

Running title: Dorsal-stream functioning in autism and dyslexia

Investigating the functional integrity of the dorsal visual pathway
in autism and dyslexia

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Abstract

Numerous reports of elevated global motion thresholds across a variety of neurodevelopmental disorders have prompted researchers to suggest that abnormalities in global motion perception are a result of a general deficiency in the dorsal visual pathway. To test this hypothesis, we assessed the integrity of the dorsal visual pathway at lower, subcortical (sensitivity to flicker contrast) and higher, cortical (sensitivity to global motion) levels in children with autism, children with dyslexia, and typically developing children, of similar age and ability. While children with autism demonstrated intact lower-level, but impaired higher-level dorsal-stream functioning, children with dyslexia displayed abnormalities at both lower and higher levels of the dorsal visual stream. These findings suggest that these disorders can be dissociated according to the origin of the impairment along the dorsal-stream pathway. Implications for general cross-syndrome accounts are discussed.

Several reports of reduced global motion sensitivity in autism (Bertone, Mottron, Jelenic, & Faubert, 2005; Milne *et al.*, 2002; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Spencer *et al.*, 2000; though see White *et al.*, 2006) have sparked discussion of whether the dorsal-stream system – the primary visual pathway responsible predominantly for processing dynamic stimuli – is disrupted (see Milne, Swettenham, & Campbell, 2005). Yet, reduced sensitivity to coherent motion has also been documented in dyslexia (Hansen *et al.*, 2001), fragile X syndrome (Kogan *et al.*, 2004), and Williams syndrome (Atkinson *et al.*, 1997), indicating that this might be a common feature of a variety of neurodevelopmental disorders. Based on these observations, Braddick, Atkinson, and Wattam-Bell (2003) suggested that reduced sensitivity to coherent motion might be an epiphenomenon of anomalous brain development. They propose that the neural systems subserving motion perception emerge later during development than those involved in form perception, rendering dynamic visual processing more susceptible to insult in developmental disorders – the so-called ‘dorsal-stream vulnerability hypothesis’. Accordingly, they postulate qualitatively similar impairments in the dorsal-stream pathway across developmental disorders. Here, we test this claim by investigating the functional integrity of the dorsal visual pathway in autism and dyslexia.

The dorsal-stream pathway is a system with progressively more complex processing occurring at higher levels. As such, reduced global motion sensitivity could arise from abnormalities at lower *and/or* higher levels along this pathway. Psychophysical coherent motion tasks, in which participants are presented with a portion of coherently moving (signal) dots, set amongst a background of randomly-moving (noise) dots, are used typically to index *higher-level* dorsal-stream functioning. The percentage of signal dots is varied from trial to trial; sensitivity to coherent motion is determined by the percentage of dots needed to perceive the direction of movement. Neuronal firing rates in higher cortical areas of the dorsal stream (area V5/MT) are related strongly to

coherent motion perception (e.g., Newsome, Britten, & Movshon, 1989), and it is in this cortical region that local directional signals are pooled to create a global representation of motion (Newsome & Paré, 1988). Sensitivity to coherent movement therefore relies on the efficiency of cooperative mechanisms at higher levels of the dorsal visual pathway.

Crucially, however, such sensitivity also relies on the quality of information fed to V5/MT from lower, subcortical levels. The dorsal cortical stream receives predominant input from magnocellular neurons in the lateral geniculate nucleus (Merigan, Byrne, & Maunsell, 1991) and area V1 (Schiller, Logothetis, & Charles, 1990). Magno neurons show high sensitivity to rapidly moving (e.g., flickering) stimuli and provide the best response to stimuli composed of low spatial and high temporal frequencies (Merigan *et al.*, 1991). Thus, it remains plausible that abnormalities at lower (subcortical) and/or higher (extrastriate) levels of the dorsal-stream pathway could contribute to difficulties detecting global motion in neurodevelopmental disorders.

In autism, researchers have begun to elucidate the origin of reduced global motion sensitivity by probing perceptual processing at different levels along the dorsal-stream hierarchy. Bertone *et al.* (2005) and Pellicano *et al.* (2005) assessed dorsal-stream functioning at both lower (sensitivity to flicker contrast) and higher (sensitivity to complex motion) levels, and found evidence of unimpaired early visual processing despite deficits in global motion perception. Their results indicate that abnormalities in complex motion perception in autism arise from higher-level dorsal cortical abnormalities, rather than from lower-level magnocellular dysfunction.

In dyslexia, dorsal-stream functioning has been the subject of intense investigation owing largely to the hypothesis that impaired magnocellular/dorsal-stream functioning is a primary etiological factor in the development of reading problems (Stein & Walsh, 1997). Some studies (e.g., Martin & Lovegrove, 1988) have evidenced

diminished contrast sensitivity in dyslexia¹, but others have since failed to demonstrate that visual deficits are specific to the magnocellular system (e.g., Amitay, Ben-Yehudah, Banai, & Ahissar, 2002). As such, it remains unclear whether motion coherence abnormalities in dyslexia are cortical or subcortical in origin.

There are, therefore, reasons to suspect that reduced sensitivity to coherent motion in autism and dyslexia might arise for different reasons, contrary to Braddick *et al.*'s (2003) proposal. It is insufficient, however, to assess the validity of the dorsal-stream vulnerability hypothesis by comparing published studies of specific developmental disorders; not only do these studies employ different visual tasks but their participants vary widely in developmental level.

In the present study, we assessed children with autism, children with dyslexia, and typical children, *of similar age and ability*, on two psychophysical tasks, one targeting lower-level (magnocellular) functioning, and another tapping higher-level dorsal-stream functioning. We investigated whether abnormalities in global motion perception in autism and dyslexia arise from a common neuropathological locus or whether these disorders might be dissociated according to the origin of impairment along the dorsal-stream pathway.

Method

We present novel analyses of combined data which were collected using the same procedures (including task scripts) in the same laboratory, and under similar experimental conditions. The autism data were reported previously by Pellicano *et al.* (2005) and the dyslexia data were reported by Gibson, Hogben, and Fletcher (2006).

Participants

¹ The stimuli used in these studies, however, may have been poorly suited to isolating magnocellular function (Skottun, 2000).

A total of 122 children within a restricted age range (8-12 years) participated: 20 with autism, 41 with dyslexia, and 61 typically developing children². These groups were well-matched in terms of chronological age, $F(2, 119)=.78, p=.46$, and nonverbal IQ, $F(2, 119)=.01, p=.94$ (see Table 1). The groups did differ in terms of verbal IQ, $F(2, 119)=3.24, p=.04$, such that typical children scored significantly higher than children with autism, $t(119)=2.34, p=.02$, and slightly higher than children with dyslexia, $t(119)=1.69, p=.09$. Importantly, the two clinical groups did not differ on verbal IQ ($p=.34$).

Children with autism received an independent clinical diagnosis, according to DSM-IV criteria (APA, 2000), and also met autism spectrum criteria on the Autism Diagnostic Interview – Revised (ADI-R; Lord et al., 1994). Children with dyslexia were recruited through local clinics and screening exercises at local schools. All children had a reading age that lagged >18 months behind their chronological age ($M=35.0$ months; $SD=13.1$), as assessed by the Neale Analysis of Reading Ability (Neale, 1999) (see Snowling, 2000). Children were included only if they had nonverbal IQ scores ≥ 85 , normal/corrected-to-normal vision, and no other medical/psychiatric conditions. Both visual tasks were completed in the same session, interleaved by measures of IQ.

Psychophysical tasks

Flicker contrast sensitivity (FCS). In line with Evans, Drasdo, and Richards (1994), we used a stimulus of low spatial frequency *and* high temporal frequency, which is known to activate preferentially magnocellular neurons (Skottun, 2000). The flickering stimulus (10Hz) was a 3.15° Gaussian blob (mean luminance 20 cd/m^2) presented in centre-screen for 1 s, surrounded by a field of matched space-averaged luminance. Children saw a central fixation cross, followed by two 1000 ms intervals (accompanied by a tone) separated by a second screen with a fixation cross. One of these intervals (chosen

² Children who failed to complete any task (typically developing: $n=3$; dyslexia: $n=3$) were excluded from this study. Thus, the mean scores reported herein (for the dyslexia sample) will differ slightly from those reported in Gibson *et al.* (2006).

randomly) contained the flickering stimulus and the other contained a zero-contrast version of the same mean luminance. Children indicated the interval containing the flickering stimulus. Initially, the contrast level was set at an easy level (5%), which was varied subsequently by a two-up/one-down staircase rule. The threshold (percentage of contrast needed to detect flicker) was transformed to contrast sensitivity (1000/threshold).

Global dot motion (GDM). Stimuli consisted of twenty 30 ms frames (total trial duration of 600 ms) composed of 100 bright dots (47.4 cd/m², individual dots subtending 0.11°), randomly plotted on a black background (<1 cd/m²). During each trial, a variable proportion of these (signal) dots moved coherently, upwards or downwards (randomly selected), while the remaining (noise) dots moved in random directions. Children identified the direction of coherent movement. The 'lifetime' of each signal dot was limited to a single animation frame to prevent children from following the trajectory of a single signal dot. The spatial step size for each dot was 0.19° (velocity 6.33 °/s). The first trial began at 20% coherence, and this level varied by a two-up/one-down staircase rule. The threshold reflected the percentage of signal dots required to perceive global motion.

Stimuli for both tasks were presented on a Cambridge Research System VSG2/3 and a Sony Trinitron GDM-20SEI monitor (388mm×292mm screen) in a darkened room (viewing distance=50cm). Each task consisted of 10 practice trials followed by two blocks of 60 trials. Audio feedback was provided. Thresholds were estimated using a two-alternative forced-choice procedure, and an adaptive PEST procedure, which converged on the 75% correct performance level. The threshold was defined as the mean level of the final four reversals.

Results

Initial data screening identified several outliers within each group. To reduce the impact of these outlying cases, scores more than 3 SDs above/below the group mean for any task (7% of data) were replaced with the value representing 2.5 SDs above/below their group mean (Tabachnick & Fidell, 2007). Split-half reliability analysis (Spearman-Brown correction) on the two blocks of each psychophysical task yielded moderately-high reliability coefficients (FCS: $r=.80$; GDM: $r=.79$). The mean of the two blocks indexed children's performance. GDM scores were recoded so that high scores reflected good sensitivity to global motion. Scores were converted to z scores using the typically developing group as the normative standard $[(\text{score} - \text{Mean}_{\text{control}}) / SD_{\text{control}}]$. All analyses are performed using the z scores for each variable (see Table 1 for untransformed mean scores). There were no significant effects of gender.

 Insert Table 1 about here

Figure 1 shows the distribution of z scores for all groups on each psychophysical task. The pattern of results is striking: children with dyslexia obtained lower scores on the FCS task compared with children with autism and typically developing children (Figure 1a), yet *both* clinical groups achieved lower thresholds on the GDM task (Figure 1b), relative to typical children.

A repeated-measures ANOVA on the z scores with task (FCS, GDM) and group (typical, autism, dyslexia) as factors revealed significant main effects of task, $F(1, 119)=16.60, p<.001$, and group, $F(2, 119)=13.02, p<.001$, and a significant task x group interaction, $F(2, 119)=5.50, p<.001$. Separate one-way ANOVAs were conducted on z scores for each task separately. For the FCS task, there was a main effect of group, $F(2, 119)=7.55, p<.001$. Pairwise comparisons revealed that children with dyslexia were less sensitive to flickering stimuli than typical children, $t(119)=3.62, p<.001, d=.72$, and

children with autism, $t(119)=2.87, p<.005, d=.85$. No difference emerged between the autism and typical groups, $t(119)=.85, ns$.

For GDM (Figure 1b), there was also a main effect of group, $F(2, 119)=10.82, p<.001$; children with autism *and* children with dyslexia obtained higher GDM thresholds than typical children, $t(119)=2.41, p<.01, d=1.01$ and $t(119)=4.54, p<.001, d=1.32$, respectively. Performance was similar across clinical groups, $t(119)=1.08, ns$.

 Insert Figure 1 about here

Notably, there was substantial variability in threshold estimates within clinical groups. To examine this further, we calculated the percentage of the clinical groups that displayed an ‘impairment’ (scored more than 1.65 SDs below the typically developing group mean; Ramus, 2004). This revealed that 24% of the dyslexia group were impaired on the FCS task (Figure 1a), and 45% and 36% of the autism and dyslexia groups, respectively, displayed poor global motion perception (Figure 1b). This variation in scores was related neither to general developmental variables, including chronological age, verbal IQ or nonverbal IQ (all $ps>.20$), nor indices of severity³ for either clinical condition (all $ps>.38$). Individual variation in FCS performance was related, however, to individual variation in GDM performance in the typically developing group, $r(59)=.36, p=.005$, and the dyslexia group, $r(39)=.37, p<.05$, but not the autism group, $p=.91$.

Discussion

Contrary to Braddick et al.’s (2003) proposal, the present results demonstrate that autism and dyslexia can be dissociated at a perceptual level: children with autism, on average, showed a sparing of early (magnocellular) levels, but a deficit in higher-level global motion perception, while children with dyslexia, on average, demonstrated atypicalities at

³ Total scores from the ADI-R (Lord et al., 1994) were used to index degree of autistic symptomatology, and the degree of reading lag (in months) was used to index severity of dyslexia.

both lower and higher levels of the dorsal visual stream. Furthermore, these deficits were present only in a minority of children within each clinical group, and were unrelated to the severity of either condition, questioning whether such deficits contribute to the pathogenesis of either condition.

To account for our findings, one could extend Braddick *et al.*'s proposal by appealing to the role of timing during development; dorsal-stream functioning could be susceptible to insult at distinct stages during development in autism and dyslexia, resulting in diverging developmental trajectories ultimately producing different effects on motion perception. Although plausible, this idea has difficulty explaining why dorsal-stream functioning remains unaffected in a significant percentage of children with autism/dyslexia.

One alternative cross-syndrome account purports to explain why not all children with autism/dyslexia displayed deficits on dorsal-stream tasks. Ramus (2004) proposes that sensorimotor difficulties (of which a dorsal-stream impairment is but one manifestation) represent non-specific markers of neurodevelopmental disorders, which result from, rather than cause, specific cortical abnormalities. Sensorimotor dysfunction is held to be an 'optional' characteristic, as opposed to a defining one, thus explaining why it is not pervasive in either disorder. Ramus argues that, for dyslexia, primary cortical anomalies in regions responsible for phonological processing disrupt the development of this cognitive function, which, under certain hormonal conditions, could trigger additional sensory dysfunction. A similar case could conceivably be made for autism. On Ramus' view, one might expect that the individuals who show greatest cortical anomalies will be those who develop sensory dysfunction. Yet a link between severity and degree of visual processing abnormality was not observed here. Certainly, these general cross-syndrome hypotheses require further specification and empirical validation.

Our results show that the nature of dorsal-stream functioning in neurodevelopmental disorders appears more complex than previously anticipated. Although considerable efforts were made to ensure that methods and procedures were commensurate across autism and dyslexia samples it remains possible that the use of different IQ measures may have lead to actual group differences on matching variables. The fact that verbal and nonverbal IQ scores were unrelated to performance on either visual task renders it unlikely that the pattern of findings is a consequence of general cognitive functioning. Also, the absence of appropriate (non-motion) control tasks raises an alternative explanation for the results: that poor performance by *some* children, particularly those with dyslexia, might be explained best by general nonsensory factors, such as poor attention (Roach, Edwards, & Hogben, 2004). The presence of a significant correlation between psychophysical tasks in the dyslexia group, however, speaks against this possibility. Nevertheless, the inclusion of analogous low- and high-level dorsal- *and* ventral-stream tasks in future work is integral to demonstrate that general cognitive factors have little impact upon performance.

Our results highlight the need to conduct systematic cross-syndrome investigations of visual functioning in children *of similar age and ability*. Longitudinal studies are necessary to trace the developmental trajectory of motion perception within each disorder, and determine, ultimately, whether reported visual processing abnormalities are a potential endophenotype of neurodevelopmental disorders.

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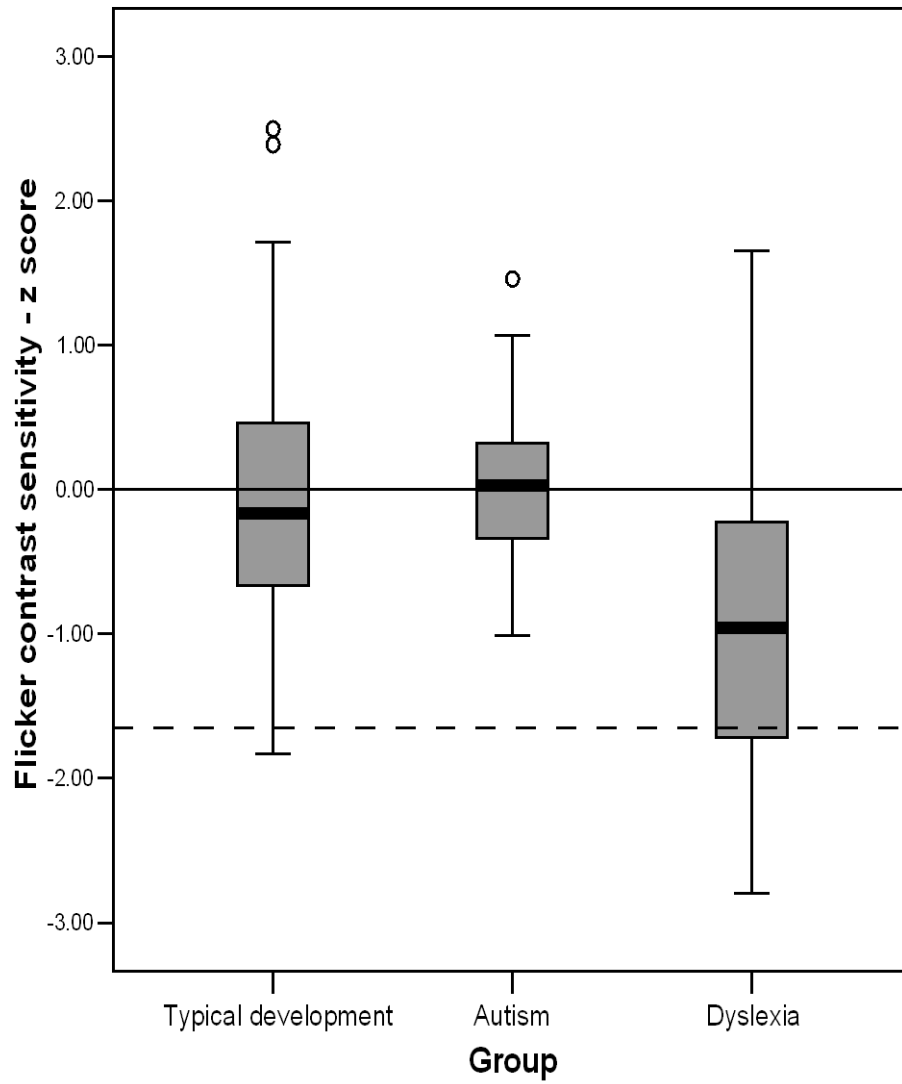
Table 1. *Participant characteristics.*

	Group		
	Typical development	Autism	Dyslexia
<i>N</i>	61	20	41
Male: female	47 : 14	18 : 2	26 : 15
Chronological age			
Mean (SD)	118.52 (13.02)	115.05 (16.55)	119.68 (13.23)
Range	96-151	97-148	97-148
Nonverbal IQ ^a			
Mean (SD)	106.82 (12.90)	107.10 (8.95)	106.76 (11.38)
Range	85-146	89-119	89-131
Verbal IQ ^b			
Mean (SD)	104.28 (11.60)	97.00 (15.29)	100.15 (11.05)
Range	77-131	76 - 121	76 – 131
Global motion task			
Mean threshold (SD)	14.02 (6.70)	22.40 (13.78)	26.38 (19.60)
Range	4.41 – 40.53	5.31 – 54.44	5.19 – 75.50
Flicker Contrast Sensitivity task			
Mean score (SD)	129.18 (42.13)	131.31 (24.06)	98.07 (49.41)
Range	52.22 – 254.28	86.73 – 190.68	11.22 – 221.99

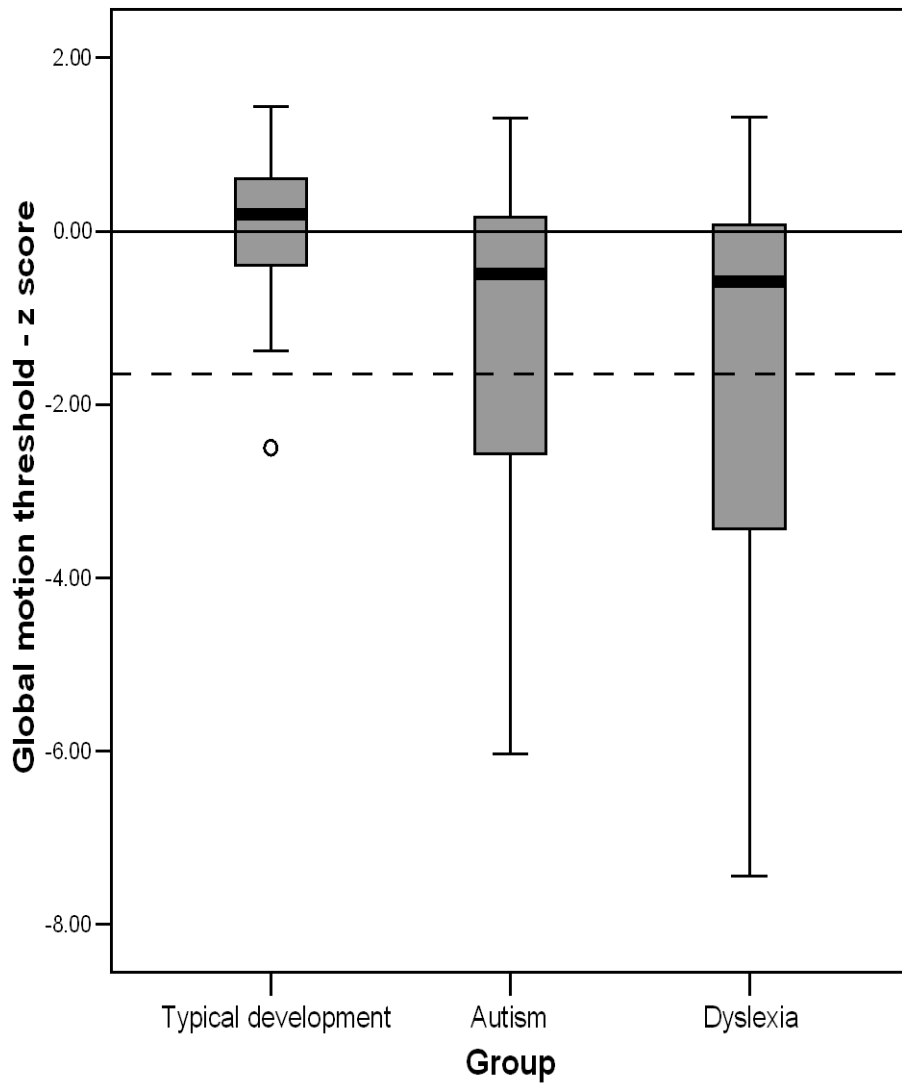
Notes: ^aNonverbal IQ was measured by either the Raven's Standard Progressive Matrices (Raven, Court, & Raven, 1992) or the Performance subscale of the Wechsler Abbreviated Intelligence Scales (WASI; Wechsler, 1999). ^bVerbal IQ was measured by either the Peabody Picture Vocabulary Test – Third Edition (Dunn & Dunn, 1997) or the Verbal subscale of the WASI.

Figure captions

Figure 1. Box plots showing performance on (a) the global dot motion task, and (b) the flicker contrast sensitivity task for typically developing children, children with autism, and children with dyslexia. Upper and lower ends of boxes represent 75th and 25th percentiles, respectively. ‘Whiskers’ attached to the boxes extend out to include 100% of the data (with the exception of outliers, represented by open circles). The median of the distribution is depicted by a solid black line bisecting the box. The solid black line intersecting the Y axis represents the mean \bar{x} score of the typically developing group, while the dotted line intersecting the Y axis represents 1.65 SDs below the mean score of the typically developing group.



(a)



(b)

Figure 1.