietal cortex for activation of attentional processing (Cheng, Eysel, & Vidyasagar, 2004; Laycock et al., 2007; Laycock, Crewther, & Crewther, 2008; Omtzigt, Hendriks, & Kolk, 2002; Vidyasagar, 1999).

We thus decided to compare good and poor readers on an abrupt onset, and also a short ramped, and a longer ramped onset flicker-defined form stimulus, with the expectation that increasing the onset duration will expose a post-striate magnocellular impairment in dyslexia that the slower temporal frequency used by Barnard et al. (1998) was not sensitive enough to show.

## 2. Methods

### 2.1. Participants

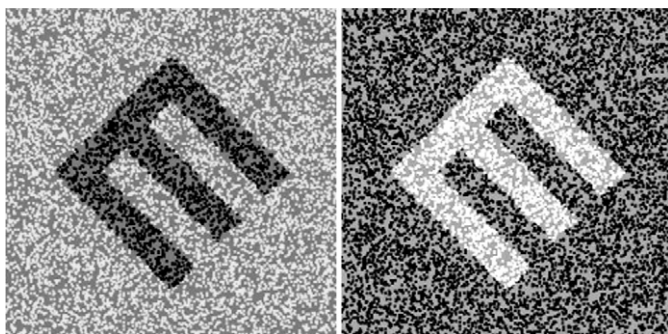
Sixty-one children aged 9–14 years participated in the study. University human ethics approval was given, and parental consent was provided prior to participation. A total of 17 children comprised a developmental dyslexia (DD) group, with 44 good readers forming a chronological age control (CA). DD children were classified on the basis of having reading performance greater than two years behind chronological age and nonverbal intelligence score within one standard deviation of the mean (see Table 1 for details of both groups).

Prior to the screening, the reading age of some children had already been assessed by the Reading Progress Test (Crumpler, De la Mare, & Vincent, 1997), which is primarily a measure of timed reading comprehension skills and a well-accepted measure of reading across the state of Victoria, Australia. Since comprehension can only follow good decoding skills, a score above the 50th percentile on this test was deemed to be a sufficient measure of reading age for readers in the control group. Those, however, who read below the expected level for their chronological age, and all other participants in the study without a current reading-age, completed the accuracy subtest of the Neale Test of Reading Analysis (1988). On this basis, reading lag behind chronological age could be determined, and classification as either a DD reader or as a good reading control was made. No children were reported by teachers to have language impairments. Nonverbal

**Table 1**

Summary of the mean (and standard deviation) chronological age, nonverbal intelligence percentile as assessed by the Raven's Coloured Progressive Matrices, and reading age for both groups of children. DD=developmental dyslexia group, CA=chronological age control group.

Group	No.	Chronological age		Nonverbal intelligence (percentile)		Reading age	
		M	(SD)	M	(SD)	M	(SD)
DD	17	11.71	(1.45)	37.59	(20.93)	7.63	(1.10)
CA	46	11.47	(0.69)	59.94	(24.31)	12.72	(1.6)



**Fig. 1.** Flicker-defined form stimulus. The boundary between light and dark dots creates an phantom contour, which when flicker from light to dark, and dark to light in counter-phase creates a self-masking flicker-defined form.



intelligence was assessed with the Raven's Coloured Progressive Matrices (RCPM) test (Raven, Court, & Raven, 1990), which has been widely used in primary education in the UK and Australia (Cotton et al., 2005).

Children were recruited for participation in the study as part of a specialist camp for children with learning difficulties, or from a school. All children completed visual assessments, and were screened for colour blindness and also excluded if a binocular vision problem or a significant refractive anomaly affecting vision was found. All children also completed a set of other psychophysics tasks which have been published elsewhere (Laycock, Crewther, Kiely, & Crewther, 2006), though this data will be referred to directly in the discussion as it provides an important control for the current study.

## 2.2. Visual stimuli

Three flicker-defined form contrast sensitivity tasks ('Flickering E' tasks) were completed on iMac computers (with CRT monitors) with a screen refresh rate of 117 Hz, and a spatial configuration of 640 × 480 pixels. Stimuli consisted of a letter E, subtending 5 deg, of four possible orientations (north-east, south-east, south-west, north-west) and of variable contrast presented at fixation. The form was created by the distribution of light and dark dots on a mid-grey background creating an illusory edge (Fig. 1). Foreground and background alternated between light and dark at a rate of 58.5 Hz, effectively self-masking the stimulus. In the abrupt task, the 'E' had a square contrast profile and an exposure time of 4 frames (34.4 ms). In the ramped tasks, stimuli were temporally ramped on and off, with onset achieved over a duration of either 4 frames (total stimuli duration lasting 8 frames, 68.8 ms) or 10 frames (total stimuli duration lasting 20 frames, 172 ms).

## 2.3. Procedure

Six children were tested at a time in one room using six computers during school hours, with a testing session lasting approximately 45 min, though children were able to take breaks whenever necessary. Between 4 and 5 investigators were present to explain, run and supervise completion of the reading and visual tasks, which were initially shown to the children in a familiarisation session. Reading and nonverbal intelligence measures were completed first, and the Flickering E tasks did not commence until the investigators were satisfied that each child understood the requirements of each task.

For each visual task, a trial involved the presentation of a flickering stimulus, with participants required to report by keyboard press which of the four alternative orientations displayed after the target disappeared they had seen in a four-alternative forced-choice match-to-sample design. Flickering E tasks utilised a PEST (parameter estimation by sequential testing) procedure that operates like a staircase, halving the step size until an estimate of threshold contrast is determined. After each trial the prior distribution from previous trials was used to estimate a confidence level, with the test terminating once the confidence level exceeded 90%.

## 3. Results

Comparisons between mean contrast thresholds for the developmental dyslexia (DD) group and the entire chronological age-matched (CA) group showed significant differences for all three tasks, as can be seen in Fig. 2 (abrupt Es:  $t(59)=4.16$ ,  $p<.001$ ; ramped-4 frame onset Es:  $t(59)=3.96$ ,  $p<.001$ ; ramped-10 frame onset Es:  $t(48)=4.15$ ,  $p<.001$ ).

A potential confound, lies in the significant difference in non-verbal intelligence established between the DD group and the CA group ( $p=.002$ ). This was deemed potentially important, due to the moderate but significant correlations between non-verbal intelligence and both ramped Es tasks ( $r=-.28$ ,  $r=-.32$ , respectively,  $ps<.05$ ), despite not correlating significantly with the abrupt Es ( $r=-.20$ ,  $p=.17$ ). The CA group was thus subdivided with a half split into higher and lower non-verbal intelligence groups (H-IQ and L-IQ, respectively), with the L-IQ group and DD groups now much better matched for non-verbal intelligence ( $p=.75$ ) (see Table 2). Data screening within each group highlighted five participants designated as extreme outliers (with threshold scores greater than three times the inter-quartile range of the median), and were consequently excluded. This included two H-IQ control participants on the abrupt task, and one from the ramped-4 frame task, and one from both the L-IQ and H-IQ control groups for the ramped-10 frame task.

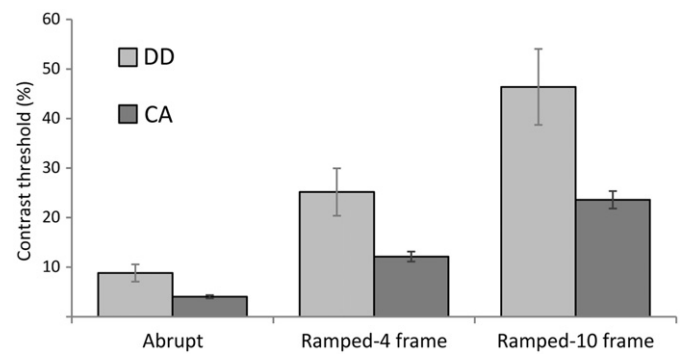


Fig. 2. Contrast threshold for the developmental dyslexia (DD) and chronological age (CA) control groups was significantly different for the abrupt onset, ramped-4 frame and ramped-10 frame onset tasks.

Table 2

Summary of the mean (and standard deviation) chronological age, nonverbal intelligence percentile as assessed by the Raven's Coloured Progressive Matrices, and reading age for the DD group, and the CA group split into a higher and a lower nonverbal intelligence group. DD=developmental dyslexia group, L-IQ=chronological age and nonverbal intelligence matched control group, H-IQ=chronological age matched but with higher nonverbal intelligence.

Group	No.	Chronological age		Nonverbal intelligence (percentile)		Reading age	
		M	(SD)	M	(SD)	M	(SD)
H-IQ	23	11.6	(0.68)	80.11	(12.58)	13.2	1.84
L-IQ	23	11.35	(0.69)	39.3	(13.44)	12.25	1.17
DD	17	11.71	(1.45)	37.59	(20.93)	7.63	1.10

A one-way ANOVA was run for each Flickering E task to compare contrast threshold performance between the DD and the two chronological age groups (L-IQ, H-IQ). For the abrupt Es task there was a significant effect of group,  $F(2, 56)=9.21$ ,  $p<.001$ , with post hoc comparisons indicating that whilst the L-IQ and H-IQ groups did not differ ( $p=.99$ ), the DD group differed significantly from the L-IQ ( $p=.001$ ) and the H-IQ ( $p=.001$ ) groups (Fig. 3).

There was a significant effect of group for the ramped-4 frame onset Es task,  $F(2, 57)=7.95$ ,  $p=.001$ , with post hoc comparisons again indicating that whilst the L-IQ and H-IQ groups did not differ ( $p=.84$ ), the DD group differed significantly from the L-IQ ( $p=.006$ ) and the H-IQ ( $p=.001$ ) groups (Fig. 3).

Finally, for the ramped-10 frame onset Es task there was a significant effect of group,  $F(2, 45)=8.45$ ,  $p=.001$ , with post hoc comparisons indicating that similar to the previous tasks, the L-IQ and H-IQ groups did not differ ( $p=.98$ ), whilst the DD group showed significantly different contrast threshold's from the L-IQ ( $p=.003$ ) and the H-IQ ( $p=.002$ ) groups (Fig. 3).

In order to assess the relative discrepancy between the DD and the two control groups on each of the Flicker E tasks a mixed design ANOVA with onset type (abrupt, ramped-4 frame, ramped-10 frame) as the within subject factors, and group (DD, L-IQ, H-IQ) as the between subject factors was run, with results indicating main effects of onset type ( $F(2, 84)=86.19$ ,  $p<.001$ ) and of group ( $F(2, 42)=9.80$ ,  $p<.001$ ) and a significant interaction between onset type and group,  $F(4, 84)=5.41$ ,  $p=.001$ . In addition the effect size (Cohen's  $d$ ) was computed for the comparisons between DD and L-IQ groups, and was found to be very large for the abrupt ( $d=1.05$ ) and the ramped-10 frame ( $d=1.09$ ) tasks, whilst a smaller, but still large effect size was found for the ramped-4 frame task ( $d=.87$ ). Similar effect sizes were also found for comparisons between the DD and H-IQ groups (abrupt=1.04; ramped-4 frame=1.09; ramped-10 frame=1.16).

We compared the contrast velocity (threshold rate of contrast change over total stimulus onset duration) between the two ramped tasks utilised here in order to investigate whether we could replicate the previous finding of a constant contrast velocity across a range of ramped stimulus onset durations. Contrast velocity was calculated by dividing each individual's threshold

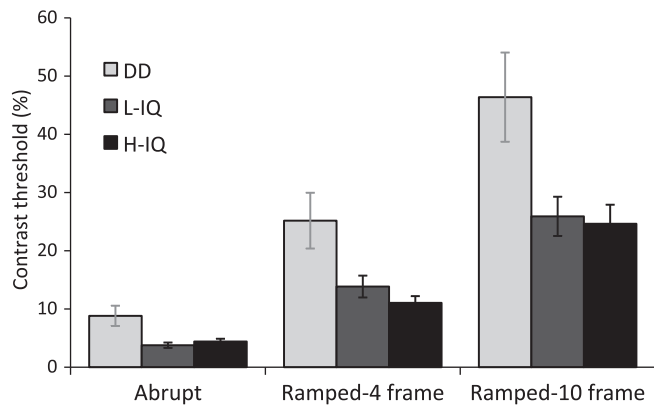
contrast by the number of frames presented during onset (i.e., either 4 or 10). Surprisingly, comparing contrast velocity between the ramped 4 frames and the 10 frames tasks demonstrated that whilst the DD group (6.8% and 4.6%,  $t(13)=2.54$ ,  $p=.025$ ) had significantly reduced contrast velocities with the longer ramped onset stimulus presentation ( $ps < .05$ ), both the L-IQ group (3.0% and 2.4%, respectively,  $t(15)=1.59$ ,  $p=.13$ ) and the H-IQ group (2.5% and 2.2%, respectively,  $t(16)=0.72$ ,  $p=.48$ ) did not show such differences.

Of further interest, was an investigation of the contrast change at the first frame of presentation, since this allows comparison of the contrast change with the two ramped onset tasks alongside the abrupt onset task (contrast change at the first frame in this case is equal to threshold performance, given that contrast remains constant over the duration of stimulus presentation). Fig. 4 demonstrates that although each group requires a slightly different contrast after the first frame depending on the task, all three groups keep within a consistent range of contrasts, with the two control groups very consistent, but not overlapping with the DD group.

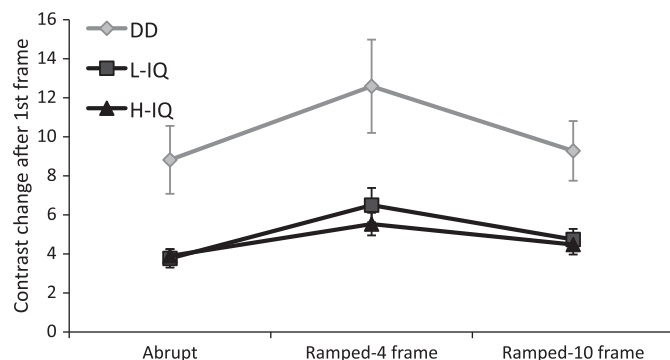
#### 4. Discussion

We have here established large and consistent differences in both abrupt and ramped onset flicker-defined form contrast sensitivity between groups of children with developmental dyslexia and age and non-verbal intelligence matched controls. This difference was also present when a higher non-verbal intelligence group was compared, thus eliminating intelligence as a likely explanation. Due to the high temporal rate of phase reversing flicker, all phantom-contour stimuli were expected to exclude the possibility of subcortical parvocellular processing and preferentially require magnocellular processing. Thus the current data indicates support for magnocellular impairment in dyslexia, which is consistent with a large body of research showing selective magnocellular deficits in dyslexia (Borsting et al., 1996; Chase & Jenner, 1993; Sperling et al., 2003; Stein, 2001; Wang et al., 2010). The very large effect sizes established here (in particular for the abrupt and 10 frame ramped conditions) are larger than most previous reports of visual deficits in dyslexia, suggesting flicker-defined form contrast sensitivity, and indeed other tasks involving high temporal frequencies are the most efficient at discriminating children with dyslexia from suitably matched controls (see Table 3).

While our previous study into the development of abrupt flicker contrast sensitivity task in children (Barnard et al., 1998)



**Fig. 3.** Contrast thresholds for the developmental dyslexia (DD) and the two chronological age-matched (CA) control groups (lower nonverbal intelligence-matched (L-IQ) and higher nonverbal intelligence (H-IQ)) were significantly different for each of the abrupt onset, ramped-4 frame and ramped-10 frame onset tasks.



**Fig. 4.** Contrast change at the first frame of the stimulus for threshold recognition performance for the developmental dyslexia (DD) and the chronological age-matched (CA) control groups. The DD group required a contrast change with means ranging between 8.8% and 12.6% depending on the task, whilst the CA group showed a mean range of between 4% and 6%.

**Table 3**  
Comparison of current and previous studies finding a difference between children with dyslexia and controls on tasks measuring magnocellular, dorsal stream or parietal visual processing.

Author	Task	Effect size (Cohen's <i>d</i> )
Martin and Lovegrove (1984)	Contrast sensitivity (spatial frequency=1c/d, stimulus duration=350 ms, screen masked to subtend 2 deg visual angle)	0.72 <sup>a</sup>
Cornelissen et al. (1995)	Motion coherence (long lifetime of each dot)	0.81
Sperling et al. (2003)	Phantom contour flicker fusion threshold	1.09
Rutkowski et al. (2003)	Change blindness (presentation time of 1st stimulus)	0.55
Pellicano & Gibson (2008)	Motion coherence (limited lifetime of each dot)	1.32
Pellicano et al. (2008)	Flicker contrast sensitivity (low spatial frequency, temporal frequency=10 Hz)	0.72
Wang et al. (2010)	Motion direction discrimination (sine gratings, 10% contrast, spatial frequency=1 C/deg, speed=54 deg/s)	0.83
Current study	Abrupt flicker defined form contrast sensitivity (DD vs. low-IQ/high-IQ groups)	1.05/1.04
Current study	Ramped-4 frames flicker defined form contrast sensitivity (DD vs. low-IQ/high-IQ groups)	0.87/1.09
Current study	Ramped-10 frames flicker defined form contrast sensitivity (DD vs. low-IQ/high-IQ groups)	1.09/1.16

<sup>a</sup> Indicates effect size was estimated based on graphs from the original publication.

did not find a significant difference associated with reading disability, it is possible that the previous use of a temporal frequency of 33 Hz did not completely exclude parvocellular responses, given that primate physiology suggests parvocellular responses in LGN may respond to temporal frequencies up to 40 Hz (Derrington & Lennie, 1984; Hicks et al., 1983). Thus the high temporal flicker rate (58.5 Hz) used here would be expected to be too high for the subcortical parvocellular system, whilst the low contrast of abrupt stimuli near threshold also makes it highly likely that magnocellular responses would be necessary.

Our current contrast sensitivity results could then be interpreted in the same light as the findings of Sperling and colleagues who used a similar phantom contour flicker-defined form task to successfully discriminate threshold flicker rates between children (Sperling et al., 2003) and adults (Sperling et al., 2006) with dyslexia and a control group in a luminant, but not in an isoluminant condition. However, although these earlier reports lend support to the notion of a deficit in dyslexia specific to some level of the magnocellular system, Sperling reached a different conclusion, suggesting noise-exclusion deficits might be responsible (this point is discussed further below).

The current study sought to extend on these findings by including a ramped onset flickering E task that was expected to provide a more taxing magnocellular based task by reducing the activation of transient attention mechanisms through the dorsal stream (Kiely et al., 2007; Kveraga, Boshyan, & Bar, 2007; Laycock et al., 2007; Laycock, Cross, Lourenco, & Crewther, 2011; Levy, Walsh, & Lavidor, 2010). Indeed, as expected the ramped Flickering E tasks proved to be more difficult for all participants, requiring far higher contrast levels for perception, indicating that ramped onset stimuli are generally less effective at 'grabbing' visual attention. Interestingly, although the largest mean difference between DD and the control group with matched chronological age and nonverbal intelligence (L-IQ) was established for the longer ramped task (46% and 25%, respectively), both this and the abrupt task (9% and 4%, respectively) produced equally large effect sizes in discriminating groups. The fact that the two non-verbal intelligence control groups did not show any differences on any aspect of the three tasks, suggests that flicker-defined form contrast sensitivity is very likely to be more sensitive in picking up visual impairments rather than other more general intelligence factors in developmental dyslexia. Given also that the tasks used here are ideally suited to targeting magnocellular processing we would argue that such a difference between children with dyslexia and control groups presents clear evidence of a magnocellular deficit in developmental dyslexia.

The inclusion of ramped onset stimuli was expected to provide information regarding the involvement of the dorsal stream in transient processing. However, the similar effect-size for discriminating between children with dyslexia and controls established across all tasks leads us to some tentative conclusions regarding the ramped onset stimuli. It is possible to conclude that removing the transience of stimuli (i.e., ramped presentation), which was expected to reduce rapid stimulus-driven dorsal stream activation of attention mechanisms, did not impact on the size of the visual deficit established in the dyslexia group.

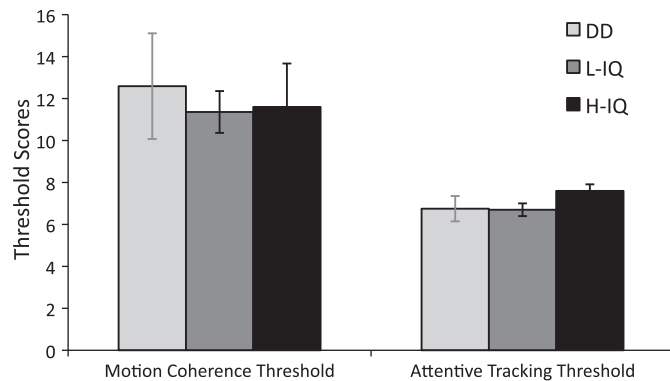
If the parvocellular system is ineffective in processing these high temporal frequency stimuli, then as onset duration increases (8.5 ms for abrupt, 68.8 ms and 172 ms for the two ramped tasks) and the abrupt transience is removed, it appears that form recognition ability is reduced (as evidenced by increased contrast required for detection). This suggests that ventral stream form recognition processing benefits from the early transient magnocellular system's ability to grab attention (Laycock, Crewther, Fitzgerald, & Crewther, 2009). Indeed current models of visual processing that incorporate sophisticated measures of temporal

sequencing of dorsal stream feedforward/feedback activation (Bar et al., 2006; Laycock et al., 2007) would predict that form recognition would become disproportionately more difficult with an impaired magnocellular activation of dorsal stream selective visual stimulus-driven attention. While the ramped onset task might be inherently unsuited to activating transient attention through the dorsal stream and instead might largely rely on ventral stream processing, the close similarity in effect size for abrupt versus ramped tasks suggests that the same neural network is likely at play. The current data cannot determine what single neural mechanism is involved.

Nevertheless, we propose that two possible explanations for the pattern of reported group differences, assuming a single neural mechanism is driving the data. First, on the assumption that ramped onset stimuli do indeed reduce the involvement of transience and dorsal stream-driven attention processes and given the similar effect observed when comparing groups on the ramped and abrupt tasks, it could be concluded that magnocellular/dorsal stream-driven attention is not the locus for the observed magnocellular deficit in the participants with dyslexia. This would leave open the possibility that direct magnocellular projections activating object recognition processes through the ventral stream, or merely the early (i.e., LGN or V1) magnocellular responses are impaired, if indeed these can be disambiguated. It is noteworthy that this interpretation is inconsistent with the interpretation coming from a body of research demonstrating dorsal stream deficits associated with dyslexia (Cornelissen et al., 1995; Eden et al., 1996; Pellicano & Gibson, 2008; Rutkowski et al., 2003).

A second possibility based on the hypothesis of a single neural mechanism rests on the comparisons made of the contrast change at the first frame for all tasks. The relative consistency of these values within each group indicates that even for the ramped tasks, which were expected to have reduced transience, a sufficient contrast increase after the first frame (i.e., an abrupt onset) might still have activated dorsal stream dominated attention processes. For example, the mean contrast change after the first frame for the dyslexia group and both control groups for the ramped-4 frame task was similar (though higher) than the contrast change for the abrupt task. In fact, 83% of all participants followed this trend. This finding also holds for the ramped-10 frame task, with the mean group change in contrast after the first frame higher (in 70% of all participants) than the abrupt contrast change at the first frame. As such, this leaves open the possibility that the contrast change at the first frame was always large enough (and therefore "abrupt" enough) to still activate transient attention through the dorsal stream. We suggest that this second explanation may be more parsimonious given the previously reported dorsal stream deficits in the literature (Cornelissen et al., 1995; Eden et al., 1996; Pellicano & Gibson, 2008; Rutkowski et al., 2003), though this remains to be tested empirically.

As alluded to earlier, Sperling et al. (2006) suggested that impaired flicker-rate threshold on a flicker-defined form task might be associated with a deficit of noise-exclusion rather than just magnocellular processing. Whether the same conclusion can be reached for the current data must be considered, even though Sperling et al.'s conclusion appears to be predominantly driven by the language impaired children with dyslexia, (see Fig. 2B in Sperling et al., 2005). Certainly our abrupt onset Flicker E task could be argued to consist of a relatively low signal-to-noise ratio, and thus may have magnified noise-exclusion deficits in our sample of children with dyslexia. However it remains difficult to determine the degree of noise in the flickering E stimuli used since the noise is necessarily required to perceive the signal (i.e., rather than being inter-dependent, the noise and signal are intrinsically dependent on each other). Furthermore, noise



**Fig. 5.** Performance of DD children and low- and high-nonverbal intelligence (L-IQ, H-IQ) groups on a motion coherence task requiring detection of the direction of signal dots moving left or right from noise dots with random directions, and an attentive tracking task requiring the tracking of three target white balls amongst a variable number of blue balls. In this task, target balls change colour to be indistinguishable from the distractor balls, but must be tracked and identified at the end of the trial. No significant differences were established between the DD and either control group (data re-drawn from Laycock et al., 2006; BioMed Central).

exclusion in other systems has been shown generally to rely on greater attention (Barutchu et al., 2010) which comes back to magnocellular driven dorsal stream attention systems. This point is particularly intriguing as Bullier (2001), Bar et al. (2006) and Laycock et al. (2007), for example, have all argued that the magnocellular system provides an initial rapid low spatial frequency signal, possibly through the dorsal stream to parietal and frontal regions. This early activation is suggested to provide an initial global analysis of the object foreground/background segregation, before feedback signals into the inferotemporal cortex fill in the details. Thus, although it is possible that dyslexia is indeed associated with noise-exclusion deficits, such noise must be related to the magnocellular pathway and its subsequent cortical connections (Barutchu et al., 2010).

A final concern with the current data, relates to the fact that children with dyslexia showed impaired performance on all tasks described here, leaving open the possibility that generalised attention rather than specific visual deficits are explanatory. However, the current sample of children in a separate experiment also completed a set of psychophysical tasks assessing dorsal stream and attentional processing (Laycock et al., 2006). In particular, we draw attention to data from a motion coherence task commonly used to assess dorsal stream functioning (Pellicano & Gibson, 2008; Talcott, Hansen, Assoku, & Stein, 2000), and a sustained attentive tracking task shown to activate parietal cortex (Culham et al., 1998). As can be seen from Fig. 5, the DD group showed no significant impairment when compared with both the high- and low nonverbal intelligence control groups on both tasks. Performance on these visual tasks thus serves as a control, indicating that deficits in this sample of children with reading impairment appears to be specific to magnocellular processing.

In summary, we have shown that groups of good and poor readers are significantly and clinically differentiated on a set of purportedly magnocellular processing tasks. Reduced contrast sensitivity for the group of children with dyslexia supports the notion of a substantial magnocellular deficit in dyslexia. The use of ramped onset flicker contrast sensitivity tasks in addition to the previously reported abrupt onset version indicates that these more difficult stimuli may be theoretically and educationally useful in assessing different aspects of magnocellular processing (subcortical, dorsal or ventral streams), which in conjunction with brain imaging and stimulation techniques might further elucidate the nature of magnocellular impairments in dyslexia.

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