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Periventricular brain injury, visual motion processing, and reading and spelling abilities in children who were extremely low birthweight

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Abstract

Among children born at *extremely low birthweight* (ELBW: <1000 g at birth) there is an association between the presence of periventricular brain injury (PVBI) and lowered performance on tests of reading and spelling ability. The present study was designed to determine if this association might be related to underlying dysfunction in the subcortical magnocellular visual pathway or its cortical targets in the dorsal stream, a prediction motivated by the magnocellular theory of dyslexia. Thirty-five ELBW children were divided into two groups based upon the presence or absence of PVBI (*no PVBI*, $n = 11$; *PVBI*, $n = 24$). The performance of these two groups was compared to that of a group of healthy full term children ($n = 12$) on a motion-defined form recognition task believed to tap into the functioning of the magnocellular pathway and/or the dorsal stream. ELBW children did, in fact, show a striking impairment on this task, with 71% of the sample performing at a level more than three standard deviations below the mean of full term controls. Surprisingly, their difficulties were not found to be related to either the presence of brain injury (verified by neonatal cranial ultrasound) or to problems with reading or spelling. An association was documented, however, between difficulties with motion processing and performance on several subtests of the Performance IQ scale of the Wechsler Intelligence Scale for Children–Third Edition. This latter finding is consistent with our earlier suggestion that magnocellular pathway/dorsal stream dysfunction may underlie problems with visuospatial and visuomotor performance in this population. (*JINS*, 2003, *9*, 440–449.)

Keywords: Extremely low birthweight, Prematurity, Academic ability, Periventricular brain damage, Magnocellular visual pathway, Dorsal visual stream

INTRODUCTION

An association between premature birth and academic difficulties in the early elementary grades has been well documented over the last decade (Herrgård et al., 1993; Klebanov et al., 1994; Rieck et al., 1996; Stjernqvist & Svenningsen, 1995). At particular risk in this area are the smallest survivors of premature birth, namely those born at extremely-low-birthweight (ELBW: birthweight <1000 g; Fletcher et al., 1997; Hall et al., 1995; Robertson et al., 1990; Ross et al., 1991; Taylor et al., 2000). Not all ELBW children are equally vulnerable to academic and cognitive difficulties, however. Those most likely to experience such problems are ELBW children who suffered periventricular brain injury (PVBI) during the perinatal period (Frisk & Whyte, 1994; Hack et al., 1994; Halsey et al., 1996; Jakobson et al., 2001; Saigal et al., 1991; Taylor et al., 1995). Recent evidence suggests that academic and cognitive difficulties persist in this subgroup into adolescence (Downie et al., 2002). In contrast, ELBW children without PVBI and who were appropriate in size for their gestational age (AGA: birthweight > 3rd percentile) perform as well as full-term children on tests of reading, spelling, phonological awareness, working memory and intelligence (Downie et al., 2002; Frisk & Whyte, 1994). These latter findings suggest that the academic problems experienced by ELBW children with PVBI result from the early disruption of neural mechanisms responsible for the normal develop-

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ment and acquisition of academic skills, and not from prematurity *per se*.

Despite the documented relationship between PVBI and academic outcome, few studies have addressed the question of which specific perceptual and/or cognitive difficulties associated with PVBI might underlie the development of reading and spelling problems in ELBW children. In formulating hypotheses about this question, we turned to the literature on specific reading disability (SRD, or dyslexia) in full-term children. Much of the research carried out with this population has suggested that reading difficulties are fundamentally linguistic in nature and associated with deficits in phonological processing and working memory, resulting in a specific inability to process and manipulate speech sounds (Brady et al., 1983; Mody et al., 1995). Other work, however, suggests that, in at least a subgroup of individuals with SRD, deficits in the ability to process rapidly changing visual information are associated with the development of reading and spelling problems. Although it remains controversial (e.g., Gross-Glenn et al., 1995; Skottun, 2000; Smith et al., 1986), this latter theory—the magnocellular theory of dyslexia—has garnered substantial support in recent years (e.g., Stein, 2001). Given this, and in the face of compelling evidence of a relationship between PVBI and visual dysfunction in children born prematurely (e.g., Bozynski et al., 1984; Jakobson et al., 2001; Jongmans et al., 1996; Olsén et al., 1998), the goals of the present study were to determine whether magnocellular deficits are (1) common in ELBW children, particularly those who experienced PVBI; and (2) associated with reading and spelling problems in this population, as they appear to be in some full-term children with SRD.

In the sections that follow, we will review briefly some of the recent literature concerning the magnocellular theory of dyslexia, and further develop the specific rationale for looking for signs of magnocellular dysfunction in ELBW children. Specific ways in which such dysfunction could contribute to reading and spelling problems will also be discussed.

The Magnocellular Theory of Dyslexia

In the primate visual system, certain subcortical and cortical pathways specialize in the processing of rapidly changing visual information. Cells in the subcortical magnocellular (M) pathway, for example, have been found to have large receptive fields and fast conduction velocities, and to respond transiently and optimally to visual stimuli of low spatial and high temporal frequency (Merigan & Maunsell, 1993). These features make them well suited to extract spatiotemporal variations in incoming visual inputs—that is, to extract information about visual flicker and motion. Numerous anatomical, physiological and psychological studies have documented M-pathway impairments in at least a subgroup of children with SRD. Psychophysical studies, for example, show that, relative to individuals without reading difficulties, many individuals with SRD show a reduced ability to detect gratings of low spatial frequency at low contrast (Felmingham & Jakobson, 1995; Lovegrove et al., 1980, 1982; Martin & Lovegrove, 1987), and to detect higher temporal frequency flicker (Martin & Lovegrove, 1987).

Although it remains unknown if magnocellular deficits play a causal role in the development of SRD or are merely associated with the disorder, several investigators have suggested ways in which such problems could directly impact on the acquisition of reading and spelling abilities. These proposals draw on the fact that the M pathway provides the predominant input to the dorsal cortical visual stream, the pathway connecting primary visual cortex to the posterior parietal lobe (Maunsell, 1992; Maunsell et al., 1990). This stream is intimately involved in visuospatial analysis, spatial attention, stereopsis, the perception of self- and object-motion, and the visual guidance of movements of the eyes and limbs (Milner & Goodale, 1995). It has been argued that disruption of one or more of these higher-order processes might contribute directly to reading failure in children with SRD. Several researchers have suggested, for example, that problems with *visual localization* and/or *visual attentional control* could explain problems with letter position encoding or nonword reading ability in children with SRD (Cestnick & Coltheart, 1999; Graves et al., 1999; Vidyasagar, 1999). Stein (2001) has argued that difficulties with *binocular stability* also affect many children with SRD. His demonstration that reading performance improves dramatically in these children following patching therapy supports the idea of a causal link. It has also been suggested that *motion-processing problems* could lead to impairments in the ability to detect retinal slip and to plan/execute corrective eye movements to compensate for it; it is argued that these problems, in turn, could interfere with the development of orthographic skills (Fischer & Hartnegg, 2000; Stein, 2001).

Numerous additional studies offer indirect support for a link between *visual motion processing deficits* and reading disability. Recent work has shown, for example, that area MT and adjacent motion-sensitive areas (MT+) in the dorsal stream are less activated during visual tasks requiring speed discriminations in dyslexic adults than in adults without reading difficulty (Demb et al., 1998; Eden et al., 1996). In one of these studies, a three-way correlation between brain activity, speed discrimination thresholds, and reading speed was observed (Demb et al., 1998).

Since the functional development of area MT appears to be critical for the development of several higher-order motion processing skills (Braddick et al., 1996), it is interesting that a substantial proportion of individuals with SRD have also been found to show elevated thresholds for recognizing motion-defined letters (Felmingham & Jakobson, 1995), and for detecting motion coherence (Cornelissen et al., 1995; Everatt et al., 1999). Cornelissen and colleagues have shown, in an unselected sample of young adults, that performance on their motion coherence task was predictive of accuracy of letter position encoding in two separate tasks (Cornelissen et al., 1998).

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A Magnocellular Deficit in ELBW Children?

Evidence is accumulating that, like full-term children with SRD, ELBW children with PVBI may be at risk for altered development of the M pathway and its cortical targets in the dorsal stream. Thus, associations have been described between PVBI and restriction of the lower visual fields (Jacobson et al., 1996), and between PVBI and deficits in stereopsis (Dowdeswell et al., 1995), oculomotor control (Cioni et al., 1997), and both visuospatial and visuomotor function (Jakobson et al., 2001). All of these functional impairments are consistent with dorsal stream damage or dysfunction. We propose, specifically, that PVBI may frequently compromise the white matter (subcortical) tracts between regions of the M pathway. These could include thalamocortical tracts and/or fibers connecting V1 with extrastriate areas in the dorsal stream. Indeed, damage to the optic radiations (Cioni et al., 1997; Olsén et al., 1998) and thinning of parietal or occipital white matter (Goto et al., 1994) are both common after PVBI. The observation that periventricular white matter injury in the premature infant is followed by reduced cerebral cortical gray matter volume at term (Inder et al., 1999) suggests that development of cortical, dorsal stream areas may be frequently compromised as well.

If, as we suspect, ELBW children are at elevated risk for damage or dysfunction in the M pathway or its cortical targets in the dorsal stream, one should be able to document problems with visual motion processing in this population. The first goal of the present study, then, was to assess the performance of a group of ELBW children on a motion-defined form recognition test (MDFT), adequate performance on which is thought to rely on normal functioning of these pathways (Braddick et al., 1996; Regan et al., 1992). It was hypothesized that the presence of PVBI would be an important predictor of success on this task. Based on the literature reviewed above describing a relationship between motion-processing deficits and reading performance, it was also predicted that deficits on the MDFT would be associated with reading problems in our ELBW sample.

METHODS

Research Participants

Forty-seven children (M = 11 years, 7 months; range: 8 years, 10 months–14 years, 5 months) volunteered to participate in this study. Thirty-five of these children were followed by the Integrated Perinatal Follow-Up Program at The Hospital for Sick Children and at Mount Sinai Hospital, Toronto, Ontario, Canada for extreme prematurity (M = 26 weeks gestation; range: 24–30 weeks) and extremely-low-birthweight status (M = 821 g; range: 650–1000 g). All were born at an appropriate size for their gestational age. The remaining 12 control children were born at term (M = 40 weeks gestation; range: 38–42 weeks) and had no history of neurological or developmental problems. Control participants were recruited through the community.

All ELBW children had received at least three head ultrasound scans during the first six weeks of life as part of their routine care. These ultrasound scans were retrospectively reviewed by two, independent physicians to determine the presence and severity of the two most common forms of PVBI, namely germinal matrix/intraventricular hemorrhage (GM/IVH) and hypoxic/ischemic (H/I) injury. Inconsistencies between raters regarding the presence or severity of PVBI were resolved through joint consultation. Physicians were unaware of a given child's medical and developmental history or test results when rating the scans. The extent and nature of GM/IVH was determined by the Papile classification system (Papile et al., 1978), with modification based on Volpe (1995), which grades severity along a four-point scale (see Table 1). Coding of H/I injury was based upon the classification system developed by Frisk and Whyte (1994) which also uses a four-point severity rating scale (see Table 1).

Children were excluded from this study if they (1) suffered from a major sensory impairment (i.e., blindness or deafness) or motor complications affecting the upper limbs that might have compromised test performance; (2) had required ventriculoperitoneal shunting for posthemorrhagic hydrocephalus; (3) had a history of neurologic disorder or disease other than PVBI, or (4) had visual acuity in either eye below 20/100 as assessed by the Snellen Visual Acuity Chart. This final criterion ensured that all of the participants would have adequate (corrected) visual acuity to perform the experimental tasks.

Table 1. Classification scales employed to assess the presence and severity of IVH and H/I

	Description	
Grade	IVH[a]	H/I[b]
I	Isolated subependymal hemorrhage	Small single anterior cyst $<$ 5 mm
II	Intraventricular hemorrhage *without* ventricular dilatation	Persistent periventricular echogenicity without subsequent cystic formation
III	Intraventricular hemorrhage *with* ventricular dilatation	Porencephaly $<$ 5 mm in size
IV	Intraventricular hemorrhage with associated parenchymal hemorrhage (Volpe, 1995)	Widespread periventricular leukomalacia or porencephaly $>$ 5 mm

[a]Papile et al. (1978).
[b]Frisk and Whyte (1994).

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Informed consent for participation was obtained from the parents of all children in this study, and the protocol received appropriate ethics review and approval.

Tests and Questionnaires

Demographic variables were collected by a questionnaire completed by a parent. Medical variables (birthweight, weeks gestation, and status regarding retinopathy of prematurity; ROP), were collected from the neonatal follow-up charts. ROP is a proliferative process that affects the developing retinal vessels in premature children and can lead to visual problems such as myopia, strabismus, or (in severe cases) blindness (Page et al., 1993). ROP severity was coded along a 4-point scale (Committee for the Classification of Retinopathy of Prematurity, 1984) with Stage 1 indicating the mildest form of this condition and Stage 4 the most severe. Visual acuity was assessed with the Snellen Visual Acuity Chart.

The Word Attack and Word Identification subtests of the Woodcock Reading Mastery Test (Woodcock, 1987) were used to assess the ability to read nonsense words and English words, respectively. The Spelling subtest of the Wide Range Achievement Test–Third Edition (WRAT–3; Wilkinson, 1993) was used to assess the ability to spell English words, while the Wechsler Intelligence Scale for Children–Third Edition (WISC–III; Wechsler, 1991) was used to measure intelligence. To reduce the possibility of subjective bias in scoring, all of the standardized tests were scored twice: initially by the first author (A.L.S.D.) and then again by a research assistant who was unaware of the child's status. When scoring discrepancies were found, scores were changed to those determined by the research assistant.

Visual motion processing was assessed by a motion-defined letter recognition test (MDFT; Regan et al., 1992). In this test, participants are asked to identify 10 different alphabetic letters (*C*, *D*, *H*, *K*, *N*, *O*, *R*, *S*, *V*, and *Z*), one at a time, presented in random order. Stimuli were generated by an IBM PC compatible with a WonderGraphics card and displayed on a computer monitor with a resolution of 640 × 480 pixels at a frame rate of 60 Hz. Dot density was kept constant at 25% across all trials.

Stimuli were presented either as motion-defined letters or as luminance-defined letters. During the motion-defined trials, the letter was stationary but the foreground of each stimulus was comprised of dots moving leftwards at *x* minutes/s, while the background dots moved rightwards at the same speed. The speed at which the dots moved was set at 10.26, 5.13, 2.56, 1.28, or 0.684 minutes/s. This range was selected as it includes speeds at which both patients with parietotemporal damage (Regan et al., 1992) and children with SRD (Felmingham & Jakobson, 1995) have been shown to exhibit a selective failure of motion-defined letter recognition. During luminance-defined trials, the background dots were turned off and the foreground dots moved at 10.26 minutes/s.

Procedure

Each child underwent 4 hr of testing, with breaks after each hour to prevent fatigue. The examiner was blind to the neurological status of the ELBW children throughout the procedure. The acuity, reading, spelling and intelligence tests were administered individually to each child in a quiet room prior to the administration of the MDFT, using standard administration procedures.

During the MDFT, participants sat in a darkened room at a distance of 2 m from the computer monitor. All were dark-adapted before testing began. Before beginning the trials involving motion-defined letter recognition, one block of 10 trials was administered with the background dots turned off (*luminance-defined condition*). These trials were administered to familiarize the children with the task and to ensure that each child could see the letters when their edges were defined by luminance contrast. Under these conditions, all children (both ELBW and full-term) were able to identify all 10 letters correctly. Given this, it seems unlikely that any observed deficits in motion-defined letter recognition could be attributed to problems with poor letter recognition or letter-name retrieval.

Participants were also given one practice block of motion-defined letter recognition trials before beginning the experimental trials, in which the dots moved at an intermediate dot speed. The onset of each experimental trial was preceded by a long beep, which notified the participants that a letter was about to appear. The letter size was set at 2.3 cm × 2.3 cm, subtending 0.66° of visual angle. During each block of trials, the 10 letters were presented in random order, each for a duration of 990 ms, and the participant was asked to name the letter once it had disappeared from the screen. Participants were instructed to guess if unsure. A list of all 10 letters used in the experiment was placed below the computer monitor to help children when it was necessary for them to guess. The examiner controlled the time between trials. Subjects were instructed to remain in the same position throughout the testing, and to keep their heads still. Each block of test trials was administered in order, from the slowest dot speed to the fastest dot speed, until children could not correctly recognize more than three letters in a given block. Each block of trials was presented once.

RESULTS

The major hypotheses in the present study were evaluated with two sets of analyses, one examining the relationship between PVBI and performance on the MDFT, and the other examining the relationship between performance on the MDFT and performance on tests of reading, spelling and intellectual ability. In the descriptions provided below, multivariate analyses meeting Wilks's Lambda criterion for significance were followed by univariate tests; significant univariate tests were followed by Tukey tests unless otherwise specified. Where there were missing data due to subject fatigue, or experimental error, data for that subject were excluded from each separate analysis.

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The Relationship Between PVBI and Performance on the MDFT

Surprisingly, thresholds (i.e., the dot speed required for 75% correct letter recognition) could only be obtained for 43% of ELBW children on the MDFT, as opposed to 100% of full-term controls. Indeed, a number of the ELBW children reported that they were unable to see letters at any dot speed although, when forced to guess, all were able to obtain chance performance (10% correct letter recognition) on at least one of the five dot speeds.

Since thresholds were not reached by such a large proportion of the clinical sample, another measure of performance was adopted for the MDFT, namely the average proportion of letters correctly identified across the five dot speeds. In computing these mean values, scores of zero were entered whenever a child was unable to identify any letters correctly at a particular speed. In examining these mean scores it became clear that only 5 of the 35 preterm children obtained a score falling within 1 standard deviation of the mean of the full term control sample. Incredibly, 25 of the preterm children (71%) achieved mean scores more than 3 standard deviations below the control mean. It is noteworthy that this level of performance is also dramatically worse than that obtained by full-term children with SRD who have been tested on this same task (Felmingham & Jakobson, 1995).

To determine whether the presence of PVBI increased a child's risk of experiencing difficulty on the MDFT the sample of ELBW children was divided into two groups: (1) ELBW children with normal head ultrasound scans (*no PVBI group*, $n = 11$); and (2) ELBW children with GM/IVH or H/I injury (*PVBI group*, $n = 24$). Chi-square analyses indicated that the full term, no PVBI, and PVBI groups were matched in terms of gender distribution, mother's educational level, and annual household income. A multivariate analysis of variance (MANOVA) indicated that the three groups were also matched for age at testing and visual acuity, and that the ELBW groups were matched for birthweight and gestational age at birth. Chi-square analyses indicated that severity of ROP was evenly distributed across the ELBW groups.

A MANOVA evaluating group differences on Verbal IQ (VIQ) and Performance IQ (PIQ) scaled scores was significant [$F(4,86) = 3.568$, $p = .01$]. Follow-up tests revealed that the full term and no PVBI groups were matched to one another but that each obtained a significantly higher score than the PVBI group on both VIQ [$F(2,44) = 5.113$, $p < .05$] and PIQ [$F(2,44) = 5.056$, $p < .05$]. A separate ANOVA showed that the Full Scale IQ (FSIQ) scores of the PVBI group were also suppressed relative to those of the other two groups [$F(2,44) = 6.599$, $p < .01$].

Given the observed group differences in IQ, the relationship between PVBI and MDFT performance was evaluated statistically using analysis of covariance (ANCOVA), with group (full term, no PVBI, and PVBI) as the between-subjects factor, PIQ as the covariate, and average MDFT score as the dependent variable. (PIQ was selected as the covariate in this analysis as, of the two IQ subscales, it showed the strongest relationship with average MDFT performance, $r = .491$, $p < .001$). The results of this analysis are presented in Figure 1. Significant main effects of PIQ

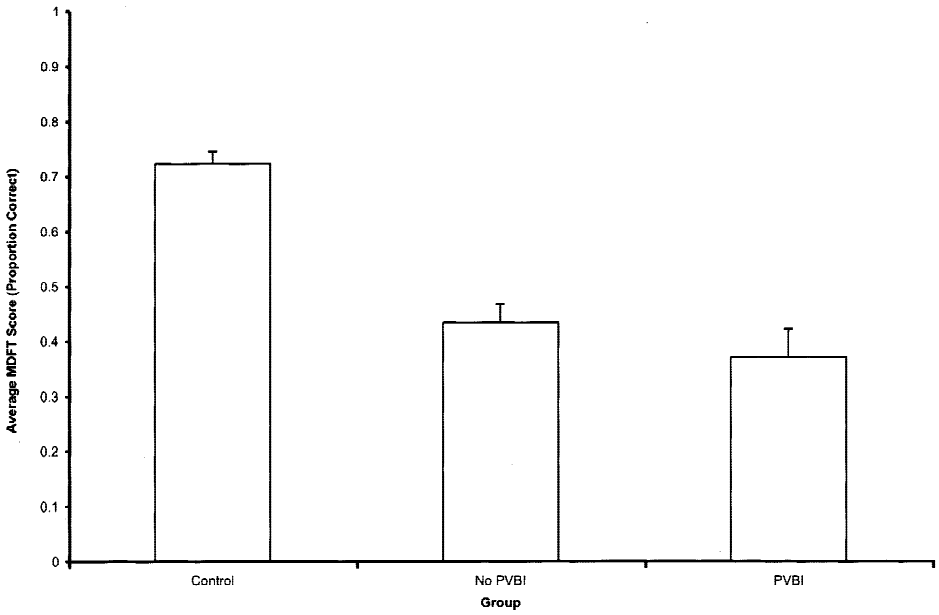

Fig. 1. Performance of each group (full term, PVBI, and no PVBI groups) on the motion-defined letter recognition test expressed as the mean proportion of letters correctly recognized across the five dot speeds. Full term control children obtained significantly higher scores on the motion-defined letter test than either ELBW group. There were no significant differences in performance between the ELBW groups. Error bars indicate standard error.

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[$F(1,43) = 6.034$, $p < .05$] and of group [$F(2,43) = 8.646$, $p < .01$] were observed. Pairwise comparisons using a Bonferroni adjustment indicated that the full term group was able to recognize a significantly larger proportion of motion-defined letters than either of the ELBW groups, but that the two ELBW groups did not differ from each other.

The Relationship Between Motion-Processing Ability and Performance on Tests of Intelligence, Reading, and Spelling

To investigate the relationship between motion-processing ability and performance on tests of intelligence, reading and spelling the ELBW children were subdivided into two groups: (1) ELBW children for whom a speed threshold for 75% correct letter recognition could be established (*ELBW threshold group*, $n = 15$); and (2) ELBW children for whom a speed threshold could not be established (*ELBW below threshold group*, $n = 20$). As outlined below, the performance of each of these groups on tests of reading, spelling, and intellectual performance was compared to that of the full term control group, all of whom reached threshold on the MDFT.

Chi-square analyses indicated that the three groups were equivalent to one another in terms of gender distribution and mother's education. A MANOVA revealed that they were also matched for age at testing, and that the two ELBW groups were matched for birthweight and gestational age at birth. In addition, chi-square analyses indicated that both severity of ROP and incidence of PVBI were evenly distributed across the two ELBW groups.

Intelligence measures

Separate MANOVAs were carried out to examine the impact of group membership on (1) VIQ and PIQ scores; (2) individual subtest scores on the Verbal scale; and (3) individual subtest scores on the Performance scale. The impact of group membership on FSIQ scores was examined separately using ANOVA. The results of the analyses are presented below and are summarized in Table 2 (which includes means and standard errors for each condition, along with relevant *F* statistics for the univariate tests).

ELBW children who were unable to attain threshold on the MDFT had lower FSIQ scores than full term controls, but did not differ from ELBW children who were able to attain threshold. The MANOVA performed on PIQ and VIQ scores was also significant [$F(4,86) = 2.634$, $p < .05$] but follow-up tests indicated that significant group differences appeared only on the PIQ scale, with the scores of the full term control group being significantly higher than those of the ELBW below threshold group.

The MANOVA examining the effects of group membership on the four subtests comprising the Verbal IQ rating was not significant [$F(8,82) = 1.958$, $p > .05$]. The MANOVA comparing performance on the four subtests

Table 2. Means and standard deviations associated with each group according to performance on the motion-defined form recognition task

	Full-term controls		ELBW–threshold		ELBW–below threshold		
Measure	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*F* statistic
Intelligence scales							
FSIQ	105.6	(4.0)	99.4	(3.5)	92.7[a]	(3.1)	$F(2,44) = 3.445$, $p < .05$
VIQ	101.7	(4.3)	96.1	(3.9)	93.0	(3.4)	$F(2,44) = 1.243$, n.s.
PIQ	109.8	(4.0)	103.2	(3.6)	93.6[a]	(3.1)	$F(2,44) = 5.569$, $p < .01$
Intelligence subtests							
Information	9.9	(1.0)	10.1	(0.9)	8.9	(0.8)	$F(2,44) = 0.727$, n.s.
Arithmetic	9.9	(0.7)	7.6[c]	(0.6)	8.2	(0.6)	$F(2,44) = 3.165$, n.s.
Similarities	10.2	(0.8)	10.1	(0.7)	9.2	(0.6)	$F(2,44) = 0.669$, n.s.
Vocabulary	10.0	(0.9)	9.3	(0.8)	8.9	(0.7)	$F(2,44) = 0.504$, n.s.
Picture Completion	10.8	(0.8)	10.8	(0.7)	9.7	(0.6)	$F(2,44) = 0.964$, n.s.
Coding	11.6	(1.0)	11.8	(0.9)	8.9[b,c]	(0.8)	$F(2,44) = 3.869$, $p < .05$
Picture Arrangement	11.5	(0.7)	9.7	(0.6)	7.7[a,d]	(0.6)	$F(2,44) = 8.865$, $p < .01$
Block Design	11.9	(1.0)	9.4	(0.9)	9.0[c]	(0.8)	$F(2,44) = 3.130$, n.s.
Reading and spelling							
Word Identification	105.3	(3.9)	94.5	(3.5)	95.8	(3.0)	$F(2,44) = 2.532$, n.s.
Word Attack	102.3	(3.6)	93.4	(3.2)	96.2	(2.8)	$F(2,44) = 1.767$, n.s.
Spelling	107.8	(3.8)	96.5	(3.4)	99.2	(3.0)	$F(2,44) = 2.641$, n.s.

[a]different from full-term controls, $p < .05$.
[b]different from ELBW–threshold group, $p < .05$.
[c]different from full-term controls, $p < .10$.
[d]different from ELBW–threshold group, $p < .10$.

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comprising the Performance IQ rating, however, revealed a main effect of group [$F(8,82) = 2.67$, $p < .05$]. Examination of mean scores on each of the four tests comprising the performance scale revealed the same trends, with the ELBW below threshold group tending to have the lowest scores (see Table 2). Significant group differences, however, were observed only on the Coding and Picture Arrangement subtests.

Reading and spelling ability

Given the finding that the ELBW below threshold group had significantly lower IQs than either of the other groups, multivariate analysis of covariance (MANCOVA) was used to examine the relationship between motion-processing ability (as measured with the MDFT) and performance on the measures of reading and spelling ability. In this analysis VIQ was used as the covariate as, of the two IQ subscales, it had shown the strongest relationship with scores on both the Word Identification and Word Attack tests ($r = .603$, $p < .001$; and $r = .459$, $p < .001$, respectively), and with scores on the WRAT–3 Spelling test ($r = .523$, $p < .001$).

It was hypothesized that significant group differences would be observed on tests of reading and spelling between ELBW children who did not achieve threshold performance on the MDFT and both of the remaining groups. The results of the MANCOVA, however, did not support this prediction. Thus, no significant group differences were observed in the omnibus test [$F(6,82) = .705$, $p > .05$]. In contrast, the covariate (FSIQ) was significant in the omnibus test [$F(3,41) = 7.248$, $p < .001$], and accounted for a significant amount of the variance in performance on each of the tests of reading and spelling ability used in the present study [Word Identification: $F(1,43) = 22.279$, $p < .001$; Word Attack: $F(1,43) = 10.362$, $p < .005$; WRAT–3 Spelling: $F(1,43) = 14.605$, $p < .001$].

Post-Hoc Analyses on ELBW Children Who Were Poor Readers

It is possible that motion-processing deficits are only related to reading difficulties in the presence of a reading disability. Given this, one final set of analyses was undertaken, focusing only on those preterm children who were considered to be experiencing reading problems.

Of the 35 children in the clinical sample, 13 obtained scores below 90 (i.e., below the 25th percentile) on one or both of the reading tests used in the present study. Of these 13 children, 10 were also considered to be poor spellers, obtaining scores that fell below the 25th percentile on the Spelling subtest of the WRAT–3. Close inspection of the characteristics of this group of poor readers revealed that all had suffered from PVBI, and that 8 of the 13 had FSIQs below 85. It is important to note, however, that only 2 members of this group had both VIQ and PIQ scaled scores below 85. In other words, 11 of the 13 "poor readers" (85%) had at least one IQ score that fell within 1 standard deviation of the mean for their age, suggesting that their reading difficulties were not a simple reflection of a global cognitive deficit.

Hierarchical multiple regression was used to investigate the relationship between motion-processing ability and performance on the three tests of reading and spelling ability in this restricted sample. Separate regression equations were computed for the two reading tests and for the test of spelling ability. In each case, FSIQ was entered first, followed by average MDFT score. Neither predictor accounted for a significant amount of the variance in test scores for any of the three tests. Thus, even in this restricted sample of poor readers there was no evidence for a relationship between performance on our test of motion-processing ability and performance on tests of reading or spelling ability.

DISCUSSION

Perhaps the most striking result of the present study concerns the performance of the ELBW children on the test of visual motion processing. More than half of the ELBW children tested were unable to achieve threshold on the MDFT, and more than 70% achieved mean scores more than three standard deviations below the mean of the full term control group. Surprisingly, and counter to our expectation, the presence of PVBI did not appear to be the crucial variable determining performance levels on this task. Thus, ELBW children who showed no evidence of PVBI in neonatal cranial ultrasound scans were as impaired as those with abnormal scans. In several cases, the impairment was so pronounced that the children reported being unable to see motion-defined letters at any dot speed, despite showing perfect recognition performance when the boundaries of the letters were defined by luminance contrast.

One possible explanation for this surprising result is that cranial ultrasound scans were simply not sensitive enough to reveal subtle injuries that may have affected the developing brains of the premature children who participated in the present study. Certainly, the use of structural magnetic resonance imaging, which provides superior soft tissue contrast and spatial resolution compared to ultrasonography, might have revealed damage even in those children whose cranial ultrasound scans appeared normal. In addition, however, it is possible that *functional* impairments might have been revealed, even in the absence of structural anomalies, had functional imaging techniques been employed. The superiority of functional over structural imaging in diagnosing damage to cortical visual areas has already been clearly documented (Duncan et al., 1994; Silverman et al., 1993). Such functional impairments might reflect the effects of the developmentally inappropriate stimulation associated with premature exposure to the extrauterine environment (Duffy et al., 1990).

Both the MDFT and tests measuring sensitivity to coherent motion require global motion processing (Regan et al., 1992). Given that elevated motion coherence thresholds have previously been shown to be related to various indices of

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reading ability in unselected samples of young adults (Cornelissen et al., 1998), a relationship was expected between performance on tests of reading and spelling ability and performance on the MDFT. No such relationship was observed, even in the subgroup of preterm children who were identified as having significant reading and spelling problems. Taken at face value, then, the present findings cast doubt on the theory that magnocellular deficits play a causal role in the development of reading problems. They suggest, instead, that while weaknesses in these two realms can be a feature of developmental disorders such as SRD, they are dissociable deficits. Before one endorses this position, however, several issues need to be considered. One concerns the manner in which reading and spelling performance was evaluated in the present study. It is important to note that the precise mechanism(s) through which magnocellular deficits might influence the normal acquisition of reading and spelling skills remain unspecified. It is possible that many of the problems thought to be related to magnocellular deficits (e.g., difficulties with visual localization, visual attentional control, oculomotor control, etc.) might affect variables such as *reading speed*, without affecting performance on untimed tests of reading and spelling accuracy such as those used in the present investigation. Indeed, as noted earlier, in their recent functional imaging study Demb et al. (1998) reported a strong correlation between reading speed, speed discrimination thresholds, and brain activity in several motion-sensitive areas in the dorsal stream. Future studies should include a broader assessment of reading performance than that used in the present study to allow for a better test of the theory.

Another issue that requires careful consideration when interpreting the present findings concerns the fact that it is unlikely that even a subgroup of the ELBW children in our sample experienced an *isolated* impairment of magnocellular/dorsal stream function. Given the high probability that those experiencing visual difficulties actually suffered more pervasive insults, affecting the development and/or function of a variety of visual (and nonvisual) pathways, it may be difficult to tease out the potential contribution of magnocellular deficits to reading and spelling problems in this population, and to establish the factors that underlie poor performance on psychophysical tests such as the one used in the present study.

It is also important to consider the potential impact of the atypical visual experiences and, in some cases, perinatal brain injuries suffered by preterm children on neurodevelopment and the functioning of particular neural systems. The degree to which preterm children are able to compensate for atypical neurodevelopment remains unknown. Given that their performance on the MDFT was dramatically worse than that exhibited even by full-term children with SRD (Felmingham & Jakobson, 1995), it is possible that compensatory mechanisms are operating in this population to support the development of reading and spelling skills. In other words, atypical neural or functional organization of the brains of these children may allow them to circumvent any problems with reading that might normally be associated with such a profound impairment in visual processing.

One final issue concerns the small sample size employed in the present study, particularly in the *post-hoc* analysis which involved only 13 children. It is possible that with a larger clinical sample, a clearer picture of the relationships between variables may have emerged.

Even if it does not contribute to reading difficulties, M pathway or dorsal stream dysfunction in ELBW children may underlie problems in other areas, such as visuospatial and visuomotor functioning (Jakobson et al., 2001). Indeed, in the present report a relationship was observed between motion-processing ability and performance on several of the subtests of the Performance IQ scale—successful completion of which depend on intact visuospatial and/or visuomotor abilities. We are presently exploring the relationship between these variables in a larger and younger sample of ELBW children.

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REFERENCES

Bozynski, M.E., Nelson, M.N., Genaze, D., Rosati-Skertich, C., Chilcote, W.S., Jr., Ramsey, R.G., O'Donnell, K.J., & Meier, W.A. (1984). Intracranial hemorrhage and neurodevelopmental outcome at one year in infants weighing 1200 grams or less. Prognostic significance of ventriculomegaly at term gestational age. *American Journal of Perinatology*, *1*, 325–330.

Braddick, O., Atkinson, J., & Hood, B. (1996). Striate cortex, extrastriate cortex, and colliculus: Some new approaches. In F. Vital-Durand, J. Atkinson, & O.J. Braddick (Eds.), *Infant vision* (pp. 203–220). Oxford, UK: Oxford University Press.

Brady, S.A., Shankweiler, D.P., & Mann, V. (1983). Speech perception and memory coding in relation to reading ability. *Journal of Experimental Child Psychology*, *35*, 345–367.

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

Cioni, G., Fazzi, B., Coluccini, M., Bartalena, L., Boldrini, A., & van Hof-van Duin, J. (1997). Cerebral visual impairment in preterm infants with periventricular leukomalacia. *Pediatric Neurology*, *17*, 331–338.

Committee for the Classification of Retinopathy of Prematurity. (1984). The international classification of retinopathy of prematurity. *Pediatrics*, *74*, 127–133.

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*, *35*, 1483–1494.

Cornelissen, P.L., Hansen, P.C., Gilchrist, I., Cormack, F., Essex, J., & Frankish, C. (1998). Coherent motion detection and letter position encoding. *Vision Research*, *38*, 2181–2191.

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

Dowdeswell, H.J., Slater, A.M., Broomhall, J., & Tripp, J. (1995). Visual deficits in children born at less than 32 weeks' gestation with and without major ocular pathology and cerebral damage. *British Journal of Ophthalmology*, *79*, 447–452.

Downie, A.L.S., Frisk, V., & Jakobson, L.S. (2002). *The impact of periventricular brain injury on reading and spelling abilities in the late elementary and adolescent years*. Manuscript submitted for publication.

Duffy, F.H., Als, H., & McAnulty, G.B. (1990). Behavioral and electrophysiological evidence for gestational age effects in healthy preterm and fullterm infants studied two weeks after expected due date. *Child Development*, *61*, 271–286.

Duncan, D., Alavi, A., Galetta, S.L., Gray, L., & Grossman, M. (1994). The value of technetium-99m HMPAO SPECT in the evaluation of visual abnormalities [Abstract]. *Journal of Nuclear Medicine*, *35*, 209–10P.

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

Everatt, J., Bradshaw, M.F., & Hibbard, P.B. (1999). Visual processing and dyslexia. *Perception*, *28*, 243–254.

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

Fischer, B. & Hartnegg, K. (2000). Stability of gaze control in dyslexia. *Strabismus*, *8*, 119–122.

Fletcher, J.M., Landry, S.H., Bohan, T.P., Davidson, K.C., Brookshire, B.L., Lachar, D., Kramer, L.A., & Francis, D.J. (1997). Effects of intraventricular hemorrhage and hydrocephalus on the long-term neurobehavioral development of preterm very-low-birthweight infants. *Developmental Medicine and Child Neurology*, *39*, 596–606.

Frisk, V. & Whyte, H. (1994). The long term consequences of periventricular brain damage on language and verbal memory. *Developmental Neuropsychology*, *10*, 313–333.

Goto, M., Ota, R., Iai, M., Sugita, K., & Tanabe, Y. (1994). MRI changes and deficits of higher brain functions in preterm diplegia. *Acta Pædiatrica*, *83*, 506–511.

Graves, R.E., Frerichs, R.J., & Cook, J.A. (1999). Visual localization in dyslexia. *Neuropsychology*, *13*, 575–581.

Gross-Glenn, K., Skottun, B.C., Glenn, W., Kushch, A., Lingua, R., Dunbar, M., Jallad, B., Lubs, H.A., Levin, B., Rabin, M., Parke, L.A., & Duara, R. (1995). Contrast sensitivity in dyslexia. *Visual Neuroscience*, *12*, 153–163.

Hack, M., Taylor, H.G., Klein, N., Eiben, R., Schatschneider, C., & Mercuri-Minich, N. (1994). School-age outcomes in children with birth weights under 750 g. *New England Journal of Medicine*, *331*, 753–759.

Hall, A., McLeod, A., Counsell, C., Thomson, L., & Mutch, L. (1995). School attainment, cognitive ability and motor function in a total Scottish very-low-birthweight population at eight years: A controlled study. *Developmental Medicine and Child Neurology*, *37*, 1037–1050.

Halsey, C.L., Collin, M.F., & Anderson, C.L. (1996). Extremely low-birth-weight children and their peers. A comparison of school-age outcomes. *Archives of Pediatric and Adolescent Medicine*, *150*, 790–794.

Herrgård, E., Luoma L., Tuppurainen, K., Karjalainen, S., & Martikainen, A. (1993). Neurodevelopmental profile at five years of children born at < 32 weeks gestation. *Developmental Medicine and Child Neurology*, *35*, 1083–1096.

Inder, T.E., Huppi, P.S., Warfield, S., Kikinis, R., Zientara, G.P., Barnes, P.D., Ferenc, J., & Volpe, J.J. (1999). Periventricular white matter injury in the premature infant is followed by reduced cerebral cortical gray matter volume at term. *Annals of Neurology*, *46*, 755–760.

Jacobson, L., Ek, U., Fernell, E., Flodmark, O., & Broberger, U. (1996). Visual impairment in preterm children with periventricular leukomalacia—visual, cognitive and neuropaediatric characteristics related to cerebral imaging. *Developmental Medicine and Child Neurology*, *38*, 724–735.

Jakobson, L.S., Frisk, V.A., Knight, R.M., Downie, A.L.S., & Whyte, H. (2001). The relationship between periventricular brain injury and deficits in visual processing among extremely-low-birth-weight (<1000 g) children. *Journal of Pediatric Psychology*, *26*, 503–512.

Jongmans, M., Mercuri, E., Henderson, S., de Vries, L., Sonksen, P., & Dubowitz, L. (1996). Visual function of prematurely born children with and without perceptual–motor difficulties. *Early Human Development*, *45*, 73–82.

Klebanov, P.K., Brooks-Gunn, J., & McCormick, M.C. (1994). Classroom behavior of very low birthweight elementary school children. *Pediatrics*, *94*, 700–708.

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

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

Martin, F. & Lovegrove W. (1987). Uniform-field flicker masking in control and specifically disabled readers. *Perception*, *17*, 203–214.

Maunsell, J.H.R. (1992). Functional visual systems. *Current Opinion in Neurobiology*, *2*, 506–510.

Maunsell, J.H.R, Nealy, R.A., & De Priest, D.D. (1990). Magnocellular and parvocellular contributions to responses in the middle temporal area (MT) of the macaque monkey. *Journal of Neuroscience*, *10*, 3323–3334.

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

Milner, A.D. & Goodale, M.A. (1995). *The visual brain in action*. New York: Oxford University Press.

Mody, M., Studdert-Kennedy, M., & Brady, S. (1995). Speech perception deficits in poor readers: Auditory processing or phonological coding? *Haskins Laboratories Status Report on Speech Research*, *119/120*, 1–24.

Olsén, P., Vainionpää, L., Pääkkö, E., Korkman, M., Pyhtinen, J., & Järvelin, M. (1998). Psychological findings in preterm children related to neurologic status and magnetic resonance imaging. *Pediatrics*, *102*, 329–336.

Page, J., Schneeweiss, S., Whyte, H.E.A., & Harvey, P. (1993). Ocular sequalae in premature infants. *Pediatrics*, *92*, 787–790.

Papile, L., Burstein, J., Burstein, R., & Koffler, H. (1978). Incidence and evolution of subependymal and intraventricular hemorrhage: A study of infants with birth weights less than 1500 grams. *The Journal of Pediatrics*, *92*, 529–534.

Regan, D., Giaschi, D., Sharpe, J.A., & Hong, XH. (1992). Visual processing of motion-defined form: Selective failure in patients with parietotemporal lesions. *Journal of Neuroscience*, *12*, 2198–2210.

Rieck, M., Arad, I., & Netzer, D. (1996). Developmental evaluation of very-low-birthweight infants: Longitudinal and cross-sectional studies. *International Journal of Behavioral Development*, *19*, 549–562.

Robertson, C.M.T., Etches, P.C., & Kyle, J.M. (1990). Eight-year school performance and growth of preterm, small for gestational age infants: A comparative study with subjects matched for birthweight and for gestational age. *Journal of Pediatrics*, *116*, 19–26.

Ross, G., Lipper, E.G., & Auld, P.A.M. (1991). Educational status and school-related abilities of very low birthweight premature children. *Pediatrics*, *88*, 1125–1134.

Saigal, S., Szatmari, P., Rosenbaum, P., Campbell, D., & King, S. (1991). Cognitive abilities and school performance of extremely low birth weight children and matched term control children at age 8 years: A regional study. *Journal of Pediatrics*, *118*, 751–760.

Skottun, B.C. (2000). The magnocellular deficit theory of dyslexia: The evidence from contrast sensitivity. *Vision Research*, *40*, 111–128.

Silverman, I.E., Galetta, S.L., Gray, L.G., Moster, M., Atlas, S.W., Maurer, A.H., & Alavi, A. (1993). SPECT in patients with cortical visual loss. *Journal of Nuclear Medicine*, *34*, 1447–1451.

Smith, A.T., Early, F., & Grogan, S.C. (1986). Flicker masking and developmental dyslexia. *Perception*, *15*, 473–482.

Stein, J. (2001). The magnocellular theory of developmental dyslexia. *Dyslexia*, *7*, 12–36.

Stjernqvist, K. & Svenningsen, N.W. (1995). Extremely low-birthweight infants less than 901 g: Development and behaviour after 4 years of life. *Acta Paediatrica*, *84*, 500–506.

Taylor, H.G., Hack, M., Klein, N., & Schatschneider, C. (1995). Achievement in children with birth weights less than 750 grams with normal cognitive abilities: Evidence for specific learning disabilities. *Journal of Pediatric Psychology*, *20*, 703–719.

Taylor, H.G., Klein, N., & Hack, M. (2000). School-age consequences of birth weight less than 750 g: A review and update. *Developmental Neuropsychology*, *17*, 289–321.

Vidyasagar, T.R. (1999). A neuronal model of attentional spotlight: Parietal guiding the temporal. *Brain Research Reviews*, *30*, 66–76.

Volpe, J.J. (1995). *Neurology of the newborn* (3rd ed.). Philadelphia: W.B. Saunders Company.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children–Third Edition manual*. Toronto: Harcourt Brace Jovanovich, Inc.

Wilkinson, G.S. (1993). *Wide Range Achievement Test administration manual*. Wilmington, DA: Wide Range Inc.

Woodcock, R.W. (1987). *Woodcock Reading Mastery Tests–Revised examiner's manual*. Circle Pines, MN: American Guidance Service, Inc.