



Why is the processing of global motion impaired in adults with developmental dyslexia?



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ABSTRACT

Individuals with dyslexia are purported to have a selective dorsal stream impairment that manifests as a deficit in perceiving visual global motion relative to global form. However, the underlying nature of the visual deficit in readers with dyslexia remains unclear. It may be indicative of a difficulty with motion detection, temporal processing, or any task that necessitates integration of local visual information across multiple dimensions (i.e. both across space and over time). To disentangle these possibilities we administered four diagnostic global motion and global form tasks to a large sample of adult readers ($N = 106$) to characterise their perceptual abilities. Two sets of analyses were conducted. First, to investigate if general reading ability is associated with performance on the visual tasks across the entire sample, a composite reading score was calculated and entered into a series of continuous regression analyses. Next, to investigate if the performance of readers with dyslexia differs from that of good readers on the visual tasks we identified a group of forty-three individuals for whom phonological decoding was specifically impaired, consistent with the dyslexic profile, and compared their performance with that of good readers who did not exhibit a phonemic deficit. Both analyses yielded a similar pattern of results. Consistent with previous research, coherence thresholds of poor readers were elevated on a random-dot global motion task and a spatially one-dimensional (1-D) global motion task, but no difference was found on a static global form task. However, our results extend those of previous studies by demonstrating that poor readers exhibited impaired performance on a temporally-defined global form task, a finding that is difficult to reconcile with the dorsal stream vulnerability hypothesis. This suggests that the visual deficit in developmental dyslexia does not reflect an impairment detecting motion *per se*. It is better characterised as a difficulty processing temporal information, which is exacerbated when local visual cues have to be integrated across multiple (>2) dimensions.

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1. Introduction

A predominant view is that human visual cortex is organised into two anatomically distinct and functionally independent processing streams or pathways, each specialised for encoding different types of visual information. The dorsal stream projects from primary visual cortex to the parietal lobes and is often referred to as the “where” pathway, as it is involved in tasks such as determining the global (overall) motion of objects, spatial cognition and visual motor planning. The ventral pathway projects from visual cortex to the temporal lobes and has been termed the “what” pathway, as it is involved in tasks such as global shape perception, visual memory and recognition of familiar objects/faces (Milner

& Goodale, 1995; Ungerleider & Mishkin, 1982). Vulnerability of the dorsal stream has been suggested as a primary origin of impairment in individuals with developmental dyslexia, and a range of other neurodevelopmental disorders (e.g. Williams syndrome, autism spectrum disorder, developmental dyspraxia). Dorsal pathway vulnerability is claimed to manifest as a selective deficit in processing global motion relative to global form (Braddick, Atkinson, & Wattam-Bell, 2003). However the selectivity of this deficit is equivocal (Grinter, Maybery, & Badcock, 2010).

Several studies have used random-dot kinematograms (RDKs) to investigate the dorsal stream vulnerability hypothesis (see Benassi, Simonelli, Giovagnoli, & Bolzani, 2010 for review). These stimuli comprise a series of discrete images, each containing a pattern of individual local dots, that when presented in succession, create the perception of apparent motion. Some of the dots are constrained to move in a common direction (*signal dots*), whilst others move randomly (*noise dots*). By changing the relative

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proportion of signal and noise dots the coherence of the stimulus is varied. Motion coherence thresholds are defined as the minimum number of signals dots needed to detect or identify reliably the global motion direction (Britten, Shadlen, Newsome, & Movshon, 1992; Newsome & Paré, 1988). To judge the overall direction of motion in a RDK local motion information has to be integrated (i.e. pooled, compared or combined) across two spatial dimensions and over time.

Cornelissen, Richardson, Mason, Fowler, and Stein (1995) were amongst the first to investigate the processing of global motion in poor readers classified as dyslexic. They administered a task originally devised by Wattam-Bell (1992). The stimuli comprised two RDKs. One of the patterns was segregated into three horizontal bands, whereas the other was spatially uniform. Signal dots in the former moved in opposite directions in adjacent bands. Those in the latter moved in a common direction. The participants' task was to detect the segregated pattern. Consistent with the dorsal stream vulnerability hypothesis, poor readers' coherence thresholds were significantly higher (1.3 times) than those of control readers. However, there was considerable heterogeneity in the performance of the two groups, a common finding in studies of developmental dyslexia (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Ramus et al., 2003; Roach, Edwards, & Hogben, 2004; White et al., 2006), that recent research suggests might reflect genotypic variation (Cicchini, Marino, Mascheretti, Perani, & Morrone, 2015; Gori et al., 2014).

The stimuli in the Cornelissen et al. (1995) study were spatially complex. To perform the task participants had to detect directional shearing between horizontal bands, rather than the direction of global motion *per se*. Thus one cannot determine whether poor readers have a difficulty processing visual motion in general or a difficulty detecting motion contrast. To address this issue, Raymond and Sorensen (1998) administered a simpler, conventional random-dot global motion task. A single RDK was presented on each trial, the participants had to judge the overall direction of the stimulus and motion coherence was varied. Poor readers' coherence thresholds were significantly higher (1.8 times) than those of controls. However, there was no group difference when the RDKs consisted of only two images (i.e. the dots underwent a single displacement). These results imply that poor readers have a particular difficulty integrating local motion signals over extended trajectories, rather than a general difficulty with motion detection.

Talcott, Hansen, Assoku, and Stein (2000) sought to determine whether the perceptual deficit in poor readers reflects anomalous spatial or temporal integration. In two separate experiments, the mean dot density and exposure duration of random-dot stimuli, similar to those used by Raymond and Sorensen (1998) were manipulated. Results showed that overall poor readers' coherence thresholds were significantly higher than those of normal readers in both experiments and there was no significant interaction between subject group and dot density nor subject group and duration, demonstrating that the spatiotemporal manipulations had similar effects regardless of reading ability. However, at the highest dot density tested (12.2 dots/deg²) the performance of readers with dyslexia approached that of the controls, suggesting a marginal improvement perhaps as a consequence of the greater motion energy present in the denser RDKs facilitating the poor readers. Talcott et al. speculated that greater motion energy might be expected to facilitate performance if motion sensors have a relatively low response gain, more inherent noise or sparser spatial sampling but no firm conclusions could be drawn.

An alternative hypothesis is that deficits on sensory tasks associated with poor reading and dyslexia are the result of impairments in external-noise exclusion (Sperling, Lu, Manis, & Seidenberg, 2005). Within this framework relatively poor perfor-

mance on RDK tasks, in which coherence thresholds are used as a measure of sensitivity, is directly indicative of an underlying problem in segregating the signal dots from the noise dots. Although this noise-exclusion hypothesis has received support (e.g. Sperling, Lu, Manis, & Seidenberg, 2006) it fails to explain why some individuals with dyslexia often exhibit relatively normal performance on analogous static global form tasks that also contain high levels of visual noise. For example, Hansen, Stein, Orde, Winter, and Talcott (2001) administered two psychophysical tasks: a random-dot global motion task and a static global form task. The latter was devised by Atkinson et al. (1997) to investigate the processing of global form in individuals with Williams syndrome. It is assumed to provide a sensitive measure of ventral stream capability because it evokes a BOLD response in cortical areas that have been implicated in the processing of global form (Braddick, O'Brien, Wattam-Bell, Atkinson, & Turner, 2000). The stimuli in the task are similar to the random-dot patterns described above, except they comprise static line segments rather than dots. They can either be orientated coherently to form a concentric target or randomly. Poor readers' coherence thresholds were significantly higher than those of controls on the random-dot global motion task but not the static global form task. This result is difficult to reconcile with a general noise-exclusion hypothesis but is consistent with the dorsal stream vulnerability hypothesis.

A related issue concerns the degree to which motion segmentation processes are normal in individuals with dyslexia. This is important because under natural viewing the visual system has to satisfy the competing requirements of integrating local motion signals that belong to a common surface or object but also segregating those arising from other objects in the world (e.g. Braddick, 1993). How the visual system achieves this delicate balance is still unknown but there is some evidence to suggest that motion segmentation mechanisms may also be impaired in poor readers. Hill and Raymond (2002) investigated this issue using transparent motion stimuli generated by constraining half of the dots in a RDK to move coherently in a horizontal direction (leftwards or rightwards) and others to move vertically (upwards or downwards). This created the perception of two segregated and transparent surfaces sliding across each other and the subjects' task was to identify the two directions of motion present on each trial. The exposure duration of the stimulus was manipulated by changing the number of images comprising the motion sequence. A transparency threshold was calculated, which corresponded to the minimum exposure duration needed to achieve 75% correct performance. Results showed that poor readers' transparency thresholds were over three times higher than those of controls in that they required an additional 339 ms to identify the two directions of simultaneous motion.

Recently it has been suggested that a deficit in the processing of global motion only occurs in a sub-group of individuals, which might explain why performance on random-dot tasks is heterogeneous (Amitay et al., 2002; Ramus et al., 2003; White et al., 2006). Approximately 10–17% of poor readers classified as dyslexic and 4% of controls have a deletion on intron 2 of the DCDC2 gene (Meng et al., 2005; Wilcke et al., 2009). Studies have shown that individuals with this genotypic deletion (hereafter referred to as DCDC2d) have altered white matter tracts in brain regions implicated in reading (e.g. Darki, Peyrard-Janvid, Matsson, Kere, & Klingberg, 2014). Interestingly, morphological changes have also been reported in extrastriate visual areas such as V5/MT (Morrone et al., 2011). Cicchini et al. (2015) administered a motion discrimination task to groups of poor readers with and without DCDC2d. The results showed that poor readers with the deletion had more profound impairments than those without DCDC2d. However, the latter performed significantly worse than controls, which suggests that factors other than genotypic variation are

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