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# EVIDENCE FOR IMPAIRED VISUOPERCEPTUAL ORGANISATION IN DEVELOPMENTAL DYSLEXICS AND ITS RELATION TO TEMPORAL PROCESSES

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An analysis of normal and dyslexic readers' reaction-time (RT) performance in a standard visual-detection task (Experiment A) and in temporally primed visual detection (Experiment B) reveals a tendency for significantly longer search and detection RTs for dyslexic relative to the performance of normal readers. Consistent with previous studies, the RTs of normal readers and fast dyslexic responders exhibited target-specific priming effects. In contrast, in addition to increased but statistically insignificant target priming, a set of slower dyslexic responders showed strong negative priming on target-absent trials. In spite of the longer detection latencies produced by these dyslexic participants, no evidence was found to suggest that negative priming occurred as a general function of increasing difficulty in task performance (Experiment C). The enhanced positive and the negative priming effects are both interpreted in the context of the possible deployment of attentional mechanisms to the priming stimulus. The extent to which this strategy is characteristic of dyslexic performance as a whole may relate to the degree to which the dyslexic responder concerned experiences some general temporal processing impairment: Attentional deployment in this instance serving to compensate a lack of the requisite temporal resolution required for coding the spatiotemporal structure of the prime.

## INTRODUCTION

### Controversies on the aetiology of dyslexia

According to the International Classification of Diseases (World Health Organisation, 1992), developmental dyslexia is defined as a specific disability in learning to read and to spell despite at

least normal intelligence, adequate instruction, sociocultural opportunity, and the absence of sensory defects in vision and hearing. By this definition, instead of aetiologically grounded criteria, the diagnosis of dyslexia rests upon a criterion of discrepancy between the reading performance expected from measures of general intelligence

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and the observed reading performance, or in other words the discrepancy between how a child is expected to learn to read and how, in fact, it does. However, the existence of partially contradicting experimental work suggests that dyslexia may be best considered a polyaetiological syndrome that is influenced by structural and functional characteristics of the central nervous system in interaction with exogenous factors.

Reading is understood as a complex cognitive technique requiring the coordination of a series of subfunctions, including visual functions such as the analyses of configural (feature) and orthographic forms, as well as language-related functions, such as phonological, semantic, and syntactic coding and decoding (e.g., Friederici & Lachmann, 2002). In this context, current models of dyslexia may be roughly divided into those assuming language-related deficits (which represent the majority view; e.g., Snowling, 2000, 2001; Vellutino, 1987) and those assuming visual deficits as an important determinant of reading disability (see, e.g., Habib, 2000). Visual impairments are often (but controversially) related to the transient visual subsystem. Typically, it is assumed that the visual processing of normal and dyslexic individuals undertakes a coarse decomposition of the visual scene into low and high spatial frequencies that are processed by independent channels, these being the transient and sustained subsystems, respectively (Breitmeyer & Ganz, 1976). The transient subsystem is believed to be most sensitive to low spatial and high temporal frequencies while the sustained visual subsystem is proposed to be most sensitive to high spatial and low temporal frequencies. Evidence of a functional deficit in transient subsystem activity is claimed for dyslexic readers on the basis of evidence indicating that magnocellular layers of dyslexic brains are disordered while the magnocells are significantly smaller in dyslexic readers than in normal readers (Livingstone, Rosen, Drislane, & Galaburda, 1991). Psychophysical studies have also shown dyslexic readers to demonstrate a general slowing of visual processing relative to normal readers: Related to the transient subsystem some studies have shown longer visual persistence to accompany

low spatial frequency stimuli (Lovegrove, Bowling, Badcock, & Blackwood, 1980a; Lovegrove, Heddle, & Slaghuis, 1980b; see also Slaghuis & Ryan, 1999), while it has also been shown that dyslexic readers experience slower flicker fusion rates (Martin & Lovegrove, 1987). There is also evidence for overall slowed responding to configural visual information, which is independent of spatial frequency (Keen & Lovegrove, 2000), although this finding has been considered in terms of a reduced capacity to process rapidly presented stimuli and has thus been related to problems with saccadic suppression, which, by extension, relates to the transient subsystem (see, e.g., Breitmeyer, 1980). Evidence for a transient subsystem deficit has also been claimed from electroencephalographic (EEG) data in which dyslexic readers' exhibit diminished visually evoked potentials to motion signals (Livingstone et al., 1991). In addition, functional magnetic resonance imaging (fMRI) studies have shown reduced activity in brain areas receiving strong magnocellular input such as areas of primary visual cortex (V1/V5) and the secondary cortical visual area MT+ (Demb, Boynton, & Heeger, 1997; 1998; Eden et al., 1996).

The ascription of abnormal visual processing in dyslexic readers to impaired transient subsystem function has been questioned (see, Johannes, Kussmaul, Munte, & Mangun, 1996) and been subject to counter-claims (see Greatrex & Drasdo, 1995, for a general analysis of the issues). The fMRI data recorded by Eden, Van Meter, Rumsey, Maisog, Woods, and Zeffiro (1996) has been argued to lack control for responses to motion stimuli evoking a response in the sustained subsystem, for example colour-global dot motion. There have also been a number of failures to replicate some psychophysical findings ascribed to impaired magnocellular processing in dyslexia (see, e.g., Hayduk, Bruck, & Cavanagh, 1996; Hulme, 1988; Skottun, 2000), in particular, studies such as that of Spinelli, Angelelli, De Luca, Di Pace, Judica and Zoccolotti (1997), who investigated contrast sensitivity thresholds, found no evidence that the performance of dyslexic participants could be specifically ascribed to abnormalities in transient

subsystem function. Concerns of a methodological nature, such as those expressed by Chase and Stein (2003; see also Skottun, 2000) refer to the validity of specific measures of contrast sensitivity or motion perception as effective indicators of magnocellular function, suggesting that a transient subsystem deficit may not emerge in all measures of contrast sensitivity and may in fact be based on the technical characteristics of the measures themselves, where measurement noise obscures mild deficits.

While weaknesses in the hypothesis of deficits in the transient subsystem have led to the proposal that dyslexic reading performance should be ascribed to impairments in the functioning of the sustained processing channels (see Skottun, 2000), there is also a more general view that dyslexic readers experience a generalised difficulty in processing sensory information that is brief or that changes rapidly over time. Generalised is here taken to indicate that dyslexia should be characterised in terms of a "temporal integration" deficit that is not specific to a given visual processing subsystem and may be neither specific to the visual (DiLollo, Hanson, & McIntyre, 1983; Talcott, Hansen, Assoku, & Stein, 2000) nor the auditory domain (Kujala, 2002; Tallal, 1984), but which has been argued to influence processing in both domains (Farmer & Klein, 1995; Habib, 2000; Klein, 2002; Lachmann, 2002; Stein & Walsh, 1997; although see also Studdert-Kennedy & Mody, 1995, for a critical review of the case for auditory deficits in dyslexia). Alternative proposals suggest that visual impairments should be better considered in terms of disturbed attentional mechanisms (Hari & Renvall, 2001; Stuart, McAnally, & Castles, 2001).

### Particular temporal aspects of visuoperceptual organisation

If activation of the transient (magnocellular) system inhibits activation of the sustained (parvocellular) system (Breitmeyer, 1980; also Burr, Morrone, & Ross, 1994), simple logic would state that this pattern of interaction occurs only under circumstances when both transient and sustained

channels are concurrently engaged in stimulus processing, with the necessary implication that activity in each channel is functionally connected and thus share some common mechanism of coactivation. Candidate mechanisms for linking transient and sustained channels are inhibitory interneuron networks: In early visual processing, inhibitory thalamic connections are known to be responsible for the generation of oscillatory neuronal activity serving as a carrier for neuronal synchronisation. An emergent structure of oscillations coupled with the cross-correlation (synchronisation) of oscillatory neuronal activity has been found in the responses of cells engaged in processing the parallel trajectories of separate (and linked) moving contours. This pattern of oscillatory synchronisation is interpreted in terms of the coding of spatio-temporal relations between (i.e., the common fate motion of) the moving stimuli, suggesting that oscillatory synchronisation is one means by which spatial and temporal information can combine to form a unitary perceptual experience (see Singer, 1999, for a recent review of the literature on this topic).

A relationship between inhibitory interneuron networks and the coding of stimulus synchrony has been suggested in an experiment conducted by Elliott, Becker, Boucart, and Müller (2000). In this task, observers were required to produce a reaction-time (RT) response to the presence or absence of a target grouping of four corner junctions (a Kanizsa-type square) presented in a regular  $5 \times 5$  matrix of distracter junctions. Presentation of this target matrix was preceded by the presentation of a flickering premask matrix comprising 25 crosses divided across four asynchronously presented image frames and organized spatially in the same  $5 \times 5$  arrangement as the target matrix. On 50% of trials, the premask matrix included an embedded target-prime, which comprised four simultaneously presented crosses (a synchronous premask), repeatedly shown below detection threshold at the location of the subsequently presented target. This "synchronous" premask condition contrasted with a random premask condition in which four crosses were presented simultaneously but in pseudorandomised spatial configuration (i.e.,

which did not correspond to a square), while for both conditions, the remaining premask matrix elements were pseudorandomly divided across the remaining three premask matrix frames (see Figure 1 and Methods and Elliott & Müller, 1998, for further details of the paradigm).

Elliott et al. (2000) discovered that responses to target (but not nontarget) presentation were significantly expedited for healthy volunteers when targets followed synchronous-premask presentation; these target "priming" effects were substantially amplified when experimental participants had been administered Lorazepam, a GABA<sub>A</sub>ergic agonist known to influence the temporal response of interneuron networks. This finding was consistent with earlier findings that suggested priming to be sensitive to the precise temporal characteristics of premask matrix presentation: Elliott and Müller (1998, 2000) discovered priming effects to be specific to premask matrices that flickered at 40 Hz, which is in the range of those frequencies accompanying neuronal synchronisation.

### Investigation of a temporal processing deficit in dyslexic readers

Based upon relations between the findings of Elliott and colleagues and those of physiologists interested in the temporal organisation of neuronal mechanisms engaged in visual processing, we considered the outcomes of synchrony priming as potentially revealing with respects to the regularity and temporal structure of synchronised neural responses to visual stimulus presentation. On this basis, our specific aim was to further explore the idea of visual temporal processing deficits in dyslexic readers on the assumption that reading, as a task requiring the organisation of graphemic information, may be related to the temporal organisation of visual-coding processes. We were particularly interested in the extent to which the coding of stimulus synchrony and, with close analogy, neuronal synchronisation, may be impaired in dyslexic relative to normal readers and, consequently, the extent to which evidence exists to suggest that difficulties in the precision of (neuronal)

temporal organisation may be a general characteristic of dyslexia.

Using both a standard target detection task (i.e., a target detection task without priming, Experiment A) and target detection supported by the premask paradigm developed by Elliott and Müller (in Experiment B), we recorded response time and response accuracy during target detection performance for both dyslexic and normal readers. By employing the premask paradigm of Elliott and colleagues we investigated the extent to which the dyslexic and normal readers may be differentially susceptible to synchronous premask presentation. Given that successful priming appears to be highly dependent upon the precise frequency of premask matrix presentation, we considered that impaired magnocellular or transient subsystem responses and/or a prolongation of visual persistence might be responsible for reducing the temporal fidelity of the neural response to premask matrix presentation. However, on the evidence of Elliott and Müller (2001), who discovered no reduction in priming as a consequence of the introduction of apparent-motion signals at target matrix onset (these signals were induced by varying the size of the target elements relative to the size of the premask crosses), it seemed more likely that variations in priming would relate to variations in the structure of prime persistence, which has been shown to match, with high fidelity, the 40-Hz structure of premask matrix presentation (see Elliott & Müller, 2000). On this basis, we expected priming effects to be reduced or absent and an overall elevation in target and nontarget detection RTs. Furthermore, if target search were influenced by prolonged and temporally ill-defined premask matrix persistence, a further expectation was of slightly slower RTs to target matrices presented after premask matrix presentation (Experiment B) relative to those presented in Experiment A.

In the present experiments, premask crosses and target elements were presented in  $5 \times 5$  element matrices and in two inducer specification conditions. Variations in inducer specification refers to variations in the premask cross-cross or target junction-junction continuances, which were

specified by two luminous contours covering in equal measures either 40% ( $2 \times 20\%$ ) or 60% ( $2 \times 30\%$ ) of the overall distance between the junction or cross vertices. On the basis of similar manipulations made by Elliott and Müller (2001), variations in inducer specification were expected to result in 60%-specified targets being faster to detect than targets specified to 40%, indicating variation in inducer specification to be analogous with variation in the "figural goodness" of the target. Consequently, this variation was intended as a means of examining whether or not target detection was differentially influenced as a function of figural goodness for the dyslexic relative to normal readers in Experiment A, while in Experiment B, the extent to which priming interacted with figural goodness could be examined with a view to assessing the extent to which synchrony priming supports figural processing if the speed of target detection is otherwise compromised relative to the performance of normal readers.

## METHODS

### Participants

A total of 23 students, 11 male dyslexic readers and 12 normal readers (11 male), participated in Experiments A and B. For Experiment C, 11 adult volunteers (4 male, mean age 25.73 years) were recruited from the undergraduate population of the University of Leipzig. The groups in Experiments A and B (dyslexic–nondyslexic) were matched according to age and intelligence (see Table 1 for details). For each participant, the level of general intelligence was measured by means of the Raven Standard Progressive Matrices (Heller, Kratzmeier, & Lengfelder, 1998) on the same day the experiment was performed. Each of the participants was found to have at least normal intelligence; no significant differences in age or intelligence were found between the groups.

The dyslexic participants were recruited from a special school for language-disabled children in Leipzig (seven students) and from a vocational

**Table 1.** Comparison of age and intelligence level between normal and dyslexic readers<sup>a</sup>

	Normal readers		Dyslexic readers		<i>t</i>	<i>p</i>
	Mean	(Min, max)	Mean	(Min, max)		
Age	14.75	(13, 17)	16.09	(13, 20)	-2.058	.052
IQ	97.17	(80, 127)	91.82	(74, 106)	1.070	.297

<sup>a</sup>Homogeneity of variances assumed following a Levene-test. Intelligence level was measured with the Raven Standard Progressive Matrices.

education centre for language-disabled adolescents in Borna, Saxony (four students). The students comprising the control group were obtained from a junior high school (*Realschule*) in Leipzig. All participants had normal or corrected-to-normal vision. The regional supervisory school authority as well as the students and their parents gave their informed consent to the participation of the children in the present study. All students received €10.00 and a reimbursement for transportation costs for their participation. The adult volunteers were paid at a standard rate of €8.00 per hour.

Q1

Q1

### Diagnostics

A diagnosis of dyslexia was given to all participants of the dyslexia group according to the discrepancy definition of the diagnostic manual ICD9/ICD10 (World Health Organisation, 1992) by a team of professional examiners some 5–12 years before the study. All dyslexic readers conformed to the definition of developmental dyslexia as opposed to acquired dyslexia. The examination team consisted of one psychologist, two specialist teachers for dyslexic children, and one specialist teacher for language impaired children. As required by federal law, the diagnosis had to be given during the second class of primary school using the test battery by Weigt (1980; see also Kossakowski, 1961) in order to send the children on a 2-year special training programme for dyslexic students during grade 3. The test battery included tests of reading and spelling for both contextualised and isolated letters and words, tests

of phonological encoding ability, phoneme segmentation, phonological and visual differentiation (Breuer & Weuffen, 1995), and visual recognition, as well as a test of the ability to learn mathematical procedures. In addition, physical development, sensory functioning (vision and hearing tested by an ophthalmologist and an otolaryngologist), evidence of mental retardation, and motivational, attentional, emotional, educational, and social factors that may have fostered the reading disability were tested. The participants of the control group were not tested in advance; however, their teachers of the German language classified their reading ability as normal or above average.

At the time of the experiment all of the dyslexic participants fitted the discrepancy definition of dyslexia according to German law. Nevertheless, and given an absence of current discrepancy data, a supplementary diagnosis of the dyslexic participants was undertaken in parallel to the experimental testing. Each participant was asked to complete four short reading tasks, a test of nonword and letter-similar nonword reading, a test of frequent word reading, and a test of text reading, all taken from the *Salzburger Lese- und Rechtschreibtest* (Salzburger Reading and Spelling Test, SLRT; Landerl, Wimmer, & Moser, 1997). The tests cannot be used as a standardised diagnostic tool, because the norms are limited to grades 1 to 4; however, all dyslexic participants performed below 1.5 *SDs* of the reading norm for grade 4 in at least one of the tests, indicating a reading performance discrepancy of some 3–4 grades at the time of testing (the dyslexic participants were at least grade 7 students at the time of testing).

## Experimental design

### *Experiment A*

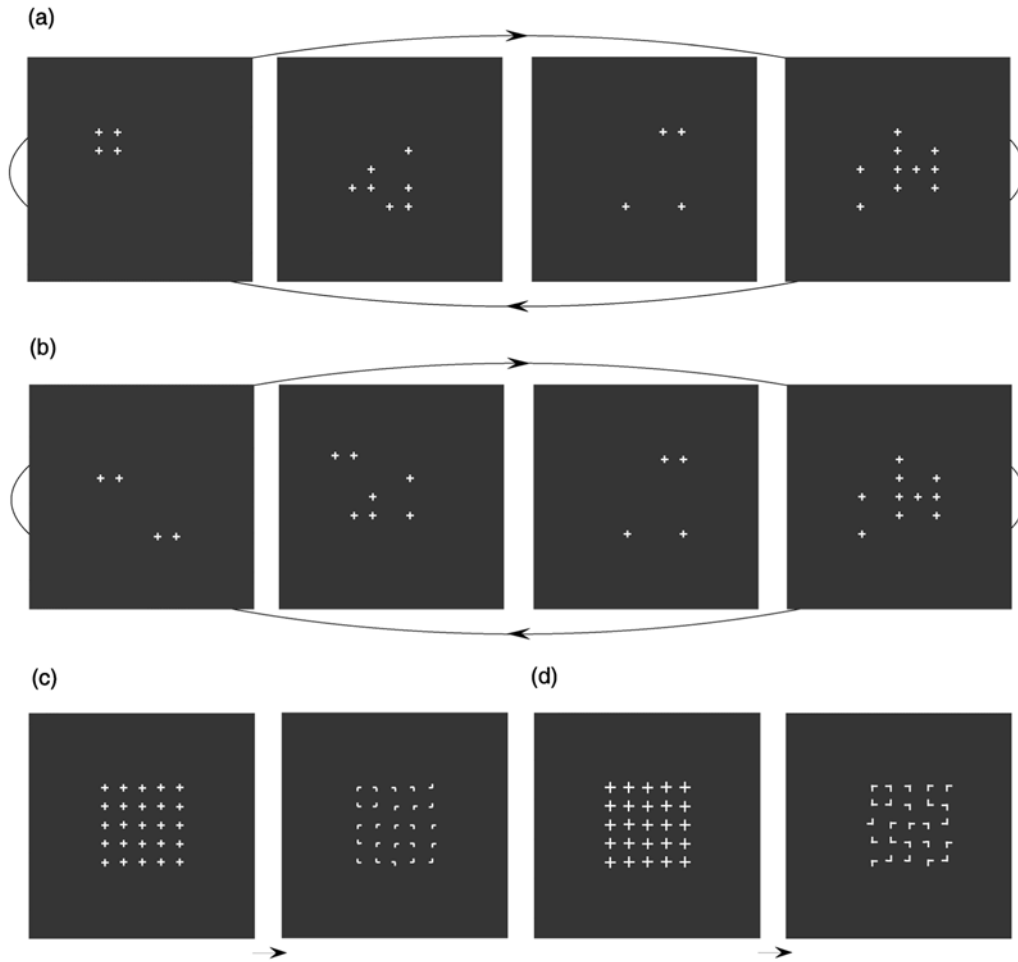
Experiment A aimed to investigate both accuracy and the time taken to detect targets for both dyslexic and normal readers in a search task of varying difficulty. On each trial a  $5 \times 5$  matrix of corner junctions (elements) was presented, which on 50% of trials included the collinear arrangement of four elements to form a (target) Kanizsa-type

square (As in Figure 1d). On a further 50% of trials no four elements were thus grouped (target absent trials). Targets could appear with equal probability in any one of the 16 possible display locations in the  $5 \times 5$  matrix.

The measure of target detection difficulty employed in Experiment A was expressed in terms of the ratio of the physically specified to the unspecified continuance between the axes of the regularly arranged junction elements: Two ratios were employed in which either 40% or 60% of the overall continuance was specified while the remaining 60% or 40% (respectively) of the continuance was not illuminated. Note that “physically specified” here refers to the representation of the collinear continuance between premask crosses or target elements by means of two luminous contours covering in equal measures either 40% ( $2 \times 20\%$ ) or 60% ( $2 \times 30\%$ ) of the overall distance between the element vertices (see Figures 1c and 1d for examples of variations in physical specification of premask—left image—and target—right image—matrices): It was expected that for better-specified targets, target detection would be both faster and more accurate than for less well-specified targets.

### *Experiment B*

Experiment B aimed to examine the extent to which the target detection performance in Experiment A varied differentially for the normal readers and dyslexic participants as a function of synchrony priming. On each trial in Experiment B, a  $5 \times 5$  premask matrix of crosses composed of a repeating pattern of four sequentially presented premask matrix frames was presented for 600 ms, after which all of the premask crosses were reduced to a semistatic display of  $90^\circ$  corner junctions. On 50% of trials, a figurally relevant “synchronous premask,” defined by four premask crosses (elements) presented simultaneously and in square arrangement, was embedded in the premask matrix as the first of the four sequentially presented premask matrix image frames (a sample sequence of four premask frames, including a synchronous premask in frame 1, is illustrated in Figure 1a). The elements com-



**Figure 1.** *The display paradigm. The series of panels in (a) and (b) give two contrasting examples of the possible spatiotemporal distributions of 25 premask crosses across the 4 premask matrix presentation frames. The panels in (a) illustrate the “synchronous” premask condition in which, in the first frame 4 premask crosses are presented simultaneously and in square arrangement. The panels in (b) illustrate a possible “random” premask condition in which the first frame consists of 4 premask crosses that did not correspond to a square arrangement. In both synchronous and random conditions the remaining 21 pre-mask crosses were pseudorandomly distributed across the remaining premask matrix frames, obeying the constraint that remaining frames comprising 4 or more elements would not include accidental arrangements of crosses in square formation. The sequences of 4 frames were repeatedly presented for an overall premask matrix presentation duration of 600 ms. During this period, each frame was repeatedly presented at a rate of 10 frames, with constant frame durations of 25 ms and with interframe intervals of < 1 ms. This pattern produced a global 40-Hz presentation frequency across the entire premask matrix, helping to ensure that the premask matrices appeared as almost static  $5 \times 5$  element matrices characterised by apparently stochastic surface flicker, within which the contents of a given frame were not discernable. After 600 ms the premask matrices were immediately replaced at the same screen location by a target matrix: To supplement panels (a) and (b), panels (c) and (d) offer some indication of what the experimental participants actually perceived for premask matrices (left-hand panel) and target matrices (right-hand panel) comprising inducers, in (c) physically specified to 40% of the element–element continuances or in (d) physically specified to 60% of the continuance. On 50% of trials the target matrix included 4 grouping corner junctions in collinear arrangement. Note that in panels (c) and (d) a target appears in the upper left-hand matrix location.*



prising the synchronous premask were presented at the matrix location that could subsequently be occupied by the four elements that defined a target Kanizsa-type square (compare the matrix location of the four elements presented in square arrangement and in the first frame in Figure 1a with the location of the target Kanizsa figure in Figure 1d). The effects of synchronous-premask presentation were controlled for in the remaining 50% of trials by presentation of four elements in the first frame of the premask presentation sequence, although the spatial arrangement of these four premask elements was pseudorandomised across the entire premask matrix and they were not presented in square arrangement (the "random premask condition," see Figure 1b). This measure constituted a specific spatial and temporal control for synchronous premask presentation in that the four-element random premask frame (as with the synchronous premask frame, always presented first in the sequence of premask frames) was designed specifically not to contain four grouping elements. An additional control concerned the distribution of the remaining 21 premask matrix elements across the three remaining premask matrix frames. These elements were pseudorandomly distributed spatially across the remaining premask matrix locations and temporally, across the three remaining frames with special attention to the restriction that for frames comprising four or more elements there would be no arrangement of four premask elements presented simultaneously and in square arrangement.

In all trials either a "target" or a "nontarget" matrix comprising 25 corner junctions (elements) was presented immediately following termination of premask matrix presentation. A target matrix (presented in 50% of trials) included presentation of a target Kanizsa-type square. Alternatively on the remaining 50% of trials the matrix of junction elements did not include any combination of elements that grouped to form an illusory square. Presentation of the premask and target conditions were varied such that both target and the nontarget matrices followed both synchronous and random premask matrices with equal probability (although presentation orders were fully randomised across trials for each experimental ses-

sion). Note in addition that both targets and synchronous premask could appear with equal probability in any one of the 16 possible display locations in the  $5 \times 5$  matrix. The presentation quadrant of the synchronous premask elements and the elements of the target was always identical. The repeated and high-frequency presentation of the premask frames lead to a phenomenal experience of a static  $5 \times 5$  matrix characterised by some apparently stochastic surface flicker (similar to that illustrated in the left image of Figure 1c), within which it was impossible to discern the spatiotemporal structure of the premask matrix (see also Elliott & Müller, 1998, Experiment 2).

As with Experiment A, in Experiment B the ratios of physically specified to unspecified information along the continuances between the axis of the collinearly arranged junction elements were varied, but in this instance both for the target junctions and premask elements. Consistent with Experiment A, two ratios were employed in which either 40% or 60% of the overall continuance was specified by an illuminated contour while the remainder (60% or 40%, respectively) of the continuance was not illuminated.

### *Experiment C*

Experiment C aimed to provide comparative data against which the pattern of dyslexic performance in Experiment B might be evaluated: Specifically, to establish whether or not target-priming effects were subject to increased variability and whether or not negative-priming effects emerge on target-absent trials as a general function of increasing RT.

The basic paradigm employed in Experiment C matched that of Experiment B, with two critical differences. First, the detection task was made more difficult overall than that undertaken in Experiment B by reducing the ratio of physically specified information presented along the continuances between collinearly arranged junction elements. In Experiment C, 20% of the overall continuance was specified by an illuminated contour while the remaining 80% of the continuance was not illuminated. This modification effec-

tively reduced the amount of information in either premask crosses or the target junctions upon which collinearity grouping could be achieved and was expected to reduce target conspicuity with a commensurate reduction in the efficiency of nondyslexic detection performance to a level equivalent to the dyslexic performance in Experiment B. On the basis of overall equivalence between dyslexic and nondyslexic performance, an evaluation was possible of negative priming as a generalisable pattern of performance beyond the performance of dyslexic participants in Experiment B. Finally, in Experiment C, adult volunteers were employed in order to provide the general estimate of the tendency for negative priming effects to emerge in the Elliott and Müller task.

### Apparatus

Stimuli were presented on a 6" Tektronix 608-oscilloscope monitor with a fast-decay P15 phosphor, which plots image frames with temporal control greater than 1 kHz. The use of a P15 phosphor ensured that the on-screen image persistence reduced to 10% of normal image intensity within 2.8  $\mu$ s (microseconds) of image termination. Stimulus-frame generation, event timing, and data collection were controlled by an IBM-PC compatible computer. Oscilloscopic image presentation was controlled by the computer through an Interactive Electronics point-plotter buffer with 8 MB frame store memory. The buffer permitted pixels to be plotted directly from memory at a rate of one pixel every microsecond. Both the oscillatory (premask) and the static (target) frames were displayed at a background presentation frequency of 1 kHz to keep the image point luminance constant. Experiments were conducted in a dark room (mean screen surround luminance 7.8  $\text{cd}/\text{m}^{-2}$ ), with stimulus luminance maintained at 40.0  $\text{cd}/\text{m}^{-2}$  upon a background field of 7.5  $\text{cd}/\text{m}^{-2}$ . Stimuli with low contrast were used to selectively stimulate the magnocellular system, which is most responsive to low contrast stimulation, especially if stimulation exhibits high temporal frequencies. Observers viewed the monitor from a distance of 57 cm main-

tained via a chin rest. Participants pressed one of two response buttons for target-present and target-absent responses (using the dominant and the non-dominant hand, respectively).

### Stimuli

The  $5 \times 5$ , 25-element displays subtended  $11^{\circ}48' \times 11^{\circ}48'$  under 40% inducer specification conditions and  $12^{\circ}29' \times 12^{\circ}29'$  under 60% inducer specification conditions. For the 20% inducer specification conditions used in Experiment C the  $5 \times 5$ , 25-element displays subtended  $11^{\circ}07' \times 11^{\circ}07'$ . Premask elements were crosses of size 38', 1'17', or 1'54' and were separated from their nearest horizontal and vertical neighbors by 1'59', 1'21', or 43' for the 20%, 40%, and 60% conditions, respectively. Junction elements in the target display subtended 20', 39', or 59' and were separated horizontally and vertically by 1'59'–2'38', 1'21'–2'38', or 43'–2'38' for the 20%, 40%, and 60% conditions, respectively. The premask matrix (appearing in Experiments B and C) comprised a repeating sequence of four image frames that was presented for 600 ms (see Figure 1). This presentation regimen allowed a frequency of premask matrix presentation to be determined in the following way: Each frame was presented for 25 ms with 24 frame presentations during the 600 ms of premask matrix presentation, which is equivalent to a global premask frame presentation rate of 40 Hz. (Note that the elements of each of the four frames actually repeat at 10 Hz).

### Experimental procedure

Experiment A consisted of eight blocks of 40 trials per block preceded by a training block. The target inducer specification (40% vs. 60%) and the target (absent vs. present) condition were varied randomly across each block. Participants were instructed to fixate the centre of the  $5 \times 5$  element matrix and avoid unnecessary eye movements. Following a brief (300 ms) computer-generated tone (250 Hz), participants were presented with the  $5 \times 5$  junction matrix and were asked to respond as quickly and as accurately as possible to the presence or absence of a

target Kanizsa-type square within the matrix of junctions. Matrix presentation was terminated immediately following response. In the event of an error, feedback was provided through a second computer-generated tone (100 Hz).

Experiments B and C consisted of 16 and 8 blocks (respectively) of 40 trials per block. As with Experiment A, the experimental blocks were preceded by a training block of 40 trials. The factors target (present, absent), premask synchrony (synchronous, random), and, for Experiment B only, the factor target inducer specification (40%, 60%) were varied randomly across blocks. Following a brief (300 ms) computer-generated tone (250 Hz), participants were presented with the oscillating  $5 \times 5$  matrix of premask crosses, which, after a presentation time of 600 ms, reduced to simple  $90^\circ$  corner junctions by the removal of redundant line segments. Participants were instructed to fixate the centre of the premask matrix and, avoiding eye movements during presentation, allow the matrix to flicker in the centre of their visual field. Following premask matrix termination/target matrix presentation, participants were asked to respond as quickly and as accurately as possible to the presence or absence of a target Kanizsa-type square within the matrix of corner junctions. Matrix presentation was terminated immediately following response. In the event of an error, feedback was provided through a second computer-generated tone (100 Hz).

Note that the presentation order of the experiments A and B was counterbalanced across groups (dyslexic and normal readers) and randomised between participants in order to control for the possibility of order effects influencing either RT or error production.

## RESULTS

### Experiment A

#### RT analysis

For each participant, RTs on trials on which a response error was made (normal readers: 3.2%, dyslexic readers: 2.4%) and RTs above 3 or below

2.5 SDs from the means of all correct observations (by condition) were removed from the data prior to further analysis. The error RTs tended to be overall slower than correct RTs and analysis of the probability correct by RT revealed no significant correlation, arguing against the correct data being contaminated by fast guess responses.

Analyses were conducted using repeated-measures analysis of variance (ANOVA) with the between-subject factor group (normal readers, dyslexic readers), and the within-subject factors target (present, absent) and inducer specification (40%, 60%). A significant main effect for group,  $F(1, 21) = 14.865$ ,  $p = .001$ , mean RTs 745 ms (controls), 1072 ms (dyslexic readers), showed dyslexic readers to be substantially slower than normal readers in this search task. Consistent with target search involving some additional matrix checking for target-absent matrices, RTs were faster to target relative to nontarget matrices (753 ms, target-present vs. 1065 ms, target-absent) and were overall faster when responding to the better-specified targets: Inducer-specification main effect:  $F(1, 21) = 62.178$ ,  $p < .001$ , mean RTs 881 ms (60% condition) and 936 ms (40% condition). A significant Group  $\times$  Inducer Specification interaction,  $F(2, 21) = 12.866$ ,  $p = .002$ , see Table 2, was based upon more pronounced differences in the RT latencies to matrices consisting of elements specified to 40% relative to those specified to 60% for the dyslexic participants. A simple main effects analysis revealed matrices with elements specified to 60% were 80 ms faster to respond to than those including elements specified to 40%,  $F(1, 21) = 6.006$ ,  $p < .025$  for the dyslexic participants; identical analyses for the normal readers revealed a smaller and nonsignificant difference of 30 ms,  $F(1, 21) = 0.9198$ , n.s. This may indicate an increased

**Table 2.** The mean RTs (and standard errors) in ms for the normal and dyslexic readers by inducer specification (40%, 60%) in Experiment A

	40%	60%
Normal readers	760 (61)	730 (57)
Dyslexic readers	1112 (64)	1032 (59)

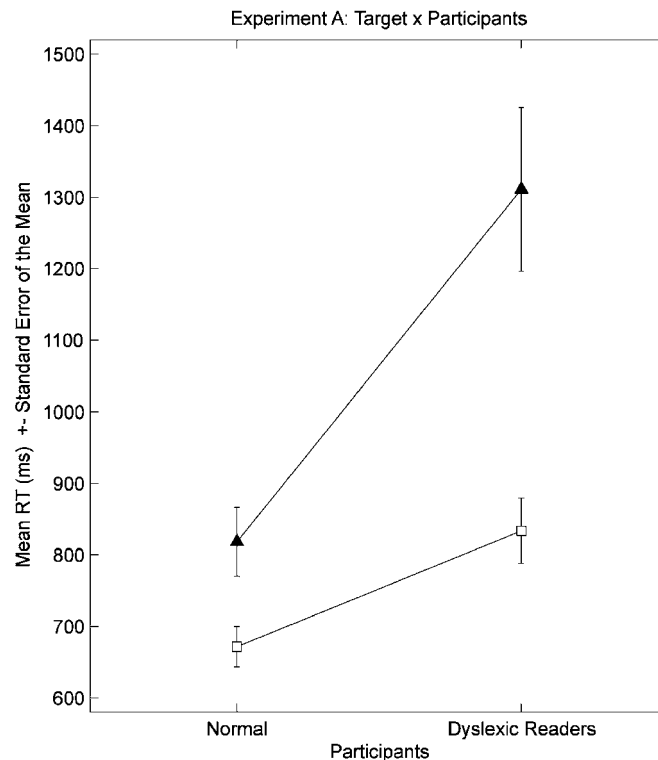
reliance upon potential “goodness” criteria given overall slower detection performance for the dyslexic participants.

However, the nonsignificant Target  $\times$  Inducer Specification and the nonsignificant three-way interactions suggest against a direct relation between improved inducer specification and the improved detectability of a “target,” suggesting that improved inducer specification is employed as a means of facilitating more efficient matrix search in general. This may also relate to, but not substantially influence, the general tendency for the dyslexic readers to produce overall increased RTs relative to normal readers, which in turn contributes to a significant interaction between group and target,  $F(1, 21) = 18.608$ ,  $p < .001$ . This interaction arose due to a smaller difference

between target present and absent RTs for normal readers relative to the difference in target present – absent RTs produced by the dyslexic readers (see Figure 2). Inspection of Figure 2 also shows that the spread of the mean RTs tended to be generally greater for dyslexic relative to the normal readers (variances of the grand means were 167,298 vs. 38,135) although on the basis of these data it cannot be ruled out that variance increased as a general function of increased RTs.

#### Error analysis

An ANOVA was performed on the arcsine-transformed error data with identical main terms to those employed in the analysis of the RT data. A significant inducer specification main effect was



**Figure 2.** The mean target present and target absent RTs (with standard errors) by the participant groups (normal and the dyslexic readers) revealed in Experiment A. Unfilled squares and filled triangles represent the mean target present and target absent RTs (respectively).

revealed,  $F(1, 21) = 14.536, p = .001$ , representing a higher degree of errors under 40% conditions (3.6% errors) relative to the 60% conditions (2.0% errors), suggesting that target detectability may have been substantively influenced by variations in inducer specification. There were no significant between-group effects.

### *Discussion of Experiment A*

The principle outcomes of Experiment A were tendencies for the dyslexic participants to show greater sensitivity to inducer specification, suggesting search to benefit more substantially for inducers specified to 60% compared with 40% specification and relative to the performance of normal readers. Consistent with Keen and Lovegrove's (2000) findings, search was overall slower for the dyslexic relative to normal readers, in addition to which the dyslexic RTs were accompanied by overall higher variance relative to that of the normal readers.

## **Experiment B**

### *RT analysis*

One aim of this study was to examine variations in synchrony-priming performance for dyslexic relative to normal readers, and in this context Experiment A sought not only to identify variations in detection performance between these groups, but also to provide a set of baseline data with which the data obtained following flickering premask matrix presentation might be compared.

As with Experiment A, for each observer the RTs on trials on which a response error was made (2.0% of all trials for normal readers, 1.4% of all trials for dyslexic readers) and RTs above 3 or below 2.5 *SDs* from the means of all correct observations (by condition) were removed from the data prior to further analysis. No speed-accuracy trade-offs were revealed from condition by condition correlations of the RTs by the associated probability of that RT being correct or erroneous. Also similar to Experiment A, the overall spread of RTs in Experiment B was substantially increased for

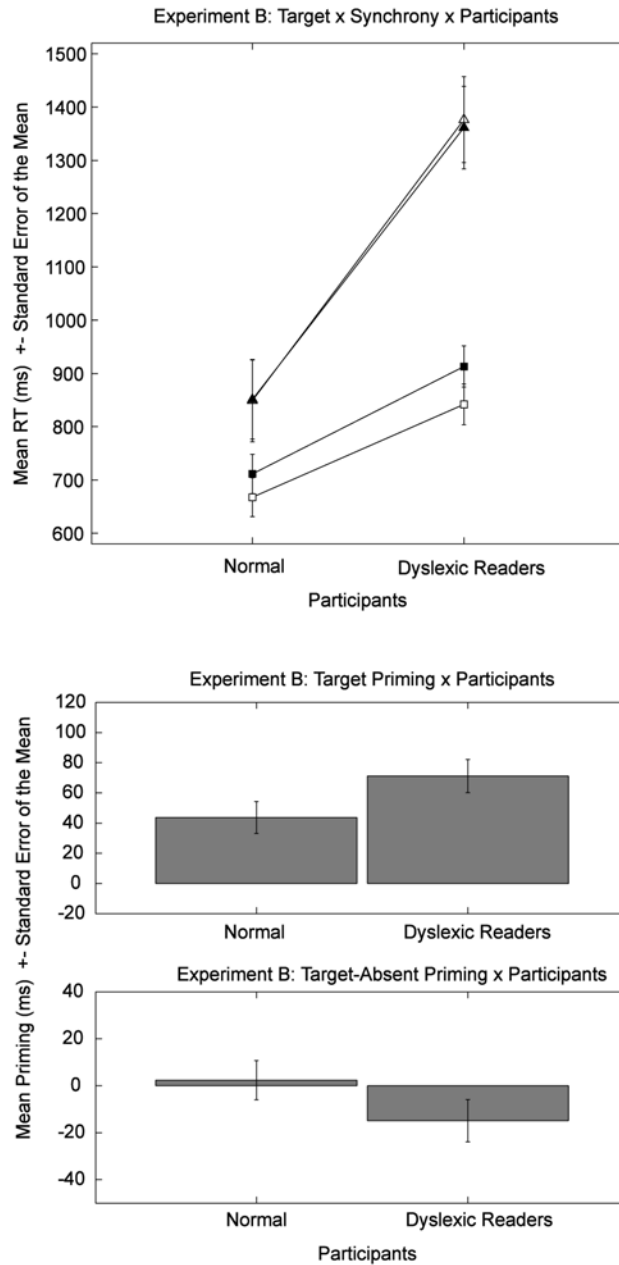
the dyslexic relative to normal readers (variances were 48,407 vs. 23,368, respectively).

The RT data were examined by means of repeated-measures ANOVA with the between-subject factor group (normal, dyslexic readers), and the within-subject factors, target (present, absent), synchrony (synchronous, random), and inducer specification (40%, 60%). A significant group main effect,  $F(1, 21) = 20.319, p < .001$ , mean RTs 770 ms (normal readers), 1123 ms (dyslexic readers), was found. Consistent with the task involving some degree of target search on absent trials, the target RTs were significantly faster than the target-absent RTs: Target main effect,  $F(1, 21) = 86.269, p < .001$ , mean RTs 783 ms (target-present) vs. 1110 ms (target-absent). As with Experiment A, a more precise assessment of the differences between groups in target-present-absent RT latencies was offered by a significant Group  $\times$  Target interaction,  $F(1, 21) = 22.215, p < .001$ , which arose due to a smaller difference between target-present and target-absent RTs for normal readers relative to the difference in RTs produced by the dyslexic readers (see Figure 3).

A synchrony main effect revealed a tendency to make more rapid RTs following synchronous—relative to random—premask presentation,  $F(1, 21) = 28.022, p < .001$ , mean RTs 934 ms (synchronous condition) and 959 ms (random condition). Consistent with the previous work of Elliott and Müller (1998, 2000, 2001), the presence of a significant Target  $\times$  Synchrony Interaction,  $F(1, 21) = 40.602, p < .001$ , was based upon the presence of significant differences on synchronous relative to random trials for target-present but not target-absent RTs (the random minus synchronous differences were 57 ms compared with 6 ms for the target-present and target-absent trials

**Table 3.** *The mean RTs (and standard errors) in ms for the normal and dyslexic readers by inducer specification (40%, 60%) in Experiment B*

	40%	60%
Normal readers	781 (59)	759 (51)
Dyslexic readers	1164 (61)	1082 (53)



**Figure 3.** The upper panel depicts the mean Target  $\times$  Synchrony RT functions (with standard errors) in Experiment B according to participant group (normal readers and dyslexic readers). The unfilled squares and triangles represent the mean synchronous target-present and target-absent RTs (respectively), while the filled squares and triangles represent the mean random target-present and target-absent RTs (respectively). The lower panels depict the mean priming effects (the random minus synchronous RTs) for the target-present (upper) and target-absent (lower) trials, respectively. Interestingly, and contrary to expectations, the dyslexic participants show a significant negative priming effect on target-absent trials.

respectively). However, the possibility that the significance of the Target  $\times$  Synchrony interaction may vary by group was raised in the context of a significant Group  $\times$  Target  $\times$  Synchrony interaction,  $F(1, 21) = 4.996$ ,  $p = .036$ . This interaction was explored by a series of simple main effects analyses, which revealed significant synchrony effects to be confined to target trials for normal readers:  $F(1, 21) = 43.649$ ,  $p < .001$  for the target-present trials and  $F(1, 21) = 0.118$ , n.s. for the target-absent trials. The mean random-synchronous differences were 43 ms and 2 ms for the target-present and target-absent trials, respectively). However, inconsistent with the trend for target-specific priming revealed for the normal readers and with the results of previous experiments, the dyslexic readers showed a substantial gain for synchronous relative to random premask presentation on target-present trials accompanied by a smaller but nonetheless significant cost for synchronous relative to random premask presentation on target-absent trials,  $F(1, 21) = 97.152$ ,  $p < .001$  (target-present) and  $F(1, 21) = 4.391$ ,  $p < .05$  (target-absent). The mean random-synchronous differences were 71 ms and  $-15$  ms for the target-present and target-absent trials, respectively; see Figure 3).

A second pattern of effects in which dyslexic participants showed differences to normal readers concerned variations in inducer specification: As in Experiment A, RTs were significantly faster for the 60% inducer-specification condition relative to the 40% condition,  $F(1, 21) = 26.455$ ,  $p < .001$ , mean RTs 921 ms vs. 972 ms, respectively, while a significant Group  $\times$  Inducer Specification interaction,  $F(1, 21) = 8.927$ ,  $p < .01$ , indicated that the extent to which inducer specification played a role in target detection (or target-absent verification) varied across participants. Examination of the interaction indicated the dyslexic participants to be more likely to employ an enhancement in potential goodness criteria as a means of expediting matrix search (i.e., they were faster to search matrices that included elements specified to 60% relative to those including elements exhibiting 40% specification, simple main effects analysis,  $F(1, 21) = 7.92$ ,  $p < .01$ , relative to normal readers,

$F(1, 21) = 0.607$ , n.s., see Table 4. A significant interaction between synchrony and inducer specification,  $F(1, 21) = 5.506$ ,  $p < .05$ , suggests nonetheless that variations in matrix search as a function of inducer specification may have been modified (albeit weakly) by a general tendency for priming to be more effective under 40%: simple main effects analysis,  $F(1, 21) = 13.262$ ,  $p < .01$ ; relative to 60%,  $F(1, 21) = 1.815$ , n.s., conditions. Neither the Group  $\times$  Synchrony  $\times$  Inducer Specification interaction nor the four-way interaction was significant.

#### *Error analysis*

An ANOVA with identical terms to that performed on the RT data was conducted on the arcsine-transformed error data. Significantly more misses (2.3%) than false alarms (1.1%) were recorded,  $F(1, 21) = 9.444$ ,  $p < .001$ . Consistent with Experiment A, observers made significantly more errors in the 40% inducer specification condition than in the 60% condition,  $F(1, 21) = 9.546$ ,  $p < .01$ , errors were 2.2% vs. 1.2%, respectively. A significant Inducer Specification  $\times$  Target interaction,  $F(1, 21) = 9.120$ ,  $p < .01$ , was due to participants making more misses than false alarms for matrices specified to 40% relative to those specified to 60%. This interaction is generally consistent with the pattern of effects in the RT data when it is considered that RTs tended to be overall faster for matrices with elements specified to 60% relative to those specified to 40%. In addition, this pattern of effects tends to argue against the possibility of the RT data being confounded by speed-accuracy trade-offs. The absence of a main effect or interactions involving group suggests that detection, while slowed for the dyslexic relative to normal readers, is nevertheless performed with approximately equal accuracy. Further and more specifically the absence of an interaction between group and synchrony, or an interaction of these factors with target, argues against the possibility that for the dyslexic readers the synchronous prime activates a tendency to respond "present." (A tendency to respond present might be expected to expedite correct target RTs [hits] while slowing

correct rejections on target-absent trials. Consequently any tendency for the dyslexic readers in particular to generate more misses would confound interpretation of the increased priming coupled with negative priming effects as recorded in the RT data.)

#### *Discussion and supplementary analysis of Experiment B*

The results of Experiment B compared very well with those of Experiment A in a number of respects, which may be taken to indicate that conclusions concerning target detection following oscillatory priming are entirely consistent with those applied to a simple target detection task: As in Experiment A, in Experiment B normal readers produced overall faster RTs than dyslexic readers. Furthermore, as in Experiment A, in Experiment B there was a trend for the dyslexic participants to make greater use of potential goodness criteria (in the form of variations in inducer specification) as a means of expediting matrix search.

In spite of large differences in overall RTs between normal and dyslexic readers, synchrony enhancements in dyslexic readers were not decreased relative to the performance of normal readers. Instead, numerically larger target-priming effects accompanied the longer RTs produced by dyslexic readers relative to the normal readers and were mainly confined to trials with weaker inducer specification (i.e., the 40% specified inducers). Of principle interest was the inverted, negative-priming effect produced by the dyslexic readers on target-absent trials. This pattern of effects is of interest as it is clearly inconsistent with the normally target-specific priming effects previously reported by Elliott and Müller (1998, 2000, 2001). It seemed possible that negative priming on target-absent trials, alongside relatively substantial target priming effects, relate to the overall longer search and detection RTs that characterised the performance of the dyslexic readers. On the other hand, dyslexic readers' performance is in general characterised by substantial variability relative to normal readers' performance and in some cases the performance of the dyslexic readers was as fast as

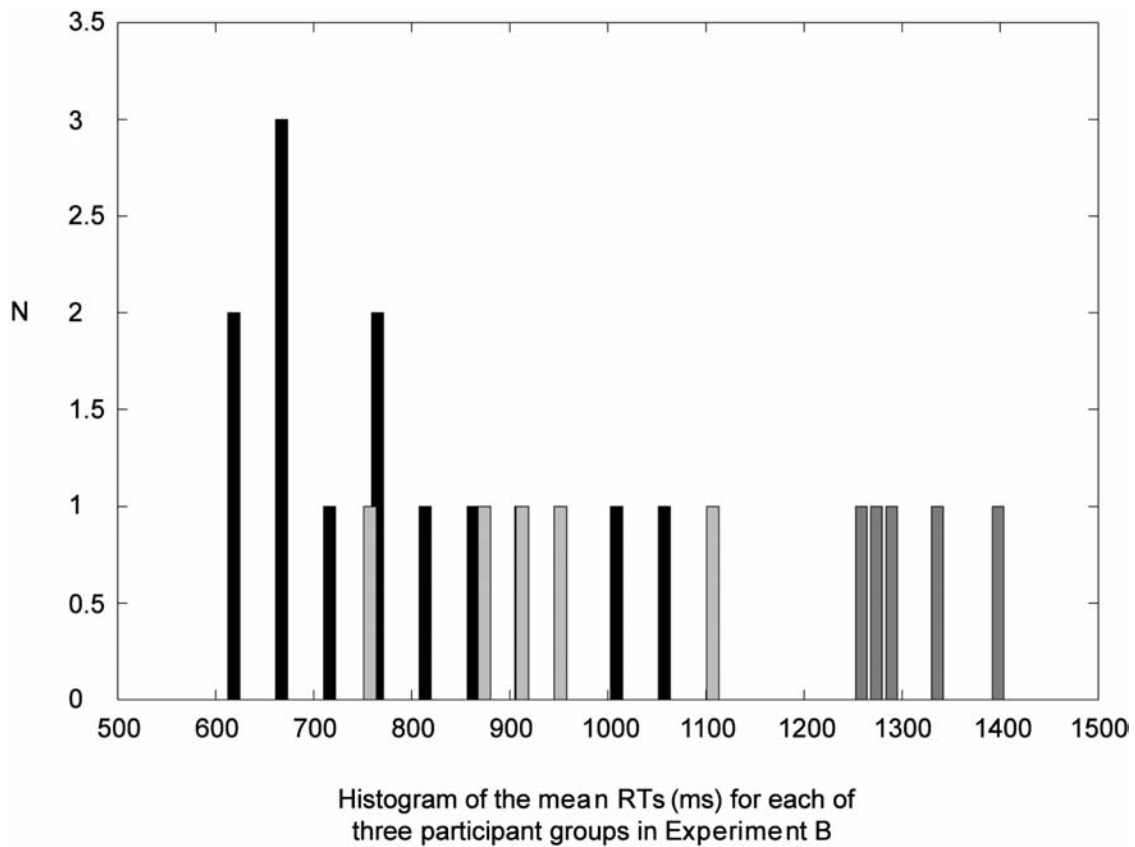
that of normal readers (see Figure 4). This raises the question of whether negative and relatively substantial target priming should be considered characteristic of the dyslexic group as a whole, or whether such effects are attendant upon dyslexic readers responsible for particularly slow search and detection performance. If negative and substantial target priming were only to be found for the slower dyslexic participants a further question is whether such effects would emerge for any participant group responding as slowly as those dyslexic readers.

The latter hypothesis, that negative and relatively substantial target priming may emerge as a general function of particularly slow search and detection performance, was examined in more detail in Experiment C. As a first step, and in order to evaluate the data from Experiment B, two groups referred to as "slow" and "fast" dyslexic responders were derived by splitting the relevant set of grand mean RTs into two sets of values across the median location at 1195 ms. Thus 10 of the 11 dyslexic readers were divided into two sets of 5 participants per set: the first set contained 5 fast RTs (mean set RT = 920 ms) while the second set contained 5 slow RTs (mean set RT = 1312 ms, see Figure 4). The sets of faster and slower responders were approximately matched according to age and intelligence (see Table 4 for details).

Conditionwise RT performance within each set of dyslexic responders was examined by means of separate, near-identical analyses of variance to that carried out on the entire data from Experiment B (without the between-subjects factor group). The general aims of these analyses were descriptions of the pattern of Target  $\times$  Synchrony interactions: The specific aims were separate estimates of the magnitude and statistical significance of target priming alongside estimates of the magnitude and statistical significance of (target-absent) negative priming for the sets of fast and slow responders, respectively.

Analysis of the set of fast RTs revealed a pattern of effects consistent with normal readers' performance and by and large characteristic of target-





**Figure 4.** The RT distributions for normal readers (black bars), the fast dyslexic responders (light grey bars), and slower dyslexic responders (medium grey bars) revealed in Experiment B. The fast and slow dyslexic responders were differentiated on the basis of a division of mean dyslexic readers' performance around the functional median. Note that while slow dyslexic readers are not reliably primed and produce negative priming on target-absent trials, the fast dyslexic readers respond in a range comparable to that of normal readers while exhibiting target-specific priming consistent with the performance of normal readers and adult controls.

**Table 4.** Comparison of age and intelligence level between fast and slower dyslexic responders<sup>a</sup>

	Fast responders		Slow Responders		t	p
	Mean	(Min, max)	Mean	(Min, max)		
Age	15.6	(13, 18)	15.8	(14, 17)	-0.191	.854
IQ	88.4	(74, 102)	97	(88, 106)	-1.474	.179

<sup>a</sup>Homogeneity of variances assumed following a Levene-test. Intelligence level was measured with the Raven Standard Progressive Matrices.

priming performance as previously reported by Elliott and Müller. The significant Target × Synchrony interaction,  $F(1, 4) = 15.887$ ,  $p < .025$ , indicated synchrony effects to be confined to target trials: The mean random-synchronous differences (and 95% confidence interval around the means in ms) were 72 (44) ms and 8 (17) ms for the target-present and target-absent trials, respectively. By contrast, analysis of the set of slow RTs revealed a significant Target × Synchrony interaction,  $F(1, 4) = 11.672$ ,  $p < .05$ . When analysed by means of a least significant difference test, this interaction

described a substantial and significant negative priming effect on target-absent trials relative to a numerically larger although nonsignificant target priming: The mean random-synchronous differences (and 95% confidence interval around the means in ms) were 63 (76) ms and 44 (33) ms for the target-present and target-absent trials, respectively<sup>1</sup>.

These supplementary analyses of the dyslexic readers' RTs are suggestive of two different modes of performance: In the first mode, dyslexic readers performed the search and detection task approximately as rapidly and in a fashion broadly similar to that of normal readers, although target priming effects tend to be somewhat larger than usual. Conversely, in a second mode, dyslexic readers who were overall slower to conduct the search and detection task in comparison with the other participants show numerically large priming effects that are, at the same time, sufficiently variable to be nonsignificant. In addition, and quite unlike normal readers and the results previously reported by Elliott and Müller, these dyslexic readers show a strong tendency for negative priming on target-absent trials.

Before considering performance in the second of these two modes to be particular to the slower dyslexic responders it was necessary to first determine whether or not target priming was inconsistent, i.e., subject to high variability, and whether negative priming emerged for any participant group responding as slowly as the slower dyslexic responders. These hypotheses were examined in Experiment C.

## Experiment C

### *RT analysis*

In Experiment C, adult volunteers were presented with a similar target detection task to that employed in Experiment B, with the difference that the premask and target matrices comprised elements with very much reduced inducer specification relative to the matrices employed in either Experiments A or B. This measure was designed to elevate baseline RTs based upon the assumption that, for very weakly specified target and nontarget matrices, the overall RT latency would come to be determined by inducer specification. It was also assumed that given a slowing of RTs to a level approximately equivalent to the RT latencies of the slower dyslexic readers in Experiment B, the appropriate conditions would be achieved for an evaluation of two hypotheses arising from Experiment B: Namely, that both unreliable target priming coupled with the negative priming effects observed on target-absent trials are specific to slower dyslexic participants, or instead occur as a general function of slow detection performance.

As with the previous experiments, the RTs on trials in which a response error was made (9.8% of all trials) and RTs above 3 or below 2.5 *SDs* from the means of all correct observations (by condition) were removed from the data prior to further analysis. No speed-accuracy trade-offs were revealed from condition by condition correlations of the RTs by the associated probability of that RT being correct or erroneous. The RT data were examined

<sup>1</sup> Given a near-identical pattern of target detection performance between Experiments A and B and in order to determine the external validity of the supplementary analysis conducted on Experiment B, an identical median split to that performed on the grand mean data of Experiment B was performed on the equivalent data from Experiment A. The resulting sets of fast and slow dyslexic responders in Experiment A were then examined in two separate analyses of their corresponding primed detection RTs from Experiment B. While the resulting sets differed with respect to two members from those sets derived from the partition of Experiment B, the subsequent analysis revealed closely matching patterns of performance to those revealed from the equivalent analysis of Experiment B (detailed in the main body of text). Notably, and in the case of both fast and slow dyslexic responders, the critical Target  $\times$  Synchrony interaction was significant, attributable in the case of the fast responders to target-specific priming with no negative priming: The mean random-synchronous RTs (and 95% confidence interval around the means in ms) were 51 (41) ms and 1 (20) ms for the target-present and target-absent trials, respectively; and in the case of the slow responders to target-priming coupled with negative priming, the mean random-synchronous RTs were 89 (73) ms and -19 (15) ms for the target-present and target-absent trials, respectively. On these bases and with respects to the validity of supplementary analyses following the partition of Experiment B, the trends in performance of individual dyslexic participants in Experiment B may be considered to closely match their performance in Experiment A.

by means of repeated-measures ANOVA with the within-subject factors target (present, absent) and synchrony (synchronous, random).

The target RTs were substantially faster than the target-absent RTs, target main effect  $F(1, 10) = 274.37$ ,  $p < .001$ . The mean RTs were 1020 ms for target-present and 1449 ms for target-absent trials, and were generally comparable with the RTs produced by the slower dyslexic responders in Experiment B (1126 ms for target-present and 1498 ms for target-absent trials). A statistical comparison of the mean RTs between Experiments B and C, assuming unequal variances, confirmed the difference to be nonsignificant:  $t(14) = 1.42$ ,  $p = .177$ . Although the synchrony main effect was not significant, a significant Target  $\times$  Synchrony interaction,  $F(1, 10) = 9.547$ ,  $p < .025$ , suggested priming effects to be confined to target trials. This suggestion was confirmed by simple main effects analyses that revealed significant priming effects on target trials,  $F(1, 10) = 13.122$ ,  $p = .005$ ; mean random-synchronous RT difference = 30 ms, but not on target-absent trials in which a small, nonsignificant RT cost was recorded,  $F(1, 10) = 0.559$ , n.s.; mean random-synchronous RT difference = -5 ms.

#### *Error analysis*

An ANOVA with identical terms to that performed on the RT data was conducted on the arcsine-transformed error data. The only effect revealed by this analysis related to the production of substantially more misses than false alarms,  $F(1, 10) = 112.737$ ,  $p < .001$ , errors 17.6% and 2.1% for the target-present and target-absent trials, respectively. This quite substantial difference confirms that participants experienced considerable difficulty in target detection as a function of reduced inducer specification, but at the same time suggest it as unlikely that the target RTs were contaminated by speed-accuracy trade-offs.

#### *Discussion of Experiment C*

As expected, a reduction in potential goodness achieved by reducing the amount of information

specified along the cross-cross and junction element continuances resulted in increased RTs that were comparable with those recorded for the slower dyslexic participants in Experiment B. However, in spite of increased RTs and a tendency for RTs on random trials to be slightly faster than those on synchronous target-absent trials, the magnitude of this difference was much smaller than the negative priming effect recorded in Experiment B and was not statistically significant. In addition, although slightly smaller in magnitude, target-priming effects were highly significant and, it is assumed, relatively stable. On this outcome, Experiment C provides no evidence to support the idea that inconsistent target priming and negative priming effects emerge as a general function of increasing task difficulty and increased RTs. Consequently, it seems reasonable to assume the negative priming effects recorded in Experiment B to be particular to the performance of the slower dyslexic readers.

## GENERAL DISCUSSION

The results of three experiments show that dyslexic readers exhibit some quite different patterns of visual-processing performance to normal readers. The performance of dyslexic readers was characterised by significantly longer search and detection latencies relative to those produced by normal readers in both standard visual search (Experiment A) and primed search tasks (Experiment B). A generalised slowing in dyslexic performance might be considered characteristic of visual processing difficulties in dyslexia given Keen and Lovegrove's (2000) findings. The lack of differences between the RTs of normal readers to 40% and 60% specified targets is a similar finding to that reported by Elliott and Müller (2001), who found the only significant difference to be between 40% and 60% specified targets and 20% specified targets. Taken together and in general, these results suggest that the influence of figural goodness upon detection RTs to the target grouping presented in these experiments would be expected to vary with increasing

inducer specification, rising to reach asymptote for targets with continua specified to around 40%. In contrast, given the same target groupings the dyslexic participants show clearly increased sensitivity to the amount of specified information, with substantially slowed RTs to 40% relative to 60% specified matrices in both Experiments A and B.

The pattern of results related to inducer specification seem to indicate that dyslexic readers rely upon figural information to facilitate target detection to a greater extent than normal readers, although the presence of substantial differences between inducer-specification conditions on target-absent trials also indicates that the dyslexic readers may not be specifically impaired in the detection of "figures." Instead, it seems reasonable to assume that these dyslexic participants may have experienced some difficulty performing local analyses of the element-element relations at target matrix quadrants, leading to overall longer detection latencies as a function of the time taken for matrix analysis rather than the time taken for the detection of a target.

This seems possible on the basis of other studies of visual-spatial disturbance in dyslexia, particularly the findings that for stimuli presented at durations similar to fixation times in reading, dyslexic readers show decreasing contrast sensitivity as a function of increasing spatial frequency (see Lovegrove et al., 1980a) and increased visual persistence (see Slaghuis & Ryan, 1999). In the former case, the extended RTs to matrices with inducers specified to 40% relative to those specified to 60% may occur due to a general difficulty in processing relatively high-frequency patterns (the less well-specified inducers occupying a smaller area of visual space than the better-specified inducers and thereby encouraging processing via high spatial frequency filters). In the case of extended visual persistence, given that the detection task is likely to include an element of serial location-by-location matrix search, visual information may persist across fixations. During matrix search this persistence could result in information at one location overlapping and becoming perceptually integrated with informa-

tion at a subsequent location. If this were the case, successful quadrant-by-quadrant search of the target matrices would require local analyses to be extended for a time equal to or greater than the period of extended persistence to avoid errors in reportage. Examination of the error rates supports this conclusion. Given that the number of errors made by dyslexic participants was not overall different from that of normal readers, it again seems plausible to consider the increased RTs to be one consequence of extended matrix analysis and not based upon a specific difficulty to distinguish a target.

Contrary to expectation, target-detection RTs for both groups benefited from the presence of a priming stimulus in the premask matrix while the priming effects for the dyslexic group were overall larger in magnitude relative to those of normal readers (Experiment B) and adult controls (Experiment C). The dyslexic readers also showed a reversal of the normal trend for target-specific priming effects, with negative priming on target-absent trials. Further analysis of Experiment B showed that a set of fast dyslexic responders exhibited priming effects somewhat larger in magnitude than normal readers but consistent in the sense that negative priming effects were absent. By contrast a set of slower dyslexic responders were not reliably primed and showed substantial and significant negative priming. The generalisability of this effect was tested in a third experiment (Experiment C), which was conducted with adult volunteers. In this experiment, by reducing the size of the premask-cross/target inducers detection became slowed to approximately the latency of the slower dyslexic responders in Experiment B. Experiment C found no evidence for substantive negative priming (even though detection RTs were of the order of those recorded for the slower dyslexic responders), while target-priming effects were of a lesser magnitude for the adult participants in Experiment C (and the normal readers in Experiment B) relative to both fast and slower dyslexic responders in Experiment B (30 and 43 ms vs. 84 and 63 ms priming effects for the adult and normal readers, fast and slow dyslexic readers, respectively).

The performance distinction between fast and slower dyslexic responders is suggestive of the hypothesis that dyslexia is characteristically multipartite. It also appeals to Boder's (1970, 1973) distinctions between dysphonetic (accounting for 55–70% of the dyslexic population), dyseidetic (10–30%), and dysphoneidetic (about 10%) dyslexia. There is some evidence to suggest that, for at least some dyslexic readers, problems in visual processing may best be described in terms of specific abnormalities in transient system activity (see Stein, Talcott, & Walsh, 2000). On these bases, it seems plausible to consider variations in performance between fast and slow dyslexic responders in Experiment B to be one indication that the dyslexic group employed in this study comprised a mixture of dysphoneidetic and dysphonetic dyslexics (i.e., slow responders) with dyseidetic dyslexics. Considerable caution is required before accepting this conclusion as given: At the very least the number of dyslexic participants in this study precludes an effective statistical case for subtyping. In addition, it must be acknowledged that the evidence for subtyping is not generally conclusive: Williams, Stuart, Castles, and McAnally (2003), for example, found no significant differences between the different dyslexic groups or between dyslexics and normal readers with respect to visual contrast sensitivity.

In Experiment B, substantially slower detection performance can combine with increased priming for the dyslexic participants. At first glance this combination of effects appears counterintuitive: If dyslexic readers are subject to difficulties in coding the relations between (target) matrix stimuli, and if relational coding is a function of neural synchronisation, then it might be expected that they are also subject to difficulties in forming a synchronised response by virtue of which stimuli may be coded as related. However, this argument ignores the possibility that some of these participants employ the synchronous premask not as a prime but instead as a cue, thereby prioritising the matrix quadrant at which the synchronous premask is presented for subsequent processing. The evidence for a cueing account relates to the negative priming effects observed under target-absent trials for the slower dyslexic

responders: Following the presentation of a matrix with no target presented at the location previously occupied by the synchronous premask, an RT cost (relative to random premask presentation) might occur as a function of the subsequent disengagement of attention (from the synchronous-premask quadrant) in order to search other matrix quadrants for a target (e.g., Posner & Petersen, 1990). The use of a cueing strategy to outweigh any response sluggishness might also account for the increased target "priming" effects compared with the priming effects at comparable detection latencies recorded in Experiment C. It may also be the case that, while both fast and slow dyslexic responders employ a combination of synchronous-premask induced cueing and priming, the slower responders come to make greater use of the synchronous premask as a cue rather than as a priming stimulus relative to the fast responders. This may account for the high variability and consequent unreliability of the apparent priming effects for this set of dyslexic responders, given that the synchronous premask is presented below detection threshold and with limited efficiency as a cue.

Nonetheless, even the set of slow dyslexic readers in Experiment B registered a twofold increase in the magnitude of priming compared with the adult volunteers (63 vs. 30 ms) suggesting that, irrespective of the costs, cueing results in a net gain, the magnitude of the benefits recorded in target-present outweighing the mean cost obtained on target-absent trials. In the context of related research, the cueing account developed here seems consistent with dyslexic readers' performance in other tasks, which show that although dyslexic readers experience sluggish attentional capture, once attention is engaged a dyslexic reader may find it difficult to subsequently disengage and relocate to another item or location (see Hari & Renvall, 2001). Moreover, the potential for the synchronous premask to cue, rather than to prime, is supported by evidence indicating that peripheral events or events of which we are otherwise unaware may nevertheless attract attention (see, e.g., Ivanoff & Klein, 2003; McCormick, 1997).

The difference between priming and attentional deployment essentially refers to the mechanisms engaged by synchronous premask presentation. On the one hand priming is assumed to refer to the outcome of an organisation (a binding or perhaps more appropriately the temporal segmentation) of synchronous-premask information, whereas cueing does not specifically refer to perceptual organisation in this fashion but instead entails the recruitment of mechanisms for subsequent processing at the synchronous premask quadrant. This then raises the question of why the slow dyslexic responders employ the synchronous premask in a fashion analogous to a cue and are not passively primed as the other participants appear to be. An answer to this question may relate to the enhanced persistence resulting from impaired function in magnocellular pathways. In the context of synchronous-premask presentation, one consequence of enhanced persistence may be an inability to successfully differentiate the contents of the synchronous frame from preceding or subsequent frames. In other words, a lack of temporal precision in the structure of premask-induced persistence might engender a degree of spatiotemporal smearing in the content structure of the prime such that it comes to include the contents of other, temporally adjacent, premask frames. What is more, using this logic an additional possibility exists; that spurious synchronisations would arise when four elements come to group in square arrangement across temporally adjacent presentation frames. Under these circumstances dyslexic readers may experience difficulties in resolving both the spatial specificity and the temporal fidelity that come to define the prime relative to activity across the remaining premask frames, while at the same time possessing sufficient spatiotemporal resolution to be able to respond, with some probability, to the repeated presentation of some task-relevant structure at a given location in the flickering premask matrix.

On this basis, attentional deployment may come to be adopted as a compensatory strategy when insufficient temporal resolution leads to the unsuccessful segmentation of the prime from other premask matrix frames. It may also be the

case that the relatively low spatial frequencies in matrices defined by 60% inducers operate independently and as a compensatory strategy—with the result of uncommon RT enhancements for the 60%- relative to the 40%-specified matrices—under which conditions cueing may represent an alternative strategy given additional problems concerned with the perceptual resolution of the matrix elements. For the cueing account to hold, some capability for resolving the prime from other premask elements must be maintained: In other words, temporal resolution is reduced or impaired, but not entirely malfunctioning in the dyslexic readers relative to the performance of related processes in a population of normal readers.

This hypothesis leads to the following processing scenario related to the engagement and operation of attentional resources given reduced or impaired priming: Responding to spatiotemporally ambiguous outputs from retinally specified pathways, which include one or more sources of potential target-related information, neurons in later processing areas may attempt to resolve ambiguity within their receptive field substrate by selectively reinforcing potentially structured clusters of activation via descending pathways. Under these circumstances, not only might activity across the synchronous premask become reinforced, but other spurious synchronisations might also benefit from reinforcement, leading to competition between candidate synchronisations for saliency and, while competition remains unresolved, increasing amplitudes across premask-evoked activity in early visual processing areas with subsequent reinforcement. While the object of reinforcement would be to enhance signal-to-noise ratios and thereby resolve activity that may relate to subsequent target presentation, one additional consequence would be an increase in the amplitude of activity signalling the synchronous premask (and other candidate synchronisations) over and above that normally encouraged by synchronous premask presentation. Given that, in synchronous premask matrices, the most regular spatiotemporal pattern of relevance to target detection is the synchronous premask frame, it seems likely that

neural activation across the synchronous-premask frame would win the competition for access to later mechanisms more often than any spurious synchronisation that may arise. However, at the same time, the neural response across the synchronous premask may achieve a level of activation sufficient to deploy processing resources (i.e., spatial attention) across the matrix quadrant at which it is presented. As has been noted, the effect of processing resource deployment manifests clearly on target absent trials, under which conditions additional RT costs might arise due to the need to disengage attention from the synchronous premask quadrant prior to search of other quadrants of the target display, which is precisely the pattern of effects revealed in the performance of the dyslexic participants.

In summary, we have shown a set of fast dyslexic responders with detection and primed detection performance similar to that of normal readers, in addition to which we have identified a set of slow dyslexic responders who display substantially elevated target detection latencies (relative to normal readers) and strong evidence of attentional deployment to a synchronous premask stimulus. We suggest that dyslexics exhibiting this mode of primed-target detection performance may be subject to extended visual persistence. This may, as has been previously suggested, result from impairment in the functioning of neurons in the magnocellular pathways. However, the precise source of this impairment remains unknown and the outcome of this study as it relates to subtyping remains suggestive. Nonetheless, it might be concluded that functional impairments in visual performance might only arise under circumstances in which the visual system is required to respond to rapid variations in stimulus activity, such as coding information across saccades during reading or while performing efficient grouping during the traversal of a crowded stimulus array. It is interesting to consider that, while some dyslexic readers experience impaired visual performance that might relate to impairments in the timing of the neural response to stimulus events, other dynamic processes, such as the synchronisation of neural

activity, may be little affected if given appropriately timed stimulus events.

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## REFERENCES

- Boder, E. (1970). Developmental dyslexia: A new diagnostic approach based on the identification of three subtypes. *Journal of School Health, 40*, 289–290.
- Boder, E. (1973). Developmental dyslexia: A diagnostic approach based on three atypical reading-spelling patterns. *Developmental Medicine and Child Neurology, 15*, 663–687.
- Breitmeyer, B. G. (1980). Unmasking visual masking: A look at the “why” behind the veil of the “how.” *Psychological Review, 87*, 52–69.
- Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychological Review, 83*, 1–36.
- Breuer, H., & Weuffen, M. (1995). *Lernschwierigkeiten im Schulanfang* [Learning difficulties at the beginning of schooling]. Weinheim/Basel, Switzerland: Beltz Verlag.
- Burr, D. C., Morrone, M. C., & Ross, J. (1994). Selective suppression of the magnocellular visual pathway during saccadic eye movements. *Nature, 371*, 511–513.
- Chase, C., & Stein, J. (2003). Visual magnocellular deficits in dyslexia. *Brain, 126*, E2.
- Demb, J. B., Boynton, G. M., & Heeger, D. J. (1997). Brain activity in visual cortex predicts individual differences in reading performance. *Proceedings of the National Academy of Sciences: USA, 94*, 13363–13366.
- Demb, J. B., Boynton, G. M., & Heeger, D. J. (1998). Functional magnetic resonance imaging of early visual pathway in dyslexia. *The Journal of Neuroscience, 18*, 6939–6951.
- DiLollo, V., Hanson, D., & McIntyre, J. S. (1983). Initial stages of visual information processing in dyslexia. *Journal of Experimental Psychology: Human Perception and Performance, 9*, 923–935.
- Eden, G. F., Van Meter, 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.
- Elliott, M. A., Becker, C., Boucart, M., & Müller, H. J. (2000). Enhanced GABA<sub>A</sub> inhibition enhances synchrony coding in human perception. *Neuroreport*, *11*, 3403–3407.
- Elliott, M. A., & Müller, H. J. (1998). Synchronous information presented in 40 Hz flicker enhances visual feature binding. *Psychological Science*, *9*, 277–283.
- Elliott, M. A., & Müller, H. J. (2000). Evidence for a 40-Hz oscillatory short-term visual memory revealed by human reaction time measurements. *Journal of Experimental Psychology: Learning, Memory and Cognition*, *26*, 703–718.
- Elliott, M. A., & Müller, H. J. (2001). Effects of stimulus synchrony on mechanisms of perceptual organization. *Visual Cognition*, *8*, 655–677.
- Farmer, M. E., & Klein, R. M. (1995). The evidence for a temporal processing deficit linked to dyslexia: A review. *Psychonomic Bulletin and Review*, *2*, 460–493.
- Friederici, A. D., & Lachmann, T. (2002). From language to reading and reading disability: Cognitive functions and their neuronal basis. In E. Witruk, A. D. Friederici, & T. Lachmann (Eds.), *Basic functions of language, reading and reading disability* (pp. 9–21). Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Greatrex, J. C., & Drasdo, N. (1995). The magnocellular deficit hypothesis in dyslexia: A review of reported evidence. *Ophthalmic and Physiological Optics*, *15*, 501–506.
- Habib, M. (2000). The neurological basis of developmental dyslexia. *Brain*, *123*, 2373–2399.
- Hari, R., & Renvall, H. (2001). Impaired processing of rapid stimulus sequences in dyslexia. *Trends in Cognitive Sciences*, *5*, 525–532.
- Hayduk, S., Bruck, M., & Cavanagh, P. (1996). Low-level visual processing skills of adults and children with dyslexia. *Cognitive Neuropsychology*, *13*, 975–1015.
- Heller, K.-A., Kratzmeier, H., & Lengfelder, A. (1998). *Ein Handbuch mit deutschen Normen zu den Standard Progressive Matrices von J. C. Raven* [Manual for the Standard Progressive Matrices by J. C. Raven with German standards]. Göttingen, Germany: Beltz.
- Hulme, C. (1988). The implausibility of low-level visual deficits as a cause of children's reading difficulties. *Cognitive Neuropsychology*, *5*, 369–374.
- Ivanoff, J., & Klein, R. M. (2003). Orienting of attention without awareness is affected by measurement-induced attentional control settings. *Journal of Vision*, *3*(1), 32–40, from <http://journalofvision.org/3/1/4/>, doi:10.1167/3.1.4.
- Johannes, S., Kussmaul, C. L., Munte, T. F., & Mangun, G. R. (1996). Developmental dyslexia: Passive visual stimulation provides no evidence for a magnocellular processing defect. *Neuropsychologia*, *34*, 1123–1127.
- Keen, A. G., & Lovegrove, W. J. (2000). Transient deficit hypothesis and dyslexia: Examination of whole-parts relationship, retinal sensitivity, and spatial and temporal frequencies. *Vision Research*, *40*, 705–715.
- Klein, R. M. (2002). Observations on the temporal correlates of reading failure. *Reading and Writing*, *15*, 207–232.
- Kujala, T. (2002). The mismatch negativity as an index of auditory dysfunction in dyslexia. In E. Witruk, A. D. Friederici, & T. Lachmann (Eds.), *Basic functions of language, reading and reading disability* (pp. 359–368). Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Kossakowski, A. (1961). *Wie überwinden wir Schwierigkeiten beim Lesen- und Schreibenlernen, insbesondere bei Lese- Rechtschreibschwäche?* [How do we overcome difficulties in learning to read and spell, in particular in the case of dyslexia?]. Berlin, Germany: Verlag Volk & Wissen.
- Lachmann, T. (2002). Reading disability as a deficit in functional coordination. In E. Witruk, A. D. Friederici, & T. Lachmann (Eds.), *Basic functions of language, reading and reading disability* (pp. 165–198). Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Landerl, K., Wimmer, H., & Moser, E. (1997). *Salzburger Lese- und Rechtschreibtest* [in German]. Göttingen, Germany: Hans Huber.
- Livingstone, M. S., Rosen, G. D., Drislane, F. W., & Galaburda, A. M. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proceedings of the National Academy of Sciences of the USA*, *88*, 7943–7947.
- Lovegrove, W. J., Bowling, A., Badcock, D., & Blackwood, M. (1980a). Specific reading disability: Differences in contrast sensitivity as a function of spatial frequency. *Science*, *210*, 439–440.
- Lovegrove, W. J., Heddle, M., & Slaghuis, W. (1980b). Reading disability: Spatial frequency specific deficits in visual information store. *Neuropsychologia*, *18*, 111–115.
- Martin, F., & Lovegrove, W. J. (1987). Flicker contrast sensitivity in normal and specifically disabled readers. *Perception*, *16*, 215–221.



- McCormick, P. A. (1997). Orienting attention without awareness. *Journal of Experimental Psychology: Human Perception and Performance*, *23*, 168–180.
- Posner, M. I., & Petersen, S. E. (1990). The attentional system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42.
- Singer, W. (1999). Neuronal synchrony: A versatile code for the definition of relations? *Neuron*, *24*, 49–65.
- Skottun, B. C. (2000). The magnocellular deficit theory of dyslexia: The evidence from contrast sensitivity. *Vision Research*, *40*, 111–127.
- Slaghuis, W. L., & Ryan, J. F. (1999). Spatio-temporal contrast sensitivity, coherent motion and visible persistence in developmental dyslexia. *Vision Research*, *39*, 651–668.
- Snowling, M. (2000). *Dyslexia*. Oxford: Blackwell.
- Snowling, M. (2001). From language to reading and dyslexia. *Dyslexia*, *7*, 37–46.
- Spinelli, D., Angelelli, P., De Luca, M., Di Pace, E., Judica, A., & Zoccolotti, P. (1997). Developmental surface dyslexia is not associated with deficits in the transient visual system. *NeuroReport*, *8*, 1807–1812.
- Stein, J., Talcott, J., & Walsh, V. (2000). Controversy about the visual magnocellular deficit in developmental dyslexics. *Trends in Cognitive Sciences*, *4*, 209–211.
- Stein, J., & Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. *Trends in Neurosciences*, *20*, 147–152.
- Stuart, G. W., McAnally, K. I., & Castles, A. (2001). Can contrast sensitivity functions in dyslexia be explained by inattention rather than a magnocellular deficit? *Vision Research*, *41*, 3205–3211.
- Studdert-Kennedy, M., & Mody, M. (1995). Auditory temporal perception deficits in the reading impaired: A critical review of the evidence. *Psychonomic Bulletin and Review*, *2*, 508–514.
- Talcott, J. B., Hansen, P. C., Assoku, E. L., & Stein, J. F. (2000). Visual motion sensitivity in dyslexia: Evidence for temporal and energy integration deficits. *Neuropsychologia*, *38*, 935–943.
- Tallal, P. (1984). Temporal or phonetic processing deficit in dyslexia? That is the question. *Applied Psycholinguistics*, *5*, 167–169.
- Vellutino, F. R. (1987). Dyslexia. *Scientific American*, *256*, 34–41.
- Weigt, R. (1980). *Zur Auswahl von Schülern für LRS-Klassen* [On the selection of students for dyslexia school classes]. Berlin, Germany: Verlag Volk & Wissen.
- Williams, M. J., Stuart, G. W., Castles, A., & McAnally, K. I. (2003). Contrast sensitivity in subgroups of developmental dyslexia. *Vision Research*, *43*, 467–477.
- World Health Organisation. (1992). *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organisation.

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