Temporal and Spatial Attention in Dyslexia

By Elizabeth Liddle, BA, BA, MA

Thesis submitted to the University of Nottingham

For the degree of Doctor of Philosophy, May 2006

Table of Contents

Table of Figures

Abstract

It was hypothesized that the deficits underlying reading impairment may arise from supra-modal deficits in temporal and spatial attention, disrupting, on the one hand, the ability to segment the temporally ordered phonemes of language and thus the acquisition of decoding skills, and, on the other, the ability to integrate spatially and temporally ordered orthographic information acquired from the fluent visual scanning of written text. Temporal and spatial attentional deficits in dyslexia were investigated using a lateralized visual temporal order judgment (TOJ) paradigm that allowed both sensitivity to temporal order and spatial attentional bias to be measured. Dyslexic and non-dyslexic participants were required to report the temporal order of two simple visual stimuli presented in either the same or different lateral hemifields. Findings indicated that dyslexic participants showed markedly impaired sensitivity to temporal order, and that the degree of impairment was correlated with the severity of their dyslexia. Furthermore, the findings suggested that at least three partially dissociated deficits may underlie both impaired TOJ task performance and reading disorder. One is a deficit associated with difficulty in reporting the temporal order of two visual stimuli, particularly when the first is presented in right hemifield; with slow word recognition and non-word reading; and with deficits in spelling and phonological skill. This constellation of deficits was interpreted as reflecting deficits in networks in left cerebral hemisphere implicated in phoneme-grapheme mapping and visual orienting. The second is a deficit that is associated with a rightward attentional bias; with inaccurate non-word reading that is worse than predicted by phonological skill or by word recognition; and with poor sustained attention. This constellation of impairments was interpreted as evidence of a deficit in right-lateralised networks implicated in the modulation of arousal, and possibly reflecting a "developmental left-neglect" syndrome. A third deficit was associated with impaired temporal order sensitivity, regardless of hemifield presentation; with symptoms of Attentional Deficit and Hyperactivity Disorder (ADHD); and with increased interference from distractor stimuli. This constellation of deficits suggests that the impaired network is implicated in executive control of attention, including conflict resolution and working memory. The results of the investigation as a whole suggests that the reading impairments of dyslexia may arise from attentional deficits that have with substantial overlap with those of ADHD, and include deficits in attentional networks implicated in orienting attention to temporally presented stimuli.

Acknowledgements

There are many, many people who contributed to this investigation, both directly and indirectly. Among them, first of all, I would like to thank Elaine Baron, Anna Plodowski, Jessica Jackson, Jennifer Cipko, Tanya Patel, Ruth Bryan and Yu Ju Chou, who were all involved in the collection of data. Secondly, I would like to thank Stephen and Georgina Jackson and Chris Rorden, not only for their support and encouragement throughout, but for giving me the opportunity and the confidence to embark on the investigation in the first place. Thirdly, I would like to thank my family, including my husband, Peter Liddle, for all he was able to teach me from the depth of his knowledge and experience in neuroscience research, my son, Patrick Liddle, for his patience, and my father, Geoffrey Poole, both for his encouragement and for his help in keeping the household running during school holidays. And lastly, I would like to thank all those who participated in the studies, not only for their willingness to undertake many lengthy tasks, but also for the wealth of insight into the nature of dyslexia that they were able to bring and to share with me.

Ethical Approval

Experiment 1a was conducted with ethics approval from the University of Wales, Bangor. The remainder of the experiments were carried out with ethics approval from ethics committee of the School of Psychology, University of Nottingham. All participants gave informed consent to take part, and where participants were aged below 16, consent was also obtained from their parent or guardian.

Dedication

I dedicate this thesis to the memory of my mother, Joyce Poole (1925-1998) who gave up her own research into the fluid replacement requirements of severely burned children in order to have me.

1. READING & READING DISORDER

1.1 Introduction

When a hunter throws a spear at a grazing antelope, his attention is focused on his target. By this we mean that for the hunter, the most salient point of space, as he prepares his attack, is between the eyes of the animal he plans to kill. However, his attention is also focused on a moment of time – the moment at which he anticipates that the spear will impact his quarry. If he misjudges the moment, his spear may miss. Similarly, a drummer selects, from many options, the precise location in space and moment in time at which each beat will fall. In the mind of both hunter and drummer, space and time are continua on which salient locations and moments that are relevant to the current goal are represented. Should an irrelevant moment or location acquire inappropriate salience, the goal will be subverted.

Literacy, unlike hunting, or even drumming, is a recent development in the timescale of human evolution; it seems certain that our brains did not evolve to read. Perhaps the most intriguing question regarding the phenomenon of reading, therefore, is not why some children have difficulty in learning to read, but rather why the majority of children learn to read so easily. In other words, when a child learns to read, which cognitive modules have been co-opted? And this leads us to the converse question: when a child fails to learn to read easily,

which cognitive modules are failing to take their role?

Written language is a remapping of the temporally ordered sounds of the spoken language into spatially ordered visual symbols. Reading thus involves the translation of spatial into temporal order, and writing involves the reverse process; thus, envisaging the way a word is spelled, and imagining the sound a written word makes, involve opposite and complementary conversion processes. To be done successfully, the sounds of spoken language must be parsed into components that are represented by their appropriate written symbols; in alphabetic languages these components are phonemes. The ability to segment language phonologically is thus required for successful reading; so too, however, is the ability to parse written symbols into a spatially coherent order that can be reconstituted into the temporally ordered language it represents.

The ability to focus attention both on locations in space and moments in time is therefore as essential to reading and writing as it is to the skills of hunting or drumming. By the same token, any failure in the ability to thus focus attention is likely to compromise the acquisition of literacy.

1.2 Models of developmental dyslexia

Reading Disorder, or dyslexia, is diagnosed when reading (and spelling) is impaired despite otherwise normal intellectual function and the opportunity to learn, or, as specified in the fourth edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM IV): “is substantially

below that expected given the person's chronological age, measured intelligence, and age-appropriate education" (American Psychiatric Association, 1994).

1.2.1 Phonology

Current evidence suggests that the proximal cause of most reading disorders, at least in English speakers, is a core deficit in phonological processing (Metsala et al., 1998; Snowling et al., 1996; Stanovich and Siegel, 1994) that disrupts awareness of the temporal order of phonemes in a word, and thus with the phoneme-grapheme mapping that is the basis of reading in alphabetic writing systems. Phonological awareness in children starting school is a good predictor of their subsequent reading progress (Caravolas et al., 2001; Marx et al., 2001; McBride-Chang et al., 1997) and programs designed to improve phonological awareness remain the best attested interventions for improving reading skills (Torgesen, 2002), although there is evidence that causal relations between phonological skill and reading-related knowledge may be bi-directional (Wagner et al., 1994).

However, some researchers have postulated subtypes of dyslexia, of which a "phonological" or "dysphonetic" subtype is only one. This subtype is postulated to be characterized by a deficit in phonological skill that preferentially impairs non-word or unfamiliar word reading, which demands sub-lexical phonological assembly, over the reading of real words, which can be read by lexical recognition (Castles and Coltheart, 1993). In contrast, an opposite "dyseidetic" or "surface" subtype is postulated in which the chief difficulty is with word

recognition, rather than with sub-lexical decoding, as well as a mixed "dysphoneidetic" subtype, manifesting both types of difficulty (Castles and Coltheart, 1993; Ridder et al., 1997; Stanovich et al., 1997).

Wolf and Bowers (1999) proposed a subtype model that, they argue, may have parallels with "surface" and "phonological" subtypes. They propose two dissociated deficits as contributing to reading disorder, one evidenced by deficits in phonological tasks, and the other by speed deficits in tasks requiring rapid automatized serial naming, such as those devised by Denckla and Rudel (1976), and found to be associated with dyslexia. Wolf and Bowers found, through their own work and a review of the existing literature, evidence to suggest that while readers who displayed deficits on either phonological measures or on naming tasks were likely to show impaired reading, those who displayed a "double deficit" were more likely to be amongst the most severely impaired. They further proposed that while phonological deficits may impair decoding skills, and thus the acquisition of fast and accurate reading, a naming deficit may reflect deficits in a different pathway or pathways, and specifically hamper the acquisition of fast word recognition and fluency.

An alternative to the "subtype" model is a conception of variance in reading disorder that views reading impairments in terms of dimensions. The apparent existence of "mixed" or "double deficit" subtypes might argue in favour of such a conception. Murphy and Pollatsek (1994) studied 65 dyslexic children, together with 65 reading-aged matched controls and 17 age-matched good readers, and

found no evidence for discrete subtypes, but rather evidence that "phonological" and "surface" patterns of deficits represent continua along separate dimensions. It may therefore be the case that a phonological deficit affecting sub-lexical phonological assembly is only one of a number of proximal causes of reading impairment; difficulties in word recognition may arise from a deficit in a different cognitive module to that implicated in non-word reading impairment. Moreover, these deficits may or may not be aetiologically related.

1.2.2 Vision

A phonological deficit might be viewed as a disruption to the sequential orienting of attention to each of the temporally ordered phonemic components of spoken language. However, in written language, this temporal sequence of auditory stimuli is represented by a spatial sequence of visual stimuli, processed initially by the visual system; successful reading therefore requires that visual attention be oriented to the text in a spatio-temporal sequence that can be reconstituted to represent the temporal sequence of the portrayed speech.

In a review of the literature on eye movements in reading, Rayner (1998) reports that mean saccade lengths during silent reading are 7-9 letter spaces; that, typically, most words are fixated, although some, particularly function words, tend to be skipped; and that about 10%-15% of saccades are regressive. Thus control of overt temporal-spatial attention during reading is complex, and likely to be critical to success (May et al., 1988; Rayner, 1998). Interestingly, the visual aspect of reading was emphasized in early accounts of reading disorder:

Pringle and Hinshelwood's original term for the condition was "word blindness", thus reflecting a view of reading disability that was essentially visual, as did Orton's later term "strephosymbolia" or "twisted symbols" (Torgesen, 1998), coined to refer to the visual disruptions reported by many dyslexic readers (Davis and Braun, 1997; Stein and Walsh, 1997). Indeed, Wolf and Bowers (1999) suggest that a possible deficit underlying their observed link between rapid automatized naming deficits and reading impairment may be in visual pathways implicated in orthographic recognition.

1.2.3 Supra-modal models

Given the debate about whether or not visual as well as phonological deficits may be associated with reading disorder, theories that account for both visual and auditory impairments in dyslexia are attractive. One such is that the core disorder in dyslexia may be a supra-modal deficit in networks implicated in the temporal ordering of rapidly presented stimuli (Farmer and Klein, 1995; Klein and Farmer, 1995; Tallal, 1980; Tallal, 1984).

In the auditory domain, Tallal (1980) found that dyslexic children required longer Inter Stimulus Intervals (ISIs) than control participants in order to correctly report the temporal order of two tones presented in rapid succession (Tallal, 1980), while Rey and colleagues (Rey et al., 2002) found the performance of dyslexic participants was poorer than that of controls when they were asked to report the temporal order of two phonemes ("p" and "s") within a

cluster, although their performance improved to meet that of the control participants when the speech sounds were artificially slowed. In contrast, manipulating the phonological complexity of the cluster did not result in improved performance. The authors interpreted their finding to support a temporal processing, rather than a linguistic deficit, as the source of the phonological disorder in dyslexia.

Goswami and colleagues (2002) proposed and tested a more specific hypothesis regarding deficits in the processing of phonological information. She postulated that the key deficit in dyslexia may be sensitivity to amplitude modulation of vowel sounds, resulting in a reduced awareness of the perceptual centre ("P-centre") of syllables, and thus of the demarcation between onset and rime. She found that sensitivity to amplitude modulation of a continuous tone was significantly attenuated in dyslexic children as compared with both chronological and reading age matched controls. Moreover, she assessed ability to discriminate temporal order on two different auditory tasks, including the Rapid Frequency Discrimination (RFD) task devised by Tallal (1973) as a way of tapping deficits in rapid frequency discrimination postulated to underlie the phonological deficit in dyslexia (Tallal, 1980). The RFD task accounted for significant variance in reading related measures, including reading, spelling and phonological short-term memory (after controlling for age and IQ); however the effect sizes for P-centre sensitivity were substantially greater, and while RFD performance accounted for only 4% of additional variance in reading over and above P-centre discrimination scores, P-centre discrimination scores accounted for 19% of

variance over and above RFD scores.

One specific supra-modal theory is that the underlying deficit may lie in the magnocellular pathways specialized for the transmission of rapid sensory information (Greatrex and Drasdo, 1995; Keen and Lovegrove, 2000; Livingstone et al., 1991; Lovegrove, 1996; Slaghuis and Ryan, 1999; Stein and Walsh, 1997). Dyslexic participants have been found to have lower fusion or higher gap detection thresholds with both auditory and visual stimuli (Farmer and Klein, 1995), consistent with the theory that pathways specialized for processing rapid temporal sensory information may be impaired in dyslexia. Such a deficit has been postulated to affect magnocellular pathways in any sensory domain (Stein and Walsh, 1997). If this is the case, the question arises as to whether the postulated magnocellular deficit affects reading solely via the auditory modality, by disrupting the development of phonemic awareness and thus the acquisition of the phonological decoding skills that facilitate successful reading; if they do, observed visual anomalies may be epiphenomena associated with reading disorder merely aetiologically. Alternatively, magnocellular deficits in the visual pathway may directly impair reading, perhaps only in some dyslexic subtypes, by disrupting recognition of letter and word forms, and the fluent shifting of visual attention across the written text.

However the answer to this question remains unclear. Ridder and colleagues (Ridder et al., 1997) found, perhaps counter-intuitively, that dysphonetic and dysphoneidetic participants showed evidence of a visual magnocellular deficit

but dyseidetics did not; however, a later study (Ridder et al., 2001) suggested that evidence of a magnocellular deficit did not distinguish between subtypes, and evidence from other researchers remains inconclusive (Cestnick and Coltheart, 1999; Davis et al., 2001a; Slaghuis and Ryan, 1999). If anything the evidence seems to suggest that impairments on tasks designed to tap magnocellular deficits is more strongly predictive of deficits in non-word reading than in word recognition (Cestnick and Coltheart, 1999). This is the opposite of what would seem to be predicted by the hypotheses advanced by Wolf and Bowers (1999) to account for the association between rapid automatized naming deficits and reading impairment, which suggest that deficits in rapid information processing should lead to word recognition, rather than non-word reading, deficits. If non-word reading impairment is regarded as evidence of impaired phonological processing, this may suggest that an apparent magnocellular deficit in the visual modality may be a marker for supra-modal deficits that also affect phonology. An alternative interpretation may be that a visual magnocellular deficit preferentially impairs non-word reading because of its dependence on sub-lexical processes, and thus on good letter-position encoding. In support of this latter interpretation, Cornelissen and colleagues found that performance on tasks designed to tap a magnocellular deficit accounted for separate variance in letter-position errors from that accounted for by phonological skill (Cornelissen et al., 1998b)

A second model that might account for supra-modal impairments is that proposed by Nicolson and colleagues (Nicolson et al., 2001), namely, that a

developmental cerebellar deficit may account for the range of deficits observed in dyslexic participants. The cerebellum is implicated in predicting the sensory consequences of action by comparing the "forward model" of an action with sensory feedback from the movement (Blakemore et al., 2001). A cerebellar deficit might therefore be expected to compromise the learning of automatized skills, whereby a temporally ordered sequence of actions must be integrated into fluency. These skills are postulated to include articulatory skill, which in turn may give rise to a phonological deficit that impacts directly on reading, but also to impairment in any skill demanding fluency, including skills underlying word recognition, spelling, and handwriting. The model also predicts that dyslexia will be associated with impaired learning of motor skills unrelated to literacy, including balance, a prediction borne out by a number of behavioural studies (Fawcett et al., 2001; Nicolson and Fawcett, 2000; Nicolson et al., 1999; Stoodley et al., 2005). Some histological and imaging evidence also supports the theory of cerebellar dysfunction in dyslexia (Eckert et al., 2003; Finch et al., 2002; Rae et al., 2002). Interestingly, Stoodley and colleagues note that the magnocellular deficit and the cerebellar deficit models may be causally linked, in that a magnocellular dysfunction might be associated with cerebellar dysfunction by way of disruption to cerebro-cerebellar loops implicated in the auditory and visual sensory systems, raising the possibility that temporal processing deficits in dyslexic may arise from disruption to complex networks implicated in perception and action.

1.2.4 Visual attention

Visual magnocellular sensory pathways terminate in parietal cortex, forming a fast "dorsal" data stream tuned to high temporal frequencies and low spatial frequencies, sensitive to low contrast stimuli, and implicated in exogenous attention; in contrast, a slower, parvocellular "ventral" data stream tuned to lower temporal frequencies but higher spatial frequencies, is specialized for object recognition, and terminates in temporal cortex (Livingstone and Hubel, 1987; Mishkin et al., 1983). However, there remains debate as to how and when the visual system selects objects for detailed processing and thence recognition.

Vidyasagar (1999) presents a neuronal model of visual attention in which the dorsal, magnocellular pathway may select areas of interest for preferential processing by the ventral, parvocellular system. He therefore predicts that dorsal stream deficits may result not only in impairments in tasks directly reflecting dorsal stream function, such as the control of sequential attention during reading, but also, paradoxically, in impairment on tasks tapping ventral stream function in contexts where the density of visual stimuli is high, as in written text. If so, letter and word recognition will be dependent on efficient dorsal stream function, even if the features required for recognition are processed ventrally. Vidyasagar thus proposes, that the dorsal stream is "necessary for the smooth flow of attentional focus that helps in the identification of individual letters and words" (Vigyasagar, 1999, page 71). He also proposes that while magnocellular stream deficits in the auditory system may directly account for poor phonemic discrimination in dyslexia, an alternative causal pathway might be via a primary deficit in the visual magnocellular pathway, leading to disruption of the

development of supramodal spatial maps in parietal cortex created from multimodal inputs, including vision and audition.

This suggestion is intriguing, as it suggests that disruption to the development of such supramodal maps, whether or not the disruption arises from deficits in the magnocellular pathway, may be key to both the visual and the phonological deficits that appear to be associated with developmental dyslexia.

1.2.5 Neurological evidence from brain imaging

Booth and colleagues (Booth et al., 2003) used functional Magnetic Resonance Imaging (fMRI) to investigate the relationship between brain activation and performance on four tasks, two intramodal (reporting whether visual words were spelled the same or whether two auditory words rhymed) and two cross-modal (reporting whether auditory words were spelled the same or whether visual words rhymed). On intramodal tasks, better performance was correlated with activation in brain areas corresponding to that modality (superior temporal gyrus for the auditory task and fusiform gyrus for the visual task). On cross-modal tasks, better performance was correlated with increased activation in the area corresponding to the target modality, but also in posterior areas of association cortex in the inferior parietal lobe, suggesting that these areas are implicated in mapping the spoken sounds of language on to the spatial representations of written text.

This finding suggests that disrupted functional connectivity between parietal regions of association cortex and other brain regions involved in the processing of visual and language stimuli, are associated with reading deficits. Indeed, functional brain imaging studies of reading disorder have revealed disruption to networks that include posterior multimodal association cortex, particularly in left hemisphere (Horwitz et al., 1998; Paulesu et al., 1996; Pugh et al., 2000a; Pugh et al., 2001; Pugh et al., 2000b; Shaywitz et al., 1998; Temple, 2002)

Paulesu and colleagues (Paulesu et al., 1996), using Positron Emission Tomography (PET), found that during a rhyming task and a short term memory task using visually presented letters, compensated adult dyslexic participants who exhibited a phonological deficit did not, unlike control participants, activate anterior and posterior left hemisphere language areas in concert, and furthermore failed to show activity in left insula. They proposed that dyslexia may arise from weak connectivity between anterior and posterior areas in left hemisphere involved in language processing. In men with persistent developmental dyslexia, Horwitz and colleagues (Horwitz et al., 1998), also using PET, found disrupted connectivity between the angular gyrus and occipital and temporal lobe regions during a single reading word reading task as compared with non-dyslexic control participants, again suggesting functional disconnection of posterior regions of association cortex as a key to reading disorder.

Using functional fMRI, and a series of tasks that tapped orthographic and phonological processing, Pugh and colleagues (Pugh et al., 2000b) found that

non-dyslexic adult readers showed bilateral patterns of functional connectivity between the angular gyrus and occipital and superior temporal cortex during a non-word rhyming task. In contrast, they found evidence of dysfunction around the left angular gyrus in adult dyslexic readers, and, like Horwitz and colleagues, found evidence of disrupted left hemisphere connectivity between the angular gyrus and occipital and temporal areas on tasks requiring phonological assembly, including non-word rhyming. In the dyslexic participants, right hemisphere connectivity during the non-word rhyming task was, if anything, stronger than in non-dyslexic participants. On the basis of their own and other imaging studies, Pugh and colleagues (Pugh et al., 2001) proposed that two left hemisphere systems support skilled reading; a dorsal (temporo-parietal) network implicated in the integration of phonology with orthography, and a ventral (occipito-temporal) network implicated in the word recognition; that the latter system develops later and underlies fluency, but that reading impairment results from disruption to the former.

Evidence of disrupted neurological function in dyslexic participants does not, however, solely implicate left hemisphere networks. Using PET, Rumsey and colleagues (Rumsey et al., 1997) found bilateral patterns of disrupted functional connectivity to be associated with reading disorder in dyslexic adult men, and several studies have found evidence of a “left-neglect” syndrome in impaired readers (Facoetti et al., 2001; Hari et al., 2001; Jaskowski and Rusiak, 2005; Sireteanu et al., 2005), suggesting that deficits in homologous right-hemisphere temporo-parietal networks involved in spatial attention may also be associated

with reading impairment, and possibly implicating impairments in spatial attention in the reading difficulties encountered by some dyslexic readers.

The evidence regarding the neurological underpinnings of reading impairment thus suggests that left-hemisphere networks implicated in the cross-modal mapping of spoken and written language components may be impaired in dyslexia, and that these may include association cortex around the temporo-parietal junction and parietal areas including the angular and supramarginal gyri. However, the evidence also suggests that disruption to possibly homologous right-hemisphere networks, including those implicated in the shifting of spatial attention, may be associated with reading impairment.

1.2.6 Temporo-spatial deficits

In the auditory domain, perception of the temporal order in which an acoustic wave front is received at each ear is the means by which the spatial location of the stimulus origin is inferred. In experimental paradigms, Inter-Stimulus Intervals (ISIs) between acoustic stimuli presented to each ear can therefore be manipulated in order to give the illusion of a single stimulus originating a particular location. At an ISI of zero, the origin of the stimulus will be perceived as originating centrally; if the left precedes the right, the stimulus will be perceived as originating to the left of midline, and if the right precedes the left, the stimulus will be perceived as originating to the right of midline. The extent to which participants perceive the stimulus as coming from a particular location will therefore be a measure of the extent to which they can perceive the temporal

order of the wavefronts. If dyslexic participants are relatively insensitive to temporal order, their ability to locate the apparent origin of the stimulus may be impaired.

Dougherty and colleagues (Dougherty et al., 1998) investigated the phenomenon of "dichotic pitch" in dyslexic participants. "Dichotic pitch" can be induced by presenting participants with identical copies of full spectrum white noise to each ear, giving the illusion of a centrally located "ball of sound". In the signal sent to one ear, a narrow band of frequencies is then phase shifted, in order to induce the illusion that it originates from a different spatial location. The result is a binaural illusion of a distinct pitch, segregated from the background noise only by its displaced perceived origin. The illusion is absent when either signal is received monoaurally, and is thus dependent on the capacity of the auditory system to detect the temporal order of arrival of the wave-fronts. By manipulating the signal-to-background intensity ratio, Dougherty and colleagues found that dyslexic children were significantly less able to detect dichotic pitch binaurally than non-dyslexic children.

Hari and Kiesila (1996), however, found that dyslexic adults were more rather than less, prone to a different binaural spatial illusion than non-dyslexic adults. Rapid trains of eight clicks were presented to both ears, with an interaural interval of 0.8 ms; for the first four clicks the signal to the left ear preceded the right; for the second four clicks the order was reversed. The effect simulated was thus of four clicks originating from the left of centre followed by four originating

from the right. ISIs were manipulated. At short ISIs, participants reported an illusory "saltation": the perception that the origin of the clicks "jumped" in discrete equidistant steps from left to right. At longer ISIs, the perception was of a larger midline jump, while at the longest ISIs many participants perceived no saltation; they merely perceived four clicks originating from a single location on the left followed by four from a single location on the right. Dyslexic participants required longer ISIs before the saltatory illusion diminished.

The authors suggest that the saltatory illusion arises from backward processing of the first four stimuli, which are interpreted in the light of the perceived location of later stimuli; it would appear that the brain integrates the stimuli over a period of time, and interpolates the origin of the intermediate clicks from the perceived origin of the first and last. If so, it would appear that the dyslexic participants integrate the information over a longer time period than the non-dyslexic participants, and thus perceive the illusion at longer ISIs.

The finding of Hari and colleagues suggests that the finding of Dougherty and colleagues may not reflect an inability to localize a sound source from the tiny binaural temporal differences in the arrival of a wave front, as apparently the dyslexic participants in the Hari experiment were able to do, but from a deficit affecting the integration of information over a longer time period and which is also implicated in the accurate localization of a sound source.

A number of researchers have investigated temporal order judgement in the visual domain, using spatial location as a variable. Brannan and Williams (1988)

presented word and non-word stimuli to right and left visual fields respectively and asked normally reading children and poor readers to point to the location of the first stimulus to appear, and found that performance was correlated with reading level across groups. May and colleagues (May et al., 1988) presented pairs of words ("box" and "fox"), one in each of two locations: either above and below fixation or to left and right of fixation. Participants were children who were good readers, children who were poor readers, and a group of adults, and were asked either to report the position of the word that appeared first, or to give the word. Threshold Stimulus Onset Asynchronies (SOAs) were obtained for each group for each condition. Adults had the lowest thresholds and poor readers the highest, the good readers having thresholds of intermediate value; poor readers, unlike good readers, had a higher threshold for reporting the word than for reporting the position.

Hari and colleagues (2001) found that dyslexic participants required longer Stimulus Onset Asynchronies (SOAs) than control participants in order to correctly report the temporal order of two abstract visual stimuli (rectangles) presented to left and right visual fields respectively. They also found a significantly greater tendency for the dyslexic participants to report the right stimulus as occurring before the left than in non-dyslexic participants, a phenomenon they termed "left mini-neglect", and interpreted as supporting the hypothesis of a right parietal deficit in dyslexia, possibly implicated in the shifting of attention, and underlying the overall poor performance of dyslexic

participants on the task.

It is possible, therefore, that at least some of the evidence adduced to support a temporal processing deficit hypothesis in dyslexic may reflect deficits in spatial attention. It is also of note that some of the apparent temporal processing deficits observed involve ISIs longer than those that would support the hypothesis of a specific impairment in processing rapid sensory, such as that predicted from a magnocellular deficit. Amitay and colleagues have suggested that the observed deficits characterised as "magnocellular" deficits, may more broadly characterized as parietal deficits, and found that while dyslexic participants were indeed impaired on tasks designed to tap magnocellular function, they were also impaired on tasks not so designed.

Of particular interest is Ben-Yehudah and colleagues' finding (Ben-Yehudah et al., 2001) that temporal contrast sensitivity, assumed by many researchers to reflect the efficiency of the visual magnocellular pathway, was apparently impaired in dyslexic participants only when the stimuli were presented temporally, and participants were asked to report whether the target stimulus had been presented first. When the stimuli were presented spatially, and participants were asked to report the spatial location of the target, no group differences were found. The authors hypothesised that if the difficulty encountered by dyslexic participants in the temporal presentation was due to demands on processing time, increasing the ISI should result in improved performance; however, if it was due to demands on visual perceptual memory, then increasing the ISI might hinder

performance. They found that while increasing the ISI improved the performance of control participants, it had no effect on that of dyslexic participants, suggesting that visual perceptual memory may have contributed to the poor performance of the dyslexic participants on the TOJ version of the task.

1.2.7 Explanatory levels of models

The hypotheses advanced to account for reading impairment thus vary in the explanatory levels at which they address the disorder: cognitive, neurological, and aetiological. Moreover, within each level, some address proximal, and some more distal causes. Phonological deficits appear to be the strongest proximal predictors of dyslexia (Ramus, 2003), but the phonological deficit hypothesis does not address questions as to the aetiology of the condition; indeed, one of its strengths as a model is that it allows for multiple aetiologies: environmental, developmental, and traumatic. It is essentially a cognitive model: if a child's phonological awareness is impaired, the phonological segmentation skills that allow the learning of the grapheme-phoneme mappings that are the basis of alphabetic orthographies are likely to be compromised.

The magnocellular and cerebellar deficit hypotheses provide potentially aetiological accounts of the disorder, although Ramus (2004) argues the case that cortical insult may result in magnocellular damage rather than vice versa. Also of interest, from a causal perspective, is the suggestion made by Stoodley and colleagues (Stoodley et al., 2005) of a causal link between that the magnocellular and the cerebellar deficits in dyslexia The cerebellar deficit model, moreover

can potentially account for the phonological deficits that appear to be the proximal cause of dyslexia in many subjects (Nicolson et al., 2001), as well as other deficits affecting literacy, and thus potentially provides a comprehensive cognitive account of reading disorder.

Similarly, the magnocellular deficit model can also account cognitively for phonological impairment if it is extended to include deficits in the auditory domain, or, alternatively, if it is postulated, as by Vidyasagar (1999), that a magnocellular deficit in the visual domain may disrupt the development of the multimodal maps in parietal association cortex that are necessary for the development of phonological skill. The magnocellular deficit model also can account cognitively for visual anomalies that might plausibly be a proximal cause of impaired reading. As the dorsal visual pathway is implicated in the suppression of retinal motion signals during saccadic eye movements (Ross et al., 2001), and thus with the orienting of overt spatial attention, deficits in this pathway may impact directly on reading by leading to visual confusion. Indeed, saccadic anomalies have been frequently found in studies involving dyslexic participants (Biscaldi et al., 2000; Biscaldi et al., 1998; Crawford and Higham, 2001; Eden et al., 1994; MacKeben et al., 2004)

The parietal deficit model provides a more distal account of reading impairment than the phonological deficit model, yet is less aetiologically prescriptive than either the cerebellar model or the magnocellular model. It potentially, however, accounts, at a both cognitive and neurological level, for the visual-spatial deficits

that are associated with dyslexia, and which may be the proximal cause, in some cases, of reading impairment; it is also is consistent with the evidence for apparently left-lateralized as well as for apparently right-lateralized loci for the observed deficits. It also has a certain explanatory parsimony, in that while a number of pathological processes might account for the aetiology of a parietal deficit, it may be that parietal deficits are the common denominator in a large number of cases of reading impairment.

There is thus evidence for auditory, visual, cross-modal and supramodal deficits in dyslexia that affect cognitive processing in both the spatial and temporal domains. Variance in phonological skill would seem to account for substantial variance in literacy, at least amongst English speaking children. However there is also good evidence to suggest that deficits affecting other modalities, including vision, may underlie the pathology of the condition referred to as developmental dyslexia, and that neurologically, these may arise from disrupted function in magnocellular pathways, and/or from disrupted connectivity between parietal areas of association cortex and other brain areas, including areas in temporal cortex implicated in language; in occipital cortex implicated in visual processing; and the cerebellum, implicated in the acquisition of fluency.

1.2.8 A working model

The literature reviewed above suggests that a viable working model of dyslexia, at the cognitive level, may be a conception of dyslexia as arising from deficits in the *orienting of attention* to both events and locations, and that these directly

impact on reading by disrupting the cross-modal representation of text in time and space. In such a model, dyslexia can be considered in terms of dimensions, rather than subtypes, of disorder. Different aspects of reading may be differentially impaired, depending on which attentional modules are affected, regardless of the aetiological process implicated. Indeed, given that dyslexia is a developmental disorder, it is likely that aetiological processes interact during development, and a given primary pathological process may lead to different profiles of impairment depending on developmental, genetic and environmental factors. Furthermore, reading impairment may arise from pathological processes that also result in epiphenomenal markers that may reflect their underlying neurology and aetiology, giving rise to co-morbid diagnoses such as Attention Deficit and Hyperactivity Disorder (ADHD) and dyspraxia. In short, cognitive deficits affecting the temporo-spatial orienting of attention may account for the actual difficulties encountered by dyslexic readers, while patterns of association between such deficits may offer clues as to the likely developmental pathologies that may underlie them.

This model of dyslexia guided the investigation reported in this thesis, which sought to determine the contribution of temporal and/or spatial attentional deficits to variance in severity and type of reading impairment in dyslexic participants.

1.3 The investigation

The core of the investigation was a lateralized visual Temporal Order Judgment

(TOJ) task, in which participants with various degrees of reading impairment as well as non-impaired readers were asked to report which of two visual stimuli, consisting of low-spatial frequency abstract shapes, and presented at different locations in peripheral vision, had appeared first. The Stimulus Onset Asynchrony (SOA) of the two stimuli was randomly varied, in order to determine the temporal interval required by each participant to achieve accurate performance. The task thus potentially tapped deficits in both temporal and spatial attention, and, because the stimuli were lateralized, allowed for the detection of any lateral attentional bias from which the lateralization of any observed deficit might be inferred.

However, given the substantial overlap between developmental dyslexia and ADHD (Kaplan et al., 2001), as the investigation progressed, it became important to distinguish between poor TOJ task performance associated specifically with reading impairment and that associated with co-morbid ADHD symptoms, particularly in view of the substantial evidence for an association between neglect of left visual hemifield and a diagnosis of Attentional Deficit and Hyperactivity Disorder (ADHD) (Epstein et al., 1997; Manly et al., 2005; Sheppard et al., 1999). The investigation therefore also sought to determine the degree to which temporal-spatial attentional deficits are a) directly associated with reading impairment *per se*, and b) associated with co-morbid attentional deficits that may or may not directly contribute to impaired reading.

1.3.1 Hypotheses

Phonological deficits are typically assessed by asking participants to segment the sounds of a given word or non-word into its constituent phonemes. One hypothesis, therefore, was that that deficits affecting temporal orienting might account for impairment on such a task, by disabling the ability to direct sequential attention to each temporally ordered phoneme; TOJ task performance should therefore be correlated with measures of phonological skill. A second hypothesis was that deficits in orienting to both time and space may contribute to visual confusion while reading, as the perception of a line of text, or even a whole word, requires the integration of information picked up through a spatio-temporal sequence of fixations; TOJ task performance was therefore predicted to correlate with performance on tasks tapping both covert and overt attention, and with measures of word recognition efficiency.

However, other factors may also play a role in determining performance on a TOJ task, for example, executive control of attention, affecting working memory capacity and the ability to produce the correct stimulus response mapping. Dyslexic participants, particularly those who also suffer from ADHD may do badly on the task, not because of low-level attentional deficits, but because of inefficient executive control of the attentional processes required. Measures of working memory, and, in later studies, co-morbid ADHD, were therefore measured and included as predictor variables in the statistical models of TOJ task performance.

Finally, it was also hypothesized that lateral bias on the task would be predictive

of particular dimensions of impairment on measures of reading and attention; as well as measures of overall accuracy on the TOJ task, therefore, measures were made of the degree to which participants showed bias towards left or right visual hemifield, and the degree of bias was also treated as a dependent variable in the analyses.

1.3.2 The investigative program

The investigation consisted of three studies, reported, respectively, in Chapters 2, 3 and 4.

1.3.2.1 Study 1

In the first study, reported in Chapter 2, three experiments (Experiments 1a, 1b and 1c) were conducted. In the first experiment, the performance of adult dyslexic participants on a simple version of the TOJ task was compared with that of non-dyslexic participants; in the second, a within group design was used, in which TOJ task performance by a heterogeneous group of reading impaired adults was investigated; in the third, using the same TOJ task, the participants were a similarly heterogeneous group of reading impaired children.

1.3.2.2 Study 2

The second study, reported in Chapter 3 consisted of two experiments (Experiments 2a and 2b). It was hypothesised that impaired performance of dyslexic participants on the TOJ task may be due to deficits in orienting visual attention to peripheral stimuli. If so, dyslexic participants should also show

atypical performance on a task designed to elicit peri-saccadic "spatial compression", a phenomenon whereby abrupt-onset probe stimuli presented close in time to the onset of a saccadic eye movement are mislocalized in the direction of the saccade target. It is thought that these peri-saccadic mislocalizations may reflect predictive and post-dictive processes implicated in maintaining spatial constancy across saccades (Lappe et al., 2000; Ross et al., 1997; Ross et al., 2001); specifically, it has been postulated that it reflects the shifting of the receptive fields of parietal neurons involved in spatiotopic mapping (Kusunoki and Goldberg, 2003).

In the Experiment 2a, dyslexic and non-dyslexic participants undertook a version of the task in which the direction of the saccade required was predictable. In the second, Experiment 2b, the paradigm was modified in such a way as to make the required saccade direction unpredictable, and thus to eliminate any effects of anticipatory covert attention. This modification also allowed saccade latencies to be measured. Participants also undertook a version of the lateralized TOJ task, so that performance on the two tasks could be correlated. In addition, measures of real word and non-word reading efficiency were made, and symptoms of Attentional Deficit Disorder were assessed using The Brown Attention Deficit Disorder Scales (Brown, 1996), in order to ascertain the extent to which variance on the two tasks accounted for symptoms of Reading and/or Attentional Deficit Disorder.

1.3.2.3 Study 3

In the third and final study, comprising Experiment 3 and reported in Chapter 4, a group of parents having at least one dyslexic child was recruited, giving a sample of participants likely be at risk of dyslexia or related disorders. In addition to a version of the lateralized TOJ task, each participant undertook the Attentional Network Task (Fan et al., 2002), a task designed to measure the efficiency of three postulated attentional networks: a right-lateralized "alerting" network implicated in sustained attention; an "orienting" network implicated in directing attention to new locations; and an "executive" network implicated in resolving conflict (Posner and Petersen, 1990). Again, measures of reading impairment, and ADHD symptoms were obtained for each participant. These were correlated with TOJ performance and with the three derived measures from the ANT task representing, respectively, the efficiencies of the postulated alerting, orienting and executive attentional networks, in order to ascertain the proportion of variance in reading impairment, ADHD symptoms, and TOJ performance attributable to variance in the efficiency of each of these three networks.

1.3.2.4 Participants and sample design

In carrying out an investigation of this kind, a range of approaches can be taken to the design of the participant samples. At its simplest, comparisons can be made between the performance of people with dyslexia and that of people without. However, there are at least three drawbacks with this approach. Firstly, dyslexia as currently defined is a portion of a continuum, not a discrete

condition; a “between groups” study design may therefore artificially dichotomize a continuous variable – reading efficiency. Secondly, dyslexia is very broadly defined, and has a high rate of overlap with a number of other disorders, including Dyspraxia; Attention Deficit and Hyperactivity Disorder, Autistic Spectrum Disorder (ASD); and Specific Language Disorder (SLD) (American Psychiatric Association, 1994; Kaplan et al., 2001; Ramus, 2004). Thirdly, a between-diagnostic groups approach may tend to suppress the emergence of dimensions within disorder that extend into the normal range. Thus while between-groups studies are potentially useful in identifying deficits that may be associated with reading disorder and thus worth further investigation, any between-group differences found may reflect neither proximal causes of reading impairment nor dimensions of the underlying pathology, but simply conditions that are more prevalent in one group than another.

An alternative approach, therefore, is to consider reading efficiency as a continuum, and to try to delineate the factors that account for variance in reading efficiency across the whole spectrum. However, a problem that arises with this latter approach is that of finding a population in which reading efficiency, and factors associated with variance in reading efficiency, are likely to have a normal distribution.

A number of sampling designs were therefore used in the course of the investigation reported here, including between-group as well as within-group studies. In two of studies the reported here, initial pilot experiments

(Experiments 1a and 2a) compared dyslexic with non-dyslexic participants, in order to establish whether there was any overall difference between diagnostic groups on the cognitive task in question. For the remainder of the experiments, three approaches were taken to the issue of treating reading efficiency as continuous rather than dichotomous. The first was to recruit a broad range of reading impaired participants from the sub-clinically impaired to the severe (Experiments 1b and 1c, reported in Chapter 2). A drawback with this approach is that at the severe end of the scale, reading ability may arise from compound causes, while the range at the mild end will be restricted by diagnostic cut-off criteria.

The challenge, therefore, was to recruit a sample in which the range of reading efficiency included the normal range, without over-loading the study with unimpaired participants; in other words to recruit a "dyslexia enriched" sample with a range that included good readers. Thus, a second approach was to recruit from a homogeneous population, such as a student body, and to invite participation from both dyslexic and non-dyslexic students. Provided key variables could be shown to have a normal distribution across the entire sample, logistic regression could be used to determine the extent to which the variables of interest predicted recruitment group membership. This approach was used in Experiment 2b, reported in Chapter 3.

A third approach was to identify a single population in which dyslexia is likely to be more prevalent than in the general population, and recruit from that

population. Because dyslexia has been shown to be heritable (Schulte-Korne, 2001), one candidate population is that consisting of adults who have a dyslexic child. A sample recruited from such a population was anticipated to include both unimpaired and impaired readers but have a mean degree of reading impairment that is greater than that of the general population. This sampling methodology was employed in the final study of the investigation, Experiment 3, reported in Chapter 4.

1.4 Summary

Dyslexia is diagnosed when reading is impaired despite otherwise normal intellectual function and the opportunity to learn. Functional imaging studies indicate that reading impairment is associated with disruption to left-lateralized networks, including association areas around the temporal parietal junction and adjacent inferior parietal lobule, implicated in mapping the spoken sounds of language on to the spatial representations of written language. It has been suggested that the core disorder in dyslexia may be a supra-modal deficit in networks implicated in the temporal ordering of stimuli, whether of the sounds of speech or of the spatially ordered graphemes by which they are represented, and which are perceived via a temporal sequence of visual fixations.

However, some studies indicate bilateral patterns of disruption, and a few studies have found evidence of a left-neglect syndrome in impaired readers, suggesting that disruption to homologous right-hemisphere networks may also be associated with reading impairment, and possibly implicating impaired spatial mapping in

the reading difficulties encountered by dyslexic readers.

The studies undertaken in this thesis investigated temporal order judgement (TOJ) in the visual domain. Temporal order judgement tasks require participants to direct attention to moments in time; moreover, when the stimuli presented are visual stimuli presented at different spatial locations, attention must be directed to both moments in time and locations in space. If dyslexia arises from deficits in temporal and/or spatial attention, it would be predicted that dyslexic participants should find such a task particularly difficult.

However, dyslexia has a high co-morbidity with Attentional Deficit and Hyperactivity Disorder; it was therefore important to distinguish any apparent association between poor TOJ task performance and reading impairment from an association simply with co-morbid ADHD symptoms. The investigation thus aimed to identify the temporal and spatial attentional deficits associated with reading impairment, and then to establish the degree to which these are directly associated with reading impairment per se, and associated with co-morbid attentional deficits that may or may not directly contribute to the reading disorder.

2. LATERALIZED VISUAL TEMPORAL ORDER JUDGMENT IN DYSLEXIA

2.1 Introduction

Difficulties with temporal processing in general (Farmer and Klein, 1995; Habib, 2000; Hari and Renvall, 2001; Van Ingelghem et al., 2001) and with temporal order judgement in particular (De Martino et al., 2001; Hari et al., 2001; Tallal, 1980) have been implicated in reading disorders. Deficits in the processing of temporally presented stimuli have been found in auditory, visual, and tactile modalities (Cacace et al., 2000; Goswami et al., 2002; Grant et al., 1999; Laasonen et al., 2000; Richardson et al., 2004; Tallal, 1980; Witton et al., 1998) as well as in cross modal temporal processing tasks (Laasonen et al., 2002; Rose et al., 1999).

However, it remains unclear whether the temporal processing deficits observed across modalities in dyslexia reflect a supramodal deficit in networks implicated in the mapping of temporally ordered sensory information, or whether they reflect related pathological development of lower level dorsal magnocellular sensory pathways specialized in each modality for the transmission of rapid sensory information. Nor is it clear as to whether temporal processing deficits in non-auditory modalities directly affect reading development, or whether they merely reflect deficits that are markers for, but not causal of, dyslexia (Frith and Frith, 1996; McAnally et al., 2000).

Possible mechanisms by which magnocellular deficits affecting the rapid temporal processing of visual-spatial information might directly interfere with reading include impaired control of saccadic eye movements in dyslexic participants (Crawford and Higham, 2001), impaired binocular stability (Stein et al., 2000), impaired letter-position encoding (Cornelissen et al., 1998a) and impaired search strategies (Cestnick and Coltheart, 1999; Iles, 2000).

However, because reading deficits are typically thought to reflect a core language disorder (Catts, 1996; Stanovich and Siegel, 1994), much investigation into the role of temporal processing deficits in dyslexia has focused on the processes by which the temporally ordered phonemes of auditory language are mapped on to the spatially ordered graphemes that comprise written language. In support of this approach, functional brain imaging studies have consistently found evidence of left hemisphere (LH) differences between dyslexic and non-reading impaired groups, both in terms of activation and functional connectivity (Horwitz et al., 1998; Pugh et al., 2000b; Shaywitz et al., 1998; Temple et al., 2001), suggesting that the primary deficit in dyslexia lies in the hemisphere specialized for language. These studies implicate the temporal, occipital and parietal cortical areas of the left hemisphere with the processing of speech, the recognition of visual patterns, and cross-modal mapping respectively.

Pugh et al. (2001) suggest that in normally developing readers, a LH dorsal (temporal-parietal) network predominates in the early stages of reading; this is implicated in the integration of phonological, orthographic and lexical-semantic

information, and is activated when reading low-frequency words and non words. With increased reading experience, a LH ventral (occipital temporal) network develops; this is activated during the processing of well-learned words, providing fast lexical access to whole word forms, and contributing to fluency.

However, others researchers have suggested that a right-to-left (Bakker, 1994) or a bilateral-to-left (Waldie and Mosley, 2000) hemisphere shift takes place as fluency is acquired, and an imaging study by Turkeltaub and colleagues (Turkeltaub et al., 2003) of children at a range of stages of reading development would appear to support features of this model. Bakker's (1994) "balance model" of dyslexia postulates an aetiology whereby some forms of dyslexia (referred to as "Perceptual" or "P-type", and associated with accurate but slow decoding) may arise from failure to transfer reading from right hemisphere networks involved in perception to more efficient left hemisphere networks involved in lexical access, while other reading disabilities (referred to as "Linguistic" or "L-type", and characterized by fast but inaccurate decoding), may arise from premature transfer.

Pugh and colleagues (2000b) found that in tasks that required participants to infer the pronunciation of non-words, non-impaired participants showed bilateral patterns of functional connectivity, while dyslexic participants appeared to rely on posterior right hemisphere networks. However, Rumsey et al. (1997) found bilateral differences in regional cerebral blood flow to temporal and parietal areas during phonological and orthographic decision making by men with

persistent dyslexia as compared with non-impaired controls. Moreover, a number of researchers have found evidence that suggest right parietal deficits in dyslexia (Facoetti et al., 2000; Hari et al., 2001; Stein and Walsh, 1997).

Of particular interest, Hari et al. (1995) compared a dyslexic group and a control group on a lateralized visual temporal-order judgement task, and found that the dyslexic participants were significantly more impaired on the task (with dyslexics less accurate at judging the temporal order of stimuli). In addition, they concluded that the dyslexic group showed a significant net rightward attentional bias, which they interpreted as indicating a "mini-neglect" of stimuli presented to left hemi-field, analogous to that exhibited by patients with right parietal lesions (Robertson et al. 1998; Rorden et al. 1997).

Deficits in dorsal temporal-parietal networks in both hemispheres have thus been implicated in reading impairment, as have deficits in the dorsal, magnocellular, visual pathway. However, attempts that have been made to link particular deficits with particular subtypes of dyslexia have produced evidence that remains inconclusive. A possible explanation for the inconclusive nature of the findings may be that particular patterns of reading impairment reflect dimensions rather than subtypes of dyslexia. A single aetiology may give rise to different profiles of impairment depending on the locus and extent of the disruption; conversely, a deficit affecting a particular network may have different aetiologies in different individuals with the same profile of disability. In particular, disruptions to left hemisphere networks might be expected to affect the linguistic phonological

organization of orthographic information, while disruptions to right hemisphere networks might be expected to cause disruption to the spatial perception of orthographic patterns.

This chapter reports three experiments that investigated the performance of dyslexic participants on a similar lateralized visual temporal order judgement (TOJ) task to those used by Hari et al (2001), Rorden et al (1997), and Robertson et al (1998). The task requires participants to report the onset order of two visual stimuli, one presented to left and one to right hemifield, in either left-first or right-first pairs. The lateralization of the stimuli means that task performance not only taps sensitivity to temporal order but also any lateralized perceptual bias, measures of these being derived from Signal Detection Theory (SDT) (Green and Swets, 1966). Although originally developed for tasks in which participants have to make a "yes" or "no" decision as to whether a stimulus has been presented or not, STD can be readily adapted to other two choice tasks, as it allows a measure of sensitivity to difference in the two conditions, or "discriminability index" ("d′" or "d prime") to be derived that is independent of any systematic tendency (response bias) to make a particular response in the face of uncertainty. In other words, it can be used to determine the "criterion" by which the participant makes or rejects a given response. Because in this experiment, the bias itself was of interest, a "criterion" index, also derived from SDT theory, was used as a measure of bias.

The purpose of the first experiment was to establish whether or not poor

performance on the task was associated with dyslexia, and whether dyslexic participants showed a particular lateral bias as compared with controls. The purpose of the second experiment was to ascertain whether lateral bias on the task was differentially associated with performance on tasks tapping different aspects of reading impairment in a sample of reading impaired adults. In the third experiment, the performance of dyslexic children on the task was investigated.

2.2 Experiment 1a

2.2.1 Method

2.2.1.1 Participants

Eleven adult developmental dyslexic and sixteen non-dyslexic control participants were recruited from the student population of the University of Wales, Bangor. The dyslexic group consisted of 8 female and 3 male participants, and the control group consisted of 11 female and 5 male participants, and all were native English speakers. All participants were paid for their participation. The mean age of the dyslexic group was 23.4 years, and that of the control group was 24.8 years (age ranges: dyslexic 19-39; controls 18-36). All participants were screened for dyslexia using ten[1] items from the Dyslexia Adult Screening Test (DAST) (Fawcett and Nicolson, 1988). These comprised four tests of written language processing[2], three tests of cognitive correlates of

[1] The Postural Stability test was not used.

[2] One Minute Reading; One Minute Writing; Two Minute Spelling; Nonsense Passage.

written language deficits[3], two word generation fluency tests[4], and a test of non-verbal reasoning. The test battery generates an At Risk Quotient (ARQ) that indicates the degree to which dyslexia is a likely diagnosis. An ARQ of 0.7 denotes the score achieved by 22.6% of the general population, and is taken to be a mild indicator of dyslexia, while an ARQ of 1 or above (scored by 14% of the population) is taken to be a strong indicator. In the dyslexic group, all participants scored an At Risk Quotient of 0.8 or more (found in 19.5% of the population), and the median score was 1.3; in the control group, all participants scored less than 0.8, and the median score was 0.3. Handedness was assessed using the Edinburgh Handedness Inventory (Oldfield, 1971); five of the control participants were found to be left handed, all of the dyslexia participants were right handed.

2.2.1.2 Procedure:

The task was programmed in MatLab[5] and presented on a 14 inch computer monitor. Each trial began with a black central fixation mark appearing in on a grey ground, accompanied by an auditory warning signal. The fixation mark was replaced after an interval of 2000ms by a white box (2.5cm x 2.5cm) appearing 8 cm to either the left or right of fixation. The first box remained on the screen, and at varying stimulus onset asynchronies (SOA), a second box appeared on the

---

[3] Rapid Naming; Phonemic Segmentation; Backward Digit Span;

[4] Verbal Fluency; Semantic Fluency

[5] The MathWorks, Inc. Natick, MA

opposite side of the fixation to first stimuli. The stimuli are illustrated in Figure 2-1:

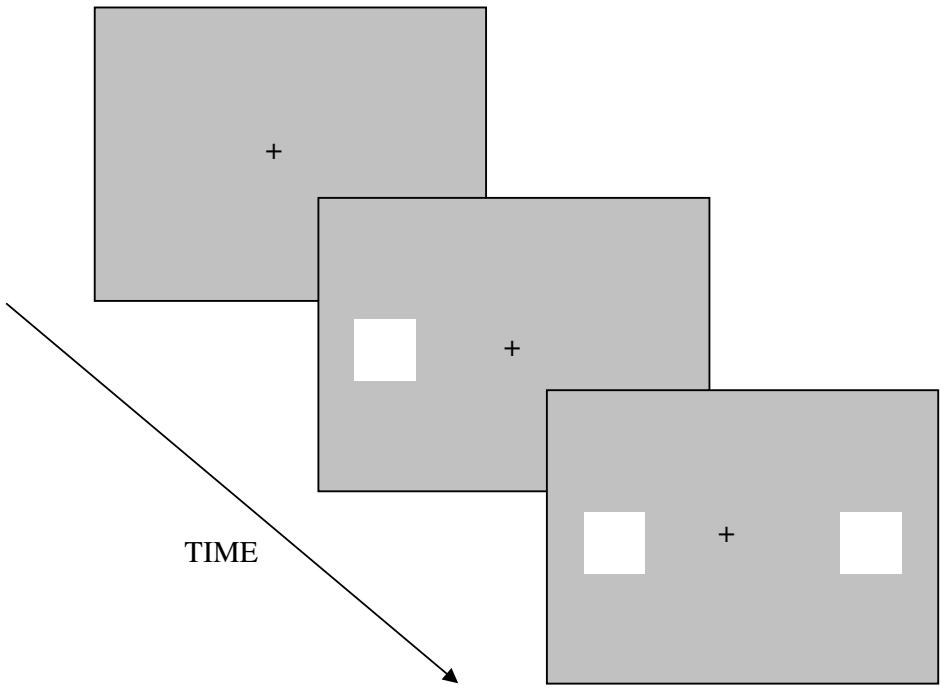

Figure 2-1: TOJ stimuli for a typical trial:

A fixation cross was followed after 2000ms by a white square either to left or right of fixation. A second square appeared in the contralateral hemifield after 0-400 ms. These Stimulus Onset Asynchronies (SOAs) were varied in 15 ms steps from 0 to 105 ms, with an additional baseline SOA of either 400 ms (Experiment 1) or 300 ms (Experiment 2). The participant was asked to report whether the left or right item had appeared first.

The SOAs between the two stimuli were randomly varied. SOAs ranged from 15 to 105ms in 15ms intervals (105, 90, 75, 60, 45, 30, 15). In addition, baseline trials with a SOA of 400ms, and catch trials in which the stimuli were presented

simultaneously, were presented. Fifty trials of each stimulus pair type (left-first pairs or right-first pairs) at each SOA were presented in random order. Participants were instructed to make an unspeeded decision as to which of the two stimuli appeared first. Participants responded using the index fingers of their right and left hands on a keypad (left key if left stimulus appeared first, right key if right stimulus appeared first).

2.2.2 Analysis and Results

2.2.2.1 Analysis

T-tests[6] were used to compare the groups for age, and for performance on individual items from the DAST. For the TOJ task, the proportion of correct responses at each SOA was computed, and STD theory used to compute d′ (prime) scores for each SOA as a measure of sensitivity, and criterion scores as a measure of lateral bias. This was done by regarding the responses as answers to the question "did the first (target) stimulus appear on the left?" A "left" response was therefore regarded as "yes" response, and a "right" responses as "no" responses. Thus, a "left" response was coded as a "Hit" for a left-first stimulus pair, and as a "False Alarm" for a right-first stimuli pair. A "right" response was coded as "Miss" for left-first stimulus pair and as a "Correct rejection" for a right-first stimulus pair.

---

[6] All statistical analyses, unless otherwise specified, were conducted using SPSS, versions 9-12. SPSS Inc. Chicago, IL.

D prime (d′) scores were computed by transforming the proportion of "Hits" and the proportions of a "False Alarm" into z scores representing the probability of such a score occurring by chance, using the "NORMSINV" function in Excel[78]. The z score of "False Alarms" was then subtracted from the z score of "Hits", to produce a d′ score, representing the participant's sensitivity to the temporal order of the stimuli, independently of any lateral bias. A d′ score of 0 would represent zero sensitivity to the temporal order of the stimuli. Criterion scores, representing degree of lateral bias, were computed by summing the two z scores. A criterion score of 0 would represent zero lateral bias; positive criterion values represented degree of leftward bias, and negative criterion values represented degree of rightward bias. These d′ and criterion scores were then entered in turn as dependent variables into a repeated-measures ANOVA with one within-subject factor (eight levels of SOA), and one between subjects factor (dyslexic versus control).

2.2.2.2 Results

There were no significant differences between the groups in age, or general cognitive function as measured by the Non-Verbal Reasoning item on the DAST. Of the remaining items from the DAST, the dyslexia group scored significantly worse than the control group on all items except for verbal fluency.

---

[7] As the z score of a perfect score is infinite, a scaling factor of .99 was used to allow d'prime and criterion scores to be computed for scores of 100%.

[8] Excel 2002 (10.3506.3501) SP-1, Microsoft Corporation, Redmond, WA.

For the d′ scores there was a main between-subjects effect of group [$F(1,25)=24.460$, $p<0.001$], indicating that sensitivity to the temporal order of the stimuli was significantly reduced in the dyslexic group relative to the control group. There was also an expected main effect of SOA, with temporal order sensitivity increasing with longer SOAs [$F(2.96,175) = 25.383$, $p<0.001$. However, there was a significant interaction between group and SOA [$F(2.96,175)$[9] $= 4.635$, $p<0.05$]. A difference contrast with the baseline SOA as the reference level indicated that sensitivity was more attenuated in the dyslexic group at all but the two shortest SOAs, and was thus evidence for a higher sensitivity threshold in the dyslexic group. The percentages of "left first" responses on the TOJ task at each SOA are shown graphically in Figure 2-2 for each group.

[9] After Greenhouse-Geisser correction for violation of sphericity.

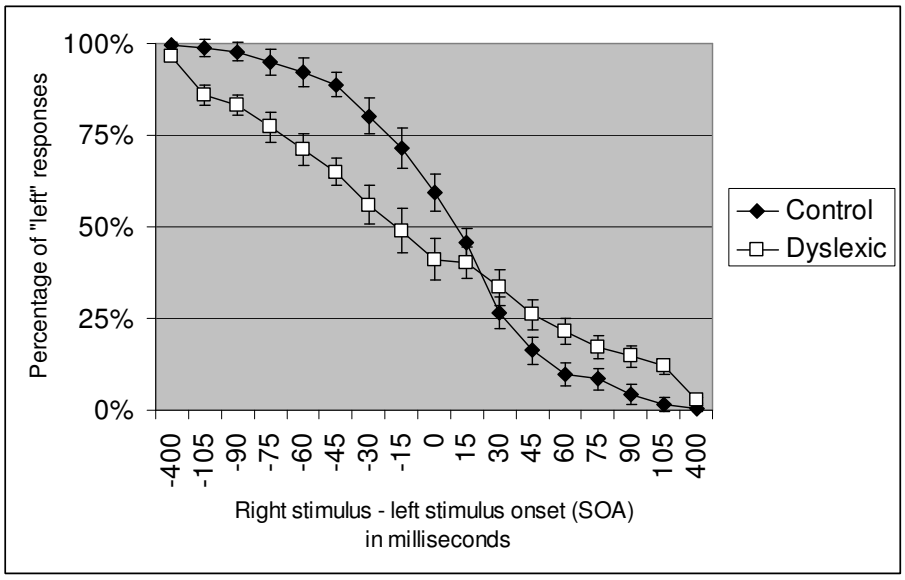

Figure 2-2: Results from experiment 1a.

Mean percentages of "right first" responses on the TOJ task for the two groups at each SOA. Left first stimuli pairs are shown as negative ms values. Error bars represent 95% confidence intervals. The dyslexic group was less accurate than the control group, but not significantly more biased.

For the criterion scores, there was no main effect or interaction, although a main effect of group trended to significance [$F(1,25)=4.119$, $p=0.053$. One-way ANOVAs conducted on each group with criterion scores as the dependent variable indicated that in neither group was the between-subjects intercept significantly different from zero, although examination of regression coefficients indicated that while the lateral bias in the dyslexic group tended to be rightwards, that of the control group tended to be leftwards. There was no main effect of SOA on lateral bias in either group. Means and standard deviations of d′ score and criterion scores are given in Table 2-1

	SOA	Control mean (St.dev) N=16	Dyslexic mean (St.dev) N=11
D prime	15	0.72(0.32)	0.11(0.76)
	30	1.63(0.61)	0.59(0.78)
	45	2.41(0.82)	1.01(0.84)
	60	2.85(0.72)	1.45(1.12)
	75	3.34(1.02)	1.85(1.21)
	90	3.88(0.73)	2.15(1.11)
	105	4.21(0.66)	2.4(1.14)
	400	4.46(0.23)	3.78(0.95)
Criterion	15	0.43(0.97)	-0.32(1.08)
	30	0.26(0.95)	-0.29(1.04)
	45	0.22(0.68)	-0.31(0.63)
	60	0.13(0.53)	-0.32(0.74)
	75	0.25(0.4)	-0.2(0.73)
	90	0.11(0.54)	-0.16(0.56)
	105	0.04(0.25)	-0.09(0.39)
	400	-0.03(0.3)	-0.08(0.26)

Table 2-1

Mean and standard deviation for d′ and criterion scores on the TOJ task for each group at each SOA.

2.2.3 Discussion

The dyslexic participants were less accurate at this temporal order task than the control participants. This finding is accord with the findings of Hari and colleagues, (2001) However, unlike Hari and colleagues, we found no significant net difference in lateral bias between the dyslexic group and control group in our study, although the trend for the dyslexic group to have a rightward bias relative to the control group was in the same direction as in the Hari study. A potential explanation for our finding lay in the inclusion of 5 (33%) left-handed participants in our control sample, as compared with only 14% non-right handers

in Hari's control sample[10] . However, our left-handed control participants tended to have a greater (although non-significant) leftward bias than the right-handed control participants. If anything, therefore, the inclusion of a greater number of left-handers in our control sample leveraged our results towards, rather than away from, the phenomenon reported by Hari et al, and cannot therefore be the explanation for our failure to replicate their findings of a net rightward bias in the dyslexic group.

An alternative explanation may be that the temporal order judgement impairments manifest in our study were due to more heterogeneous deficits than those present in Hari's sample of dyslexic readers. If these included bilateral and/or left-lateralized deficits, any net rightward bias due to a right-lateralized deficit would have tended to have been masked. Indeed, there is a large body of research indicating that English-speaking dyslexics represent a heterogeneous population, possibly because of the particular difficulty posed to readers of English by its opaque orthography, which includes many "exception" or "irregular" word such as "yacht" whose pronunciation cannot be inferred from a sublexical phonological assembly. In contrast, the pronunciation of non-words, such as "torlep" cannot be derived from a whole word recognition strategy, and must be tackled sublexically according to phonological rules.

However, Bakker's P- and L-subtypes (Bakker, 1994), defined with reference to readers of the orthographically transparent Dutch language, distinguishes reading

---

[10] No left-handers were included in either our dyslexic sample or in that of Hari et al.

impairments not in terms of non-word versus irregular word reading deficits, but in terms of problems with speed versus problems with accuracy. Using Bakker's subtypes, researchers have found evidence, in dichotic listening tasks, of right-ear advantage in L-type participants, as compared with P-type participants (Masutto et al., 1994) and of lack of right-ear advantage in P-type participants as compared with L-type participants and non-impaired control participants (Patel and Licht, 2000), thus providing some support for Bakker's model of "hemisphere imbalance", and suggesting that, at least in speakers of the orthographically transparent languages, inaccurate reading may be associated with a right-hemisphere deficit.

If so, right-lateralized deficits may be more prevalent in dyslexic groups recruited from orthographically transparent language groups than in English speaking groups, and left-lateralized deficits more prevalent in English speaking groups. This might account for the apparent "left-neglect" phenomenon found in both Hari and colleagues' (Hari et al., 2001) Finnish dyslexic readers and in Facoetti and colleagues' Italian readers (Facoetti et al., 2001), but the failure to find such a phenomenon in the group of English-speaking dyslexic participants in Experiment 1a. Such a deficit may be masked in an English-speaking group by the prevalence either of individuals with predominantly left-lateralized deficits, or of individuals with both.

It was postulated, therefore, that a heterogeneous range of deficits may have contributed to the overall profile of performance on the lateralized TOJ task

found in the dyslexic group in Experiment 1, with different individual profiles of impairment giving rise to different and opposing patterns of lateral bias. To test this hypothesis, rather than compare dyslexic with non-dyslexic groups, a within-group investigation into the performance of a heterogeneous group of reading impaired adults was conducted. Two hypotheses were tested. First of all, it was hypothesized that if sensitivity to temporal order on the TOJ task was impaired in dyslexic participants, as suggested by the findings from Experiment 1, and if the impairment on the task was directly associated with reading impairment, then in a within-group study of adult dyslexic participants, sensitivity to temporal order should be correlated with overall severity of impairment. Secondly, it was hypothesized that direction of bias on the task might account for variance in non-word reading accuracy over and above variance in accounted for by variance in irregular word reading, and by variance in phonological impairment.

2.3 Experiment 1b

2.3.1 Method.

2.3.1.1 Participants:

Thirty-eight reading-impaired adults (age range 16-60), 15 female, 23 male, were recruited, by means of a newspaper advertisement, to take part in the study. Four of the female participants were non-right-handed; 6 of the male participants were non-right-handed. All volunteers were accepted into the study, whether or not they had been previously screened for dyslexia, and were paid for their participation. As the study formed part of an investigation into the effects of a

biofeedback intervention for dyslexia (Liddle et al., 2005), the participants were also randomly allocated to either a treatment or a placebo group.

2.3.1.2 Procedure:

Participants were assessed for likelihood of dyslexia using the DAST, and were also given an untimed test of regular word, irregular word and non-word reading (Castles and Coltheart, 1993), scored for accuracy only. Handedness was determined by the Edinburgh Handedness Inventory (Oldfield, 1971).

The stimuli for the lateralized spatial temporal order judgment task were as for Experiment 1, except that the baseline SOA was 300ms, and there was no auditory warning signal. The task was presented on a laptop computer screen in four sessions consisting of 10 trials at each SOA, in random order. For each trial in which the SOA was greater than zero, the first stimulus appeared randomly in left or right hemifield, the randomization procedure generating approximately equal representations of each stimulus pair type. Unspeeded responses were made using the left and right cursor keys of an auxiliary keyboard, positioned so as to ensure that both keys and computer screen were directly aligned with the centre of the participant's body. The four blocks sessions took place within a two week period; before each session, participants undertook a session of either a placebo or a treatment version of the biofeedback intervention[11].

[11] details given in Liddle E, Jackson GM, Jackson SR. An evaluation of a visual biofeedback intervention in dyslexic adults. Dyslexia 2005; 11: 61-77.

2.3.2 Analysis and Results

2.3.2.1 Analysis

Individual deficit scores and "At Risk Quotient" (ARQ) scores on the DAST scores were computed using the age norms supplied (Fawcett and Nicolson, 1998). Responses on the TOJ task were pooled across the four sessions, the proportion of correct responses for each task condition was computed, and d′ and criterion scores computed as for Experiment 1. Pearson correlation coefficients were computed between four covariates of interest, Irregular Word Reading accuracy, and Non-Word Reading accuracy from the Castles and Coltheart task (Castles and Coltheart, 1993), and Backward Digit Span, and Phonemic Segmentation items from the DAST. Backward Digit Span was considered to be a measure of interest in that it is, like Phonological Segmentation, a measure of phonological or verbal short term memory that makes substantial demands on working memory capacity (Waters and Caplan, 2003).

Hierarchical multiple regression models were then tested in order to determine whether either Phonemic Segmentation scores, or Backward Digit Span scores accounted for a significant portion of variance unshared between Non-Word Reading and Irregular Word Reading accuracy.

For the TOJ task data, d′ and criterion scores were computed as for Experiment 1a. Repeated measures ANOVAs were then performed on d′ and criterion scores in turn, with one within-subjects factor (8 levels of SOA), and one between-subjects factor (treatment group); these were followed by a series of

ANCOVAs with Irregular Word Reading, Non-Word reading and a Phonological Short Term Memory (STM) score as between-subjects covariates.

2.3.2.2 Results

Analyses of the DAST scores indicated that the recruitment procedure succeeded in sampling a heterogeneous group of participants, with a mean and median ARQ of 1.3, ranging from 0.4 to 2.5. The incidence in the sample of scores at or above the moderate dyslexia indicator of 0.7 was 89% (34 participants). Of the four participants with an ARQ of below 0.7, all scored below the 10th percentile for the population on at least one strong indicator reading impairment[12]. Scores on all baseline test items manifested an approximately normal distribution except for Regular Word Reading scores which showed a ceiling effect.

Scores on the Non-Word Reading task were positively correlated with scores on the Irregular Word Reading task ($r = .64$, $p = <0.001$); this correlation remained significant after controlling for scores on the Backward Digit Span and the Phonemic Segmentation items from the DAST. Backward Digit Span and Phonemic Segmentation scores were also significantly correlated ($r=0.37$, $p<0.05$).

After accounting for variance shared between Non-Word Reading accuracy and Irregular Word Reading accuracy, Backward Digit Span accounted for significant further variance [R^2 change = 0.11, F change (1,35) =7.995, $p<0.01$],

[12] Rapid Naming, One Minute Reading, Phonemic Segmentation, or Two Minute Spelling

but Phonemic Segmentation scores did not, although it trended to significance. Neither Backward Digit Span scores nor Phonemic Segmentation scores accounted for additional variance in Irregular Word Reading scores over and above variance accounted for by Non-Word Reading scores. As Backward Digit Span and Phonemic Segmentation were scores were themselves significantly correlated, it was hypothesized that variance shared between the two measures represented a latent phonological short term memory (STM) factor and that this latent variable was accounting for the additional variance in Non-word reading scores. Principle Components Analysis was therefore used to extract a single component from Backward Digit Span and Phonemic Segmentation scores. The component had an eigenvalue of 1.373 and accounted for 68.7% of the total variance. Component scores for this "Phonological STM" factor were computed. When these component scores were entered into the hierarchical regression model to predict Non-word scores, Phonological STM accounted for a significant increase in R^2, over and above variance accounted for by Irregular Word Reading [R^2 change =0.13, F change(1,35)=8.993, $p<0.01$], indicating that Phonological STM was accounting for additional variance in Non-Word Reading scores.

In a repeated-measures ANOVA performed with d′ scores and criterion scores in turn as the dependent variable, and treatment group as a between-subjects factor, there was no main effect of treatment group, nor was there any interaction between treatment group and SOA[13]. For d′ scores, there was a significant main

[13] In subsequent analyses of treatment effects, some significant treatment group x session effects were found regarding lateral bias on the TOJ task; however, these did not affect findings when

effect of SOA [$F(4.17, 182.10)^9 = 150.166$, $p<0.001$], indicating, as expected, that temporal sensitivity increased with SOA. However, of interest was the between-subjects intercept for the ANOVA performed with criterion scores, which was not significantly different from zero ($F<1$), indicating no significant lateral bias across the pooled groups. However there was a significant interaction with SOA [$F(5.06.156.21)^9 = 2.910$, $p<0.05$], with a significant linear contrast [$F(1,37) = 257.709$, $p<.001$]. The signs of the regression parameters indicated that this effect represented a significant linear trend for a more rightward bias at shorter SOAs than at longer SOAs. Means and standard deviations for D′ and criterion scores are given in Table 2-2.

---

the TOJ performance scores were collapsed across sessions. See: Liddle E, Jackson GM, Jackson SR. An evaluation of a visual biofeedback intervention in dyslexic adults. Dyslexia 2005; 11: 61-77.

	SOA	Mean (St.dev)
D prime	15	-0.03(0.75)
	30	1.16(1.08)
	45	2.01(1.38)
	60	2.62(1.26)
	75	3.08(1.28)
	90	3.42(1.12)
	105	3.6(1.02)
	300	4.36(0.71)
Criterion	15	-0.08(0.59)
	30	-0.11(0.62)
	45	-0.05(0.42)
	60	-0.01(0.43)
	75	0.03(0.44)
	90	-0.07(0.28)
	105	0.12(0.35)
	300	0 (0.15)

Table 2-2

Means and standard deviations for d′ and criterion scores on the TOJ task for each SOA.

Non-Word Reading scores, Irregular Word Reading accuracy scores and Phonological STM scores were entered separately, and then together into a series of ANCOVAs, with d′ scores and criterion scores from the TOJ task in turn as dependent variables. For d′ scores, all three covariates were significant predictors of d′ scores; regression coefficients were positive, indicating that higher scores on each covariate was associated with higher d′ scores. For both Non-Word Reading scores and Phonological STM scores there was a significant interaction between SOA [Non-Word Reading scores: $F(4.34, 151.72)^{9} = 4.346$, $p<0.01$; Phonological STM scores: $F(4.53, 158.68)^{9}=3.507$ $p<0.01$]; polynomial contrasts confirmed a significant quadratic component, indicating that the

association between the covariate and TOJ d′ scores was maximal at mid-value SOA. When all three covariates were entered together, only Phonological STM score remained significant (Table 2-3).

		df	F	Sig.
Separate entry	Irregular words	(1,35)	9.439	<0.01
	Non words	(1,35)	6.932	<0.05
	Phonological STM	(1,35)	14.527	<0.001
Entered together	Irregular words	(1,33)	2.242	NS
	Non words	(1,33)	<1	NS
	Phonological STM	(1,33)	5.769	<0.05

Table 2-3

F values for the three between-subjects predictors of d′ TOJ scores. The upper part of the table represents covariates when entered separately, the lower part when entered together. Note that all three measures are significant predictors of d′ scores. However, the F values in the lower table indicate that it is variance shared between the reading measures and the phonological STM factor that is the major predictor of temporal order sensitivity on the TOJ task.

For the ANCOVAs with criterion scores as dependent variable, none of the three covariates was significant when entered separately. However, when all three were entered together, both Non-word Reading and Irregular Word Reading scores were significant predictors of lateral bias, but the prediction made by phonological STM factor scores remained non-significant. The signs of the regression coefficients indicated that Non-word reading was positively correlated with leftwards bias, while Irregular Word Reading was negatively correlated with leftwards bias. These results remained significant when phonological STM factor scores were removed from the model (Table 2-4).

		df	F	Sig.	Sign
Separate entry	Irregular words	1	1.128	NS	
	Non words	1	1.997	NS	
	Phonological STM	1	<1	NS	
Entered together	Irregular words	(1,33)	7.126	<0.05	-ve
	Non words	(1,33)	12.648	<0.01	+ve
	Phonological STM	(1,33)	2.976	NS	

Table 2-4

F values for the three predictors of criterion TOJ scores. The last column gives the sign of the regression coefficient representing the relationship between the predictor and leftwards bias. The upper part of the table represents the F values for the covariates when entered separately, the lower part when entered together. Note that when entered separately, none of the three measures is a significant predictor of criterion scores. However, when all three predictors are entered together, both Irregular word and Non word scores are significant predictors of leftward bias, but in opposite directions. Low Irregular word scores are therefore associated with leftward bias, and low Non-word scores with rightward bias ("left mini-neglect").

2.3.3 Discussion

The findings from Experiment 1b are consistent with the findings of Experiment 1a, in that impaired temporal order sensitivity on the TOJ task was associated with a number of indicators of dyslexia. Moreover, the portion of variance in reading measures most strongly associated with temporal order sensitivity scores was that shared with scores on a latent variable underlying Backward Digit Span and Phonemic Segmentation scores, and postulated to represent a measure of phonological STM, a deficit which is characteristic of dyslexia. Moreover, as with the findings of Experiment 1a, the findings of Experiment 1b fail to support the findings of Hari et al (2001) of an overall "left mini-neglect" phenomenon

associated with dyslexia, although the group as a whole tended to show a more rightward bias at shorter than at longer SOAs.

However, the findings of Experiment 1b support the hypothesis that different patterns of deficits in the TOJ task are associated with different patterns of impairments on Non-word reading, Irregular Word Reading, and tasks tapping phonological STM. Scores on the phonological STM factor predicted impairment of temporal order sensitivity at all SOAs, as well as accounting for variance in Non-Word Reading accuracy that was not accounted for by a measure of word recognition. This finding suggests that a key contributor to selectively impaired non-word reading in dyslexia, namely phonological STM, is also associated with raised temporal order sensitivity thresholds in this visual TOJ task.

Counteractive patterns of lateral bias were predicted by Non-word and Irregular Word Reading accuracy measures respectively. After controlling for a phonological STM factor, non-word reading accuracy that was worse that predicted on the basis of irregular word reading predicted a more rightward bias on the task, while irregular word accuracy that was worse than that predicted on the basis of non-word reading accuracy was associated with more leftward bias on the task.

This result is consistent with the hypothesis that word recognition deficits (as indexed by irregular word reading accuracy) may be associated, in an English-speaking sample, with a left-hemisphere deficit that also induces a leftward

attentional bias, but that additional variance in non-word reading is accounted for by a right-hemisphere deficit that induces a counter-active rightward attentional bias.

Non-word reading may be more likely than real word reading to be disabled by any form of visual-spatial disruption, such as that arising from a right-lateralized deficit affecting orienting to left hemifield. Such a deficit might impair letter position encoding, or alternatively, the development an efficient left-to-right strategy for tackling unknown words. This hypothesis is supported by evidence from lesion studies that suggest that right-sided lesions can produce a form of dyslexia in which lexical access is spared, but non-word reading is disproportionately impaired (di Pellegrino et al., 2001). A case study by di Pellegrino and colleagues (di Pellegrino et al., 2001) found that a patient with severe left neglect resulting from a right-sided brain-damage was more inclined to fixate the contralesional side of a real word than a non-word, while Ladavas and colleagues (Ladavas et al., 1997) found that real word reading, but not non-word reading, was improved if patients with neglect dyslexia first were first asked to decide whether the word was or was not a real word.

It is, of course, possible that the apparent "left-neglect" factor found to be associated with non-word reading accuracy in Experiment 1b may have been amplified by the effects of the treatment. Nonetheless, as the reading measures were all made before treatment was commenced, the inference of a trait association between rightward attentional bias and non-word reading score

remains valid. What, however, needs to be considered, is the possibility that the treatment program may have induced a state in which a propensity for a rightward attentional bias was enhanced in those participants who received the treatment. Nonetheless, the fact that the association between non-word reading accuracy and rightward bias was found even when group membership was modeled suggests that the finding is robust.

The findings of Experiment 1a therefore raise the possibility that either the postulated dorsal stream deficits, or other unspecified deficits that contribute to poor non-word reading in dyslexia, may include disruptions to right hemisphere networks implicated in spatial attention. If such deficits were more prevalent in non-English speaking dyslexic populations sampled by Hari et al (2001) and by Facoetti et al (2001), this might account for their findings of a net rightwards attentional bias in their samples. `

2.4 Interim summary

The results of Experiments 1a and 1b suggest that visual temporal order sensitivity is impaired in dyslexia. Results from Experiment 1b suggest that this impairment may associated with a latent variable that contributes to impairment on two tasks tapping phonological short-term memory, namely backward digit span and a phonemic segmentation task, as well as to non-word reading that is worse than predicted by irregular word reading. These results are consistent with the possibility that a deficit in the temporal ordering of auditory stimuli (digits, phonemes) that is characteristic of dyslexia is also reflected in impaired temporal

ordering of visual stimuli. However, the evidence presented here is also consistent with the hypothesis that non-word reading may also be selectively impaired by a right-lateralized deficit that also impairs attention to stimuli presented in left hemifield. If so, the evidence also suggests that in a heterogeneous group of English-speaking dyslexic adults the effects of such a right-lateralized deficit may be masked by the prevalence in the sample of participants with a left lateralized deficit, impairing attention to stimuli presented in right hemifield, and affecting word recognition, or of participants with bilateral deficits.

2.5 Experiment 1c

Studies of English-speaking dyslexic children suggest that irregular word reading performance is a measure of "print exposure" that may be a secondary consequence of a fundamental deficit that primarily affects non-word reading (Griffiths and Snowling, 2002; Stanovich et al., 1997). Griffiths and colleagues (Griffiths and Snowling, 2002) found that while measures of phonological and STM skills were significant predictors of non-word reading in a sample of children aged 9-15, neither accounted for significant variance in irregular word reading when a measure of reading experience was controlled for. Given the well-established relationship between phonological deficits and left-lateralized anomalies in dyslexia (Paulesu et al., 1996; Pugh et al., 2001; Temple, 2002), this is potentially at odds with the finding of Experiment 1b that poor irregular word reading, after controlling for non-word reading and a measure of

phonological STM was associated with leftward attentional bias, and thus with a likely left-hemisphere locus for the associated deficit, whereas poor non-word reading was associated with a right-ward attentional bias, and thus with a likely right-hemisphere locus for the associated deficit.

However, for dyslexic adults, irregular word reading may bear a different relationship to print exposure than it does in children, for whom print exposure is likely to be fairly closely coupled to current reading level and thus to the severity of any underlying phonological or other deficit. In adults, print exposure is likely to be a function of many factors, including educational level and opportunity for remediation. Experiment 1c was therefore conducted in order to ascertain whether the pattern of results found in Experiment 1b would be replicated in a sample of reading impaired children.

2.5.1 Method

2.5.1.1 Participants

Thirty-seven children (10 girls and 27 boys) were recruited by means of a newspaper advertisement seeking volunteers with dyslexia aged 6.5 to 16 years. A monetary reward was offered as an incentive, with a bonus payment on completion of the study. Not all children had been previously assessed for dyslexia. Caregivers of children were asked about vision impairment, and known likely physical cause of dyslexia. If any of these were reported, the child was excluded from the study.

2.5.1.2 Procedure

Children were assessed for likelihood of dyslexia using the DST, (Fawcett and Nicolson, 1996). They also took the untimed test of regular word, irregular word and Non-Word Reading (Castles and Coltheart, 1993) used in Experiment 2, scored for accuracy only, and the phoneme deletion task devised by McDougall et al (McDougall et al., 1994), also scored for accuracy only. In the phoneme deletion task, the experimenter reads out a non-word, and asks the child to repeat the word with one phoneme deleted (for example: "say *bice* without the *b*"). In each case the deletion of the phoneme produces a real word (for example, "bice" without the "b", becomes "ice"). IQ was assessed using the two-item form of the WASI (Wechsler, 1999). Handedness was determined by the Edinburgh Handedness Inventory (Oldfield, 1971). Children attended two sessions, at each of which they undertook one block of the same version of the TOJ paradigm employed in Experiment 1b.

2.5.2 Analysis and results

The age range of the children was from 8 to 16, with a mean age of 11 years and 8 months (standard deviation = 2 years and 3 months). All children had IQ scores within or above the normal range (>85). Twenty nine had an ARQ on the DST of 0.7 or higher, indicating that they were "at risk" of dyslexia, and both the mean and median ARQ score was 1.1. The remaining eight children fell within the normal range on the DST. Seven of the children were non-right-handed.

To generate factor scores on a phonological STM factor comparable to those

used in Experiment 2, scores from the Backward Digit Span and Phonemic Segmentation scores from the DST as well as on scores from the Phoneme Deletion task were subjected to Principle Components Analysis. One component had an eigenvalue >1 (=2.128), accounting for 67.9% of the total variance, and component scores on this component were computed. The component matrix is given in Table 2-5.

Item	Correlation with component score
Backward Digit Span	0.803
Phonemic Segmentation	0.831
Phoneme Deletion	0.891

Table 2-5:

Correlation coefficients between the Phonological STM component scores and the three variables from which it was derived. The component was most strongly correlated with Phoneme Deletion scores, and least strongly with Backward Digit Span scores.

Non-word reading and Irregular Word Reading were strongly correlated ($r=0.84$, $p<0.001$). Age in Months was also strongly correlated with each of the two reading accuracy measures (non-words: $r=0.40$, $p<0.05$; irregular words: $r=0.43$, $p<0.01$), but the correlation between Irregular Word Reading and Non-Word Reading remained high after controlling for age (partial correlation: $r=0.8$, $p<0.001$). A series of hierarchical regressions were then performed, as in Experiment 1b. To predict Non-Word Reading scores, Irregular Word Reading scores were entered first into the regression, followed by Age in Months, and Phonological STM scores. To predict Irregular Word Reading scores, Non-Word Reading scores were entered first into the regression, followed by Age in

Months, and Phonological STM scores. Age accounted for no additional variance in either of the two reading score that was not accounted for by the variance shared between them. Phonological STM factor scores accounted for significant additional variance in Non-Word Reading accuracy over and above that accounted for by Irregular Word Reading accuracy [R2 change=0.09, F(1, 33) change=14.014, $p<0.01$]; however the Phonological STM factor score accounted for no significant additional variance in Irregular Word Reading over and above that accounted for by Non-Word Reading.

TOJ task performance scores were pooled across both blocks, and d′ and criterion scores were computed for each SOA as for Experiments 1a and 1b. Analyses carried out as for Experiment 1b, except that Age in Months was entered as an additional covariate in all analyses.

For the repeated measures ANOVA on d′ scores, there was a significant and expected main effect of SOA [$F(4.47, 169,96)^{9}= 53.413$, $p<0.0001$], with a significant linear contrast indicating increasing temporal order sensitivity with increasing SOA. For the ANOVA with criterion scores, the between-subjects intercept was not significantly different from zero ($F<1$), indicating no significant lateral bias across the group. There was no significant main effect of SOA for criterion scores. Means and standard deviations for d′ and criterion scores are given in Table 2-6.

	SOA	Mean (St.dev)
D prime	15	0.17(0.55)
	30	0.48(0.68)
	45	0.63(0.89)
	60	1.19(1.03)
	75	1.13(0.92)
	90	1.63(1.14)
	105	1.92(1.04)
	300	2.68(1.31)
Criterion	15	-0.03(0.22)
	30	-0.04(0.25)
	45	0.04(0.22)
	60	-0.02(0.2)
	75	0.01(0.21)
	90	-0.04(0.16)
	105	0.04(0.17)
	300	-0.06(0.16)

Table 2-6

Means and standard deviations of d′ and criterion scores at each SOA.

For ANCOVAs performed with d′ scores, when Non-Word Reading, Irregular Word Reading and Phonological STM scores were entered separately (together with Age in Months), each was a significant predictor of temporal order sensitivity over and above any prediction made by age. However, when entered together, each covariate failed to make a significant independent prediction, indicating that variance shared between the three predictors was accounting for variance in temporal order sensitivity on the TOJ task. A second Principle Components Analysis was therefore performed, in order to isolate a component representing variance shared between the two reading measures (Non-word Reading and Irregular Word Reading) and the three measures contributing to Phonological STM scores. One component had an eigenvalue >1 (=3.395),

accounting for 67% of the total variance, and factor scores for this component were computed. The component was regarded as a "Composite Reading Skill" component. The component matrix is shown in Table 2-7.

Item	Correlation with component score
Backward Digit Span	0.727
Phonemic Segmentation	0.754
Phoneme deletion	0.879
Non Word Reading accuracy	0.910
Irregular Word Reading accuracy	0.836

Table 2-7:

Correlation coefficients between "Composite Reading Skill" component and the three variables from which it was derived. The component was most strongly correlated with Non-Word Reading scores, and least strongly with Backward Digit Span scores.

Composite Reading Skill scores were entered as a covariate together with age into the ANCOVA. Composite Reading Skill scores a significant predictor of temporal order sensitivity [$F(1.33)=11.779$, $p<0.01$], Age in Months accounting for significant additional variance [$F(1,33)=5.566$, $p<0.05$]. The prediction made by Composite Reading Skill remained significant after controlling for IQ scores.

In the ANCOVAs performed with criterion scores, Non-Word Reading, Irregular Word Reading and Phonological STM component scores were entered separately into the model, together with Age in Months. None of the three covariates made any significant prediction regarding lateral bias, after controlling for age, and Age in Months itself made no significant prediction. However, both Non-Word Reading accuracy scores and the Phonological STM factor interacted significantly with SOA. [Non Word Reading: $F(5.39, 183.34)^{9}=2.481$, $p<0.05$;

Phonological STM: $F(5.49,186.82)^{9}=2.268$, $p<0.05$]. Deviation contrasts indicated that in both cases, the relationship between covariate and leftward bias was more positive at shorter SOAs.

When all four covariates were entered together into the model, these interactions ceased to be significant, suggesting that collinearity between Non-Word Reading and Phonological STM component scores was masking the SOA effect. To test the interaction, whilst avoiding confounds due to collinearity, two further ANCOVAs were performed on criterion scores at the two shortest SOAs only (15ms and 30ms). In the first, Irregular Word Reading, Non-word Reading and Age in Months were entered as covariates; in the second, Irregular Word Reading, Phonological STM and Age in Months were entered as covariates. In both these ANCOVAs, Irregular Word Reading was a significant predictor of criterion scores, the sign of the regression coefficient indicating that low scores were associated with a more leftward bias at short SOAs. In contrast, rightward bias was significantly predicted by Non-word Reading scores in the first ANCOVA and by Phonological STM in the second. These results are given in

Table 2-8. As a final check, IQ scores were entered into both ANCOVAs as an additional covariate. Both findings remained robust, and IQ was not a significant predictor of lateral bias.

		df	F	Sig.	Sign
Model 1	Age in Months	(1,33)	0.001	NS	
	Irregular Word Reading	(1,33)	5.472	0.026	-ve
	Phonological STM	(1,33)	8.276	0.007	+ve
Model 2	Age in Months	(1,33)	0.567	NS	
	Irregular Word Reading	(1,33)	5.639	0.024	-ve
	Non Word Reading	(1,33)	6.026	0.020	+ve

Table 2-8:

Results for the two ANCOVA models conducted on criterion scores for the two shortest SOAs (15ms and 30ms). The last column gives the sign of the regression coefficient representing the relationship between the predictor and leftwards bias. Note that in each model, Irregular Word Reading is negatively correlated with leftward bias, indicating that low scores are associated with a leftward bias. In contrast, both Non-Word Reading scores and phonological STM scores are positively correlated with leftward bias, indicating that low scores on both are associated with a more rightward ("left neglect") bias.

2.5.3 Discussion

The results from Experiment 1c regarding temporal order sensitivity thus support the results from Experiment 1a and b. Temporal order sensitivity on the TOJ task was positively correlated with measures of reading and phonological STM, after controlling for age, suggesting that impaired temporal order sensitivity on the TOJ task is associated with impairments typical of dyslexia. Moreover, by using Principle Components Analysis to compute scores on a factor representing variance shared between reading measures and measures of phonological STM, and by using scores on this factor to predict TOJ task performance, Experiment 1c replicates the finding from Experiment 1b that it is the portion of variance in

reading accounted for by phonological STM task performance that is associated with impaired TOJ task performance.

Given the strong association between reading impairment and phonological deficits (Ramus, 2003; Snowling et al., 1996; Stanovich et al., 1997), the finding that the association between phonological skill-related variance in reading, and temporal order sensitivity on the TOJ task was robust after controlling for both age and IQ thus supports the hypothesis that dyslexia, whether defined in terms of reading that is discrepant with IQ and age, or in terms of a phonological deficit, is associated with impaired temporal order sensitivity on a visual, non-phonological, task.

Regarding lateral bias, the results from Experiment 1c are consistent with results from Experiment 1b, at short SOAs. As with Experiment 1b, impaired Non-Word Reading was associated with a rightward lateral bias, while impaired irregular word reading was associated with a leftward lateral bias. However, unlike the adults in Experiment1b, the biases in the children were only apparent at short SOAs (15ms-45ms), and moreover, the variance in Non-Word Reading that was predictive of a rightward bias was also shared with variance on tasks tapping phonological STM.

2.6 General Discussion

Evidence from all three experiments reported in this chapter support the hypothesis that dyslexia is associated with a supramodal temporal ordering

deficit that is manifest in impaired temporal order sensitivity in a temporal order judgement task utilizing visual, non-letter, stimuli, as well as in impairment in tasks tapping phonological STM.

In Experiment 1a, a between-groups comparison indicated reduced temporal order sensitivity on the TOJ task in dyslexic participants as compared with control participants. Experiments 1b and 1c investigated signs of dyslexia within heterogeneous groups of reading impaired participants, and in both, a deficit in phonological STM that was associated with selectively impaired Non-Word Reading was positively correlated with impaired temporal order sensitivity on the TOJ task. In Experiment 1c, both age and IQ were controlled for, and the effect remained robust. Thus, whether dyslexia is conceived of in IQ-achievement discrepancy terms, or in terms of a phonological deficit model, its severity would appear to be reflected in the degree of impairment on this visual TOJ task.

The evidence is thus consistent with several supramodal models of dyslexia. For example, as the stimuli utilized were low spatial frequency peripheral stimuli, and the task required rapid temporal processing, the findings are consistent with the magnocellular deficit model (Greatrex and Drasdo, 1995; Stein and Walsh, 1997); however it is equally consistent with models that postulate more generalized perceptual deficits (Amitay et al., 2002), or indeed deficits in automatization, as in the cerebellar deficit model (Nicolson et al., 2001).

Regarding lateral bias in dyslexia, all three experiments reported in this chapter fail to replicate Hari's (2001) finding of an overall rightward bias, or "left mini-

neglect", in dyslexic participants as a group. However, both Experiment 1b and Experiment 1c provide evidence of counteractive biases associated with impairments in non-word reading accuracy and irregular word reading accuracy respectively. The first of these may be congruent with Hari et al's "left mini-neglect" and associated with a characteristic feature of dyslexia, namely non-word reading that is more inaccurate than would be expected on the basis of word recognition skills. However, in the adult sample, this deficit was not associated with variance in non-word reading accuracy accounted for by a phonological deficit, whereas in the sample of children it was. Moreover, the effect in children was only observed at short SOAs.

A number of possibilities may account for these differences. One is that in children, non-word reading and performance on phonological tasks may affect each other. While deficits in phonological skill may hamper decoding, any additional deficit affecting decoding, for example, poor letter position encoding, and/ or an unstable left-to-right decoding strategy, may in turn hamper the acquisition of phonemic segmentation skills. Thus, even a non-phonological deficit in non-word reading may be reflected in impaired performance on a phonological task, and account for the relationship observed between a variable representing performance on task tapping phonological skill and a rightward attentional bias. Wagner and colleagues (Wagner et al., 1994) speculate that as children become more able to visualize a written word, they may use such a visualization to manipulate its phonemes; if so, a deficit that affected such

visualization might contribute to difficulties on phonological tasks.

A second possibility is that processes subserving reading, including phonological analysis, may be less strongly lateralized in children than in adults. Thus a right lateralized deficit may be manifest in impairment on phonological tasks in children but not in adults. This would be consistent with the finding of Turkeltaub and colleagues (Turkeltaub et al., 2003) in a study of children at varying stages of reading development, that greater activity in the left superior temporal sulcus was associated with maturation of phonological processing abilities, and that an increase in left lateralized activity and a decrease in right-lateralized activity was associated with reading development.

Regarding the finding that significant bias was only observed at short SOAs in children, one possibility, borne out by the high correlation between non-word and irregular word reading scores in Experiment 3, is that for children, reading exposure, and thus word recognition skill, may bear a much closer relationship to current decoding skill level than it does in adults. In the children, the proportion of variance unshared between the two reading measures was less, and the study may therefore have had less power to detect the counteractive effects of any lateralized deficits underlying each scores on each measure.

A further possibility is that the children found the task harder (their scores were lower than in the adult study), resulting in a greater proportion of random noise in the data. Lateralized effects may therefore only have been observable at short SOAs where they may have produced "extinction" of the onset of second

stimulus. At longer SOAs, where both stimulus onsets were observed, but the temporal order was not recorded, children may simply have randomly guessed.

Nonetheless, to the extent that the results of the children's study replicates the findings in the adult study regarding the relationship between non-word reading and a rightward lateral bias, the possibility is raised that reading impairment that is characterized by a specific non-word reading deficit may arise from more than one cause. One of these might be an underlying supramodal deficit in sensitivity to rapid rise-time stimuli, leading to phonological deficit by the mechanism proposed by Goswami and colleagues, (2002), but also manifest, epiphenomenologically, in raised temporal order sensitivity thresholds on this visual TOJ task.

A second may be a right-lateralized deficit characterized by a "mini-neglect" of stimuli presented in left visual field, particularly at short SOAs, as postulated by Hari and colleagues (2001). Such a deficit might account for poor non-word reading accuracy in a task where the word can neither be recognized lexically, nor divined from context. Possible mechanisms by which such a deficit might impair non-word reading might be either disruption to networks implicated in letter position encoding, or the undermining of an efficient left-to-right reading strategy.

If so, the failure to find a net lateral bias in the three dyslexic samples studied may be explained by the presence, in all three samples, of participants with left-lateralized deficits in word recognition, either with or without co-morbid non-

word reading deficits that produce a counteractive leftward attentional bias. Candidates for such a deficit include more general language deficits, or a specific word recognition deficits affecting left-lateralized areas implicated in word recognition (Cohen et al., 2000; Pugh et al., 2000a).

These experiments leave open the question as to the extent to which the deficits predictive of lateral bias also contribute to impairment in temporal order sensitivity. The use of Signal Detection Theory enabled temporal order sensitivity to be separated from the issue of lateral bias, but by the same token, necessarily masked any relationship between the two. One possibility is that deficits in bilateral networks implicated in the orienting of attention to stimuli in time and/or space may underlie both reduced temporal order sensitivity and lateral bias on this visual, lateralized TOJ task, but that the extent to which deficits in such networks are left-lateralized, right-lateralized or bilateral may be reflected in different profiles of reading impairment, and in different patterns of lateral bias on the TOJ.

Perhaps a more fundamental question raised by these three experiments is whether the decreased temporal order sensitivity to the onsets of lateralized visual stimuli reflects a deficit that is best viewed as a deficit in temporal order judgement per se or a deficit in orienting attention towards a peripheral stimulus. One possibility is that either or both of the apparently dissociated deficits observed in the dyslexic participants in Experiments 2 and 3 are related to the shifting of attention in space as well as in time.

Coull and colleagues (Coull et al., 2000) proposed, and found evidence to support, the hypothesis that orienting to time and orienting to space are separate systems that involve cortical networks located in parietal areas of left and right cerebral hemispheres respectively. Rushworth and colleagues (Rushworth et al., 1997) similarly hypothesized separate attentional systems implicating homologous left and right lateralized parietal networks in, respectively, motor attention – preparation of a limb for movement, and spatial orienting – preparation for an eye movement, a hypothesis supported by evidence from patients with parietal lesions, and the effects of Transcranial Magnetic Stimulation (TMS) (Rushworth et al., 2001). Nobre (2001a) has proposed that attentional systems relevant for any given task co-opt brain areas specialized for the execution of that task. Thus left-lateralized networks implicated in the planning of limb movements or articulatory gestures are likely to be implicated in temporal orienting, while right lateralized networks implicated in the planning of eye movements are likely to be implicated in representing spatial locations.

The next stage of this investigation therefore turned to the issue of overt spatial orienting in dyslexia, and to its relationship, if any, with the impaired patterns of performance on the lateralized TOJ task found in dyslexic participants in the experiments reported in this chapter.

3. SPATIAL COMPRESSION AND TEMPORAL ORDER JUDGEMENT IN DYSLEXIA

3.1 Introduction

The results from the TOJ experiments reported in Chapter 2 indicate that people with dyslexia display a deficit in judging the temporal order of pairs of visual stimuli presented one to each visual hemifield. It was postulated that unilateral or bilateral deficits in networks implicated in the orienting of attention may underlie the reduced temporal order sensitivity and lateral bias observed in dyslexic participants. One possibility, therefore, is that saccades – eye movements by which salient features of the visual scene are brought to subtend the foveal region of the retina where acuity is greatest – are poorly specified in dyslexia. If so, this might be manifest in abnormalities in both covert attention – attention to peripheral areas of the visual field – and overt attention – the act of foveating a salient location.

Networks involved in covert orienting are likely to overlap with those involved in planning a saccade (Corbetta et al., 1998; Findlay and Gilchrist, 2003; Nobre, 2001a). Godijn and Theeuwes (2002) present a model of saccade planning according to which potential saccades are represented by locations on a neuronal "saccade map". Salient stimuli, to which a potential saccade may be made, are postulated to be represented by increased neural activation at their location on this map. When activation at any one location on the map reaches a threshold, a

saccade is triggered, while activation at a sub-threshold level represents covert orienting to that location. One possibility, therefore, is that low levels of activation on such a saccade map may underlie the deficits in performance by dyslexic participants on the TOJ experiments reported in Chapter 2.

The TOJ task requires the localization of an abrupt onset stimulus that is rapidly followed by a competing stimulus. As saccade latencies are typically longer than the SOAs utilized in the task, no overt orientation to the target is likely before the arrival of the competing stimulus. Accurate performance may therefore depend on the magnitude of sub-threshold levels of activation at the stimuli locations on saccade maps. If salient stimuli are poorly specified on the map, accuracy on the TOJ task may be impaired. If so, the deficit that underlies impaired TOJ task in dyslexic participants may also be reflected in poorly specified saccades, leading not only to impaired judgement of the temporal order of peripherally presented visual stimuli, but to impaired spatial constancy across saccades.

When we make a saccade, the world appears to stay still, even though the images of objects in the visual field have been displaced on the retina. The fact that spatial constancy is maintained across saccades implies that with each saccade, the spatiotopic representation of a visual stimulus is translated from a coordinate system with the initial fixation point as origin, to one with the upcoming fixation point as origin. Evidence that this remapping process is at least partially predictive is provided by single neuron recordings from lateral intra-parietal (LIP) neurons in macaques indicating that the receptive field properties of these

neurons show anticipatory shifts in their receptive fields immediately prior to a saccadic eye movement (Colby et al., 1996; Kusunoki and Goldberg, 2003).

If saccades are poorly specified in dyslexia, one consequence may be that this predictive remapping process fails. As predictive remapping is thought to be necessary to enable the perceptual system to distinguish between retinal displacement that is due to an eye movement from retinal displacement that is due to the movement of the object itself, failure of this system might be expected to lead to illusory movement of elements in the visual field during a task such as reading. Poorly specified saccades might therefore account for the visual anomalies reported by some dyslexic readers, such as letters and words appearing to change position on the page (Stein, 2003).

Eden and colleagues (Eden et al., 1994; Eden et al., 1995) investigated eye movements in dyslexic children and found in a preliminary study (Eden et al., 1994) that dyslexic children had poor fixation control at the end of saccades. In a subsequent study with a larger sample (Eden et al., 1995) they found that dyslexic children were significantly impaired as compared with non-disabled control children on tasks tapping fixation control, vergence control, and the tracking of static stimuli down a page. They speculate, *inter alia*, that this may indicate a deficit in right-hemisphere networks implicated in stereoscopic vision. However, in a study of dyslexic and normally reading adults, Moores and colleagues (Moores and Andrade, 2000) found no significant differences between groups on a task requiring vergence control across saccades, although they did

find that when a sequential element was introduced into the task, practice resulted in improved performance in the control group, but decrements in performance in the dyslexic group, suggesting that learning effects may have been outweighed by fatigue effects in the dyslexic group. The authors postulate that this may be due to deficits in learning a sequential motor task, consistent with the hypothesis of a cerebellar deficit; alternatively it may be that making voluntary saccades is more fatiguing for dyslexic than for non-dyslexic participants. If so, one possibility is that this may be due to a deficit in saccade programming.

Biscaldi and colleagues (1998) found that saccadic reaction times (saccade latencies) were more variable in dyslexic participants, who made more late saccades (>700ms) and more "express saccades". They speculate that the their findings may be accounted for by deficits in attentional selection processes, and may result in dyslexic readers failing to utilize peripheral or parafoveal cues to direct their attention across the visual field while reading.

Of interest in this regard is an investigation by Crawford and Higham (2001) into saccade trajectories to single and double stimuli in dyslexic and non-dyslexic participants. When more than one saccade target is presented, a "global" or "centre-of-gravity" effect is sometimes observed, whereby, if the two targets are relatively close, saccades tend to terminate midway between the two targets (Walker et al., 1997). Crawford and Higham (2001) found that dyslexic participants and non-dyslexic control participants were equally accurate on a task

requiring a saccade to be made to a single stimulus presented at either 5° or 10° eccentricity. However, when stimuli were presented at both locations simultaneously, the control participants showed the expected "centre-of-gravity" effect, but in the dyslexic participants this effect was attenuated, the endpoints of the saccades of the dyslexic participants being close to the near stimulus.

The model proposed by Godijn and Theeuwes (2002) can account neatly for the centre-of-gravity effect, as well as a for second effect, by which distractor stimuli remote from the saccade target result in longer latencies, but do not appear to distort the endpoint of the saccade (Walker et al., 1997), by postulating that neural representations of nearby salient locations have mutual lateral excitatory effects, while remote locations exert mutual lateral inhibitory effects. Their model is shown schematically in Figure 3-1.

Interpreted in the light of Godijn and Theeuwes' model, Crawford and Higham's (2001) finding of an attenuated centre-of-gravity effects in dyslexic participants could indicate reduced lateral excitation from neural representations of salient peripheral stimuli, possibly from a deficit in the processes by which salient peripheral stimuli, and thus potential saccade vectors, are represented on a neural visual-spatial map. Furthermore, peri-saccadic processes by which spatial constancy is maintained across saccades are also likely to be compromised. If so, this might account for visual confusion during reading, and may lead to disruption to the processes by which written text is recognized.

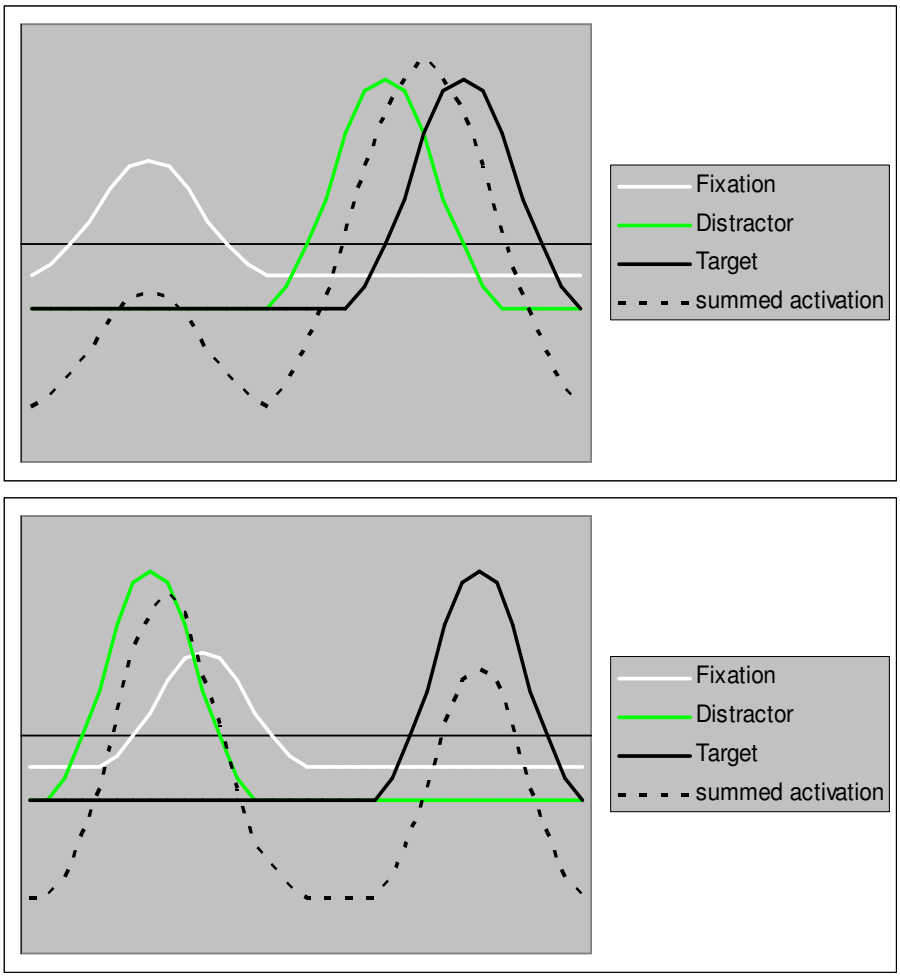

Figure 3-1: Godijn and Theeuwes's model of distractor effects:

Godijn and Theeuwes's model of distractor effects: In the top figure, a distractor is presented close to the saccade target. The two salient locations (probe and target) result in summed activation that reaches threshold more rapidly, reducing saccade latency, and peaking at a location between the two, distorting the intended saccade trajectory. In the lower figure, a distractor is presented distant from the saccade target. The salience of the distractor location inhibits activation at the target sight, slowing the achievement of threshold and thus increasing latency, but leaving the saccade trajectory unaffected.

One paradigm designed to tap the efficiency of the mechanisms involved in maintaining spatial constancy across saccades, is, ironically, a paradigm designed to elicit peri-saccadic *mis*-localization – the mislocalization of stimuli presented briefly within a small time window in which a saccadic eye movement occurs. A typical paradigm used to elicit peri-saccadic spatial mislocalization is that developed by Ross and colleagues (Ross et al., 1997). In this paradigm, a brief probe stimulus is presented around the time of a saccade, and participants are asked to report the position of the probe. The saccade is made between two stimuli, positioned several degrees of visual angle apart. Participants are asked to fixate the first stimulus, and to make a saccade to the second stimulus when it appears. At a variable Stimulus Onset Asynchrony (SOA) from the onset of the saccade cue, a probe stimulus is briefly presented in one of three positions: contralateral to the saccade target; beyond the saccade target; and at a position midway between the initial fixation point and the saccade target. On completion of the saccade, participants are asked to indicate the location at which the probe appeared. One version of the paradigm is illustrated in Figure 3-4.

Typically, participants make accurate judgments about the position of the probe as long as it appears substantially before, or substantially after, the onset of the saccade (Lappe et al., 2000; Ross et al., 1997; Ross et al., 2001). However, if the probe appears within 50 milliseconds of saccade onset, it tends to be mislocalised. Two patterns of mislocalization occur. In one pattern, the probe

tends to be mislocalised in the direction of the second fixation point. In other words, probes either contralateral to the saccade direction, or presented between the two fixation points, are mislocalised in the direction of the saccade. In contrast, probes presented beyond the second fixation point tend to be mislocalised against the direction of the saccade. This pattern of mislocalization has thus been termed "peri-saccadic spatial compression", as the perceived distance between probe position and saccade target is compressed relative to actuality Figure 3-2.

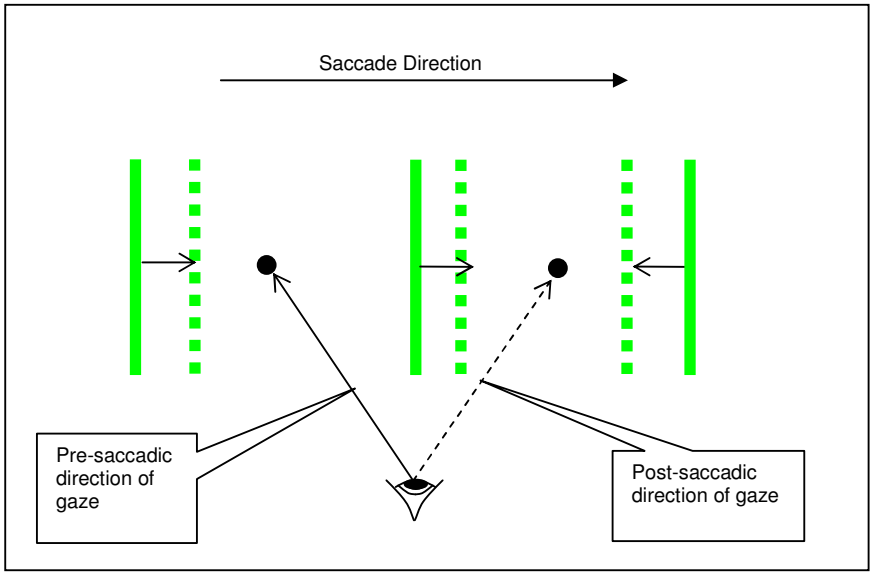

Figure 3-2: Compression of probe stimuli towards saccade target

This diagram illustrates the pattern of mislocalization referred to as "spatial compression". Actual probe positions are shown as solid green lines; apparent positions are shown as dotted green lines. All probes are mislocalized in the direction of the saccade target.

In a second pattern of mislocalization, probes are simply mislocalized in the direction of the saccade. Which of the two patterns predominates appears to depend at least partly on the availability of visual references immediately post-

saccade (Lappe et al., 2000). When no visual references are available on saccade completion, the "shift" pattern predominates: peri-saccadic mislocalization, even of probes presented beyond the saccade target, tend to be in the direction of the saccade.

Peri-saccadic mislocalization of briefly presented, abrupt onset stimuli is therefore a phenomenon found in unimpaired participants. It would thus appear to reflect peri-saccadic processes subserving normal vision. One possibility is that it may, paradoxically, implicate processes implicated in maintaining spatial constancy across saccades: it is likely to prove advantageous for the visual system to make a predictive working model of the way the world will look on completion of a saccade, requiring only minimal confirmatory re-calibration on saccade completion (Findlay and Gilchrist, 2003).

As part of this investigation, a computation model of pre-saccadic mislocalization phenomena was developed, an account of which is given in Appendix A, which assumes that, brief, salient, sudden onset stimuli, rare in the natural world, evade the re-calibration process and are thus erroneously re-mapped. Briefly, the model postulates that both predictive and post-dictive processes give rise to the phenomenon. A saccade map, as proposed by Godijn and Theeuwes (2002) is implicated in the predictive process. Probes presented close to saccade target, and ipsilateral to the direction of the saccade, are postulated to exert a "centre-of-gravity" effect on the saccade map, as in Godijn and Theeuwes' model, briefly representing a potential saccade to a location

somewhere between the saccade target and the probe. This is postulated to result in an underestimate of the retinotopic vector that the probe will have on saccade completion. When, post-saccade, this Predicted Retinotopic Vector (PRV) is remapped on to the re-calibrated spatiotopic co-ordinates of the intended target, the probe is mislocalized in the direction of the target thus giving rise to the pattern of mislocalization referred to as "spatial compression" (Figure 3-3). It was also postulated that additional mislocalization of probes in the direction of the saccade, including those presented contralaterally to the saccade direction, would occur as a result of anticipatory estimates as to the eye position at the time of probe onset, and giving rise to the pattern of peri-saccadic mislocalization referred to as "shift" .

Given Crawford and Higham's (2001) finding of an attenuated centre-of-gravity effect in dyslexic participants, and Godijn and Theeuwes' (2002) proposed model to account for the effect, if the model developed here to account for peri-saccadic spatial compression is correct, dyslexic participants should also show attenuated spatial compression effects. However, no difference would be predicted between groups regarding peri-saccadic "shift".

These predictions were tested in an experiment (Experiment 2a) in which the performance of dyslexic participants was compared that of non-dyslexic control participants on a task designed to elicit peri-saccadic spatial compression, In a second experiment (Experiment 2b), in order to ascertain whether atypical peri-saccadic mislocalization arose from the same deficits as those underlying the

atypical performance of dyslexic participants on the TOJ task, the performance of dyslexic participants was not only compared with that of non-dyslexic participants, but the relationship between performance on a peri-saccadic mislocalization paradigm and performance on the TOJ task was investigated.

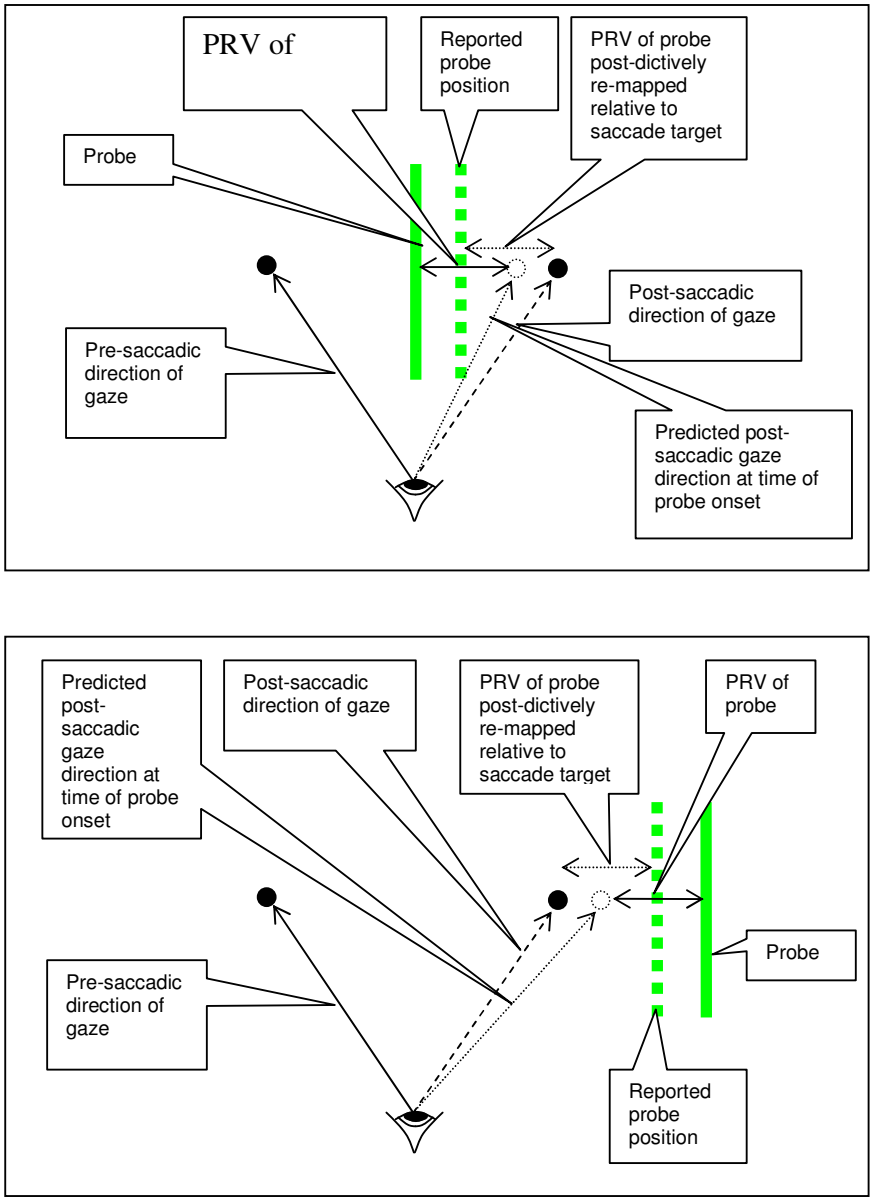

Figure 3-3: Schematic representation of spatial compression model

The upper diagram represents a probe presented fixation and saccade target; the lower diagram represents a probe presented beyond fixation. In both cases, the predicted post-saccadic gaze direction of is deviated briefly in the direction of the probe, leading to an underestimate of the Predicted Retinotopic Vector (PRV) of the probe relative to the intended saccade target. When these underestimated vectors are remapped relative to a spatio-topic representation of the saccade target, recalibrated in light of post-saccadic visual references, the probe is recalled as having appeared closer to the saccade target than it was in fact presented. Thus the space between target and probe is apparently "compressed".

It was also hypothesized that atypical performance on tasks tapping overt and covert spatial attention in dyslexic participants might be due to co-morbid symptoms of an Attentional Deficit Disorder (ADD) such as Attentional Deficit and Hyperactivity Disorder (AHDH). In the second experiment, therefore, measures of symptoms of ADD, using the Brown ADD Scale (Brown, 1996) were made, in order to determine the extent to which atypical performance on either paradigm could be attributed to a co-morbid ADD.

3.2 Experiment 2a

3.2.1 Method:

3.2.1.1 Participants:

A total of forty nine students at the University of Nottingham participated in the experiment, recruited in two batches. In the first batch, twenty-three participants were recruited, eleven by means of an advertisement requesting participants who had been diagnosed with dyslexia, and twelve were recruited by a general advertisement. In the second batch, a further twenty six participants were recruited, twelve by means of an advertisement requesting participants who had

been diagnosed with dyslexia, and fourteen by a general advertisement. The first batch undertook a version of spatial compression paradigm in which they were required to make a saccade from left to right. The second batch performed the same task, but in a version in which they were required to make a saccade from right to left.

All participants were screened for dyslexia using the DAST (Fawcett and Nicolson, 1998). A cut off score of .07 was used to confirm whether participants were, or were not, likely to suffer from dyslexia, and any control participant scoring .07 or greater was excluded from the study.

3.2.1.2 Stimuli:

The experimental stimuli were programmed in E prime[14] software, and presented on a 14 inch computer monitor with a screen refresh rate of 16.6ms. Stimuli were presented on a grey ground. Each trial began with a fixation point (a black circle subtending 1°) located either 5° to the left (for the left to right version) or to the right (for the right-to-left version) of the vertical midline of the screen. After 1104 ms, this fixation point offset, and was replaced by a second identical target stimulus 5° to the right (for the left-to-right version) or to the left (for the right-to-left version) of the vertical midline of the screen. At a randomly varied stimulus onset asynchrony (SOA)[15] after the onset of the saccade target, a bright green vertical bar appeared in one of the following three positions: 10° to the left

[14] E Prime 1.1.4.1, Psychology Software Tools, Inc. Pittsburgh PA

[15] with a quasi-Gaussian distribution: mean = 174ms; standard deviation = 66ms

of the midline; at the midline; and 10° to the right of the midline. This bar remained on screen for 17ms (one screen refresh rate), and was immediately replaced by a horizontal ruler, marked in multiples of 5° degrees. The midline position was marked "0", positions to the left being marked as negative (-5°, -10° and -15°) and positions to the right were marked as positive (5°, 10° and 15°). The three probe locations were randomized. Participants were asked to fixate on the first fixation point, and to make a saccade to the target as soon as it appeared. They were requested to try not to fixate the bar, but to note its position, and when the ruler appeared, to give an estimate of the probe's position relative to the ruler. The experimenter notated their verbal responses, and the next trial was initiated by the participant, using the spacebar of a the computer keyboard. A schematic representation of the experimental stimuli is given in Figure 3-4.

Eye movements were recorded using Biopac[16] electro-oculography (EOG) eye tracking equipment. Two electrodes were taped to the skin of the temples, over the lateral rectus muscle (which produces lateral movement of the eye), and a ground electrode was taped above the nose. Voltage differences between the electrodes were sampled at 200 Hz, and data were acquired using Acknowledge[16] software. Separate EOG recordings lasting 3.5 seconds each were made of each trial, and were triggered at the onset of the first fixation point.

---

[16] Biopac Systems inc., Goleta, CA

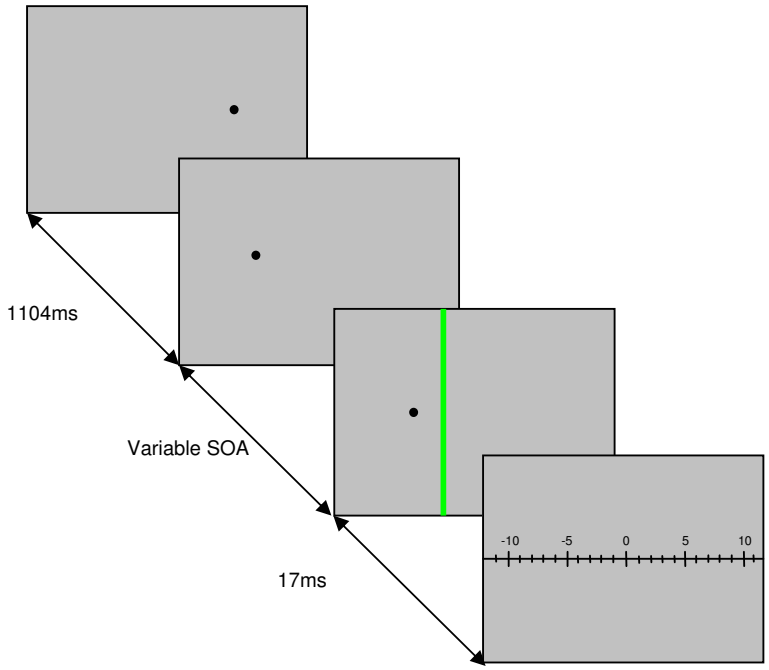

Figure 3-4: Stimuli for spatial compression task

In each trial, a fixation stimulus of 1104ms duration is presented, after which a saccade target (2nd frame) is presented. The participant is asked to make a saccade to the target. After a varying SOA, a brief green probe stimulus (3rd frame) is presented for 17ms, immediately followed by a ruler stimulus. Participants are asked to report the observed position of the probe relative to the ruler (4th frame). The ruler remains on screen until the participant's response is recorded by the experimenter. The participant then initiates the next trial with a by pressing the space bar of the computer keyboard.

3.2.2 Analysis and results

3.2.2.1 Analysis

EOG data: For the first batch of subjects, saccade onsets were determined by visual inspection of the EOG voltage samples, graphed in Acqknowledge[16] software. For the second batch of subjects, saccade detection was partially automated using an algorithm written in LabView[17] software. The algorithm took a moving window of eight consecutive voltage samples, and compared the mean of the first four samples with the mean of the second. When the difference between the two means exceeded a threshold value, ac possible saccade was deemed to have been initiated; visual inspection of the plot was used to distinguish between saccades and artefacts. Thresholds were set for each subject by trial and error, using visual inspection of a few sample plots.

All trials in which no saccade could be discerned were discarded, as were trials in which no response was made. The time interval in milliseconds between probe onset and saccade onset was computed for each trial. The apparent positions of each probe were coded relative to the direction of the saccade. "Pro-saccadic" mislocalizations of the probe (in the direction of the saccade) were coded with a positive value, and "anti-saccadic" mislocalizations (against the direction of the saccade) were coded with a negative value. Thus, a probe

---

[17] Labview 7.1; National Instruments Corporation, Austin, TX. The algorithm was programmed in LabView by the author.

perceived to be to the left of its veridical position was coded with a negative value for left-to-right saccades and with a positive value for right-to-left saccades, while a probe perceived to be to the right of its veridical position was coded with a positive value for left-to-right saccades and with a negative value for right-to-left saccades.

For each participant, valid trials (those in which a response was made and a saccade was detected) were allocated to one of two time "bins". The first bin consisted of trials in which the saccade was initiated within 50 ms of the onset of the probe; these were designated "Peri-saccadic" trials. The second consisted of trials in which the saccade was made 200ms before or after probe onset; these were designated "Fixation" trials. For each subject, the mean reported position of the probe was computed for each real position (central; 10º to the right of the midline; or 10º to the left of the midline) for each trial type (peri-saccadic and fixation). Then, for each trial type, indices of "shift" and "compression" were computed.

Measures of "shift" and "compression" were derived from those proposed by Lappe and colleagues (Lappe et al., 2000). "Shift" is defined as the tendency to mislocalize all probe types in the direction of the saccade, regardless of their position relative to the saccade target. One way of quantifying "shift" therefore, is to compute the mean apparent target position, coded relative to saccade direction, for each time bin of interest. If there is no "shift", the mean of the probe positions will tend to equal zero, the position of the central probe. As the

saccade direction is coded with a positive value, a mean of greater than zero will indicate a net tendency to mislocalize the probe in the direction of the saccade; a mean of less than zero would indicate a tendency to mislocalize the probes in the contrary direction to the saccade. "Compression", on the other hand, is defined as a tendency to mislocalize the probe in the direction of the target. If there is no compression, the standard deviation of the reported target positions will be 10°, the distance between the central probe and the two outer probes. However, if the apparent space between the probes is compressed, the standard deviation will be less than 10°. "Shift" for each time bin was therefore measured as the mean of the reported probe positions, and "compression" for each time bin as the standard deviation of the reported probe positions.

A 2x2x2 repeated-measures ANOVA was then conducted on both the "shift" and the "compression" measure, with two between-subjects factors and one within-subjects factor. "Time-bin" was the within-subjects factor, with two levels, "Peri-saccadic" and "Fixation". Between-subjects factors were "diagnostic group" (two levels: dyslexic and non-dyslexic) and "direction group" (two levels: left-to-right and right-to-left).

3.2.2.2 Results

In the case of two participants, no responses were recorded for any trial in which their saccade was initiated more than 200 before or after the onset of the probe. Both had performed the right-to-left paradigm; one was a dyslexic participant and one was non-dyslexic. These participants were therefore deleted from this

part of the analysis, leaving 11 dyslexic participants in each condition, 12 non-dyslexic participants in the left-to-right group, and 13 non-dyslexic participants in the right-to-left group.

For the dependent variable "compression", the interaction between "diagnostic group" and "bin" was significant [$F(1,43)=4.500, p<0.05$], indicating that the non-dyslexic participants displayed a greater increase in compression during the peri-saccadic trials than the non-dyslexic participants (Figure 3-5). The two diagnostic groups were then analyzed separately in order to ascertain whether a main effect of time bin was found in either group. Both groups showed a significant main effect of time bin, indicating that both the dyslexic [$F(1,20)=22,892$, $p<0.001$] and the non-dyslexic [$F(1,23=46.832$, $p<0.001$] participants showed significantly more compression on peri-saccadic than on non-peri-saccadic trials. In neither diagnostic group was saccade direction a significant predictor of compression, nor did it interact significantly with time bin.

There was also a significant interaction between "direction group" and "bin" [$F(1,43)=4.800$, $p<0.05$], which indicated that increase in compression in peri-saccadic trials was greater in the right-to-left group than in the left-to-right condition. There was no significant 3-way interaction between group, direction and time bin.

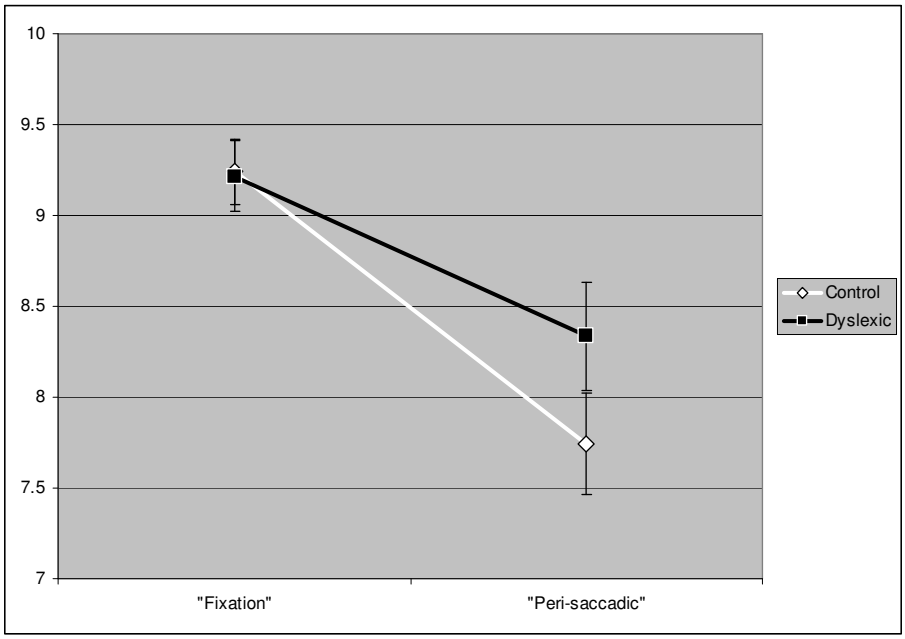

Figure 3-5: Spatial compression in dyslexic and non-dyslexic participants

"Compression" is indexed by the standard deviation of the three apparent positions, which is shown on the vertical axis. The smaller the standard deviation the greater the degree of apparent spatial compression. While both groups of subjects showed significantly greater compression (smaller standard deviation) on peri-saccadic than on fixation trials, the difference was significantly greater in the non-dyslexic group than the dyslexic group. (Error bars = standard error).

For the dependent variable "shift", the interaction between "direction" and "bin" was significant [$F=(1,43)=13.101$, $p<0.001$] and indicated a greater increase in shift on peri-saccadic trials in the right-to-left condition than in the left-to-right condition. There was no interaction or main effect of diagnostic group. A simple effects analysis conducted on each direction condition indicated that for the left-to-right group, shift was not significantly greater in the peri-saccadic condition than in the fixation condition, whereas in the right-to-left group, a significant main effect of time bin indicated that participants showed greater mean shift on

peri-saccadic than on fixation trials [$F(1,22)=21.919$, $p<0.001$]. Again, there was no main effect of diagnostic group, nor interaction between diagnostic group and any other group. Means and standard deviations are given in Table 3-1

Measure	Bin	Directional Group	Control Mean(St.dev)	Dyslexic Mean(St.dev)
Shift	Perisaccadic	left to right	0.04(0.62)	0.05(0.54)
		right to left	0.87(0.84)	1.01(0.82)
	Fixation	left to right	0.09(0.46)	0.05(0.48)
		right to left	0.65(0.71)	0.77(0.6)
Compression	Perisaccadic	left to right	7.84(1.51)	9(1.2)
		right to left	7.64(1.2)	7.67(1.68)
	Fixation	left to right	9.19(0.79)	9.4(0.83)
		right to left	9.29(0.9)	9.04(1.09)

Table 3-1

Means and standard deviations for "Shift" and "Compression" for each group, for each time bin. For "Shift", a positive value indicates mislocalization in the direction of the saccade, where a value of 1 would indicate 1° of visual angle. For "compression" a value of 10 would indicate no compression; lower values indicate increased compression.

3.3 Discussion

Although both dyslexic and non-dyslexic participants showed significantly greater spatial compression on peri-saccadic trials as compared with fixation trials, the dyslexic participants, as predicted, showed significantly less increase in compression on peri-saccadic trials, relative to fixation trials, than did the non-dyslexic participants. Interpreted in terms of the model of peri-saccadic compression reported in Appendix A, this is consistent with the finding of Crawford and Higham (2001) of attenuated centre-of-gravity effects in dyslexic

participants, and is also consistent with the hypothesis that peripheral stimuli are less clearly specified in terms of potential saccade targets in dyslexic than in non-dyslexic participants, resulting in reduced distortion to the estimate of the planned saccade by probes presented during the peri-saccadic period.

There was no difference between diagnostic groups in terms of the degree of "shift" observed. The model given in Appendix A proposes that "shift" effects are more dominant when post-saccadic re-calibration is more difficult or impossible. The finding of no significant difference between dyslexic and non-dyslexic participants in the degree of peri-saccadic "shift" they showed on the task, may suggests that dyslexic participants do not differ from controls in the efficiency of the post-saccadic recalibration process.

However, participants who participated in the "right-to-left" version of the paradigm, whether dyslexic or non-dyslexic, showed both greater peri-saccadic "shift" of probe locations in the direction of the saccade than those who undertook the left-to-right version of the paradigm. As this was a between-subjects comparison, interpretation needs to be made with caution; however, one possible explanation of this effect may be that as readers of English, all participants in the experiment were more practiced at making left-to right saccades. It is possible, therefore, that parietal neurons postulated to shift their receptive fields in anticipation of a saccade (Colby et al., 1996) may be less well tuned to right-to-left saccades, resulting in greater saccade-wise error when saccades are made in this direction.

It may be worth noting at this point is that the model postulates that peri-saccadic mislocalization arises when probes are both abrupt in onset, and brief in duration, and reflects the operation of a mechanism in the visual system postulated to make a model of the way the world will look after a saccade is completed that requires only minimal recalibration post-saccade (Findlay and Gilchrist, 2003). While the mechanism is postulated to result in the mislocalization of a kind of stimulus that is rare in the natural world (brief, salient, abrupt onset stimuli), this would be a small price to pay for a system that facilitates the maintenance of spatial constancy across saccades. If this system is deficient, or noisy, in dyslexia, possibly because salient locations are not well specified on the postulated saccade map, spatial constancy across saccades may be compromised, resulting in visual confusion during a task such as reading, in which the relative spatial position of fine-grain visual stimuli is crucial to word recognition, and indeed to perceiving the correct order of words on the page.

Such a deficit might also be expected to lead to a deficit in performance on the visual TOJ task, a task that requires covert, rather than overt, visual attention to salient peripheral stimuli, and to the temporal order in which they occur. Again, if the proposed model is correct, the peripheral stimuli employed on the task will be represented on the saccade map by an increase in activation corresponding to the location of the first stimulus, and thus a potential saccade to that location, followed by an increase in activation corresponding to the location of the second stimulus, and thus a potential saccade to the second location. Weak or noisy patterns of activation on the saccade map would therefore be predicted to lead to

poor performance on the task. Thus, the finding of attenuated peri-saccadic "compression" effects in dyslexic participants found in Experiment 2a suggests that weak or diffuse representations of salient stimuli, particularly those at high eccentricity, may be the common cause of attenuated peri-saccadic spatial compression effects and impaired TOJ task performance. This, again, would be consistent with the hypothesis that a deficit in sensitivity to rapid rise-time visual stimuli may be a counterpart to the deficits in rapid rise time auditory stimuli found to be associated with reading deficits by Goswami and colleagues (Goswami et al., 2002; Richardson et al., 2004).

It was therefore hypothesized that the atypical patterns of peri-saccadic spatial compression found in dyslexic participants in Experiment 2a would be associated with impaired performance on the TOJ task, and also with longer saccade latencies. This hypothesis was tested in a second experiment (Experiment 2b) in which dyslexic, non-dyslexic, and borderline dyslexic participants performed both a spatial compression task and a TOJ task.

3.4 Experiment 2b

Given the apparent dissociation, reported in Chapter 2, between deficits associated with temporal order inaccuracy in general, bias to left hemifield, and bias to right hemifield, the TOJ task was modified in order to ensure that lateralized effects were not confounded with motor response bias. This was done by differentiating the first and second stimuli by randomly assigning a different shape (a circle or a triangle) to each, and asking participants to report the shape

of the first stimulus rather than its position. Thus on approximately half the trials, the stimulus presented in left hemifield would require the response: "circle", while on the remaining trials, the left hemifield stimulus would require the response: "triangle". Any motor bias for one response (for example "circle") rather than the other would therefore be cancelled out across hemifield, allowing any residual lateral bias to be attributed to a perceptual rather than a motor bias.

The spatial compression paradigm was also modified. The theoretical model of spatial compression proposed, and reported in Appendix A includes a computational model of mislocalization of probe stimuli presented between the presentation of a saccade target and the execution of the saccade; while it might be expected that mislocalizations would also occur of probes immediately after the onset of a saccade, including the duration of the saccade itself, as found in studies by Ross and colleagues (Ross et al., 1997; Ross et al., 2001), precise modeling of such mislocalizations is very much more complex, as the angle subtended by the probe will also vary depending on eye-position at the time of probe onset; for this reason the model given in Appendix A only addresses pre-saccadic mislocalization. However, in Experiment 2a, a large number of anticipatory saccades by both dyslexic and non-dyslexic participants meant that there were insufficient trials in which the saccade occurred after probe onset for the analysis to be confined to these trials. In order to increase the number of trials in which the saccade onset would occur after probe presentation, in this experiment, the location of the saccade target was made unpredictable. This was done by presenting subjects with a central fixation point, and presenting saccade

cues randomly to either left or right of fixation. This was not only expected to reduce the number of anticipatory saccades, but would allow saccade latencies to be measured; it also allowed within-subjects comparisons to be made of leftwards versus rightwards saccades.

The experiment also offered the opportunity to investigate further the association between the direction of lateral bias exhibited by participants on the TOJ and impairments in non-word relative to irregular word reading. It was hypothesized that irregular word reading impairments reflected impaired networks implicated in word recognition, and thus would also be tend to be manifest as attenuated single word reading latencies, while non-word reading impairments, particularly those postulated to be due to visual confusion, might be manifest as accuracy deficits. A modified version of the single word reading task devised by Castles and Coltheart (1993) was therefore designed, in which regular, irregular and non-words were presented on a computer screen, so that reading latencies could be recorded by use of a voice key.

A number of studies have found atypical saccadic phenomena in Attention Deficit and Hyperactivity Disorder (ADHD). Feifel and colleagues found that adults with ADHD made more anticipatory saccades and more directional errors on an anti-saccade than adults without ADHD (Feifel et al., 2004), while Munoz and colleagues found that participants with ADHD, including both children and adults showed longer saccade and more variable saccade latencies on a pro-saccade task than unimpaired participants, and made more errors and had longer

saccade latencies on an anti-saccade task. Moreover, Berger and Posner (2000) have postulated that ADHD may be associated with deficits in two attentional networks, both of which are implicated in visual attention: an "executive network", postulated to govern the selection of relevant stimuli; and an "alerting" network, which they suggest is right-lateralized, postulated to modulate arousal levels, and activated in response to abrupt onset stimuli. In support of the latter hypothesis, Rothlind and colleagues (Rothlind et al., 1991) found that saccade latencies in children with ADHD failed to show a left-hemifield advantage under conditions in which the saccade target was not preceded by warning cue, an asymmetry found non-impaired children and adults.

There is substantial evidence that diagnoses of dyslexia have a high overlap with diagnoses of ADHD, although it remains unclear as to whether the overlap represents true co-morbidity, or alternatively, different dimensions of a common underlying pathology (Friedman et al., 2003; Kaplan et al., 2001; Shaywitz et al., 1995). It was therefore hypothesized that co-existing ADHD may account for some of the abnormalities of visual attention observed in dyslexic participants Experiments 1a, 1b, 1c and 2a. In particular, given the postulated right-lateralization of the "alerting" network, it was hypothesized that deficits in such a network might be reflected in a bias against left hemifield stimuli. Participants in the study were therefore screened using the Brown Attentional Deficit Disorder Scale for Adolescents and Adults (Brown's ADD scale) (Brown, 1996), and symptoms scores recorded.

3.4.1 Method

3.4.1.1 Participants

Fifty two participants were recruited by means of an advertisement seeking both dyslexic and non-dyslexic volunteers. All participants were screened for dyslexia using the Dyslexia Adult Screening Test (DAST)[18] (Fawcett and Nicolson, 1998) and the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 1999) to ensure that each participant's IQ was in the normal range (greater than 85), and all participants completed the Brown ADD Scales questionnaire for adolescents and adults (Brown, 1996).

3.4.1.2 Tasks

Single word reading

A computerized single word reading task was devised, based on the list of regular, irregular and non-words produced by Castles and Coltheart (Castles and Coltheart, 1993). Thirty regular English words, thirty irregular English words and thirty non-words were presented on a 14 inch computer monitor using E Prime[14] presentation software. Stimuli were presented in a fixed random order and are given in Appendix B. Regular and irregular words were matched for length, frequency, and number of syllables using the MRC psycholinguistic database (University of Western Australia: School of Psychology, 2003); non-

[18] The Non-Verbal Reasoning subtest was not included; as all participants scored in the normal range on the WASI, ARQ on the DAST was computed assuming a normal score this subtest.

words were matched for length, and also for number of syllables, which ranged from one to three. Words were presented in black, lower case 28 point Courier font in the centre of a white screen, preceded by a fixation cross (+) in the same font at centre screen. Participants were told to read the word aloud when it appeared on screen. They were also told that some words would be "straightforward", some would be "tricky" and some would "not be real words at all", but that they should pronounce all words as best they could. Seven practice items were given before the beginning of the task. Accuracy was recorded by the experimenter after each trial by means of a mouse, and word latencies were recorded using a voice key linked to a microphone placed a few inches away from the participant.

Temporal Order Judgement Task

Stimuli for this task were programmed in E Prime[14] presentation software and presented on a 14 inch computer monitor with a refresh rate of 16.67ms on a grey ground. Each trial began with a blank grey screen; after 600ms a black central fixation cross subtending 1º appeared and remained on screen for the remainder of the trial. After 1000ms a white, black-bordered, shape, either a circle or a triangle, appeared either to the right or to the left of fixation. After a variable stimulus onset asynchrony (SOA), a second shape (a circle if the first shape had been a triangle, and vice versa) appeared in the opposite hemifield. The maximum dimension of each shape subtended 5.5º and was centred 10º either to the right or left of the midline of the screen. Participants were asked to make a

forced choice as to whether the circle or the triangle had appeared on screen first, and the stimuli remained on screen until the response was made.

Participants used both index fingers (on the 'v' and 'n' keys of a qwerty keyboard) to indicate "triangle", and both middle fingers (on the 'c' and 'm' keys) to indicate a circle. To aid stimulus mapping, raised circle and triangle shapes were affixed to the appropriate keys, and, as a further *aide memoire,* participants were invited to consider that the shape made by their thumbs and index fingers formed a "triangle shape". A block of 10 practice trials were given and the participant had the option of repeating the practice block as many times as they felt they needed. SOAs ranged from zero to 116ms in multiples of 16.67ms (one screen refresh rate) with an additional baseline SOA of 331 milliseconds.

Spatial compression.

Stimuli for this task were programmed in E Prime[14] presentation software (E Prime 1.1, Psychology Software Tools, and were back-projected from a data projector with a refresh rate of 12ms onto a translucent screen[19]. Each participant was seated at a table in front of the screen; a fixed distance of 57cm was maintained between the participant and the display by means of a chin rest. Stimuli were presented on a grey ground. At the beginning of each trial a black fixation point (a black circle subtending 0.5°) was presented at the midline of the

[19] Back projected screen presentation was chosen so as to enable stimuli to subtend the substantial angles to either side of the midline required to accommodate the bi-directional paradigm.

screen for 100ms. Simultaneous with the offset of the fixation point, a saccade target (a second black circle, also subtending 0.5°) was presented randomly either 10° to the left or 10° to the right of the midline for 200 milliseconds. This was immediately followed by the presentation of a horizontal ruler, marked off from right to left in fives from 5 to 25, the 15 mark being at the midline of the screen, and each unit representing 2° of visual angle.

On 86% of trials a probe stimulus consisting of a bright green vertical bar, subtending 2° horizontally and extending from the top to the bottom of the screen was presented at randomly varied Stimulus Onset Asynchronies (SOAs) consisting of multiples of a single screen refresh rate (12ms) after the onset of the saccade target and before the onset of the ruler. The probes were presented in three positions relative to the direction of the saccade: beyond the saccade target ("Beyond"); between the central fixation point and the saccade target ("Between"); and contralateral to the saccade ("Behind") (Figure 3-6).

For each trial type ("Beyond", "Between" and "Behind"), the precise position of the probe was randomly "jittered" between one of two positions: for "Beyond" trials, the probe was presented either 4° or 6° beyond the saccade target (in other words, between 14° and 16° from fixation), while for "Between" and "Behind" trials, the probe was presented 4° or 6° to left or right of fixation. Relative to the

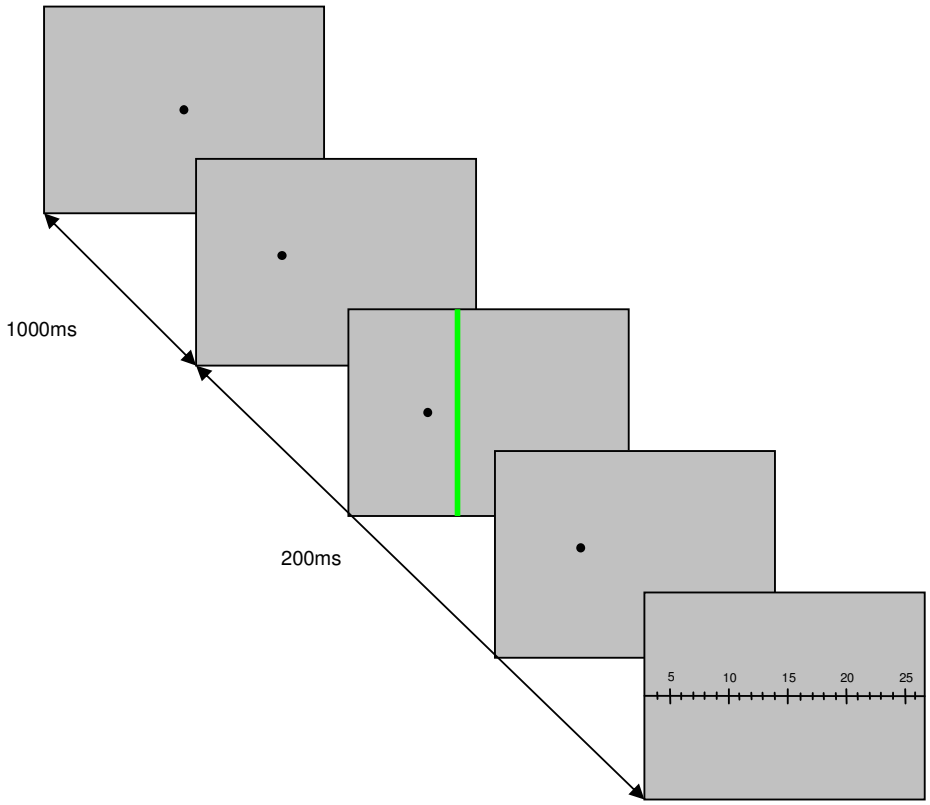

Figure 3-6: Stimuli for spatial compression task

In each trial, following a fixation stimulus of 1000ms duration, a saccade cue (second frame) is presented for 200 ms. During this interval, a brief green probe stimulus (third frame) is presented at varying positions and at varying SOAs from the onset of the saccade cue. (In this illustration, the "Between" condition is portrayed, in which the probe is presented between fixation and saccade target.) Participants are asked to report the observed position of the probe relative to the ruler (5^{th} frame). The ruler remains on screen until the participant's response is recorded by the experimenter. The participant then initiates the next trial with a mouse click. A proportion of trials were "catch" trials in which no probe was presented.

ruler, therefore, probes were presented at positions 6, 7, 12, 13, 17, 18, 22 and

23, and participants were asked to report the position of the probe relative to the ruler.

On the remaining 14% of trials ("catch trials") no bar was presented, in order to discourage guessing. Participants were informed that on some trials there would be no bar. A total of 336 trials were presented in four blocks of 84 trials, and rest periods were provided between each block. Two practice blocks preceded the experiment proper. In the first, no probes were presented and the participants simply practiced making saccades to the target stimulus; this block was repeated until the participant felt confident about making the saccades. In the second practice block the trials were as for the experiment proper; probes were presented and the participant was asked to report the position of the probe; again this practice block was repeated if the participant was not confident of the procedure. Each trial was initiated by a mouse-click by the participant. Rest periods were provided between each block, and participants were also permitted to take extra rest periods as required. Eye movements were recorded as for Experiment 3.1 using Biopac[20] electro-oculography (EOG) eye tracking equipment and AcqKnowledge[20] software.

[20] Biopac Systems Inc., Goleta, CA

3.4.2 Analysis and results

One participant was excluded as her full-scale IQ (FSIQ) was 75; a second participant was excluded as although his ARQ on the DAST was borderline (=0.07) his Performance IQ (PIQ) was 29 points lower than his Verbal IQ (VIQ) and below the normal range (84) suggesting an atypical cause for his literacy difficulties; he subsequently reported having had a serious head injury as a child. This left 50 participants in the study: 36 had been recruited as "dyslexic", 24 of whom were female (66%); 15 had been recruited as "control" participants, 11 of whom were female (27%). Seven participants in the dyslexic group (20%) were left handed, as were two participants in the control group (13%). The mean age of the dyslexic group was 30.7 years (sd=12.5) and that of the control group was 29.0 years (sd-11.3). The mean ages of the two groups were not significantly different.

3.4.2.1 Assessment measures

The mean full scale IQ (FSIQ) scores on the WASI were significantly higher [$t(47)=3.320$, $p<0.01$) in the control group (mean=125, sd= 9) than in the dyslexic group (mean= 115, sd=10) , as were Verbal IQ (VIQ) scores [$t(47)=2.384$, $p<0.05$] and Performance IQ (PIQ) scores [$t(47)=2.413$, $p<0.05$]; however the groups were not significant on either the Similarities sub-test nor on Block Design. On the DAST, At Risk Quotient (ARQ) scores in the dyslexic

group ranged from 0.3 to 2.2, and in the control group from zero to 0.9[21]. Six participants from dyslexic group scored an ARQ of less than 0.7, although all had an educational psychologist's diagnosis of dyslexia; it appears therefore that they had subsequently been remediated to the extent of screening negative for dyslexia on the DAST. One participant from the control group had an ARQ of 0.9, indicating possible dyslexia. Non-parametric tests (Mann Whitney U) indicated that the diagnostic groups were significantly different each other on all the sub-tests of the DAST apart from Verbal and Semantic fluency. Mann Whitney U tests also indicated that the dyslexic group scored significantly higher the Brown's ADD scale, on all symptom clusters. These results are given in Table 3-2.

Nineteen dyslexic participants scored above the screen-positive threshold of 55 for on the Brown's ADD scale, and sixteen participants scored below the threshold. No control participants scored above the screen-positive threshold, and there was no significant difference between the control group and the sub-ADD threshold dyslexic groups on ADD symptom scores, on either a non-parametric (Mann-Whitney U) or a parametric test (t-test).

[21] An ARQ of 0.7 or greater is deemed to indicating that the participant is mildly At Risk of dyslexia; a ARQ of 1.0 or greater is deemed to indicate that the participant is strongly At Risk (Fawcett AJ, Nicolson RI. The Dyslexia Adult Screening Test (DAST). London: The Psychological Corporation, 1998.).

Instrument	Item	Mann-Whitney U	Asymp. Sig. (2-tailed)
DAST	ARQ	17	0.000
	Rapid Naming	95.5	0.000
	1 Minute Reading	74	0.000
	Postural Stability	151	0.017
	Phonemic Segmentation	64	0.000
	2 Minute Spelling	39	0.000
	Backward Digit Span	103	0.001
	Nonsense Passage Reading	9	0.000
	1 Minute Writing	46.5	0.000
	Verbal Fluency	193	0.140
	Semantic Fluency	196.5	0.161
Brown's ADD scale	Activation	94	0.000
	Attention	65	0.000
	effort	85.5	0.000
	affect	137	0.008
	memory	58	0.000
	ADD score	59.5	0.000

Table 3-2: Non-parametric between-groups tests

Mann-Whitney U values for comparisons between diagnostic groups on the DAST and ADD Brown's scale scores and subtests. The participants from the dyslexic group ranked significantly more impaired than participants from the control group on all subtests and screening scores, except for the Verbal and Semantic fluency items from the DAST.

In subsequent analyses, therefore, as well as comparing dyslexic participants with control participants, dyslexic participants with sub-threshold ADD scores ("Dyslexia Only": DO) were also compared with those who screened positively for ADD symptoms ("Dyslexia + ADD": D+ADD). There were no significant differences between the DO group and the D+ADD group in FSIQ scores, nor on any WASI subtest.

However, the D+ADD group ranked significantly more poorly than the DO group in ARQ scores [Mann Whitney $U(16,19)=75.5$, $p<0.01$], indicating greater disability in the D+ADD group. Non-parametric tests on subtest items of the DAST indicated that the D+ADD group ranked significantly more poorly than the DO group on Two Minute Spelling [Mann Whitney $U(16,19) = 65.5$, $p<0.01$] and Nonsense Passage Reading [Mann Whitney $U(16,19) = 91$, $p<0.05$]. Interestingly the DO and D+ADD groups were not significantly different on Phonemic Segmentation or on Backward Digit Span. Median scores for each group on these measures are given in Table *3-3*.

DAST measure	Control (N=15)	DO (N=16)	D+ADD (N=19)
ARQ	0.1(0.3)	0.9(0.75)	1.3(0.6)
Two Minute Spelling	37(6)	30(7)	24(9)
Nonsense Passage Reading	95(4)	81(14)	72(10)
Backward Digit Span	9(4)	5(2)	5(4)
Phonemic Segmentation	15(0)	13(5)	12(4)

Table 3-3: Measures of reading impairment in the three groups

This table shows the median (interquartile range) for five measures of risk for dyslexia disability from the DAST: the At Risk Quotient (ARQ), and scores on four subtests. The D+ADD group ranked significantly more at risk than the DO group on Two minute spelling and Nonsense Passage Reading, but not Phonemic Segmentation or Backward Digit span.

3.4.2.2 Single word reading

Accuracy scores were computed for each of the three word types (regular, irregular and non-words) for each of the three word lengths. Median reading latencies for all correctly read words were also computed for each word type and

length (one, two and three syllables)

Because many participants scored at ceiling on accuracy, non-parametric tests were used to compare participants' performance. Non-parametric tests indicated that the dyslexic group read significantly less accurately than the control group on all three word types [Mann Whitney U(15,35) for regular words = 135, $p<0.01$; for irregular words = 136, $p<0.01$; for non-words=28, $p<0.001$]. When the DO group was compared with the D+ADD group, there was no significant difference between real word reading accuracy in the two groups. However, the D+ADD participants ranked significantly less accurate than the DO group when reading non-words [Mann Whitney U(16,19) = 73, $p<0.01$].

Median[22] reading latencies were similarly computed for the three word types and lengths for all words read correctly. Four participants failed to read any three-syllable non-word correctly; they were therefore excluded from analyses of latencies for this word type. Median latencies were positively skewed across the sample, and so natural logs of the median were computed; this produced distributions that were not significantly[23] different from normal. T tests were then used to compare mean latencies for each group. Even at a strictly adjusted alpha level for multiple t tests[24], the mean dyslexic group word reading latencies

---

[22] Because latencies do not have a normal distribution, being bounded at the lower extreme, median values were taken as a measure of central tendency.

[23] Shapiro Wilks test: $p>.05$

[24] For 8 tests, dividing 0.05 by the number of tests gives an alpha of 0.006. This is conservative as it does not allow for the any correlation between latencies on each trial type.

were found to be significantly longer for each word type and length than in the control group (Table 3-4). Mean latencies are illustrated in Figure 3-7

Word type	Number of syllables	t(47)	Sig. (2-tailed) Alpha=0.006
Regular words	1	3.608	0.001
	2	3.391	0.001
	3	4.358	0.000
Irregular words	1	3.056	0.004
	2	3.653	0.001
	3	3.547	0.001
Non-words	1	3.975	0.000
	2	5.426	0.000

Table 3-4:

T values for between-groups comparison of single word reading latencies. Even after a stringent Bonferroni correction for multiple comparisons, dyslexic participants had longer single word reading latencies for all word types and lengths.

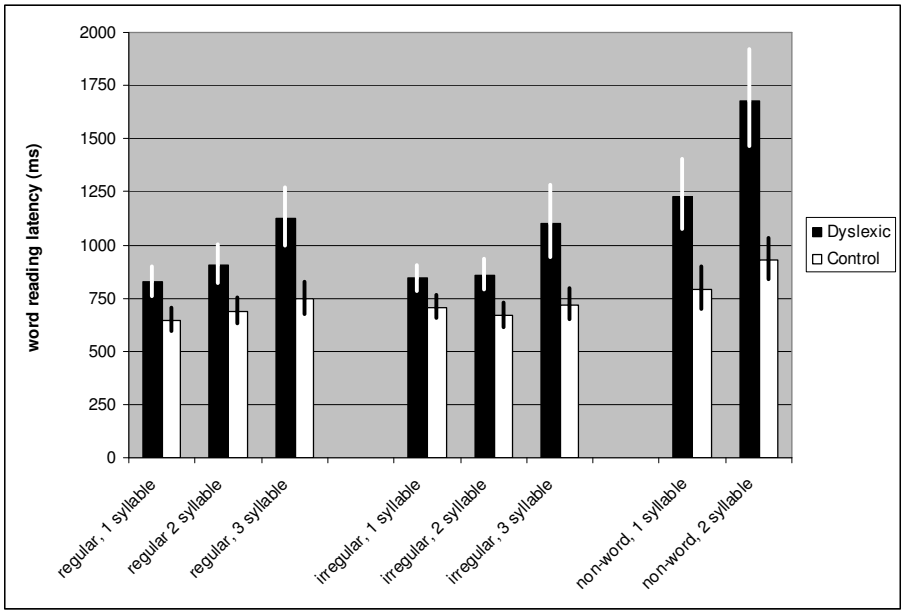

Figure 3-7: Mean latencies for single word reading

Dyslexic participants read single words significantly more slowly than control participants, regardless of word type and length.

Word reading latencies were analyzed with a 3 x 2 x 2 ANOVA, with two within-subjects factors: three levels of word type and two levels of word length (one and two syllables), and one between-subjects factor: diagnostic group. There was no significant three way interaction but the interaction between word type and length was significant [$F(1.62,77.54)^{25}=28.468$, $p<0.001$] (Figure 3-8: Mean word reading latencies for 1 and 2 syllable words.

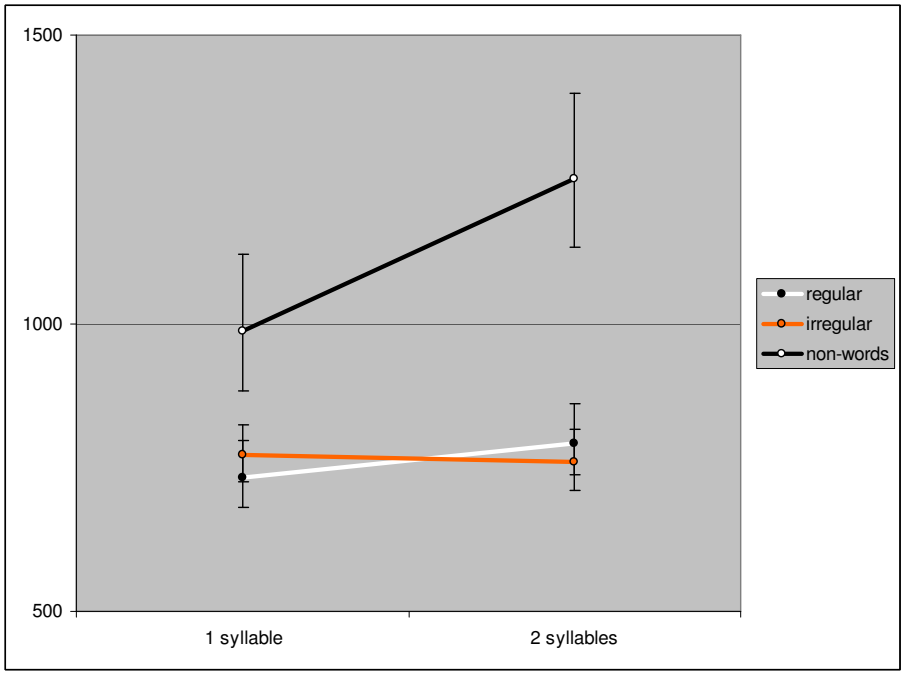

Figure 3-8: Mean word reading latencies for 1 and 2 syllable words

These latencies are pooled across both dyslexic and non-dyslexic participants. The cost of length was significantly greater for non-words than for real words, and significantly greater for regular than for irregular words. These findings remained robust when data from each group was analyzed separately.

[25] After Greenhouse-Geisser correction for violation of sphericity.

Orthogonal contrasts were therefore performed: length costs for non-words were compared with those for real words, and those for irregular words with those for regular words. Both contrasts were significant. The cost of length was significantly greater for non-words than for real words[$F(1,48)=34.091$, $p<0.001$]; and for regular words than for irregular words [$F(1,48)=14.056$)]. These results are shown graphically in Figure 3-8: Mean word reading latencies for 1 and 2 syllable words. When data from each group was analyzed separately, the effects remained significant.

There was a significant interaction between diagnostic group and word type [$F(1.18, 56.72)^{25} =15.822$, $p<0.001$]. A contrast between real words and non-words by group indicated that in dyslexic group the cost of non-lexicality was significantly greater than the control group [$F(1,48)=16.925$, $p<0.001$]. The cost of irregularity was not significantly greater in the dyslexic group than in the control group.

There was also a significant interaction between group and word length [$F(1, 48)=12.671$, $p<0.001$], indicating that in the dyslexic group the cost of length was significantly greater in than in the non-dyslexic group. When the DO group was compared with the D+ADD group, there were no significant differences in reading latencies between the groups.

3.4.2.3 Interim summary and discussion

These results indicate that the participants recruited as “dyslexic” differed from

the control group both on measures of reading and on the degree to which they reported symptoms of ADD (Table 3-5). Although the two groups also differed in mean FSIQ scores, there was no difference between the groups on the Similarities subtest. As scores on Vocabulary have been found to be depressed in reading disabled subjects (Wechsler, 1999), whilst Similarities scores are preserved, this may indicate that underlying verbal intelligence was not different in the two groups, and that their lower scores on reading were likely to be due to a specific reading disorder. The D+ADD group appeared to have more serious impairments than the DO group (Table 3-3).

	Control (N=15)	**Dyslexic (N=35)**	**Dyslexic (N=35)**	
			DO (N=16)	**D+ADD (19)**
Activation	7(4)	14(6)	9(5)	18(4)
Attention	7(4)	16(6)	11(4)	20(4)
effort	5(5)	13(6)	7(4)	18(4)
affect	4(3)	8(5)	5(4)	11(4)
memory	4(3)	11(5)	8(4)	14(3)
add	27(15)	62(24)	40(14)	81(11)

Table 3-5

Mean Brown ADD scale scores. This table shows the mean (standard deviation) Brown ADD scale scores for the dyslexic and control groups, and for the dyslexic subgroups after those with scores above a threshold of 55 had been allocated to the "D+ADD" group, ,and those below had been allocated to the "DO" group.

3.4.2.4 TOJ performance

For the TOJ task data, accuracy scores were computed for trials in which the first stimulus appeared on the left, and for trials in which the first stimulus appeared

on the right, for each SOA, regardless of shape type (circle or triangle). One D+ADD participant was excluded from all further analyses of TOJ scores as his accuracy scores indicated that he had not correctly understood the task[26]. D prime scores and criterion scores were then computed, as for Experiments 1a, 1b, 1c. As all participants achieved a perfect score on trials in which the SOA was at the baseline value of 331 ms, data from these trials were discarded, as was data from trials with an SOA of zero. Means and standard deviations for d′ and criterion scores are given in Table 3-6.

	SOA	Control mean (St.dev) N=16	Dyslexic mean (St.dev) N=11
D prime	17	2(1.41)	0.47(0.81)
	33	1.76(1.24)	0.57(0.66)
	50	3.06(1.07)	1.3(0.9)
	66	3.88(0.78)	1.79(1.11)
	83	3.9(0.88)	2.3(1.14)
	99	4.67(0.54)	3.9(0.66)
	116	4.36(0.7)	3.07(1.05)
Criterion	17	-0.19(0.71)	-0.18(0.65)
	33	0.08(0.5)	-0.01(0.43)
	50	0.46(0.72)	-0.11(0.55)
	66	0.21(0.77)	0.02(0.59)
	83	0.19(0.51)	-0.04(0.62)
	99	0.48(0.54)	1.25(0.66)
	116	0.14(0.62)	-0.13(0.62)

Table 3-6

Means and standard deviations of d′ and criterion scores by SOA for the TOJ task, for each group.

[26] He consistently reported the shape that had appeared in the left hemifield regardless of SOA.

D prime scores, as a measure of sensitivity to temporal order, were then entered into a 7 x 2 ANOVA, with seven levels of SOA as a within-subjects factor, and 2 levels of diagnostic group as a between subjects factor. The dyslexic group were significantly less sensitive to temporal order than the control group (between subjects effect: $F(1,47)=45.527$, $p<0.001$); there was also a significant interaction between group and SOA [$F(2.71, 127.14)=4.368$]. Orthogonal contrasts (comparing d prime scores at each SOA relative to the mean of the shorter SOAs) indicated that the effect size of the difference between the groups was maximal where the SOA was 66ms [$F(1,47)=24.317$, $p<0.001$), and a polynomial contrast indicated a significant quadratic component [$F(1,47)=4.796$, $p<0.05$], thus confirming that difference between the groups was maximal at mid-value SOAs. These findings remained significant when FSIQ scores from the WASI were entered as a covariate into the model, and FSIQ scores did not account for significant additional variance in d prime scores. Finally, they remained significant when scores from Matrix Reasoning and from Vocabulary, the two WASI subtests on which the groups differed significantly, were entered into the model, and neither subtest score accounted for significant additional variance in d prime scores.

Criterion scores, as a measure of the extent to which participants tended to respond “left first” regardless of accuracy, were then entered into 7 x 2 ANOVA, with seven levels of SOA as a within-subjects factor, and diagnostic group

(dyslexic and control) as a between-subjects factor. There was no main effect of group, but a significant group by SOA interaction [$F(6, 282)=6.473$]. To investigate the nature of the interaction, the two groups were analyzed separately. There was no significant main effect of SOA in the control group; however, the intercept was significantly greater than zero [$F(1,14)=7.561$, $p<0.05$], indicating a slight left-hemifield advantage, irrespective of SOA. In the dyslexic group the intercept was not significantly different from zero; however there was a significant main effect of SOA [$F(4.34, 143.18)=31.060$, $p<0.001$]. Orthogonal contrasts (mean criterion scores at each SOA compared with the mean of shorter SOAs) indicated that at the second longest SOA (99ms) the dyslexic group had a significantly greater tendency to respond "left first" [$F(1, 33)=114.405$, $p<0.001$] than at shorter SOAs . There were no significant differences between the Dyslexia Only group and the Dyslexia + ADD group.

3.4.2.5 TOJ and single word reading

In an attempt to replicate the findings from the Experiment 2, reported in Chapter 1, total accuracy scores for irregular words and non-words, and backward digit span scores for the dyslexic group were entered as predictor variables into a one-way ANOVA with d′ scores as the dependent variable and 7 levels of SOA. None of the predictor variables accounted for significant variance in d′ scores, and there were no significant interactions with SOA. The ANOVA was then repeated with criterion scores as the dependent variable; again none of the predictor variables accounted for significant variance in criterion scores, and

there was no significant interaction with SOA.

An alternative analytical approach

It was postulated that failure to replicate directly the findings from Experiments 1b and 1c might be due to the fact that the participants in this experiment were recruited from an able, university educated population, whereas participants in Experiments 2a and 2b were drawn from the general public. It was therefore postulated that the dyslexic participants' reading performance was more likely to resemble that of the non-dyslexic participants; print exposure, for example, was likely to be greater amongst the dyslexic participants in this study, potentially masking word recognition differentials, as measured by irregular word reading accuracy, between the dyslexic and non-dyslexic groups (Griffiths and Snowling, 2002; Stanovich et al., 1997).

Scores on the One Minute Reading, Two Minute Spelling, and One Minute Writing subtests of the DAST were therefore pooled across both the dyslexic and control participants, and examined for departures from normality. All distributions were slightly but insignificantly negatively skewed, and none were significantly platykurtic (z score of kurtosis <1.96). There was thus no suggestion of a bi-modal distribution. A second series of analyses was therefore conducted in which performance of control and dyslexic participants on the TOJ task were considered together, and, in an alternative approach to that derived from SDT, Principal Components Analysis was used to extract factors accounting for both accuracy and bias on the task.

Accuracy scores on that TOJ task were at ceiling for many participants at SOAs greater than 99ms. For SOAs of 99ms, accuracy scores were negatively skewed, the degree of skew decreasing with increasing SOA. These were therefore normalized where required by raising to an appropriate power[27], and converted to z scores, thus giving a series of measures, for each condition (SOA and hemifield order), of each participant's performance in standard deviations from the group mean ($z = 0$). These were then entered into a principal components factor analysis in order to determine any patterns of association between performance on different conditions.

The scree plot indicated a pronounced decrease in slope after the second factor, and a two-factor solution was therefore obliquely rotated (Direct Oblimin). Factor scores were obtained for the two factors which were significantly correlated (Pearson's correlation coefficient= 0.53, $p<0.001$). Factor loadings given by the pattern matrix are shown graphically in Figure 3-9.

[27] 99ms: scores raised to power of 10; 83ms; scores raised to power of 8; 66ms: scores raised to power of 5; 50ms: scores raised to power of 2.

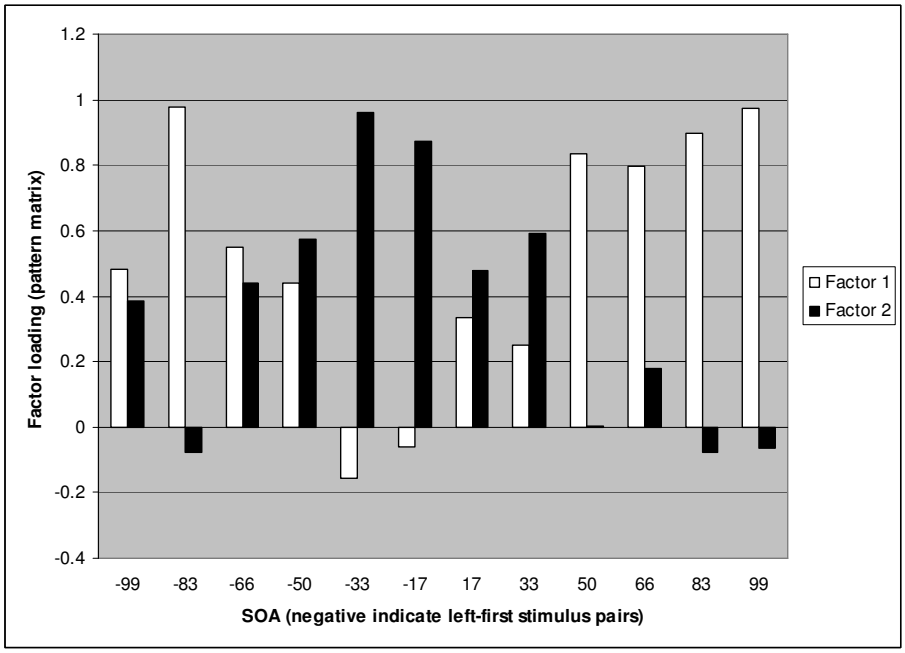

Figure 3-9: TOJ factor loadings

The first factor had a significantly greater loading on right-first stimulus pairs than on left-first pairs, and on longer SOAs. The second factor loaded significantly more heavily on right-first pairs than on left-first pairs, and on shorter SOAs.

Visual inspection indicated that the first factor (eigenvalue = 7.295) loaded most heavily on trials with long SOAs, and more heavily on trials in which the right stimulus preceded the left. The second factor (eigenvalue =1.414) loaded most heavily on trials with short SOAs, and more heavily on trials in which the right stimulus preceded the left. To ascertain whether this characterisation of the two factors had quantitative validity, z scores for the trials for which the factors had been extracted (SOAs from 17ms to 99 ms) were entered as dependent variables into a 6 x 2 ANOVA with six levels of SOA and two levels of "hemifield" (the hemifield in which the first stimulus was presented). The two factor scores were

entered as between-subjects factors. There were significant interactions between each factor and SOA [Factor 1: $F(3.97, 182.38)^{25} = 36.724$, $p<0.001$; Factor 2: $F(3.97, 182.38)^{25} = 28.223$, $p<0.001$]. Polynomial contrasts indicated that the most significant component of the relationship between SOA and factor was a linear component [$F(1,46)=89.952$, $p<0.001$], and examination of the regression parameters indicated that Factor 1 accounted for significantly more variance (more positive regression coefficient) in accuracy as SOA increased. The most significant component of the relationship between Factor 2 and SOA was also linear [$F(1,46)=68.335$, $p<0.001$]; in contrast, however, examination of the regression parameters indicated that this factor accounted for significantly more variance (more positive regression coefficient) in accuracy as SOA decreased.

There were also significant interactions between each factor and hemifield [Factor 1: $F(1,46)=18.738$, $p<0.001$; Factor 2$(1,45)=25.072$, $p<0.001$]. Examination of the regression coefficients confirmed that this indicated that Factor 1 accounted for significantly more variance in accuracy (more positive regression coefficient) for trials in which the target stimulus appeared in right hemifield than for trials in which it appeared in the left, while the reverse was true for Factor 2.

Factor 1 therefore appeared to represent performance at long SOAs in which good performance was associated with a right hemifield advantage, while Factor 2 appeared to represent performance at short SOAs in which good performance was associated with a left-field advantage. It was postulated that the leftward

lateral bias associated with Factor 1 may represent variance in the efficiency of left-hemisphere networks, and that its loading on longer SOAs suggests that these networks may be implicated in recall of temporal order, and deficits in which may result in a tendency to find right hemifield stimuli more salient. It was postulated that, in contrast, the rightward lateral bias associated with Factor 2 may represent variance in the efficiency of right hemisphere networks, and that its loading on short SOAs suggests that these networks may be implicated in response to an alerting stimulus. If so, variance in the efficiency of these networks might account for separate variance in degree and kind of reading impairment.

Factor scores on each factor were therefore entered as predictor variables into a series of hierarchical regression models, with four measures of reading as dependent variables. The four reading measures were: total non-word reading accuracy; total irregular word reading accuracy; and a factor (extracted using principal components factor analysis[28]) representing single real word reading latency. Factor 1 was entered into the model first, followed by Factor 2. Factor 1 accounted for significant variance in non-word reading accuracy [R^2=0.278, $F(1,47)=18.063$, $p<0.001$,], and the regression coefficient was positive; Factor 2 accounted for significant additional variance [R^2 change = 0.075, $F(1,46)=5.361$, $p<0.05$]; again, the regression coefficient was positive. The prediction made by Factor 1 remained significant when Factor 2 was added to the model. Factor 1

[28] Scree plot indicated a single factor with an eigen value of 5.391, accounting for 89.8% of the variance in real word reading latencies.

also accounted for significant variance in irregular word reading accuracy [R^2=0.140, F(1,47)=7.622, p<0.01], with a positive regression coefficient; Factor 2 accounted for no significant additional variance in irregular word reading accuracy. When factor scores for real word reading latencies were entered as dependent variable, Factor 1 accounted for significant variance in latency [R2=0.199, F(1,47)=11.701, p<0.01], with a negative regression coefficient, and Factor 2 accounted for no significant additional variance.

In order to test whether the postulated dissociated deficits might account for variance in ADD symptoms, the two TOJ factors were then entered as predictor variables in to a MANOVA with the five cluster scores from the Brown's ADD scale as dependent variables. This indicated that Factor 1 was a significant predictors of a single variate [Factor 1: F(5,42)=4.861, p<0.01], while Factor 2 did not account for significant additional variance on this variate. Examination of univariate output indicated that Factor 1 accounted for greatest variance in the Activation [F(1,46)=15.747, p<0.001] Memory [F(1,46)=7.810, p<0.01] and Affect clusters [F(1,46)=5.562, p<0.05]. However, Factor 2 accounted for significant variance in the Attention [F(1,46)=10.735, p<0.01) and Effort [F(1,46)=7.644, p<0.01] clusters. As a follow-up, therefore, two further MANOVAs were conducted; in the first, the dependent variables were: Activation, Memory and Affect, while in the second, the dependent variables were Attention and Effort. These indicated that Factor 1, after controlling for Factor 2 scores, was a significant predictor of a single variate extracted from Activation, Memory and Affect scores [F(3,44)=5.304, p<0.01], Factor 2

accounting for no significant additional variance. In contrast, Factor 2, after controlling for Factor 1 scores, was a significant predictor of a single variate extracted from Attention and Effort scores [$F(2,45)=5.355$, $p<0.01$].

Finally, while there was no discrete difference between the diagnostic groups in terms of functional literacy, they had been recruited from two different populations: those who considered themselves to have dyslexia, and those who did not. It was therefore postulated that factor scores on the two TOJ factors might have power to predict participant membership of each population. The two TOJ factors were therefore entered as predictor variables into a binary logistic regression models, with group as outcome variable (34 Dyslexic and 15 Control). The outcome variable was coded in such a way that the odds ratio was for membership of the Control group. Both sets of factor scores were entered in the same block using a forward entry procedure (Likelihood Ratio). Factor 1 was entered first, and resulted in significant increase in chi square for the model [increase in chi square $(1)=20.127$, $p<0.001$]. When Factor 2 was entered into the model, chi square again increased significantly [increase in chi square $(1) = 9.042$, $p<0.01$]. The sign of the regression coefficient for both factors was positive, indicating that the higher the scores on each factor the greater likelihood that a participant was recruited from the non-dyslexic group. Thirty-one of the 34 dyslexic participants were successfully classified by the model (91.2%), and 12 of the 15 control participants were successfully classified (80.0%).

3.4.2.6 Interim summary and discussion:

These results replicate the findings from Experiments 1a, 1b, and 1c, that dyslexic participants were significantly less accurate than control participants in reporting the temporal order of onset of two stimuli presented to opposite lateral hemifields. Furthermore, there was no significant difference in accuracy between dyslexic participants who screened positively for ADD and those who did not, suggesting that the association between reading impairment and TOJ performance observed was not solely due to co-morbid ADHD. While the groups did differ significantly in FSIQ, Matrix Reasoning and Vocabulary scores on the WASI, the differences between the groups remained significant when these were controlled for.

The results did not, however, directly replicate the findings of Experiments 1b and 1c, of an association between direction lateral bias, as measured by criterion scores, and deficits on non-word reading and irregular word reading, respectively, when the dyslexic group was analysed separately. One possible explanation for failure to replicate this result may have been insufficient statistical power, as the sample size was smaller than in the two earlier experiments. However, the associations between the two factors extracted by the principal components analysis from data collected from all participants and measures of both reading and ADHD symptoms may nonetheless be consistent with the findings from the experiments reported in Chapter 2 (Experiments 1b and 1c).

The first factor extracted loaded on trials with long SOAs and right-first stimulus presentation order. Bearing in mind that in this version of the TOJ paradigm, a more complex stimulus-response mapping was required than the one-to-one mapping required in the paradigm utilized in Experiments 1a, 1b and 1c, one possibility is that scores on this factor may represent the efficiency of networks involved not so much in determining the temporal order of the stimuli but in either retrieving the appropriate mapping or inhibiting an inappropriate mapping. This might account for its loading on longer SOAs, where temporal ordering *per se* is likely to have been easier, for both right-left and left-right stimulus pairs. Moreover, if the mapping was achieved through verbal labeling of the stimuli, this might also account for its heavier loading on right-first pairs. Scores on this factor may therefore reflect the efficiency of networks implicated in executive control, including verbal working memory and/or inhibition of inappropriate response, and/or efficiency of lexical retrieval. The fact that scores on this factor were associated with three measures of reading impairment, namely, word recognition latencies; real-word reading accuracy, and non-word reading accuracy, suggests that low scores on this factor may reflect deficits associated with the core features of dyslexia.

However, low scores on Factor 1 were also associated with symptoms of ADHD, particularly those concerning activation and organizing for work ("Activation"); managing affective interference ("Affect") and Memory, supporting the hypothesis that it reflects the efficiency of networks implicated in executive function. In an investigation into the co-morbidity of dyslexia and ADHD,

Wilcutt et al. (Willcutt et al., 2005) found that low scores on a verbal working memory factor were common to children with both dyslexia and ADHD, although the weaknesses were more consistent and severe in the dyslexic children. The findings of this study are thus consistent with the hypothesis that poor performance on the TOJ task by dyslexic participants, particularly at long SOAs and when the right hemifield stimulus is presented before the left, is indicative of a deficit in a factor that is associated most strongly associated with reading impairment but also with symptoms of co-morbid ADHD, and may be a manifestation of deficits in executive function that are common to both conditions. In this respect, it was of interest that the DO group did not differ from the D+ADD group on either of the two DAST items likely to tap phonological working memory capacity, namely Phonemic Segmentation and Backward Digit Span.

In contrast, the second factor loaded on trials with short SOAs and left-first stimulus presentation order, and thus may represent variability in the deficit associated with rightwards bias and poor non-word reading accuracy in Experiments 1b and 1c. The finding that this factor loaded not only on right-first stimuli pairs but also on short SOAs may suggest that scores on this factor may represent the efficiency of networks implicated in general vigilance as well as specific sensitivity to the onset of stimuli presented in left hemifield. The finding by Robertson and colleagues (Robertson et al., 1998) that phasic arousal ameliorated left-neglect in patients with right lateralized lesions is therefore of interest, and suggests that Factor 2 may reflect the efficiency of a right localized

network common to the modulation of arousal and the attention to left hemispace, as proposed by Berger and Posner (2000).

This hypothesis is supported by the finding that the postulated right hemisphere factor was a significant predictor of scores on the ADD clusters representing "Attention" and "Effort" both of which are measures of sustained attention to a task (Brown, 1996), and thus may be a measure of vigilance. This would be consistent with findings by other researchers of a "left neglect" syndrome associated with ADHD, including that of Rothlind and colleagues (1991) cited earlier.

Moreover, George and colleagues (2005) found, in a neat inverse of Robertson et al's finding in adults with hemispatial neglect, that left neglect in children with ADHD was increased with time on task, and Sheppard et al. (Sheppard et al., 1999) found that stimulant medication normalized the significantly rightward bias found in unmedicated ADHD children. Carter et al. (Carter et al., 1995) found that unmedicated children with ADHD showed a significant cost difference for invalidly exogenously cued targets, whereby invalid cues presented in right hemifield attenuated reaction times more than invalid cues presented in left hemifield. In contrast they found that invalid endogenous cues produced reduced costs for left hemifield targets than for right hemifield targets, and concluded that their findings were consistent with a disruption to right hemisphere attentional mechanisms.

The finding of the experiment reported here is thus consistent with the hypothesis

that ADHD symptoms of impaired sustained attention and effort may be due to deficits in right-lateralized networks implicated in both vigilance and awareness of left space.

The finding that this postulated right hemisphere factor was also predictive of non-word reading accuracy, over and above the variance accounted for by the postulated left-hemisphere factor, would be also be consistent with the findings reported in Chapter 2 of an association between rightward bias and inaccurate reading of non-words. Neglect patients can show a form of reading impairment known as "neglect dyslexia" which is more manifest in non-word than in real word reading (Sieroff et al., 1988), possibly because attention to the left-most letters of a word is more critical for the accurate reading of non-words than of real words (Auclair and Sieroff, 2002). Such reading errors have also been found in ADHD children who show left neglect (George et al., 2005; Manly et al., 1997). The findings from the current experiment are thus consistent with the hypothesis that specific difficulty in reading unfamiliar words accurately may arise directly from a right-lateralized deficit associated with "left neglect" and found in association with ADHD.

A hypothesis that could account for the findings of this experiment, therefore, is that the TOJ task in the form used here tapped two partially dissociated networks, deficits in both of which are associated with both reading impairment and ADHD. One is a possibly left lateralized network that is implicated in the stimulus-response mapping process, the core symptoms of dyslexia and some of

the symptoms of ADHD, and possibly implicated in executive function. This may be manifest in inefficient verbal working memory, and slow lexical retrieval. The other is a right-lateralized deficit that is implicated in insensitivity to right hemifield stimuli, impaired vigilance, and the inaccurate reading of unfamiliar words.

To summarize: two partially dissociated factors may account for individual differences in performance on the version of the TOJ paradigm: the first reflects the efficiency of networks involved in response mapping and/or verbal working memory; where the efficiency of these networks is compromised, single word reading latencies are slow, word and non-word reading is inaccurate and scores on symptoms clusters reflecting poor response inhibition are high. The second reflects the efficiency of networks involved in spatial awareness and/or vigilance; where the efficiency of these networks is compromised, additional problems are experienced in reading non-word stimuli in which word recognition strategies are of limited use, and scores on symptom clusters reflecting difficulty in sustaining attention are high.

3.4.2.7 Spatial Compression

For thirteen subjects, the EOG recordings proved to be too noisy[29] for saccades onsets to be reliably determined; 9 of these were DO participants, and 4 were D+ADD participants. For the remainder of the participants, saccade onset times

[29] Interference from mobile phones was subsequently found to be the source of the noise.

were determined using the same algorithm as for Experiment 2a. Latencies were computed as the time difference between the onset of the saccade target stimulus and the onset of the saccade, and Probe-Saccade Asynchronies (PSA) were computed as the time difference between the onset of the probe and the onset of the saccade. Trials in which the saccades had a latency of less then 50ms were discarded. Inspection of a histogram of the remaining saccade latencies indicated a separate population of trials in which the saccade occurred at more than 550ms after the onset of the saccade target, and may thus have been made as a response to the onset of the ruler rather than to the onset of the cue. These trials were also discarded. From the remainder, trials were selected in which the onset of the probe occurred before the onset of the saccade.

These were sorted as in Experiment 2a into two time bins: a "Fixation" time bin, and a "Peri-saccadic" time bin. Trials in which saccades occurred from 300 and 101ms bin were allocated to the "Fixation" bin, and trials in which saccades occurred from 50 to 0ms before saccade onset were allocated to the "Peri-saccadic" bin. As for Experiment 2a, for each trial, for the mislocalization of the probe was computed by subtracting the reported position from the actual position, and coded relative to the direction of the saccade in such a way that a positive value represented a mislocalization in the direction of the saccade, and a negative value represented a mislocalization against the direction of the saccade.

Mean mislocalization values were then computed for each subject in each of the three time bins for each of the three position categories: "Beyond", "Between"

and “Behind”, and for each two saccade directions: “left-to-right” and “right-to-left”. A nominal “apparent” position” for each probe was then computed by adding the mislocalization for each trial from the mean of the two jittered actual positions for each position category, again coded relative to saccade direction. An index of “compression” for each time bin and saccade direction was then computed by taking the standard deviation of the three nominal mean apparent positions (one for each position category). An index of “shift” for each time bin and each saccade direction was computed by taking the mean of the three (one for each position) mean mislocation values.

For a few participants, some categories contained no viable trials. In order to preserve statistical power, the following conservative procedure was followed regarding missing values: where trials for one saccade direction were missing, but intact for the same condition for the opposite saccade direction, the value for the opposite saccade was substituted. This would tend to weaken lateralized effects, but should not bias other effects. Where conditions for both saccade directions were missing, the grand mean (pooled across all participants) for each condition was substituted for the missing data.

To test whether the diagnostic groups differed in the degree of peri-saccadic compression and shift, two 2x2x2 ANOVAs were performed, with “compression” and “shift” indices in turn as as dependent variables. The within-subjects factors were “time bin”, with two levels: “Fixation” time bin and “Peri-saccadic” time bin, and “Saccade Direction” with two levels: left-to-right, and

right-to-left. "Diagnostic Group" (dyslexic and control) was a between-subjects factor.

There were no significant interaction between group and either of the within-subjects in either ANOVA, nor were there any main between-subjects effects of group. Furthermore, there was no significant interaction between saccade direction and time bin for compression, and no main effects of either saccade direction or time bin on compression. However, there was a significant main effect of time bin on shift [$F(1,36)=11.926$, $p<0.01$] indicating that greater shift in the direction of the saccade in the peri-saccadic time bin than in the fixation time bin, but there was also a significant main effect of saccade direction [$F(1,26)=10.603$, $p<0.01$], indicating, as in Experiment 2a, that shift in the direction of the saccade was more marked when saccades were made from right-to-left.

Exploratory analyses

The failure to find a significant peri-saccadic compression effect was of intrinsic interest, given the robustness of the finding in previous studies in which the saccade direction was predictable, including Experiment 2a. A series of exploratory analyses were therefore carried out.

One sample t tests were conducted on each mislocation score for each probe position, for each time bin, for each group, with zero as the test value. All three groups mislocated the "Beyond" probe significantly *against* the direction of the saccade in both the fixation and peri-saccadic time bins. All three groups also

mislocalized the "Between" probes presented during the "Fixation" time bin, significantly *against* the direction of the saccade, but in no group was the mislocalization of the "Between" probes significantly different to zero when the probe was presented in the pre-saccadic period. All three groups significantly mislocalized the "Behind" probe *in the direction of the saccade* in both time bins. Results are given in Table 3-7.

	Probe position	Time bin	Mean (sd)	t	Sig. (2-tailed)
Dyslexic	Beyond	Fixation	-0.73(0.67)	t(22)5.427	0.000
		Peri-saccadic	-0.85(0.84)	t(22)4.908	0.000
	Between	Fixation	-0.72(0.38)	t(22)8.904	0.000
		Peri-saccadic	-0.02(0.63)	t(22)0.159	0.875
	Behind	Fixation	0.96(0.61)	t(21)7.371	0.000
		Peri-saccadic	0.83(0.82)	t(22)5.043	0.000
Control	Beyond	Fixation	-0.34(0.59)	t(14)2.263	0.040
		Peri-saccadic	-0.6(0.54)	t(14)4.311	0.001
	Between	Fixation	-0.61(0.37)	t(14)6.386	0.000
		Peri-saccadic	0.13(0.44)	t(14)1.169	0.262
	Behind	Fixation	0.64(0.32)	t(14)7.835	0.000
		Peri-saccadic	0.89(0.85)	t(14)4.040	0.001

Table 3-7: One sample t tests for mislocalization of probe

Both groups mislocated the "Beyond" probe significantly against the direction of the saccade in both time bins (positive value) and mislocated the "Behind" probe in the direction of the saccade (negative value) in both time bins. However, both groups mislocated the "Between" probe significantly *against* the direction of the probe, i.e. towards fixation, in the "Fixation" time bin, but neither group mislocated the "Between" probe significantly in either direction in the "Peri-saccadic" time bin.

A series of one-way repeated measures ANOVAs was then performed for each group, each position in turn, with time bin (two levels) as a within subjects factor. For none of the groups was there a significant interaction between mislocalization of the "Beyond" probe and time bin, and only for the D+ADD

group was there a significant interaction between mislocalization of the "Behind" probe and time bin [$F(1,14)=5.871$, $p<0.05$]; for this group the mislocalization was *less* markedly in the direction of the saccade for trials in the peri-saccadic time bin than for the fixation time bin. However, for the "Between" probe, for all three groups, the mislocalization was more contra-saccadic (against the direction of the saccade) for trials in the fixation time bin than in the peri-saccadic time bin [Control: $F(1,14)=31.951$, $p<0.001$; D+ADD: $F(1,14)=23.272$; DO: $F(1,7)=16.585$, $p<0.01$]. These results are illustrated graphically in Figure 3-10.

It was postulated during discussion of the results of Experiment 2a that saccade latencies may be longer in dyslexia. Saccade latencies were therefore computed for each trial in the spatial compression task, and median latencies computed for each saccade direction. These were entered as dependent variable into a one way ANOVA with two levels of saccade direction, and group (Dyslexic and Control) as a between group factor. There was no significant main effect of saccade direction, nor any significant interaction between group and direction. However there was a significant main effect of group [$F(1,36)=17.821$] indicating that mean saccade latencies for the dyslexic group were significant slower than for the control group.

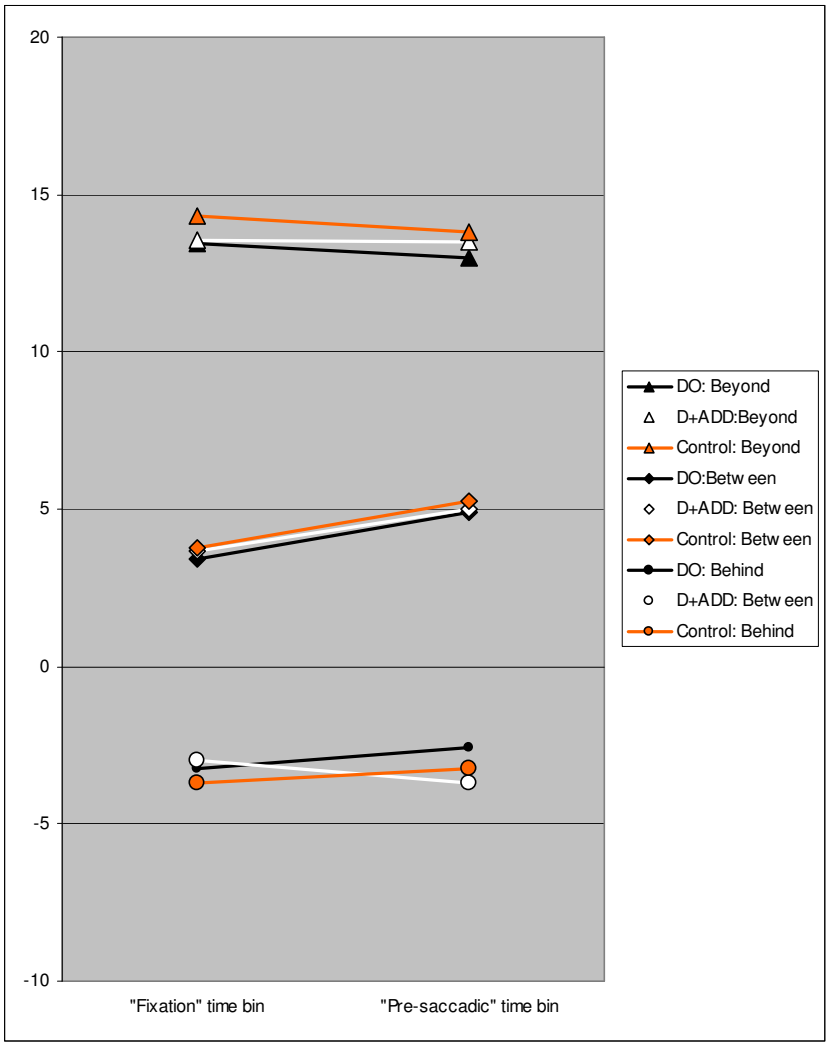

Figure 3-10: Mislocalization of probes at three positions for two time bins.

All three groups showed the same general pattern of mislocalization: “compression” towards fixation when the probe was presented more than 100 before saccade onset, and compression towards the saccade target when the probe was presented 50ms or less before saccade onset.

The ANOVA was then repeated on the dyslexic participants’ data only, with the two sub groups (DO and D+ADD) as a between-subjects variable. Again, there was no significant main effect of direction, nor any interaction between group and saccade direction. There was also no main effect of group.

3.4.2.8 Interim summary and discussion

The striking finding from these results is that while marked "compression" of space between probe positions was observed in all participants, neither the dyslexic nor the non-dyslexic control group demonstrated any greater "compression" in the "pre-saccadic" time bin than in the "Fixation" time bin. Instead, the *focus* of the compression appeared to shift, in both groups, from current fixation towards the saccade target. This finding raises important questions as to the role of predictability regarding the location of the saccade target in producing the "peri-saccadic compression" increases reported by other researchers (Burr et al., 2001; Lappe et al., 2000; Ross et al., 1997; Ross et al., 2001), and found in Experiment 2a.

The fact that, using a paradigm in which the saccade target was unpredictable, the pattern of mislocalization observed in Experiment 2b, was found in both diagnostic groups suggests that it is robust. One possibility, therefore, is that increases in peri-saccadic compression found in uni-directional paradigms, such as that employed in Experiment 2a, are a function of the degree to which endogenous covert attention is shifted to the expected saccade target location or hemifield; if so, the results of Experiment 2a may reflect a reduced ability in dyslexic participants to engage in such an anticipatory attentional shift, the opportunity for which, in this version of the paradigm, was denied to all participants.

Evidence for a peri-saccadic shift in the *focus* of compression, rather than a change in its magnitude, is given by the pattern of mislocalization of the "Between" probe. The pattern of mislocalization of the outer ("Behind" and "Beyond") probes cannot distinguish between compression towards the saccade Target and compression towards Fixation. However, the pattern of mislocalization of the "Between" probes allows these patterns of mislocalization to be disambiguated; where the "Between" probe is mislocalized *against* the direction of the saccade, compression must be towards fixation, rather than towards the saccade target. In both diagnostic groups, the "Between" probe was mislocalized significantly *against* the direction of the saccade in the fixation time bin. The "Beyond" probe, similarly, was mislocalized against the direction of the saccade, while the "Behind" probe was mislocalized in the direction of the saccade. Thus the "focus" of the compression effect was the current fixation point.

However, for probes presented 50ms or less before saccade onset, both diagnostic groups displayed a more ambiguous pattern of compression. No net mislocalization of the "Between" probe in either direction was observed in either group, although the two outer probes were both "compressed" towards each other. However the participants as a whole showed significantly greater shift in this peri-saccadic time bin, an effect largely leveraged by the less contra-saccadic mislocalization of the "Between" probe.

Thus, conceptually, the pattern observed may be more aptly described as a

pattern whereby probes presented substantially before saccade onset are compressed in the direction of fixation, whereas for probes presented within 50ms of saccade onset, the focus of the compression shifts in the direction of the saccade target.

The model reported in Appendix A postulated that compression arises because the probe itself is a salient stimulus that produces activation on saccade map, as postulated by Godijn and Theeuwes (2002), and is thus represented as a potential saccade vector. The model predicts that this will distort the Predicted Retinotopic Vector (PRV) that the probe will shortly have; in other words it will tend to make a too-foveal estimate of the probe's predicted retinotopic co-ordinates (illustrated in Figure 3-3). The model assumed that a saccade to a new target was impending, and that this too-foveal PRV would be post-dictively re-mapped on to recalibrated spatio-topic co-ordinates of the new fixation point post-saccade.

However, if no saccade is immediately executed, either to the probe location or to an alternative saccade target, the PRV may be remapped, not to the spatiotopic co-ordinates a new fixation point, but to those of the current fixation point. The term "Peri-saccadic" thus becomes moot. In this interpretation of the phenomenon of "Peri-saccadic" mislocalization, it is the fact that the probe results in a brief, unexecuted saccade that causes it to be mislocalized in the direction of whatever reference is subsequently available; in the case of a probe presented substantially before a saccade onset, this reference will be the current

fixation point; in the case of a probe presented immediately before a saccade onset, the reference will be the new fixation point. The former condition is illustrated schematically in Figure 3-11.

Such a model would be consistent with the data presented from Experiment 2a. It would also be consistent with data cited by Kerzel (2002) as evidence for "foveal bias": even when fixation is maintained, briefly presented salient stimuli tend to be recalled as having occurred as closer to fixation than their veridical position.

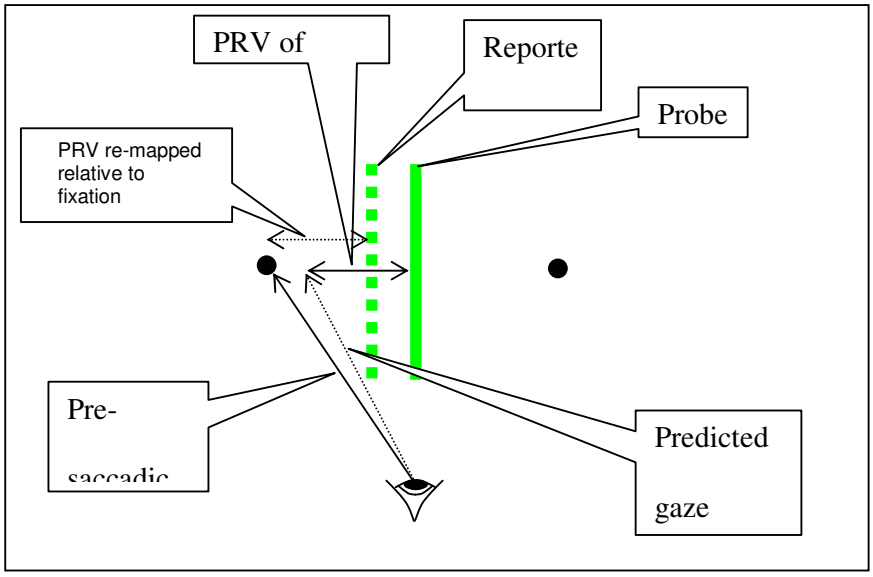

Figure 3-11: Compression of space towards current fixation

When the probe onset occurs substantially before saccade onset, it is postulated that the salience of the saccade target will still be weak. The model predicts that, as in the peri-saccadic condition, the probe will distort the predicted future gaze direction in the direction of itself, leading to an underestimated PRV for the probe that is too foveal. After probe offset, this too-foveal PRV is remapped relative to spatiotopic co-ordinates of the current fixation, producing "compression" of space between the probe and current fixation.

The question then arises as to why compression towards fixation has not been observed when using in paradigms in which, as in Experiment 2a, saccades were made in a predictable direction (Burr et al., 2001; Lappe et al., 2000; Ross et al., 1997; Ross et al., 2001). These studies appear to indicate, contrary to the above model, that compression is a peri-saccadic phenomenon, and that the focus of the compression is the saccade target. However, the major difference between those paradigms and that utilized in Experiment 2a is that the visual hemifield in which the saccade target will appear was predictable. It may therefore be most useful to seek an explanation for the difference between the pattern of mislocalization observed in Experiment 2b, and that observed in uni-directional paradigms not in terms of failure to find "peri-saccadic compression", but rather in terms of failure of the uni-directional paradigms to elicit "compression" towards fixation when probes were presented substantially before saccade onset.

In the paradigm employed in Experiment 2b, unlike that employed in Experiment 2a, no anticipatory lateral attentional bias was possible. This might have had two consequences, and these may have combined to attenuate, in uni-directional paradigms, "compression" effects towards fixation. The first consequence may be that when the saccade direction is predictable, anticipatory eye position estimate errors occur earlier during the time course of saccade preparation. If so,

the model presented in Appendix A predicts that this would tend to counteract the tendency to mislocalize "Between" and "Beyond" probes in the direction of current fixation when the probe is presented substantially before saccade onset ("Fixation" condition). However, this would not account for the relatively veridical localization of the "Behind" probe under these conditions. A second consequence of directing attention to the visual hemifield in which the saccade target is expected may be to reduce the salience of the "Behind" probe. Again, according to the model presented in Appendix A, this would result in reduced distortion of the PRV, and thus to a smaller magnitude of mislocalization.

In this interpretation, brief, salient, abrupt onset probes elicit "foveal" bias, resulting in subsequent mislocalization of the probe towards the point in space for which the visual system has good spatiotopic references. When a probe is presented substantially before a saccade, this point will be the location currently fixated; when a probe is presented immediately before a saccade this will be the new fixation point. Thus, uni-directional paradigms induce top-down attention to the hemifield in which the saccade target is expected, attenuating compression towards fixation, and throwing "Peri-saccadic" compression towards the target into relief.

Such an explanation must remain speculative, however; one problem common to both the uni-directional and the bi-directional spatial compression paradigms is that the probe-saccade asynchrony is not a direct result of experimental manipulation; the probe itself is likely to affect the latency of any subsequent

saccade, and, additionally, the population of trials in which the probe was presented early is likely to contain a greater proportion of trials with long saccade latencies, relative to the population of trials in which the probe was presented late. Moreover previous studies have compared "Peri-saccadic" trials, in which probes were presented immediately before, during, and immediately after saccades with trials in which the probes were presented both substantially before and substantially after saccade onset (Burr et al., 2001; Lappe et al., 2000). Further research is required in which saccade latency is controlled for, and the localization of pre-saccadic probes is distinguished from that of post-saccadic probes.

In this light, the finding from Experiment 2b that it was only in the more severely impaired D+ADD group that some form of attenuation of peri-compression was found may indicate that by depriving non-impaired participants of the opportunity to anticipatorily shift attention to the saccade target hemifield differences, the power of the study to reveal group differences was confined to the comparison between the most severely impaired group and other participants.

However, the question arises as to whether the attenuated pre-saccadic compression observed in the D+ADD group did or did not reflect the same attenuation observed in dyslexic participants in Experiment 2a. The finding was not that the D+ADD showed an attenuated increase in compression in the "pre-saccadic" conditions, but that they actually showed a decrease. Subsequent exploratory analyses located the source of this difference to reduced pro-saccadic

mislocation of the "Behind" probe only. A possible explanation for the finding may be that this group were less likely to shift attention to the "Behind" hemisphere once a saccade to a target in the contralateral hemisphere had been fully programmed.

"Sluggish" shifts of attention have been postulated as a marker for dyslexia in general (Ben-Yehudah et al., 2004; Facoetti et al., 2005; Facoetti et al., 2003a; Hari and Renvall, 2001; Hari et al., 1999; Wimmer et al., 2002) and for impairment on the TOJ task (Hari et al., 2001) in particular, and might also account for the attenuated peri-saccadic compression effects observed in Experiment 2a. In this interpretation, the attenuated compression effect is postulated to arise not from a failure in exogenous attention to the probe stimulus, or to faulty post-saccadic spatio-topic recalibration, but rather from failure to either attend, endogenously, to the expected saccade target location, or from sluggish exogenous attentional shift to a contralateral probe once covert attention to the target has been engaged.

This interpretation is supported by the finding that saccade latencies were significantly longer in both the DO and the D+ADD groups than in the Control group. A series of analyses was therefore carried out to determine whether the atypical performance of dyslexic participants, both in terms of peri-saccadic compression and saccade latency were predictive of TOJ performance.

3.4.2.9 TOJ and Spatial Compression

For each participant, a single "peri-saccadic compression" index was computed by taking the natural log of ratio between the compression index for fixation trials and the compression index in peri-saccadic trial (fixation trials as the denominator). Thus a value of 0 would indicate no change in compression, a positive value would indicate an increase in compression in pre-saccadic trials and a negative value a decrease in compression in pre-saccadic trials. As there was no significant difference between latencies for leftward saccades and rightward saccades, median latencies for all trials in which a valid saccade was made with a latency of between 50 and 550ms were computed for each participant. Pearson correlation coefficients were computed between median latencies, the peri-saccadic compression index, the two TOJ factor scores; non-word reading accuracy, real-word reading latencies and ADD symptom score. Median latencies and the peri-saccadic compression index were also were regressed on the two TOJ factors in a multiple regression.

The Pearson Correlation coefficient between peri-saccadic compression and median saccade latency was negative, and thus in the predicted direction (r=-.11) but not significant. Any real tendency for peri-saccadic compression to be more greatly attenuated in participants with long saccade latencies was, if present, too small an effect to be detected with the power available in the study. There was also no significant zero-order correlation between the peri-saccadic compression index and either of the two TOJ factors, with non-word reading accuracy, or with

real word reading latencies or with ADD symptom score. Neither TOJ factor was a significant predictor when the peri-saccadic compression index was regressed on both factors.

However, median latencies were significantly and negatively correlated with the two TOJ factors (Factor 1: $r=-0.63$, $p<0.001$; Factor 2: $r=0.44$, $p<0.01$). When saccade latencies were regressed on the two TOJ factors in a multiple regression, only Factor 1 was a significant predictor; it remained a significant predictor when Factor 2 was entered to the model, and Factor 2 accounted for no significant additional variance. Saccade latencies also had significant zero order correlation with real word reading latency ($r=0.50$, $p<0.01$); non-word reading accuracy ($r=-.51$, $p<0.01$) and ADD symptom score ($r=.44$, $p<0.01$).

3.4.3 Discussion

Experiment 2b replicates the findings from the Experiment 1a, 1b and 1c d of impaired TOJ task performance in dyslexic participants; it also indicates that TOJ task performance is impaired, relative to control participants, even when the dyslexic participants have screened negatively for symptoms of an Attentional Deficit Disorder, and when measures of IQ are controlled for. The experiment also partially replicated the findings from Experiment 2a of attenuated peri-saccadic compression in dyslexic participants, although only in more severely impaired participants with co-morbid symptoms of an Attentional Deficit Disorder.

The association between lateral bias and differential difficulty with non-word

reading accuracy as compared with irregular word reading accuracy was not directly replicated. This may have been due to reduced statistical power, as the number of dyslexic participants was smaller in the current study than in Experiments 1b and 1c. However an alternative explanation is that the more complex response mapping required in this version of the paradigm may have produced a more complex pattern of performance deficits. This interpretation is supported by the finding of two partially dissociated, partially lateralized factors underlying TOJ performance: a pattern of performance in which accuracy at long SOAs was associated with a right hemifield advantage and a pattern of performance in which accuracy at short SOAs was associated with a left hemifield advantage.

The finding that the first of these factors was associated with slow reading latencies for real words, inaccurate non-word reading, and slow saccade latencies on the TOJ task after controlling for factor scores on the second factor suggests that a left lateralized deficit underlying the key symptoms of dyslexia may also account not only for difficulty in response mapping during the TOJ task, but also for the slow saccade latencies observed during the Spatial Compression task. This last finding suggests that poor TOJ task performance at long SOAs in the dyslexic participants may have been due not merely to difficulty with response mapping but with shifting covert, as well as overt, attention to abrupt onset stimuli. However collinearity between the variables means that this interpretation must be made with caution.

3.5 General Discussion

The findings from Experiment 2b confirm a strong association between reading disorder and poor performance on a lateralized visual TOJ task, and suggest that poor performance on the TOJ task may be due to at least two partially dissociated, and cerebrally lateralized deficits. One of these appears to be lateralized to left hemisphere and is associated with the core features of dyslexia, including slow word recognition and inaccurate non-word reading. It also appears to be associated with long saccade latencies to a peripheral target. One possibility, therefore, is that the association is between a visual domain marker for a supramodal deficit in sensitivity to rapid-rise time stimuli, that affects reading by way of disruption to the development of phonemic segmentation skills necessary for effective decoding (Goswami, 2003; Richardson et al., 2004). An alternative possibility is that such a deficit in the visual modality directly impacts on reading accuracy and speed by disrupting the fluency with which visual attention is oriented across written text (Vidyasagar and Pammer, 1999).

However a second factor appears to be lateralized to right hemisphere. One interpretation is that this may reflect impairment of a right-lateralized "alerting" network postulated by Posner and colleagues to be impaired in Attention Deficit and Hyperactivity Disorder (Berger and Posner, 2000; Posner and Fan, in press; Posner and Petersen, 1990). This inference is supported by its apparent association with high scores on ADD symptom scale scores reflecting impaired sustained attention. If so, it would appear also to be associated, in this sample,

with additional deficits in non-word reading.

The final part of the investigation, reported in the next chapter, therefore attempted to related patterns of performance on a TOJ task to patterns of performance on the Attentional Network Task, a task designed by Fan and colleagues (Fan et al., 2002) to elicit separate measures of the efficiency of Posner's three postulated attentional networks, an alerting network, an orienting network and an executive network (Fan et al., 2002; Posner and Fan, in press; Posner and Petersen, 1990).

It was postulated that deficits in attentional "orienting", as tapped by the capacity to benefit from a spatially informative cue, might account for separate variance in TOJ task performance from deficits in "alerting", as tapped by the capacity to benefit from a cue that merely warned of an impending stimulus. As the Attentional Network Task also involves the manipulating of flanker stimuli, it was postulated that its capacity to tap the ability to resolve competing stimuli, an ability also postulated to be impaired in Attentional Deficit Disorder (Barkley, 2003; Berger and Posner, 2000), might also account for variance on the TOJ task.

4. TEMPORAL ORDER JUDGMENT & THE ATTENTIONAL NETWORK TASK

4.1 Introduction

The findings of the experiments reported in chapters 2 and 3 indicate that not only do dyslexic participants perform poorly on a lateralized visual TOJ task but that their performance on the task is correlated with their degree of severity. Moreover, while co-morbid symptoms of an ADD account for some variance on TOJ task performance, reading impairment *per se* is predictive of task performance, even after controlling for ADD symptoms.

However, the findings also suggest that at least two partially dissociated factors underlie TOJ task performance, and it was postulated that one of these factors might represent a right-lateralized deficit in an attentional network implicated in "alerting" (Fan et al., 2002; Posner and Fan, in press; Posner and Petersen, 1990). Berger and Posner postulate that deficits in an "alerting" network may typify ADHD (Berger and Posner, 2000). In chapter 2 of this thesis it was also postulated that such a deficit might constitute a "left mini-neglect" syndrome, found by Hari et al (2001) to be associated with dyslexia.

"Developmental left-neglect" has also been postulated to be associated with ADHD, worsening with time-on-task and improving with stimulant medication (Dobler et al., 2005; Epstein et al., 1997; Manly et al., 2005; Sheppard et al., 1999). However, like Hari, a number of other researchers have found an

association between rightward attentional bias and reading disorder (Facoetti and Turatto, 2000; Facoetti et al., 2001; Sireteanu et al., 2005; Stein and Walsh, 1997). In particular, Facoetti and colleagues (Facoetti et al., in press) found that degree of rightward attentional bias in dyslexic children was associated with degree of non-word reading impairment after controlling for age, IQ and phonological skill. This would be consistent with the findings from experiments 1b, 1c and 2b of an association between non-word reading impairment and rightward lateral bias, and supports the hypothesis that while variance on non-word reading may be accounted for by phonological deficits arising from impaired left-lateralized networks, additional variance in non-word reading arises from impairment to right-lateralized networks implicated not only in arousal but also in directing attention to left hemifield.

However, of interest was the finding from Experiment 2b of a strong association between saccade latencies to targets presented in either hemifield, and factor scores on a TOJ task performance factor that loaded on long SOAs, and preferentially on right-first stimuli pairs, suggesting that participants with longer saccade latencies were more likely to report the right stimulus as occurring before the left. This factor was also associated with long reading latencies, and with low accuracy scores on non-word as well as irregular-word reading.

It is worth noting that the factors were oblique, and strongly correlated; it would appear therefore that they represent dimensions of impairment rather than subtypes of participants. It was therefore postulated that a single aetiology or

genotype may underlie both deficits, the phenotype depending on the distribution of affected network, and the extent of the disruption. Furthermore, there is substantial evidence that ADHD is likely to involve disruption to a frontal network implicated in executive control of attention (Barkley, 2003; Berger and Posner, 2000; Nigg, 2001). Berger and Posner (2000) propose that ADHD is likely to be associated with disruption to their proposed "executive" attentional networks, as well as to their proposed right-lateralized "alerting" network. It was therefore postulated that, given the high prevalence of ADD symptoms in the dyslexic participants studied in Experiment 2b, and the association of ADD symptoms with both TOJ factors, disruption to networks involved in conflict resolution may constitute a third factor, in addition to the two lateralized factors identified, that also contributes to impaired TOJ performance in the dyslexic participants.

Experiment 3 was therefore designed to test the hypothesis that impaired TOJ performance can arise from disruption to each of the three attentional networks proposed by Posner and colleagues (Berger and Posner, 2000; Fan et al., 2002; Posner and Fan, in press; Posner and Petersen, 1990), disruption to each of which may also be associated with a particular profile of reading impairment, and at least two of which (the "alerting" and "executive" networks) may also be associated with symptoms of ADHD. In order to further disambiguate the findings from Experiments 1a, 1b, 1c and 2b, the TOJ task was modified to include conditions in which pairs stimuli were presented within the same lateral visual hemifield as well as in opposite lateral hemifields. This allowed,

potentially, for spatial or temporal order judgment, *per se*, to be assessed independently of any lateralized attentional bias.

Fan, Posner and colleagues devised a task to tap the efficiency of their three postulated attentional networks: the Attentional Network Task (ANT) (Fan et al., 2002). In this task, participants are asked to report the direction of an arrowhead, which may be flanked by arrowheads pointing in the same direction (congruent condition); arrowheads pointing in the opposite direction (incongruent condition) or neutral flankers, such as headless arrow shafts (neutral condition). The target may appear above or below fixation. This task is performed under four cue conditions. In 75% of trials a cue is given. On 50% of trials this is a non-spatially informative cue is given (either a central cue, or a cue indicating both potential locations of the target), and on 25% of trials it is a spatially informative cue (a 100% valid cue presented at the upcoming target location). On the remaining 25% of trials, no cue is given.

The rationale behind the ANT is that measures of cue benefits or flanker costs will indicate the efficiency of each of the three postulated attentional networks. The RT benefit of a non-spatially informative cue over no cue is postulated to reflect the efficiency of the "alerting" network; the RT benefit of a spatially informative cue over a non-spatially informative cue is postulated to reflect the additional benefit of spatial information, and thus the efficiency of an "orienting" network; while the cost of incongruent, as compared to congruent, flankers is postulated to reflect the efficiency of the "executive" network, a smaller cost

reflecting greater efficiency.

It was hypothesized that in a sample of participants that included those with symptoms of dyslexia and ADHD that a measure of the efficiency of the alerting network efficiency derived from ANT performance would be positively correlated with degree of leftward bias on the TOJ task, and with ADHD symptoms; that a measure of executive network efficiency would correlate with overall inaccuracy on the TOJ task, and also with ADHD symptoms. "Orienting" efficiency was also predicted to be associated with accuracy on the TOJ task. No specific prediction was made regarding its association with lateral bias.

The next challenge was to find a single population in which impairments associated with dyslexia would be prevalent, and yet encompass the normal range. Because there is fairly strong evidence for the heritability of reading disorder (Davis et al., 2001b; Schulte-Korne, 2001), it was decided that parents with at least one dyslexic child would constitute such a population. A sample of such parents was therefore recruited. They were asked to perform not only a lateralized visual TOJ task, but also to undertake the Fan and colleagues' Attentional Network Task (ANT) (Fan et al., 2002). In addition, participants undertook a test of regular, irregular and non-word reading, and a number of standardized tests designed to ascertain the degree to which they suffered from a reading disorder, or from an Attentional Deficit Disorder. They also undertook a task designed to tap phonological skill.

4.2 Experiment 3

4.2.1 Method

4.2.1.1 Participants

Participants were 40 adults, all with at least one dyslexic child. Twenty two participants were parents of children who had participated in Experiment 1.3, and who had screened positively for dyslexia on the Dyslexia Screening Test (DST) (Fawcett and Nicolson, 1996) despite an IQ in the normal range. Eight participants were recruited through the either Nottinghamshire Dyslexia Association or the Nottingham branch of the Dyslexia Institute, and 10 through personal contacts. All of these participants had at least one child who had been assessed as dyslexic by a chartered educational psychologist. The age range of the participants was 35 to 59, with a mean age of 45 (st.dev. = 5.6 years). The sample included fifteen couples, both of whom were parents of the same dyslexic child; of the remaining 10 participants, eight were mothers, and two were fathers. Handedness was assessed using the Edinburgh Handedness Inventory (EHI) (Oldfield, 1971). Thirty-three participants were right-handed (EHI>15) and seven participants were non-right-handed (EHI score range: -22 to +8). All participants attended two sessions of approximately 2 hours, and were paid for their participation.

4.2.1.2 Standardized measures

All participants were assessed on the DAST (Fawcett and Nicolson, 1998) as a

general screening for dyslexia. Standardized scores on untimed tests of single word reading and spelling were obtained using the Tan forms from the Wide Range Achievement Test (WRAT) (Wilkinson, 1993). Verbal, Performance and Full-Scale IQ scores were obtained using the four sub-test form[1] of the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 1999). Evidence of an Attention Deficit Disorder (ADD) was obtained through the use of the Brown ADD scale for Adolescents and Adults (Brown, 1996), a 40 item self-rating questionnaire. Where the One Minute Reading item from the DAST proved inappropriate (either because of test familiarity or in the case of very low scores), the Test of Word Reading Efficiency (TOWRE) (Torgesen et al., 1999) was administered.

4.2.1.3 Non-standardized literacy measures

Phoneme Swap:

As the Phonemic Segmentation item on the DAST has a fairly low ceiling, a further "Phoneme Swap" task was devised for the purposes of producing a test of phonological skill that would have a more normal distribution in the sample. This tested was piloted on 5 graduate psychology students and none achieved a perfect score. It consisted of twenty trials in which the experimenter read a real word aloud, and the participant was required to produce a non-word by swapping the initial and final phonemes. "Mat" thus became "tam", "shine" became

[1] Time constraints meant that the Similarities subtest was not given to 6 participants.

"nysh" and "trend" became "drent". The items were graded in difficulty by adding initial and/or final consonant blends and additional syllables to later items. Two practice items were given before the test, with feedback. Participants were asked to concentrate on sounds not letters, and to be aware that some words would have more than one sound at the beginning and the end (protocol and stimuli are given in Appendix C). One point was scored for each correct answer; any answer that was given after more than seven seconds was counted as an error. The test was discontinued after 5 consecutive errors.

Regular, irregular and non-word reading:

This task was identical to that employed in Experiment 2b. based on that devised by Castles and Coltheart (1993) (Appendix B). Participants undertook this task at their second session only.

4.2.1.4 Psychophysics tasks

All these tasks were programmed using E Prime[2] and presented on a 14 inch computer monitor with a refresh rate of 16.7 ms. Participants sat approximately 57cm from the screen for each task.

Attentional Network Task:

This task was a version of the Attentional Network Task devised by Fan and colleagues (Fan et al., 2002), slightly modified to increase the discriminability of

---

[2] E Prime 1.1.4.1, Psychology Software Tools, Inc. Pittsburgh PA

the stimuli for the age group of the sample. Stimuli were presented on a white ground, on which a black central fixation cross (subtending approximately 0.8º of visual angle) remained throughout the experiment. The target was a black chevron subtending approximately 1º, which appeared either directly above or directly below the fixation cross, and pointed either to the left or to the right (< or >). The participants' task was to indicate in which direction the chevron was pointing. The angular distance from the centre of the fixation cross to the centre of the chevron was approximately 1.5º. Participants indicated their response by means of a computer mouse held in both hands, with a thumb poised over each of the two mouse buttons. A left button press indicated a left-pointing chevron and a right button press indicated a right pointing chevron.

The target was always flanked by two irrelevant stimuli on either side. Three flanking conditions were used: in a neutral condition the four flankers consisted of simple hyphen marks; in a congruent flanker condition, the flankers consisted of chevrons pointing in the same direction as the target chevron; in an incongruent flanker condition, the flankers consisted of chevrons pointing in the opposite direction to the target chevron. The target and flankers remained on screen until the participant responded, and for a maximum of 1700ms. If no response was made within 1700ms, an accuracy score of 0 was recorded. In 75% of trials, the target followed 400ms after a cue100ms in duration.

Three types of cue were presented, with equal frequency. Two of these cues were spatially neutral: a central cue consisting of an "equals" (=) sign that briefly

replaced the fixation cross; and a double cue in which two "equals" signs appeared briefly in the two possible target locations above and below fixation. A third cue type was spatially informative, and appeared at the location of the upcoming target with 100% validity. Flanker and cue conditions were varied randomly. In addition, target onsets were designed to be temporally unpredictable by means of a randomized inter-target interval. Each trial nominally lasted for 4000, but the inter-target interval was randomly varied by between 2569 and 4831ms, by means of a formula that used the pre-trial fixation duration and response time to calculate the post-trial fixation duration. Participants undertook a total of 288 trials divided into 3 blocks of 96 trials. Details of stimuli are given in Table 4-1.

The task was explained verbally to each individual beforehand. Participants were warned that sometimes there would be symbols either side of the target that might sometimes "put them off", but that they should ignore them. They were also told that there would on some trials be a "hint" as to where or when the target would appear. They were told to ignore these as well, but informed that "your brain will make use of the hint, whether you pay attention to them or not".

Cue type:		Central	Double	Spatial	None
Cue function:		Alerts only	Alerts	Alerts and is spatially informative	N/A
Fixation	Variable duration	+	+	+	+
Cue	100ms	=	= + =	+ =	+
Fixation	400ms	+	+	+	+
Target	Max 1700ms	>><>> +	>>>>> +	+ – –>– –	+ <<<<<
		Types of target			
Congruent		> > > > >		< < < < <	
Neutral		– – >– –		– – <– –	
Incongruent		< < > < <		> > < > >	

Table 4-1: Stimuli for ANT task

Three cue types were employed: two alerting cue types that were not spatially informative (a central and a double cue), and a spatially informative cue that indicated the location of the target with 100% validity. On 25% of trials no cue was presented. Participants were asked to indicate the direction of the central arrowhead by clicking on the appropriate mouse button. On 33% the trials the direction flanker arrowheads were congruent with that of the central arrowhead; on 33% they were incongruent, and on 33% the flankers were simple arrowshafts. Participants had up to 1700ms to respond, and the target offset on response. The inter-trial interval was varied; response time was included in calculating inter-trial interval in order to produce a Gaussian distribution of intervals regardless of reaction time. Participants were asked to maintain fixation on the central cross throughout the experiment if possible, and were also encouraged to respond as quickly and accurately as possible by being told that there was a time limit "of about one and a half seconds" to their responses. Ten practice trials were given, and participants were given the option of repeating the practice block if they were unconfident of their performance. Participants undertook this task at their second session only.

Temporal Order Judgement Task:

As with the version of the task employed in Experiment 2b, participants were

required to judge which of two shapes, presented in rapid succession, appeared on screen first. One stimulus was a white, black-bordered circle, the other a white, black-bordered triangle. However, in order to ascertain whether difficulty with the task was specific to integrating the temporal order information across hemifields, or, alternatively, whether the difficulties lay in determining temporal order within the same lateral hemifield, the first of each stimulus pair was presented in one of four quadrants of the computer screen, and the second was presented in either in either the opposite horizontal or the opposite vertical hemifield. Examples are illustrated in Figure 4-1.

Each trial began with a blank grey screen, on which a black central fixation cross appeared after 600ms. After a further 1000 one shape appeared in one quadrant of the screen. The second shape appeared either in the same opposite vertical hemifield or the opposite lateral hemifield after a variable SOA. SOAs were randomly selected from six multiples of a screen refresh rate, giving values of 17ms, 33ms, 50ms, 66ms, 83ms and 100ms. The pairs of quadrants in which the shapes appeared were also varied randomly.

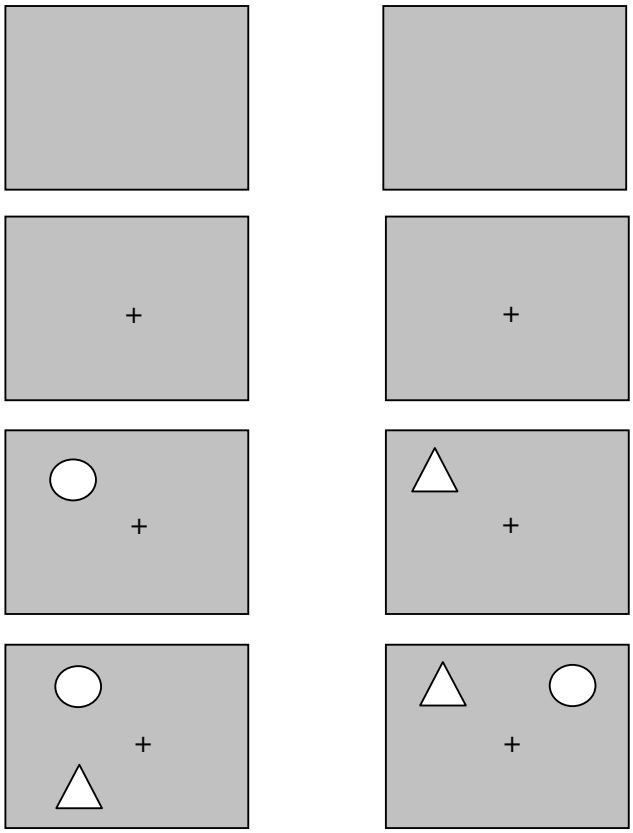

Figure 4-1: Sample stimuli pairs for the TOJ task

Each trial began with a blank screen presented for 600ms followed by the onset of a black central fixation cross. After 1000ms either a circle or a triangle appeared in one quadrant of the screen. After a variable SOA the alternate shape appeared either in the opposite horizontal quadrant in the quadrant above or below the first. Participants were asked to report which shape had appeared first. The shapes remained on screen until the participant had responded. The trial was terminated on response. In the figure, the screens portrayed on the left represent a typical within-hemifield presentation, while those on the right portray a typical between-hemifield presentation.

Dimensions of the stimuli were as for Experiment 2b and were centered 8º of visual angle from the fixation cross. Both shapes remained on screen until the subject responded. Participants were asked to make a forced choice as to which shape appeared first, and indicated their responses as in Experiment 2b. They

were informed that on many occasions the shapes might appear virtually simultaneous, in which case they should simply guess which came first.

Each participant undertook the task on two separate occasions, on which the task was presented in a total of four blocks of 192 trials, preceded on each occasion by a practice block of 16 trials. Participants were invited to repeat the practice block if they felt unsure as to how to respond. They were also told that speed of response was not important, but they were simply to respond as accurately as possible.

4.2.2 Analysis and Results

Each task and set of measures will be considered in turn, with findings summarized and interpreted briefly where appropriate. The standardized psychometric measures will be considered first, as these enable the study sample to be compared with the samples on which the measures were standardized. Next to be considered will be the non-standardized measures of single word reading and phonological skill. This will be followed by analyses of performance on the ANT, which will relate performance on task to performance on the measures of literacy. Finally, performance on the Temporal Order Judgment task will be considered, and related to performance on both the ANT and the literacy measures.

4.2.2.1 Characteristics of the sample on standardized measures

WASI:

Where all subtest scores were available, full Scale IQ (FSIQ) was estimated using the four-subtest norms from the WASI manual; in other cases the two-subtest norms were used. Where Similarities sub-test scores were not available, a proxy Verbal IQ (VIQ) was calculated by converting the T score for Vocabulary to a standardized score. The mean FSIQ for the sample was 115, with a standard deviation of 14, indicating that while the variance in the sample was similar to that in the general population (standard deviation = 15), the mean FSIQ was a full population standard deviation above the population mean (mean = 100). Only one subject scored below the normal range (85-115). Similar results were obtained for the four subtests and the Verbal (VIQ) and Performance (PIQ) subscales. Means and standard deviations for subtests, VIQ, Performance IQ (PIQ) and FSIQ are given in Table 4-2. VIQ scores were significantly and positively correlated with PIQ scores ($r=0.576$, $p<0.0001$).

Instrument/subtest		N	Mean		Std. Deviation	
			Study	Population	Study	Population
WASI	Vocabulary	40	61	50	10	10
	Similarities	34	55	50	10	10
	Block Design	40	56	50	9	10
	Matrix Reasoning	40	57	50	8	10
	VIQ	40	115	100	15	15
	PIQ	40	111	100	13	15
	FSIQ	40	115	100	14	15
WRAT	Reading	40	99	100	13	15
	Spelling	40	99	100	16	15

Table 4-2

Standardized scores Means and standard deviations of WASI and WRAT scores in the study sample. Population means and standard deviations are given for comparison.

Single sample t-tests were used to compare each subtest and subscale with the population mean: all were significantly higher than the population mean ($p<0.001$ in all cases).

WRAT scores:

The mean standard score on the both the Reading and Spelling subtest was 99, with standard deviations of 14 and 16 respectively. Single sample t-tests indicated that this mean was not significantly different from the population mean of 100. Standard deviations were close to the population standard deviation of 15, and paired sample t-tests indicated that mean differences between Reading and Spelling scores were not significantly different from the population mean. Means and standard deviations for WRAT scores are also given in Table 4-3.

Means, and maximum and minimum scores for WASI VIQ, PIQ and FSIQ scores, and WRAT Reading and Spelling scores are shown graphically in Figure 4-2, expressed in standard deviations from the population mean.

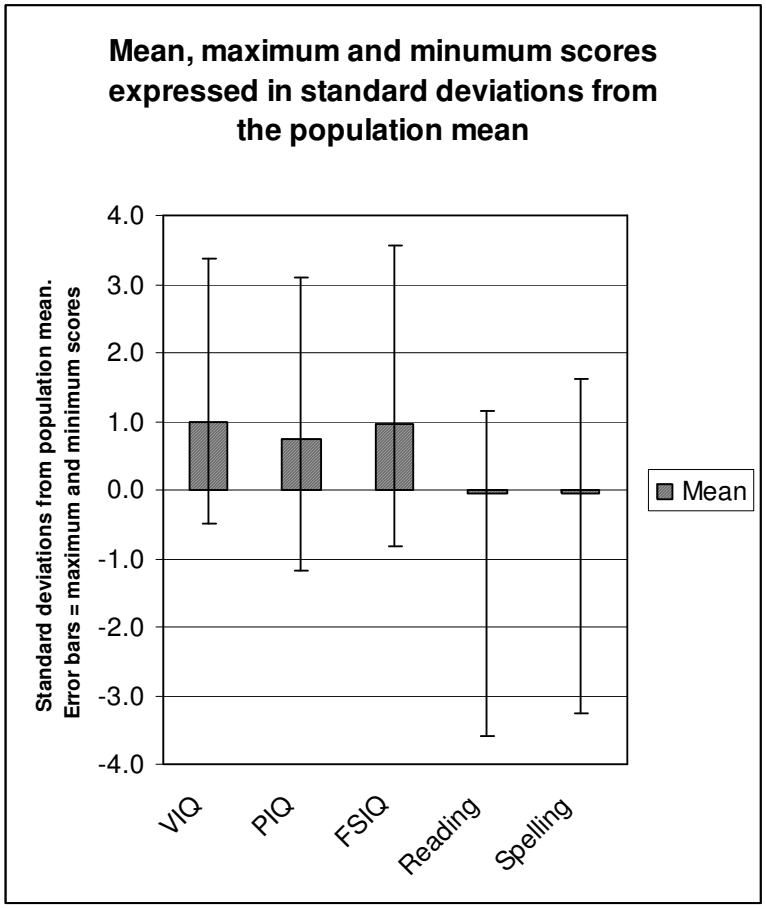

Figure 4-2: Distribution of standardized scores relative to population mean

Hatched bars in this figure indicate mean VIQ, PIQ and FSIQ scores on the WASI, and WRAT Reading and Spelling scores. Error bars indicate maximum and minimum scores. Scores are expressed as z-scores, based on population means and standard deviations. Mean WASI scores were well above the population mean, while WRAT scores were not; minimum WRAT scores were over three standard deviations below the population mean.

Paired sample t-tests were used to compare WASI full scale and subscale scores with WRAT scores. These indicated that WASI standard scores were

significantly higher than the WRAT scores ($p<0.001$ in all cases). The mean difference between FSIQ scores and both Reading and Spelling scores was 15, indicating a discrepancy equal to a full population standard deviation between these measures. Mean differences between FSIQ and subscales and each WRAT measure are given in Table 4-3.

	Paired Differences	t	df	Sig. (2-tailed)
VIQ - WRAT Reading	15.5	9.957	39	0.000
PIQ - WRAT Reading	12.1	6.441	39	0.000
FSIQ - WRAT Reading	15.4	10.979	39	0.000
VIQ - WRAT Spelling	15.4	9.303	39	0.000
PIQ - WRAT Spelling	12.0	5.290	39	0.000
FSIQ - WRAT Spelling	15.3	9.179	39	0.000

Table 4-3

WASI versus WRAT scores in sample. Results from paired-sample t tests comparing WASI scores with WRAT scores. Mean discrepancy between FSIQ score and WRAT Reading and Spelling scores was 15, a full standard deviation.

DAST scores:

The DAST generates an At Risk Quotient that is derived by taking the average number of deficit points scored on each of its 11 items. Cut-off scores for calculating deficit points are provided in the DAST manual (Fawcett and Nicolson, 1998), and were derived from a sample of 600 participants drawn from the general public on which the test was standardized (Fawcett and Nicolson, 1998). They are based on stanine scores for each item in each of several age bands. A score that falls within the lowest stanine for the participant's age band

scores 3 deficit points, a score in the second lowest stanine scores two deficit points, and a score in the third lowest stanine scores 1 deficit point. Of the 11 items of the DAST, only nine were administered in this study. Postural Stability was not included in the battery, owing to the physical fragility of a number of participants. The Non-Verbal Reasoning item was also excluded, as a fuller estimate of general cognitive function was obtained for each participant from the WASI. In order to compare the prevalence of any given ARQ score in our sample with the population on which the DAST was standardized, it was therefore important to compensate for the omission of these two items. As a proxy for the Non-Verbal reasoning item, stanine scores on the Matrix Reasoning subtest from the WASI were calculated and used to generate deficit points to substitute for Non-Verbal Reasoning deficit points on the DAST. To compensate for the omission of Postural Stability from the battery, one third (0.33) of a deficit point was added to each subject's total deficit point score, as this represents the average deficit point score in the DAST standardization sample. The total deficit score for each participant was then divided by 11 to generate ARQ scores that could be used to compare the prevalence of ARQ scores in the study sample with the prevalence in the DAST standardization sample[3].

---

[3]Alternative forms for Phonemic Segmentation (words matched for written frequency and phonemic complexity), Rapid Naming (picture items reshuffled), and Backward Digit Span were devised for two participants who were familiar with the test items. The TOWRE Sight Word reading efficiency score was substituted for One Word Reading, and the Phonemic Decoding Efficiency score for Nonsense Word reading (stanines calculated from norms for the highest age band available). Two Minute Spelling was replaced by stanine scores from WRAT Spelling scores. One Minute Writing, Verbal Fluency, and Semantic Fluency were omitted, and ARQ scores calculated on the basis of the remaining items. One further subject did not take the spelling and writing measures on the DAST owing to a hand injury. For this subject, a deficit

The proportion of participants in the study sample who scored an ARQ of 0.7 or more (indicating at least a mild "at risk" score) was 32.5% (18 participants) as compared with a prevalence of 22.6% in the DAST standardization sample. A chi square test indicated that this was not a significantly greater prevalence. Study sample prevalence of scores in each of seven ARQ bands was then compared with the prevalence of scores in that band in the DAST standardization sample (Figure 4-3). This comparison indicated that the study sample exhibited a greater prevalence (23%) of scores in the borderline.7-.9 range ("mildly at risk") than the standardization sample (8.6%) A chi square test indicated that this higher prevalence of borderline scores in the study sample was significantly higher [chi square after Yates' continuity correction = 6.520, df=1, $p<0.05$] than its prevalence in the standardization sample[4]

---

score to replace the One Minute Spelling was calculated from her WRAT Spelling score, and One Minute Writing was omitted from her ARQ calculation, which was then based on 10 items.

[4] This remained true when pro-rated ARQ scores based on 10 items (excluding the notional Postural Stability scores) were used.

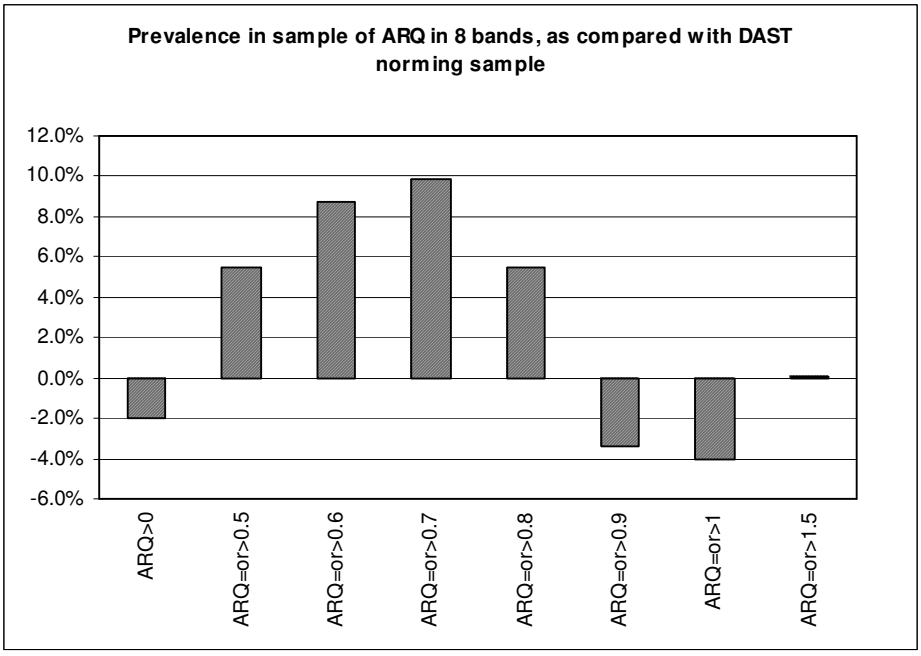

Figure 4-3: Prevalence of ARQ scores in sample

Hatched bars in this figure indicate the percentage greater or lesser prevalence of scores in each ARQ band as compared with the DAST standardization sample. The study sample showed a greater prevalence of borderline ARQ scores than would be expected from a random sample from the population.

The DAST does not distinguish between ARQ scores that are due to specific reading disabilities (dyslexia) and those that are due to more general cognitive deficits. This is because at least two contributing subtests, Semantic Fluency and Non-Verbal Fluency, are designed to tap general cognitive function. Given the high mean IQ in the sample, one possibility was that the high prevalence of borderline, rather than "strongly at risk" ARQ scores in the sample might be accounted for by downward pressure from high general cognitive function on the ARQ scores of participants who nonetheless displayed dyslexic traits. The profile

of the sample on individual DAST subtests was therefore compared with the profile of the DAST standardization sample.

Prevalence rates in the study sample for 1 or more deficit points were calculated for each DAST subtest, and compared with prevalence rates in the standardization sample using chi square tests. Chi square values for individual items showed a significantly greater prevalence of deficit scores on two indicators typical of dyslexia, namely Nonsense Passage Reading (Chi square = 15.916, df=1, $p<0.0001$) and Phonemic Segmentation (chi square = 4.472, df=q, $p<0.05$). Conversely, the study sample showed a significantly lower prevalence of deficits on the two indicators of general intelligence, Non-Verbal Reasoning (as indicated by the proxy measure of the Matrix Reasoning subtest from the WASI) (chi square=8.283, df=1, $p<0.01$) and Semantic Fluency (chi square =13.255, df=1, $p<0.001$) than the standardization sample (Figure 4-4)

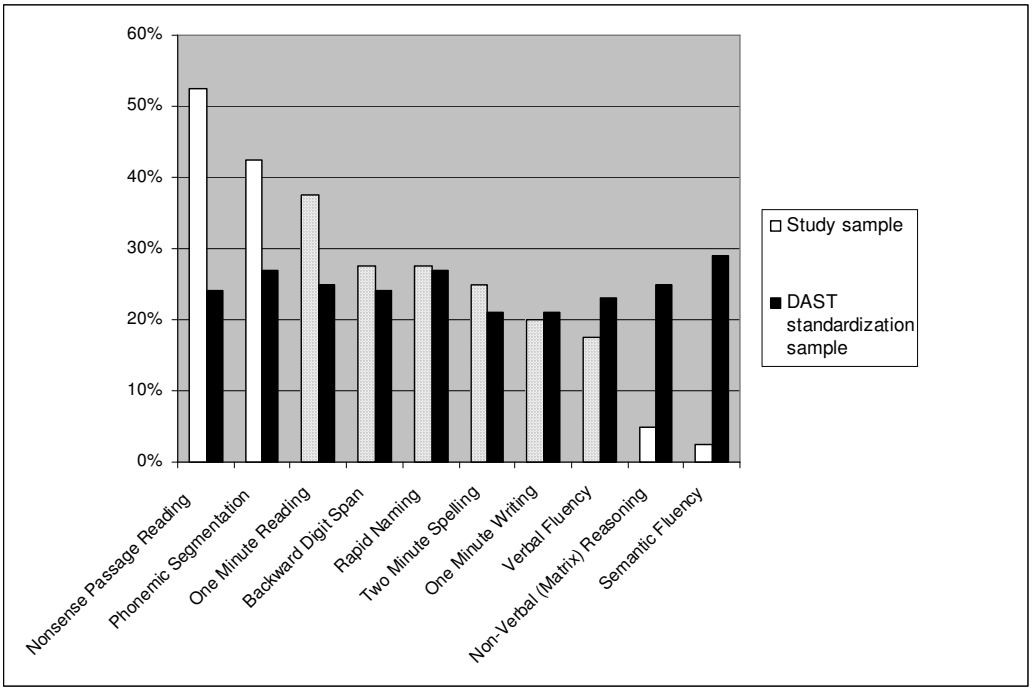

Figure 4-4: Prevalence of DAST subtest deficits in sample

Prevalence of deficits in the study sample (white bars) and in the DAST standardization sample (black bars) for each DAST subtest. Study sample prevalences that are significantly different from prevalences in the DAST sample are shown unshaded. The profile of deficit prevalence in the sample is resembles the pattern of deficits seen in a well-compensated dyslexic subject. Despite normal general cognitive function as indicated by a lower than average prevalence of deficit scores on the Semantic fluency and Non-verbal Reasoning/Matrix Reasoning, reading, writing and spelling scores deficits have the same prevalent as the general population, and deficits on the Phonemic Segmentation and Nonsense Passage Reading items are more prevalent.

Brown ADD scale:

The Brown ADD manual gives the mean and standard deviation of raw scores for their non-clinical phase 2 normalization sample (Brown, 1996). This comprised 93 participants drawn from the general population, who had a mean score of 30.01 (sd=17.48). The mean raw score in the present study sample was 37.38 (sd=18.52). An independent samples t-test (with degrees of freedom adjusted for unequal samples) indicated that the study sample mean was

significantly higher than that of the non-clinical standardization sample [t (70)= 2.138, $p<0.05$].

The Brown ADD scale also generates five cluster scores representing five aspects of attentional deficit, namely Activation (organizing and activating to work); Attention (sustaining attention and concentrating); Effort (sustaining energy and effort); Affect (Managing affective interference), and Memory (utilizing working memory and accessing recall). A series of chi square tests were used to determine whether significantly more than half the study sample scored above the median of the ADD non-clinical standardization combined sample (n=143) on each cluster. On all clusters except for Affect, more than half the study sample scored above the standardization median, but only in the case of Memory (70%) was the proportion significantly higher than the standardization sample median (Table 4-4).

		Proportion	Chi square	df	Sig.
activation	Organizing and activating to work	63%	1.962	1	ns
attention	Sustaining attention and concentrating	55%	0.313	1	ns
effort	Sustaining energy and effort	60%	1.255	1	ns
affect	Managing affective interference	35%	2.829	1	ns
memory	Utilizing working memory and accessing recall	70%	5.047	1	<.05

Table 4-4: Brown ADD scales: prevalence of cluster scores in sample.

This table shows the proportion of the study sample whose scores were higher than the Brown ADD scale standardization sample median on each cluster. Although on four out of the five clusters more than half the sample scored higher than the standardization sample median, only on the Memory cluster was this proportion significantly higher.

As the recruiting strategy in this study was primarily designed to generate a sample of individuals from a single population that would be likely to exhibit a higher than average prevalence of traits associated with dyslexia, it was important to check that the variance on the standardized measures within each family represented was not less than the between-family variance, or that participants who participated as a couple were not different to participants who participated alone. Analysis was carried out on the 30 participants who had a partner in the study. Each of the fifteen couples was given a family ID code, which was then entered as a random variable in a series of univariate ANOVAs. Dependent measures were, in turn, all the above measures that were measured on an interval scale, namely: VIQ, PIQ, FSIQ, WRAT Reading, WRAT Spelling and ADD. In addition, an IQ-Achievement discrepancy variable was computed by subtracting the mean WRAT scores from FSIQ scores and also treated as

a dependent variable. In none of these analyses did the F test indicate that the within-family variance was significantly less than the total between-family variance. A similar series of ANOVAs was carried out on the whole sample, with a code indicating whether the participant was one of a "couple" or a "single" entered as the random factor. In no case did these ANOVAs indicate a significant difference in the mean scores of those who participated as a couple, and those who participated alone. Furthermore, Levene's test of equality of variance indicated that the variance was not significantly different in these two groups.

4.2.2.2 Standardized scores: Interim summary and interpretation:

The parents of dyslexic children who made up this study sample displayed, as a group: higher than average IQ; reading and spelling scores that were significantly lower than their IQ scores; cardinal but mild dyslexic traits (namely those manifest in poor nonsense word reading and phonological processing); and higher than average scores on a measure of Attentional Deficit Disorder. These results suggest that the sample included a number of participants with an underlying phonological deficit that was largely compensated for in real-word reading and spelling tasks, but remained manifest in reading tasks involving unfamiliar words. The results also suggest that attentional deficits, whether or not directly or aetiologically related to any phonological deficits, were more prevalent in this sample of parents of dyslexic children than would be expected in a sample drawn from the general population.

Because of the finding that between-family variance was not greater than the within-family variance on measures of IQ, Achievement, IQ-Achievement Discrepancy, or ADD, participants were regarded as independent for the purposes of subsequent analyses.

4.2.2.3 Non-standardized literacy measures:

Phoneme Swap:

This task, which was designed to be difficult, unsurprisingly had a floor for participants who performed poorly on the Phonemic Segmentation item from the DAST. The Phonemic Segmentation item conversely, as expected, had a ceiling for more able participants. A composite "Phonemic Manipulation" score was therefore derived by adding half the score achieved on Phoneme Swap task to the total score on the DAST Phonemic Segmentation score. This composite score had neither of the truncated range problems of the two separate items, but had a distribution that departed significantly departure from normality [Shapiro-Wilks (40)= 0.906, $p<0.01$]. As the distribution was negatively skewed (-.991), scores were raised to the power of 2 for the purposes of parametric analyses and this produced a distribution in the sample that did not depart significantly from normality [Shapiro-Wilks (40)= 0.955, $p=.114$].

Regular, irregular and non-word reading

Accuracy scores: The distribution of accuracy scores for all regular and irregular words exhibited a ceiling effect, as did scores on one-syllable non-words. Total

non-word scores did not show a ceiling effect, but the distribution of scores departed significantly from normal [Shapiro-Wilks (40) =0.831, p<0.001]. As they were negatively skewed (-1.716), scores were raised to a power of 2. This produced a normal distribution in the sample [Shapiro-Wilks (40) = 0.957, p=0.128], and the normalized scores were used in all parametric analyses. The normalized Phonological Manipulation scores correlated significantly with the normalized non-word reading accuracy scores (Pearson correlation coefficient=0.497, p<0.001).

Word reading latencies: Two participants failed to read any three syllable non-word correctly, and one participant failed to read any two or three syllable non-word correctly. These participants were therefore excluded from all analyses of reading latencies. Median reading latencies were normally distributed for all types and lengths of words across the remaining participants. A repeated-measures ANOVA was performed with three levels of word type (regular, irregular and non-word) and three levels of length (one, two and three syllables). This analysis indicated a significant main effect of word type [F(1.21, 43.53)[5] =57.009, p<001]. Examination of the means revealed, somewhat counter-intuitively, that irregular words were read significantly faster than regular words [contrast: F(1,36)=12.429, p<0.001]. These in turn were read significantly faster than non-words [contrast: F(1,36)=66.726, p<0.001].

[5] After Greenhouse-Geisser correction for violation of sphericity

There was also a main effect of length [$F(1.22,44.23)^{5}$ =97.347, p<0.001]. Contrasts revealed that two-syllable words were read significantly faster than three-syllable words [F(1, 36)=52.347, p<0.001], and that one-syllable words were read significantly faster than two-syllable words [F(1,36)=76,661, p<0.001). However, there was also a significant interaction between type and length [$F(1.39,49.92)^{8}$=32.429, p<0.001]. Contrasts revealed that this was accounted for by significantly greater effect of length on non-word reading latencies, at both the two-syllable and three-syllable level, as compared with the effect of length on both types of real words. The interaction is plotted in Figure 4-5 and statistical tests of the interaction contrasts are given in Table 4-5.

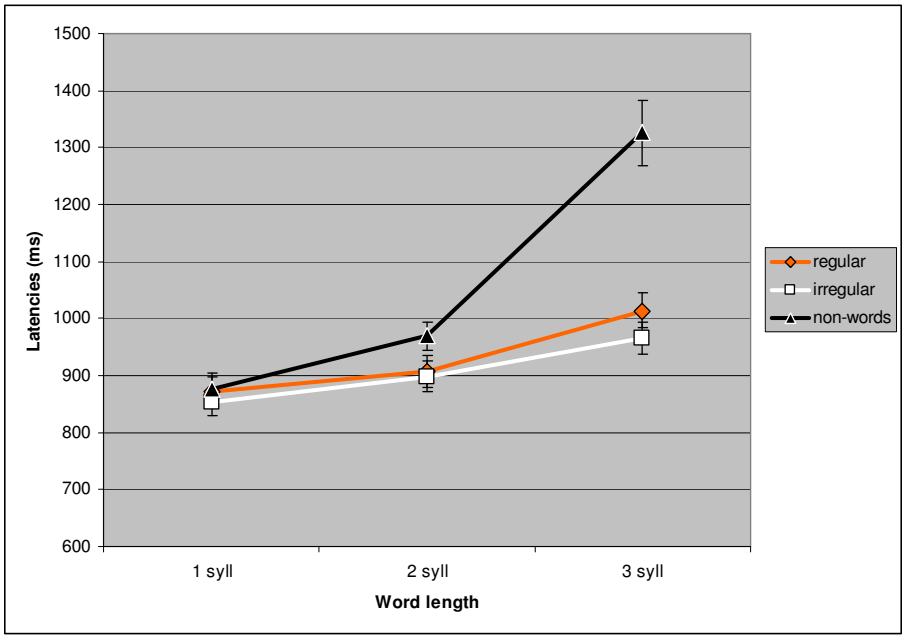

Figure 4-5: Word reading latencies: type x length interactions

Irregular words were read faster than regular words; non-words were read most slowly. The type x length interaction indicates that the cost of length for long words was greater than for real words.

A further ANOVA carried out on real words alone, revealed no significant interaction between real word type and length, although the length effects remained significant, one-syllable words being read significantly faster than two-syllable words, which in turn were read significantly faster than three syllable words.

interaction		**df**			
type	**length**	**Hypothesis**	**error**	**F**	**sig.**
regular v. non-words	1 syllable v. 2	1	36	13.132	0.001
	2 syllable v. 3	1	36	24.840	0.000
	1 syllable v. 3	1	36	46.063	0.000
irregular v. non-words	1 syllable v. 2	1	36	7.950	0.008
	2 syllable v. 3	1	36	31.310	0.000
	1 syllable v. 3	1	36	43.794	0.000
regular v. irregular	1 syllable v. 2	1	36	ns	
	2 syllable v. 3	1	36	ns	
	1 syllable v. 3	1	36	ns	

Table 4-5:

Word reading latencies: type x length interactions

Significance of contrasts for word type x word length interactions in predicting reading latencies. The effect of length on reading latencies was significant for all three word types, but was significantly greater for non-words than for real words (both regular and irregular).

To summarize these reading latency findings: latencies were slowest for non-words, faster for regular words and fastest for irregular words; latencies for all word types were slower for two-syllable than for one-syllable words, and for three-syllable than for two syllable words; the effect of length was greater for non-words than for real words.

Normalized Phonological Manipulation scores were then entered as a covariate into a 3 x 3 ANCOVA of reading latencies, with three levels of word type (regular, irregular and non-words) and three levels of length (one, two and three syllables), and also into an ANCOVA non-words alone, with the three levels of length only. Unlike this measure's strong positive correlation with non-word reading accuracy, the measure made no significant prediction regarding overall word reading latencies, nor was there any significant interaction between Phonological Manipulation and word-type, word-length, or word-type x length. Phonological Manipulation scores made no significant prediction for non-word reading latencies alone, nor did it show any significant interaction with word length.

4.2.2.4 Interim summary and discussion: literacy measures

In this study sample, the majority of participants read real words with a high degree of accuracy. Non-words, on the other hand, tended to cause considerably more problem. This finding is consistent with the results from the DAST battery, indicating that while the sample as a whole showed a prevalence of deficits real-word reading that was not different to that in the general population, the prevalence in the sample of deficits on a nonsense-word reading task was significantly greater than that in the general population. Phonological skill, as measured by the composite Phonological Manipulation score, accounted for 25% of the variance in non-word reading. This finding supports the interpretation given above regarding the DAST profile found in the sample, namely, that a

number of participants in the sample displayed a phonological deficit that manifest itself in poor non-word reading.

Word reading latencies were longer for long words than for short, and this length effect was more pronounced for non-words than for real words. Fastest latencies were found for irregular words, as compared not only with non-words but for regular real words matched for frequency. Phonological Manipulation scores made no significant prediction for reading latencies of words of any type or length.

4.2.2.5 Attentional Network Task

Across the sample, median accuracy for all target types was 95%. However, two of the participants proved problematic. One participant was incorrect for all but one of the incongruent trials, indicating that she had consistently responded to the direction of the flankers rather than the direction of the target. As this suggested that she had not correctly understood the task, this subject was also deleted from all analyses involving this task. Of the remainder, one participant's mean accuracy score (76%) was more than three times the interquartile range from the group mean, with very little variance in accuracy across all trial types. Her data was therefore deleted from all analyses of performance on this task. Mean accuracy in the remaining participants was 97%. Reaction Times (RTs) to correct responses were therefore used as the dependent measure in the initial analyses. Median RTs (because of the likelihood of RT distributions being positively skewed in some participants) for each participant for each trial type

were calculated.

Across participants, the distribution of median RTs for each trial type tended to be positively skewed. Natural logs were therefore taken of median RTs for all trial types. Kolmogorov-Smirnov (K-S) and Shapiro-Wilks tests indicated that the distribution of these transformed values did not depart significantly from normal. A four-by-three repeated-measures ANOVA was conducted on these normalized RTs for the remaining 38 participants, with four levels of cue type (single, double, spatial and none) and three levels of target flanker type (congruent, incongruent and neutral). This analysis indicated significant main effects of cue type [$F(3,111)= 53.280$, $p>.001$].

In order to obtain measures of the "efficiency" of the three postulated attentional networks, planned comparisons were therefore made of the effect of a double cue, v. no cue (to test the proposed "alerting" effect) and of the spatial cue v. the central cue (to test the proposed "orienting" effect). These confirmed a significant "alerting" effect (RTs on trials with a double cue were significantly faster than RTs for targets with no cue, $p<0.001$), and a significant "orienting" effect (RTs for trials with a spatial cue were significantly faster than RTs for trials with a central cue, $p<0.001$). Additional *post hoc* comparisons between RTs for the spatial cue and RTs for the double cue, assumed to be as spatially uninformative as the central cue, showed that the spatial cue gave only a

marginal benefit over the double cue[6], and that the double cue also gave a marginally greater benefit than the central cue[7].

The ANOVA also showed a main effect of flanker type [$F(1.25, 46.268)^{5}$= 296.407, $p<0.001$). Fan and colleagues (Fan et al., 2002) suggest that a comparison between RTs to targets with congruent and incongruent flankers indicate the strength of the "executive" attentional network. A planned comparison between mean RTs to these two flanker types revealed a significant RT cost for incongruent flankers ($p<0.001$). There was no significant difference between RTs to targets with congruent and neutral flankers, even before post hoc corrections.

The ANOVA also indicated a significant cue by flanker type interaction [$F(6,222)=17.218$, $p<0.001$]. Examination of simple contrasts and post hoc tests of simple effects delineated the nature of the interaction effects, which are shown graphically in Figure 4-6. Firstly, for targets with incongruent flankers, spatial cues conferred significantly ($p<0.001$) more benefit than either of the other cue types, which, in contrast to their effect on RTs to trials with other flanker types, did not confer any benefit significant benefit over the no-cue condition. Fan and colleagues (Fan et al., 2002) also observed this interaction.

---

[6] p=.014, p=.078 after Sidak's adjustment for multiple comparisons

[7] p=.016, p=.093 after Sidak's adjustment for multiple comparisons

[8] After Greenhouse-Geisser correction for violation of sphericity

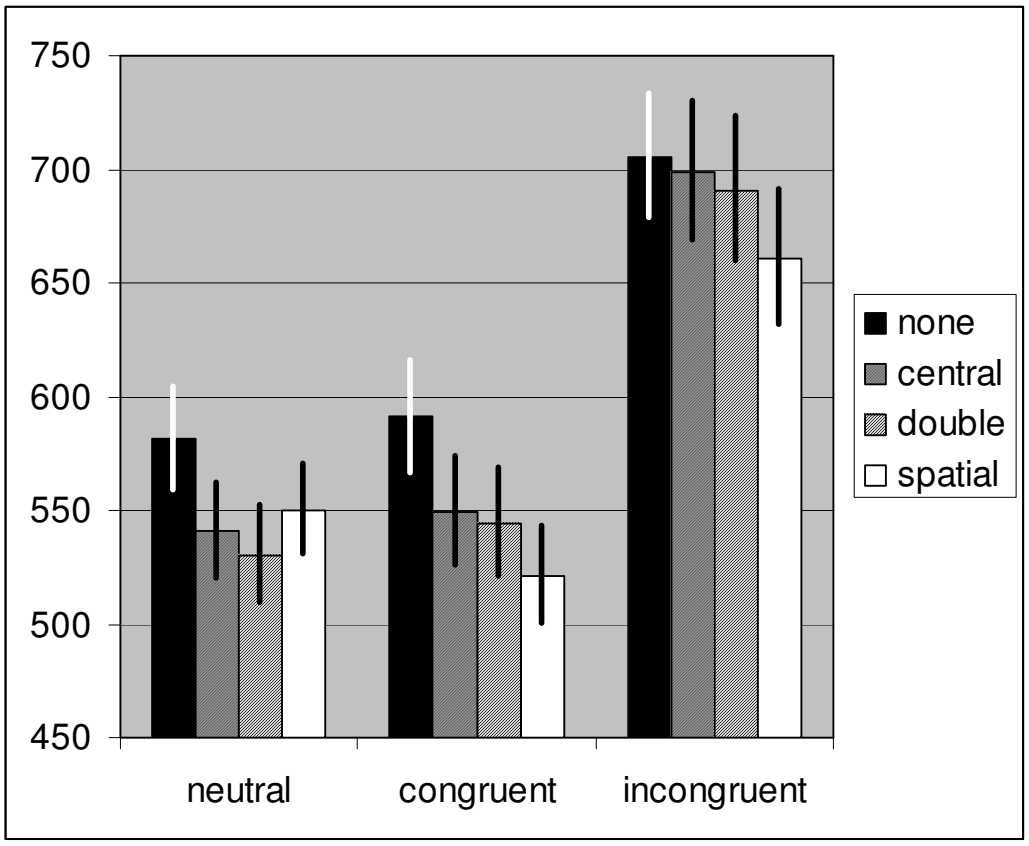

Figure 4-6: Reaction times for each trial type in the ANT.

Error bars represent 95% confidence intervals. Natural logs of median reaction times were analyzed, and have here been transformed back into milliseconds.

RTs were greatest for trials in which the flankers were incongruent with the target. For these trials, only the spatial cue conferred a significant RT benefit. For targets with congruent targets, the alerting cues conferred an advantage over no cue, and the spatial cue conferred a further benefit. For targets with neutral flankers, all three cues conferred an advantage over no cue, however, the spatial cue conferred no additional benefit over the alerting cues; indeed it conferred less benefit than either of the two alerting cues. Of these, the double cue conferred significantly greater benefit. In contrast, for targets with congruent flankers, all

cues conferred greater benefit than the no-cue condition, but again, the spatial cue conferred significantly greater benefit than either of the spatially neutral cues.

However, in this sample, a second interaction effect is apparent in the neutral flanker condition. For targets with neutral flankers, while all cue types conferred a significant benefit over the no-cue condition, the spatial cue conferred significantly less benefit than either the central or double cue. This interaction indicates that any net benefit of the spatial cue was confined to the two types of trials in which targets had to be located in the centre of a row of similar distractors, regardless of whether the distractors were congruent or incongruent with the target. Where the target was surrounded by dissimilar neutral flankers, the spatial cue conferred no net benefit – indeed less benefit – than either a central or double cue (Figure 4-7).

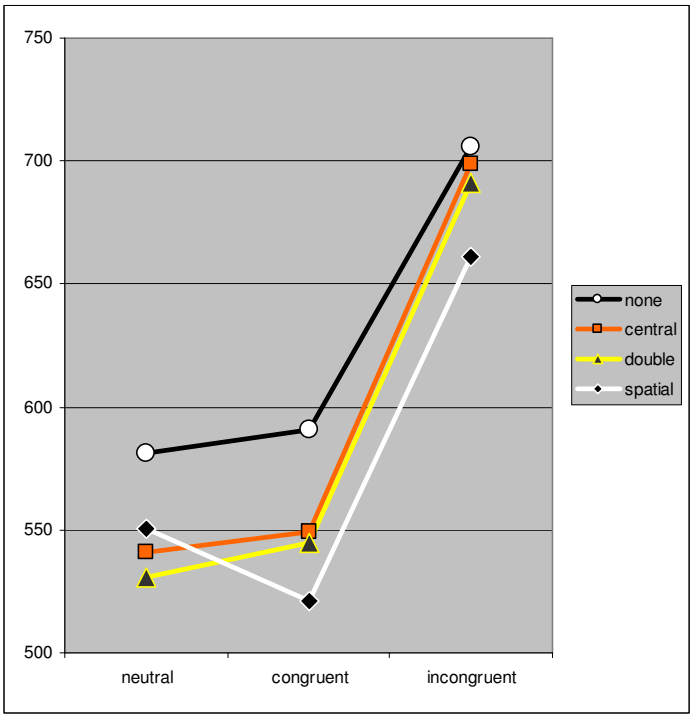

Figure 4-7: Interactions between cue type and target type.

RTs were longer for incongruent trials regardless of cue type. However the cost of incongruency was less for spatially cued targets than for other cue conditions. The Spatial cue conferred benefits over and above the alerting effect for both congruent and incongruent targets but not for neutral targets.

In a second series of analyses, raw median RTs were used to generate the three attentional parameters described by the Fan and colleagues (Fan et al., 2002). These were computed as follows: for the "Alerting" parameter, the mean of each participant's median RTs for all trial-types with double cues were subtracted from the mean of median RTs for trial-types with no cue. For the "Orienting" parameter, the mean of each participant's median RTs for all trial-types with a spatial cue was subtracted from the mean of median RTs for trial-types with central cues. For the "Executive" attention parameter, the mean of each

participant's median RTs for trial-types with congruent flankers was subtracted from the mean of median RTs for trial-types with incongruent flankers.

The very high median accuracy scores across the sample meant that ceiling effects precluded the parametric analysis of accuracy scores alone. However, the distribution of accuracy scores in the incongruent flanker condition was markedly greater than in other conditions with 12 out of the 38 (32%) of participants scoring below 95% accuracy. This raised the possibility that for these subjects, interference for targets with incongruent flankers may have been reflected in lower accuracy scores instead of, or as well as, longer RTs, and that therefore RT difference scores for this comparison might tend to underestimate the extent of flanker interference for these subjects. For estimates of "Executive" attention therefore, an "inverse efficiency score" (Townsend and Ashby, 1983) was computed by weighting the RT differential between trials with congruent and incongruent flankers by the congruent/incongruent accuracy ratio.

The means, ranges and standard deviations of all three attentional parameters are given in Table 4-6

	N	Minimum	Maximum	Mean	Std. Deviation
alerting	38	-13	109	37	27
orienting	38	-29	75	19	25
executive (weighted by accuracy ratio)	38	36	318	145	63

Table 4-6:

Estimates of efficiency in three attentional networks

Means, standard deviations, minimum and maximum values for the three attentional parameters, expressed in milliseconds: Alerting: No cue RTs -double cue RTs; Orienting: central cue RTs-spatial cue RTs; Executive: (Incongruent flanker RTs-congruent flankers RTs) x congruent flanker accuracy/incongruent flanker accuracy. No participant had a negative value for the Executive parameter; one participant had a negative Alerting parameter value; 10 participants had a negative Orienting parameter value.

It was of note that while only one participant had a negative Alerting parameter score, and no participants had a negative Executive parameter score, 10 participants scored less than 0 on the Orienting parameter. One explanation for this last finding could be that low Orienting parameter scores simply reflect a weak or absent orienting, and that negative scores are simply due to noise. However, another explanation could be that for some participants, the central cue, presented at fixation, is of greater salience than the spatial cue. This explanation would predict that participants who fail to show a benefit for the spatial cue over the benefit conferred by the central cue, would also tend to gain less benefit from double cue, which, like the spatial cue, is presented off-fovea. If so, scores on the Orienting parameter should correlate with difference scores calculated by subtracting RTs for double cue trials from RTs from central cue trials. Both cue-types are spatially uninformative, but the first is presented above

and below fixation, the second at fixation.

RTs for trials with central cues were subtracted from RTs for trials with double cues, and correlation coefficients calculated between these difference scores and the Alerting[9] and Orienting parameters respectively. There were significant zero-order correlations between the central-double difference scores and both the Alerting parameter ($r=450$, $p<0.01$) and with the Orienting parameter ($r=.452$, $p<0.01$); when both Alerting and Orienting were entered as covariates into a multiple regression model, each accounted for significant separate sources of variance [Alerting: Beta=0.457, $t(35)=3.533$, $p<0.01$; Orienting: Beta=0.459, $t(35)=3.549$, $p<0.01$].

This result indicates that the higher either the Alerting or Orienting score, the greater the benefit from the double cue relative to the central cue; the lower either the Alerting or Orienting score, the lower the benefit of a double cue over a central cue. This finding is consistent with the hypothesis that low or negative Orienting scores are associated with a reduced benefit from the other off-foveal cue presented, namely, the double cue[10]. In short, RTs for double and spatial

---

[9] A K-S test for normality on the Alerting parameter scores indicated a distribution that departed significantly from normality [K-S(38)=..145, $p<0.05$], although the S-W test did not. As these scores had a marginally significant positive skew ($z=1.84$), they were normalized by raising the scores to a fractional power. As one participant had a negative score on this parameter, the scores normalized by adding this subject's score to the scores of each participant and raising the result to the power of .5. This produced a distribution of scores that was not significantly different from a normal distribution [K-S (38)=.092, $p>.200$].

[10] To check that this result was not an artefact of the same baseline being used in two of the subraction variables, a further series of multiple regressions were conducted, with mean median rts for each of the three cue condition, with the no-cue condition as control for overall RT. Each cue condition in turn was entered as the dependent variable, with the remaining two as predictors. These regression models confirmed the results found with subtraction variables, and indicated

cues share variance that is over and above that accounted for by the alerting effect alone.

Fan and colleagues (Fan et al., 2002) suggest that the three attentional parameters generated by the ANT represent three independent attentional networks. Bivariate correlations were performed on the Alerting, Orienting and the accuracy-weighted Executive [11]parameters, and no significant correlations were found (Table 4-7)

---

that RTs for the double and central cue accounted for significant and separate variance in RTs to the spatial cue. In contrast, RTs to both the double cue and central were only significantly predicted by the RTs to the spatial cue. See Appendix ?? for regression coefficients and tests of significance.

[11] Both the raw and weighted Executive parameter scores were significantly different from normal on the S-W test, and positively skewed. These were successfully normalized by taking natural logs, and the log of the weighted score was used subsequent parametric analyses. This had a normal distribution in the sample [S-W (38)=.964, p=.263].

	N	Pearson Correlation Coefficient	Sig.
Alerting : Orienting	38	-0.015	0.928
Alerting : Executive	38	-0.174	0.296
Orienting : Executive	38	-0.125	0.454

N=38

Table 4-7

Correlations between ANT parameters. Pearson correlation coefficients computed between each of the three attentional parameters from the ANT. The lack of any significant correlations supports the inference that the parameters reflect independent attentional networks.

4.2.2.6 ANT: Interim summary and discussion

Significant Alerting, Orienting and Executive effects were found in this sample. Orienting effects were smallest, and in some cases were negative, particularly in the case of targets with neutral flankers, leading to the hypothesis that for some subjects and some conditions, an alerting cue at fixation conferred as much, if not more, benefit than the spatial cue. This in turn led to the prediction that lack of benefit from the spatial cue, as compared with the central cue, might be mirrored in a similar lack of benefit from the double cue, also presented off-fovea, as compared the benefits conferred by a central cue. This prediction was confirmed, and suggests that in this sample, low Orienting scores may arise from an inability to benefit from a valid single spatial cue, that is also reflected in a reduced benefit from a double cue that is a) presented off-fovea and b) cues the two possible locations of the target.

4.2.2.7 The ANT and ADD:

Berger and Posner (2000) suggest that Attention Deficit and Hyperactivity Disorder (ADHD) is may arise from deficits in two of the postulated independent attentional networks designed to be tapped by the ANT, namely executive control, and alerting. They argue that there is little empirical support for an orienting deficit in ADHD. As the three attentional parameters were not significantly correlated with each other, zero-order correlations were computed between each attentional parameter, and both the total and cluster scores on the Brown's ADD scale. The resulting correlation coefficients are shown in Table 4-8.

	Alerting	Orienting	Executive
activation	0.074	-0.051	0.342*
attention	0.112	-0.344*	0.167
effort	0.203	-0.138	0.319
affect	-0.083	0.047	0.535**
memory	0.045	-0.252	0.168
ADD	0.133	-0.207	0.382*

N=38. *significant at alpha=.05, ** significant at alpha=.01

Table 4-8

Correlations between ANT parameters and ADD scores. Pearson correlation coefficients between the three attentional parameter scores from the ANT and scores from the Brown ADD scale. Only Executive parameter scores correlated significantly with total ADD score. Orienting parameter scores correlated significantly with the Attention cluster, while the Alerting parameters scores did not correlate significantly with any cluster.

These indicate that the Executive parameter had a significant positive correlation with overall ADD scores, and also with the Activation and Affect clusters. As the Executive parameter is a measure of the extent to which incongruent flankers disrupt task performance, a high score on this parameter is likely reflect poor

executive control of attention. This finding is therefore consistent with Berger and Posner's (2000) hypothesis regarding the efficiency of executive attentional networks in Attentional Deficit disorder. However, the Alerting parameter did not correlate significantly with total ADD scores, nor with scores on any individual cluster in this sample, while the Orienting parameter had a significant positive correlation with the scores on the Attention cluster.

It was important, therefore to test whether any of the attentional parameters from the ANT were predictive of variance shared by the five cluster scores that might represent a single underlying dimension. Multivariate ANCOVAs were therefore conducted on the five cluster scores, with each ANT parameter entered in turn as covariate. Of these, only the Executive parameter was a significant predictor on multivariate tests [$F(5, 32)= 2.832$, $p<0.05$]. This result remained significant when all three ANT parameters were entered as covariates into the model [$F(5,30)=2.960$, $p<0.05$].

This result indicated that at least one underlying variate from the five cluster scores was associated with scores on the Executive parameter. To further elucidate the nature of this variate, the sample was split by median Executive score, generating a "high cost" group, in which incongruent flanker resulted in greater performance decrement than in a "low cost" group, in which incongruent flankers were less disruptive to performance. A discriminant function analysis was then carried out, to ascertain the power of the five cluster scores to ascribe participants into either the "high cost" or the "low cost" group. This analysis

indicated that a variate with loadings from each of the five ADD clusters significantly [Wilks's Lambda (5)=0.658, $p<0.05$] discriminated between those in the "high cost" group and those in the "low cost" group. The Structure Matrix indicating the factor loadings are given in Table 4-9.

	Factor loading
affect	0.920
activation	0.549
effort	0.443
attention	0.361
memory	0.235

Table 4-9

Loadings of incongruency costs on ADD clusters. Structure matrix showing loadings for the Brown ADD scale variate that best discriminated between participants showing a "high cost" of incongruent flankers (high Executive parameter score) and a "low cost" group (low Executive parameter scores).

These indicate, as the zero order analyses suggested, that the relevant variate is a single factor that loads most strongly on Affect ("managing affective interference") and on Activation ("organizing and activating for work"), with a lesser contribution from Effort ("sustaining energy and effort"), and very little from either Attention ("sustaining attention and concentrating") or from Memory ("utilizing working memory and accessing recall").

4.2.2.8 ANT and dyslexia:

The IQ scores (VIQ, PIQ and FSIQ) were normally distributed in the sample, and so parametric statistics were appropriate to investigate the relationship between these and the ANT parameters. Correlation coefficients between IQ scores and the ANT scores were computed and are given in Table 4-10. These indicated that

the Executive parameter correlated significantly and negatively (r=-.46, p<0.01) with Performance IQ. The sign of the correlation indicates that low PIQ was associated with a high cost of incongruent flankers on the ANT. There were no other significant correlations.

	Alerting	Orienting	Executive
VIQ	0.22	0.20	0.03
PIQ	0.22	0.21	-0.46**
FSIQ	0.26	0.23	-0.22

N=38. *significant at alpha=.05, ** significant at alpha=.01)

Table 4-10

Correlations between ANT parameters and IQ. Zero-order correlation coefficients between ANT parameters and WASI IQ scores. Executive parameter scores on the ANT significantly predictor of PIQ.

Correlation coefficients were then calculated between ANT parameters and the two PIQ constituent subtests, Block Design and Matrix Reasoning. However, subtest scores on were negatively skewed in the sample, and as these scores are based on a normal distribution in a large standardization sample, normalization procedures were considered inappropriate. In order to determine which, if any, of these subtests had a significant zero-order correlations with any of the ANT parameters, a re-sampling (Fay and Follmann, 2002) technique was devised and programmed in LabView[12]. For each pair of variables to be correlated, 1,000,000 samples of 38 pairs of scores were randomly selected (with replacement) from the 38 pairs of scores in the data set. Pearson correlation coefficients were computed for each of these samples, and the median value of these was deemed

[12] Labview 7.1; National Instruments Corporation, Austin, TX. Re-sampling algorithm programmed by the author.

the optimum estimator of the true value. If both the coefficients representing the 5th and 95th percentile from the 1,000,000 samples were either both positive or both negative, this median coefficient was considered to be significant at an alpha of .05. If the coefficients representing both the 1st and 99th percentile from the 1,000,000 samples were either both positive of both negative, the median coefficient was considered to be significant at an alpha of .01. Median coefficients for these pairs are given in given in Table 4-11.

	Alerting	Orienting	Executive
Matrix Reasoning	.08	.06	-.22*
Block Design	.21	0.32*	-.43**

*significant at alpha=.05. **significant at alpha = .01

Table 4-11

Correlations between ANT parameters and PIQ subtests Median correlation coefficients between the three attentional parameters from the ANT and scores on the two PIQ subtest, Matrix Reasoning and Block Design. Executive parameter scores were significantly correlated with both subtests. Orienting parameter scores were significantly correlated with Block Design.

These indicated that both Matrix Reasoning and Block Design, the two subtests contributing to PIQ scores in the WASI, were both significantly correlated with the Executive parameter, and thus that the high cost of incongruent flankers associated with low PIQ is also associated with low scores on both its constituent subtests. Block Design scores, but not Matrix Reasoning scores, were also positively and significantly (at an alpha of 0.05) correlated with the Orienting parameter scores, indicating that a low benefit from the spatial cue is associated with low scores on Block Design. These correlations were also significant when tested with Spearman's Rho, a less powerful non-parametric test.

The same resampling technique was used to compute confidence intervals for correlation coefficients between the ANT parameters and the two WRAT achievement tests. Scores on these tests again were not normally distributed, being negatively skewed in the sample, despite being normally distributed in the population.

Median correlation coefficients are given in Table 4-12, and indicate that while the Alerting and the Executive parameters were not significantly correlated with either of the WRAT achievement measures, the Orienting parameter was significantly and positively correlated the WRAT Spelling measure ($r=.26$, $p<0.05$).

	Alerting	Orienting	Executive
WRATRD	0.10	0.08	-.00
WRATSP	-0.02	0.26*	0.03

*significant at alpha=.05.

Table 4-12

Correlations between ANT parameters and WRAT scores. Median correlation coefficients between the three attentional parameters from the ANT and scores on the two WRAT achievement subtest, Reading and Spelling. Orienting parameter scores were significantly correlated with Spelling scores

4.2.2.9 ANT, Non-word reading accuracy and Phonemic Manipulation:

Zero-order Pearson correlation coefficients were computed between the three ANT parameter scores and the normalized scores on Non Word Reading Accuracy and Phonemic Manipulation. No significant correlations were found at

an alpha of 0.05, although the Orienting parameter had a correlation with Phonemic Manipulation scores that approached significance[13] .

Expressing the ANT parameters as difference scores has a number of benefits; however use of difference scores as predictors of other variables involves the assumption that difference scores between RTs are uncorrelated with either of the raw RTs themselves, which may not be the case. For example slow speed-of-processing may result in greater trial-type costs as well as greater overall RTs. If so, regressing both RTs on the relevant dependent variable may give a better model fit by allowing independent regression coefficients for each of the two RT scores, at a cost of only one more degree of freedom. Because of the need to weight Executive parameter scores by accuracy, this approach was restricted to the orienting and alerting effects from the ANT.

Given the trend towards significance of the zero-order correlation between the Orienting parameter scores and Phonemic Manipulation scores, this regression technique was used to re-analyze their relationship. RTs to centrally-cued ANT targets and RTs to spatially cued ANT targets, both of which were normally distributed, were entered as separate predictors, with Phonemic Manipulation and Non-Word reading accuracy score in turn as dependent variables. The predictor of interest was RTs to spatially-cued ANT targets, after suppressing (by covarying out) irrelevant variance shared between this predictor and RTs to centrally-cued ANT targets. On this analysis, there was a significant partial

---

[13] Orienting: r=.307, p=.061

correlation [r=-0.42, p<0.01] between RTs to spatially-cued ANT targets and Phonemic Manipulation scores, after covarying for RTs to centrally-cued ANT targets, indicating that for a given RT to the centrally-cued ANT targets, longer RTs to spatially-cued ANT targets were associated with lower Phonemic Manipulation scores. There was no significant partial correlation between RTs to spatially-cued ANT targets and Non-Word reading accuracy.

Finally, it was postulated that even if the alerting and orienting networks are indeed independent as indicated in this and other studies (Fan et al., 2002; Rueda et al., 2004), spatial attention may involve the co-ordination of both networks. It was therefore hypothesized that performance on some tasks might be predicted by an Alerting x Orienting interaction. Two pairs of hierarchical multiple regressions were therefore performed, with Phonemic Manipulation and Non-word Reading Accuracy as respective dependent variables. For each model, Alerting and Orienting parameter scores were entered together in the first block, followed by an interaction variable representing Alerting x Orienting scores in the second block. For dependent variable Phonemic Manipulation, the addition of the interaction term resulted in no significant change to the total R^2. However, for dependent variable Non-Word Reading, for which neither parameter had proved a significant predictor, the addition of the interaction term resulted in a significant increase in the total R2 of 0.12 [F(1,34)=4.809, p<0.05]. In the larger model, both the Orienting and the interaction term were significant (p<0.05 in both cases). However, somewhat unexpectedly, while the regression coefficient for the Orienting term was positive, that of the interaction term was negative.

This negative sign for the interaction term indicates that rather than a synergistic relationship between the two variables, where high scores of one variable interact to enhance the effect of high scores in the other, and vice versa, a reciprocal, relationship between the two predictor variables appears to operate in this sample, whereby at low levels of one variable, the prediction made by the other becomes more positive.

4.2.2.10 Interim summary and discussion: ANT and literacy measures:

Executive parameter scores derived from performance on the ANT were significantly predictive of ADD scores, in particular of an ADD factor that loaded most strongly on clusters associated with resolving conflict between competing impulses, both in the emotional ("managing affective interference"), and cognitive ("organizing and activating for work") domains (Brown, 1996). It was also a significant predictor of measures of non-verbal reasoning, being a significant correlate of Performance IQ scores, and of both PIQ constituent subtests. It made no significant prediction for Verbal IQ scores, for either the reading or spelling WRAT achievement scores, or for scores on either Phonological Manipulation or Non-Word reading.

The orienting effect, in contrast, was a significant predictor of spelling and Phonemic Manipulation scores. The alerting effect was not significantly predictive of any IQ score, achievement score, or either of the non-standardized literacy measures. However, Non-word reading accuracy was significantly predicted by a reciprocal interaction between Orienting and Alerting scores,

whereby Alerting scores became more positively predictive of Non-word reading scores at low levels of Orienting scores and vice versa. A possible interpretation of this interaction is that the alerting network may serve as a compensatory network in participants in whom the orienting network is ineffective.

4.2.2.11 Temporal Order Judgment Task:

Data preparation: One of the purposes of this paradigm was to address the question as to whether participants displayed a lateral bias in trials where they were required to judge the onset order of two stimuli presented in contralateral. If so, it was of further interest to discover whether a bias against a particular hemifield was reflected in lower accuracy for trials in which both stimuli were presented within that hemifield (ipsilateral trials). On the other hand, no *a priori* hypotheses were made concerning bias between upper and lower hemifield presentations; results were therefore pooled across these conditions. Mean Accuracy scores were therefore computed for trials in which the target was presented in left or right hemifield respectively and for those in which the distractor was in the same (ipsilateral) or different (contralateral) hemifield respectively, for each SOA.

Criterion values[14], derived from Signal Detection Theory, were computed as a measure of lateral bias for contralateral trials, as for Experiments 1a, 1b, 1c and

---

[14] Criterion value =[z(hit)+z(false alarm)]/2. Before calculating z scores, a constant of .01 was subtracted from each score, to avoid values = 1.

2b, using the convention by which positive criterion values reflected a leftward attentional bias, and negative criterion values represented a rightward attentional bias. As before, a criterion value of 0 would indicate no bias. Criterion values were computed for overall responses (pooled across all SOAs), as well as to trials at each SOA. These criterion values were normally distributed in the sample. However, two participants were found to have extreme overall criterion values that exceeded three standard deviations from the sample mean. On further investigation, one of these two participants (z=3.03) was discovered to have a right visual field defect resulting from an old eye injury, and she was therefore deleted from further analyses. No known cause was found for the extreme score of the other participant, who showed an extreme bias in the opposite direction (z=-3.22), but as his score was even further from the mean than that of the participant with the known visual field defect, he was also deleted from further analyses. This left 38 participants in the analysis. Mean d′ and criterion scores for these participants for these trials are given in Table 4-13.

	SOA	Mean (St.dev)
D prime	17	0.93(0.56)
	33	0.94(0.56)
	50	1.83(0.81)
	66	2.48(0.94)
	84	2.89(1.05)
	100	3.16(1.16)
Criterion	17	-0.06(0.3)
	33	-0.04(0.34)
	50	0.02(0.33)
	66	0.03(0.25)
	84	0.03(0.23)
	100	0.06(0.21)

Table 4-13

Means and standard deviations for d′ and criterion scores for trials in which the stimuli were presented to different lateral hemifields.

Scores for the remaining participants were negatively skewed, this skewness becoming systematically greater for longer SOAs, as an increasing proportion of participants approached the accuracy ceiling Median scores and interquartile ranges are given in Table 4-14.

	Left first	Right first
	Median (interquartile range)	Median (interquartile range)
Contralateral	0.66(0.24)	0.71(0.21)
	0.66(0.18)	0.66(0.24)
	0.84(0.15)	0.82(0.21)
	0.92(0.11)	0.89(0.11)
	0.95(0.08)	0.93(0.11)
Ipsilateral	0.67(0.17)	0.67(0.16)
	0.67(0.17)	0.65(0.13)
	0.80(0.15)	0.81(0.15)
	0.91(0.13)	0.89(0.09)
	0.95(0.06)	0.94(0.11)

Table 4-14

Median and interquartile ranges of untransformed accuracy scores for each condition on the TOJ task.

Scores were normalized using Fisher's r to z transform[15] which had its greatest effect on the most skewed distributions. This succeeded in normalizing all scores except those at the longest SOA of 99ms. Scores for trials with SOAs of 99ms were therefore excluded from further analyses. Scores for trials at the remaining SOAs were entered into a 2 x 5 x 2 repeated-measures ANOVA, with two levels of trial type (ipsilateral or contralateral), 5 levels of SOA (17ms, 33ms,50ms, 67ms and 83ms), and two levels of target hemifield (left or right).

This ANOVA indicated a significant main effect of SOA [$F(1.57,58.07)^5 = 200.683$, $p<0.001$], indicating (as expected) that participants accuracy increased

[15] Transformed score = $[\log_e(1+\text{score})/(1-\text{score})]/2$. A constant = .01 was subtracted from each score before the transform to avoid values =1

with increasing SOA. There was also a significant effect of trial type [$F(1,37)=5.102$, $p<0.05$) indicating that participants were more accurate for contralateral trials than for ipsilateral trials.

Overall criterion values for the contralateral trials were then analyzed using a one-sample t-test to address the question as to whether they were significantly different from zero, which would indicate a consistent overall lateral bias across participants. This t-test indicated that criterion scores were not significantly different from 0. In other words, the sample did not show any overall lateral bias when responses were pooled over all SOAs. Criterion values for each SOA, were then entered into a repeated-measures ANOVA with five levels of SOA. This analysis indicated that criterion values were significantly lower (thus indicating a rightward bias) at short SOAs than at long SOAs [$F(2.59,95.86)^{5}$ $=2.008$, $p<0.05$]. Polynomial contrasts indicated a significant linear relationship between criterion values and SOA, with the lowest (most right-biased) values occurring for trials with the shortest SOAs [$F(1,37)=5.612$),$p<0.01$]. One-sample t-tests conducted each on criterion value separately indicated that at the shortest SOA (17ms) mean criterion values across the sample were significantly less than 0 [$t(37)=2.136$, $p<0.05$]; in other words, at the minimum SOA, the sample showed a significant rightward bias. Thus, the more difficult the trial, and therefore the greater the degree of uncertainty, the more rightwards was the bias, across the sample.

To determine whether the degree and direction of lateral bias on contralateral

trials was reflected in accuracy differentials between left-hemifield ipsilateral trials and right-hemifield ipsilateral trials, a repeated measures ANCOVA was performed on the ipsilateral trials, with two levels of hemifield presentation (left and right) and five levels of SOA. Overall criterion values from the contralateral trials were entered as a covariate. There was no significant interaction between the criterion values[16] and hemifield presentation, indicating that the degree of lateral bias on the between-hemifield trials was not reflected in better (or worse) scores on trials in which both stimuli were presented to the "preferred" hemifield, as compared trials in which both stimuli were presented to the "neglected" hemifield.

4.2.2.12 Interim summary and discussion: TOJ :

SOA was, as expected, a significant predictor of performance on all trial types. Participants were also more accurate when stimuli were presented within either the right or left hemifield than when the stimuli were presented in opposite lateral hemifields. On these contralateral trials, participants showed, as a group, a significant rightward bias at the shortest SOAs. This bias lessened as SOAs increased and the task became easier. The degree and direction of bias shown by each participant on contralateral trials was not predictive of their performance on within-left hemifield trials relative to their performance on within-right hemifield trials.

---

[16] This was also true when the criterion values for each SOA were entered.

This pattern of results raises the possibility that the right-field advantage observed for stimuli in which the target was closely followed by a distractor in the opposite hemifield may be a callosal transfer phenomenon: if the locus of the networks involved in making the temporal order judgment is in left hemisphere, this would tend to lead to a short right hemifield advantage for between-lateral hemifields without seriously compromising performance on trials in which both stimuli were presented to left hemifield. As the particular form of the TOJ paradigm employed in this study requires the target to be labeled ("circle" or "triangle"), rather than simply located spatially, a left (i.e. language) hemisphere locus for the decision making process is plausible.

4.2.2.13 TOJ task, dyslexia and ADD.

The transformed TOJ accuracy scores were entered into a series of repeated measures ANCOVA with three levels of trial type (within-right-hemifield trials, within-left-hemifield trials and between-lateral-hemifield trials) and five levels of SOA. Phonemic Manipulation, Non-word reading accuracy, ADD and FSIQ scores were entered in turn as covariates. Significant predictions regarding overall accuracy on the TOJ task were made by Phonemic Manipulation [$F(1,36)=6.866$, $p<0.05$], Non-word reading accuracy [$F(1,36)=18.702$, $p<0.001$] and FSIQ [$F(1,36)=14.897$, $p<0.001$]. ADD scores did not make a significant prediction[17]. The three significant predictors had positive regression coefficients with the TOJ accuracy scores, indicating that poor performance on the predictors

[17] $F(1,36)=3.118$, $p=.086$

was associated with poor overall performance on the TOJ task, and vice versa. Significant interactions were found between SOA and both Non-word reading [$F(1.74,62.74)^5$ =9.140, $p<0.001$] and FSIQ scores [$F(1.71,61.59)^5$=6.816, $p<0.01$.]. Polynomial contrasts indicated a significant linear relationship with SOA with the prediction being strongest at the longest SOAs [Non-word reading accuracy: $F(1,36)=12.228$, $p<0.01$; FSIQ: $F(1,36)=9.253$, $p<0.01$].

To establish whether performance on the TOJ task was accounted for by variance that was shared by the significant predictors or separate, each significant covariate was entered in combination with one other, followed by a model containing all three. When Phonemic Manipulation and Non-word reading scores were entered into the model together, Phonemic Manipulation scores failed to reach significance, while the prediction made by Non-word reading accuracy scores remained significant [$F(1,35)11.565$, $p<0.01$], indicating that it was variance shared by both Phonemic Manipulation and Non-word reading accuracy that was associated with poor performance on the TOJ task. Similarly, when both FSIQ and Non-word reading were entered together into the model, Non-word reading scores remained a significant predictor [$F(1,35)=6.702$,$p<0.05$], while FSIQ scores did not; to put this differently, non-word reading scores accounted for variance in TOJ performance even after controlling for FSIQ. When all three covariates were entered, again, only Non-word Reading scores accounted for a significant proportion of the variance in TOJ performance [$F(1,34)=6.889$,$p<0.05$].

To establish whether any of the indicators of dyslexia accounted for variance in bias on the contralateral trials, criterion values for these trials, as a measure of lateral bias, were entered into an ANCOVA with five levels of SOA. Again, Phonemic Manipulation, Non-word reading accuracy, ADD and FSIQ scores were entered in turn as covariates. None accounted for overall bias across SOAs but there was a significant SOA by Non-word reading interaction [$F(2.78,100.03)^{5} = 4.186, p<0.01$]. This interaction indicated that Non-word reading scores accounted for significantly more variance at the three shortest SOAs than at longest. The sign of the regression coefficients indicated that lower Non-word accuracy scores were associated with more leftward bias. None of the other covariates accounted for significant variance in criterion values.

4.2.2.14 Interim summary and interpretation: TOJ, dyslexia, and ADHD

Variance shared by Non-word reading accuracy scores and Phonemic Manipulation scores was a significant predictor of overall accuracy on the TOJ task, most strongly at long SOAs. Variance in TOJ performance accounted for by this source was over and above any variance accounted for by attentional deficit symptom scores or by full scale IQ scores. This source of variance therefore has the characteristics of the core phonological cognitive deficit associated with dyslexia.

In contralaterally presented trials, Non-word reading accuracy scores also accounted for significant variance in lateral bias at shorter SOAs, namely those at which earlier analyses had shown the most marked overall right-field advantage.

Poor Non-word reading accuracy scores were associated with a more leftward bias. If the right-field advantage at short SOAs seen at group level is indeed accounted for by a predominantly left-lateralized locus in the sample for the networks involved in making the temporal order judgment involved in this trial type, this finding suggests that poor non-word reading in this sample of participants may be associated with a more bilateral, or even right-lateralized, locus for the temporal order judgment process.

4.2.2.15 TOJ and the ANT:

Executive attentional network:

To explore the relationship between the flanker interference effects on the ANT and TOJ performance, the normalized, accuracy-weighted Executive parameter difference scores were used. TOJ accuracy scores were entered into a 2 by 5 by 2 ANCOVA with two levels of trial type (contralateral and ipsilateral), five levels of SOA and two levels of target location (left or right hemifield). Executive parameter scores were entered as a covariate. This ANCOVA indicated that Executive parameter scores were a significant predictor of overall accuracy [$F(1,34)=4.583$, $p<0.05$]. There was also a significant interaction between Executive parameter scores and SOA [$F(1.65,56.13)^5 =4.433$ $p<0.05$]. Examination of regression parameters and contrasts indicated a negative correlation between Executive parameter scores and TOJ performance that was greater at longer SOAs. Bearing in mind that higher Executive parameter scores reflect a greater magnitude of interference from incongruent flankers, this

indicates that poor performance on the TOJ task, particularly at longer SOAs, is associated with less efficient inhibition of response to incongruent distractors on the ANT.

As previous analyses had shown a similar prediction from Non-word reading accuracy scores, these were entered as an additional predictor to check whether Executive function scores accounted for separate additional variance in TOJ performance, as would be expected from the non-significant correlation between Executive parameter scores and Non-Word reading scores. This ANCOVA confirmed that the two covariates accounted for significant and separate variance in TOJ task performance [Executive parameter: $F(1.33)=6.604$, $p<0.05$; Non-word reading accuracy: $F(1,33)=21.642$, $p<0.0001$]. As PIQ was a significant correlate of Executive parameter scores, PIQ scores were entered as an additional covariate. Both Non-word reading accuracy and Executive parameter scores remained significant predictors of overall TOJ task accuracy [Executive parameter: $F(1.32)=4.902$, $p<0.05$; Non-word reading accuracy: $F(1,32)=16.127$, $p<0.001$]; PIQ scores did not account for any additional variance. Finally FSIQ scores were added as a covariate; again, both Non-word reading accuracy scores and Executive parameter scores remained significant predictors [Executive parameter scores: $F(1.32)=5.054$, $p<0.05$; Non-word reading accuracy: $F(1,32)=10.277$, $p<0.01$], and FSIQ did not account for any additional variance.

Next, to check for laterality effects, contralateral trials were entered into a 5 x 2 repeated measures ANCOVA, with five levels of SOA and two levels of

hemifield (within-left, and within-right). Executive parameter scores were entered as a covariate. There were no significant interactions between Executive parameter scores and hemifield. Criterion values for between-lateral-hemifield trials were then entered into a repeated measures ANCOVA with five levels of SOA. Executive parameter scores did not predict overall lateral bias, neither did they interact significantly with SOA.

Orienting attentional network:

As the orienting effect on the ANT was not reflected in accuracy scores, RT scores alone could be used to explore the relationship between the orienting effect and TOJ task performance. RTs to centrally-cued ANT targets and RTs to spatially cued ANT targets, both of which were normally distributed, were entered as separate predictors of TOJ task performance, rather than RT difference scores, as this method allows more flexible modeling of any variance shared between the two predictors (see above). The predictor of interest was RTs to spatially-cued ANT targets, after suppressing (by covarying) irrelevant variance shared between this predictor and RTs to centrally-cued ANT targets.

A 2 x 5 x 2 repeated measures ANCOVA was therefore performed with two levels of trial type (ipsilateral hemifield and contralateral hemifield), five levels of SOA and two levels of target location (left or right hemifield). RTs to spatially-cued ANT targets and RTs to centrally-cued ANT targets RTs to ANT trials with central cues and RTs to ANT trials with spatial cues were entered as covariates. After thus covarying for RTs to centrally cued trials, RTs to spatially

cued trials accounted for significant variance in mean TOJ task performance ($F(1,33)=5.310$, $p<0.05$). Regression coefficients for RTs to spatially cued trials were negative[18], indicating that slower RTs to spatially cued targets, relative to RTs to centrally cued targets, were associated with lower TOJ task accuracy.

RTs to centrally cued interacted significantly with target location and trial type [$F(2.86, 95.22)^5 =4.065$, $p<0.05$]. Trial types were therefore analysed separately. First, ipsilateral trials were analyzed in a repeated-measures 5 by 2 ANCOVA with five levels of SOA and two of target hemifield. In this analysis, RTs to spatially cued trials were again predictive of overall accuracy [$F(1,33)=5.015$, $p<0.05$] and interacted significantly with hemifield [$F(1,35)=6.030$, $p<0.05$], regression coefficients being significantly more negative for right-hemifield presentations than for left. Left hemifield and right hemifield trials were then analyzed separately in repeated-measures ANCOVA with five levels of SOA, and RTs to centrally and spatially cued targets entered as covariates. RTs to spatially-cued ANT targets accounted for no significant variance in accuracy for left hemifield trials, but accounted for significant variance in accuracy for right hemifield trials [RTs to centrally cued targets: $F(1,33)=5.709$, $p<0.05$; RTs to spatially cued targets: $F(1,33)=8.668$, $p<0.01$]. There was no significant interaction with SOA.

---

[18] Those of the suppressor variable were, as expected, of opposite sign to that of the predictor of interest.

As the Orienting parameter was significantly predictive of Phonological Manipulation scores, which in turn accuracy, it was of interest to discover whether the variance in TOJ task performance on right hemifield stimuli pairs predicted by the orienting response on the ANT overlapped with the variance accounted for by Phonemic Manipulation scores. This last analysis was therefore repeated, with Phonemic Manipulation scores as a covariate, both separately and in addition to the two RT variables. Phonemic Manipulation scores alone accounted for significant variance in TOJ task performance in these right-hemifield ipsilateral trials ($F(1,34)=4.906$, $p<0.05$); when the two RT covariates were added to the model, Phonemic Manipulation scores were no longer significant ($p=317$), while the prediction made by RTs to spatially cued ANT trials remained significant ($F(1,32)=5.236$, $p<0.05$), indicating that it was variance shared by Phonemic Manipulation scores and the orienting effect on the ANT that was predictive of TOJ task performance in right hemifield, ipsilateral trials.

Contralaterally presented TOJ task trials were then analyzed in a 5 x 2 repeated-measures ANCOVA with 5 levels of SOA and 2 levels of target location, and with RTs to centrally and spatially cued targets as covariates. Again, RTs to spatially cued targets were a significant predictor of mean accuracy on these trials [$F(1,33)=4.551$, $p<0.05$] but did not interact significantly with hemifield. When Phonemic Manipulation scores were added as an additional predictor, the F value for the orienting effect dropped, and failed reach significance [$F(1,32)=2.352$, $p=0.153$], again suggesting that shared variance between the two

predictors was what accounted for variance in TOJ performance on contralateral trials. Criterion values for contralateral trials were then entered into a repeated-measures ANCOVA with five levels of SOA, and RTs to centrally cued targets and RTs to spatially cued targets were entered as covariates. Neither covariate was a significant predictor of overall bias, nor did either covariate interact with SOA.

Alerting attentional network:

As the alerting effect on the ANT, like the orienting effect, was not reflected in accuracy scores, RT scores alone could again be used to explore the relationship between the orienting effect and TOJ task performance. RTs to double-cued ANT targets and RTs to uncued-cued ANT targets, were both normally distributed, and were entered as separate predictors of TOJ task performance. The predictor of interest was RTs to double-cued ANT targets, after suppression of irrelevant variance shared between this predictor and RTs to uncued ANT targets.

A 2 x 5 x 2 repeated-measures ANCOVA was performed, as before, with 2 levels of trial type (ipsilateral or contralateral stimuli pairs), 5 levels of SOA and 2 levels of target location (left or right hemifield). RTs to uncued ANT targets and RTs to double-cued ANT targets were entered as covariates. RTs to double-cued targets did not account for a significant portion of mean variance on the TOJ

task[19]. However, they interacted significantly with target hemifield [$F(1,32)=4.260$, $p<0.05$]. Regression coefficients for the prediction made for left hemifield TOJ targets were significantly more negative than those for right hemifield TOJ targets .

There was also a significant three-way interaction between RTs to double-cued targets and SOA x target hemifield [$F(2.42, 79.55)^5 =3.223$, $p<0.05$]. However, polynomial contrasts that this interaction did not reflect significant linear or quadratic components (cubic and quartic components only were significant) indicating that there was no plausible systematic interaction with increasing SOA.

The laterality effect was explored further by entering criterion values for the contralateral TOJ trials into an ANCOVA with five levels of SOA, and RTs to uncued trials and RTs to double cued trials as covariates. There was no significant main effect of double-cued trials, indicating that this variable did not significantly predict overall bias, nor was there a significant interaction with SOA. However the main effect approached significance [$F(1,33)=3.490$, $p=.71$], and indicated a tendency for longer RTs to double-cued ANT targets to be associated with a more rightward bias. This was consistent with the finding from the full ANCOVA that longer RTs to double-cued ANT targets were associated more negatively with left-field targets, and more positively with right-field

---

[19] Both covariates made insignificant predictions.

targets. As earlier analyses had suggested that for some participants the double-cue had had less of an alerting effect than the central cue, the ANCOVA on criterion values was repeated. Mean RTs to all trials with a non-spatially informative alerting cue (namely, double and central cues) was computed, and substituted for RTs to double-cued trials in the last ANCOVA, which became the predictor of interest. This covariate made a significant prediction for overall bias [$F(1,33)=4.174$, $p<0.05$], suggesting that the overall lateralized prediction of the alerting found when all TOJ task trial types were included in the analysis was reflected in a lateral bias on the contralateral trials.

4.2.2.16 Interim summary and discussion: TOJ and ANT

Executive attentional network

Executive parameter scores were a significant predictor of TOJ task performance, particularly at long SOAs; there were no laterality effects associated with this predictor. Executive parameter scores accounted for separate and additional variance in TOJ task performance to that already accounted for by Non-word reading accuracy scores. Both these predictors accounted for variance in TOJ task performance that was over and above any prediction made by VIQ, PIQ or FSIQ. This suggests that poor TOJ task performance may arise from at least two dissociated deficits. One of these is associated with difficulty resolving conflict between target and distractor stimuli. The other is associated with Non-word reading that is worse than would be predicted by IQ. As this latter deficit was earlier shown to be associated with

lack of benefit from a spatially informative stimulus over and above the benefit gained from a non-spatially informative alerting stimulus – the "orienting" parameter on the ANT, it is to this we now turn.

Orienting attentional network

The magnitude of the orienting effect in the ANT, as measured by RTs to spatially cued targets after covarying for RTs to centrally cued targets was a significant predictor of mean TOJ task accuracy; however, interactions indicated that the variance in TOJ task accuracy accounted for by the orienting effect on the ANT task was confined to TOJ task trials in which both target and distractor were presented in right hemifield, and to trials in which target and distractor were presented in contralateral hemifields. Trials in which both target and distractor were presented to left hemifield were not significantly predicted by the orienting effect, nor did it account for variance in lateral bias on contralateral trials. In trials in which both target and distractor, appeared in right hemifield, variance in TOJ task performance by appeared to be shared by variance common to the ANT orienting effect and Phonemic Manipulation scores.

Alerting attentional network

The greater the magnitude of the alerting effect observed in the ANT, the greater was accuracy on TOJ task trials in which the target was presented in left hemifield, and the more leftward was the bias on contralateral trials. To put this differently – the lower the magnitude of the alerting effect, the less accurate were participants on trials involving left-field stimuli, and the more rightward was

their bias. This finding is consistent with Posner and colleagues' hypothesis (Fan et al., 2002; Posner and Fan, in press; Posner and Petersen, 1990) of a right-lateralized locus for an alerting network, and is consistent with the hypothesis that where the efficiency of such a network is compromised, a "left-mini-neglect" phenomenon may arise.

4.3 General Discussion

Experiments 1a, 1b, 1c, 2b reported this thesis indicated that dyslexia is associated with impaired performance on a temporal order judgement task in which participants are required to judge the temporal order of the onset of visual stimuli presented to left and right of fixation. Moreover the evidence amassed suggested that while little or no overall lateral bias was found in any given group, individual variance in lateral preference was to be associated with different profiles of both attentional and reading impairment. The main purpose of this study was to use Posner's model of three independent attentional networks to elucidate the nature of the attentional impairments that may be associated with ADD symptoms, dyslexia symptoms, and TOJ task impairment. The findings are illustrated diagrammatically in Figure 4-8: Diagram of main findings

The recruiting strategy employed in the study resulted in a sample of adults with a marginally but significantly greater prevalence of dyslexic and attentional deficit traits than would be expected from a random sample, and sufficient variance in both to generate significant correlations with performance on the two

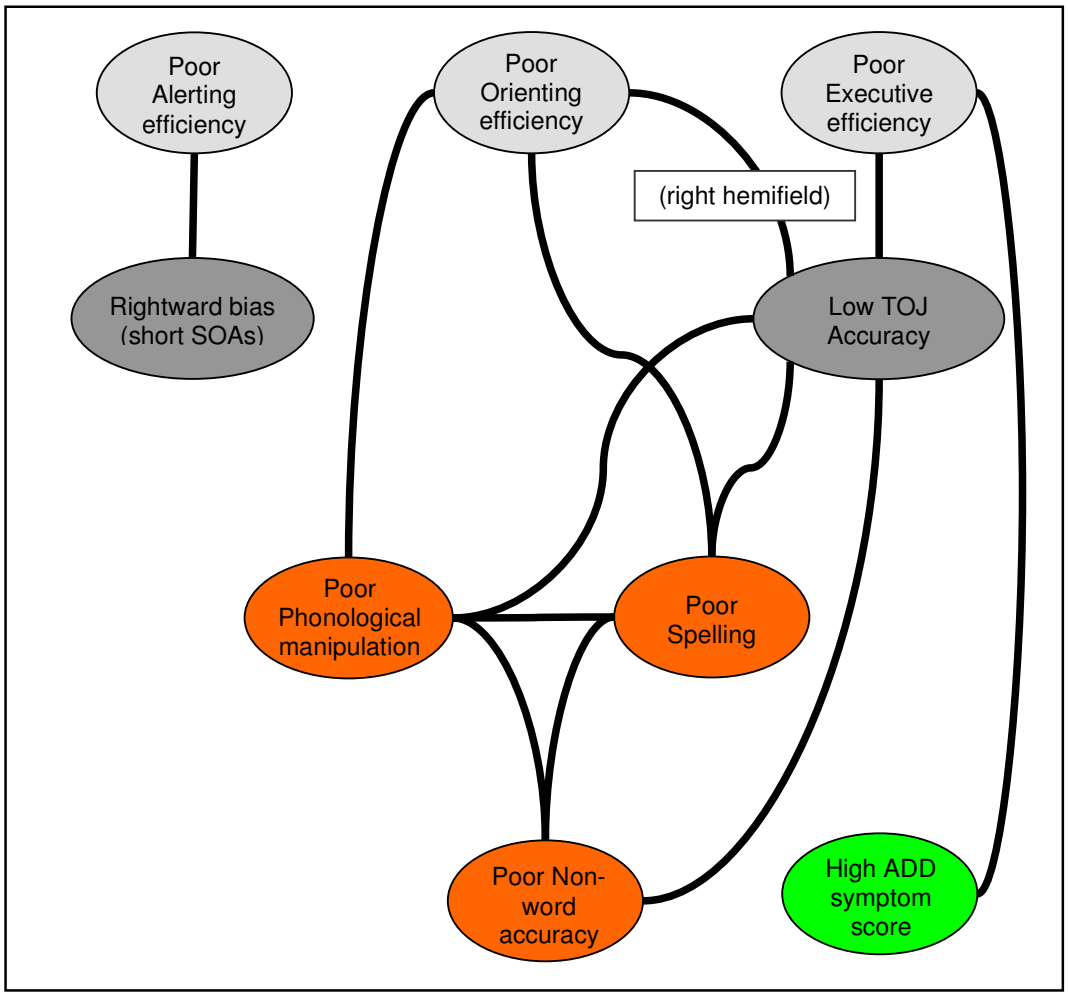

Figure 4-8: Diagram of main findings

The light grey ovals represent the ANT, the dark grey ovals represent the TOJ, the amber ovals represent scores on literacy measures, and the green oval represents ADD symptoms. Black connecting lines represent associations found to be significant; where no connector is shown, no significant association was found. Poor alerting efficiency (indexed by the small RT difference between cued and uncued targets) was associated with rightward bias on the TOJ task. Poor Executive attentional efficiency (indexed by high incongruency costs) was associated with ADD symptoms and with low accuracy scores on the TOJ. Poor Orienting efficiency was associated with poor spelling and phonological manipulation scores, and with difficult with TOJ task pairs where the first stimulus was presented to right hemifield. Low overall TOJ accuracy was associated with poor phonological manipulation, spelling and non-word reading accuracy.

psychophysics tasks, namely the ANT and the TOJ task, employed in the study. Consistent with findings from the earlier studies, poor overall performance on the TOJ task was associated with indicators of dyslexia. In this study, in which the main indicator of dyslexia was poorer non-word reading than would be expected on the basis of IQ, non-word reading accuracy was associated with poor overall TOJ performance, an association that remained significant after covarying for IQ.

In Experiment 3, poor overall TOJ performance was also found to be associated with ADD symptoms as measured by the Brown ADD scale. This sample of participants had largely sub-clinical levels of ADD symptoms, and their Brown ADD scale scores were not significantly predictive of TOJ performance. However, a measure of executive control, postulated by Posner (Berger and Posner, 2000) to be compromised in ADHD symptoms was found to be correlated with ADD scores, as well as with sensitivity to temporal order on the TOJ task and with Non word reading accuracy. Both these associations were independent of IQ.

Given the high prevalence ADD symptoms in the dyslexic participants recruited for Experiment 2b, and in the participants in Experiment 3, the tentative conclusion can be drawn that poor performance on the TOJ by participants with dyslexia may be partially accounted for by a deficit in conflict resolution that is associated with co-morbid ADD, but also by a deficit that is specific to dyslexia, being associated with one of the key indicators of dyslexia, namely, non-word

reading accuracy that is worse than that predicted by IQ.

Posner also postulates that ADD may be associated with deficit in a right-lateralized alerting network (Berger and Posner, 2000). This study provides no evidence that low levels of alerting as measured by the ANT are associated with ADD, and only evidence that high levels of alerting may compensate for low levels of orienting on the non-word reading task. It therefore is possible that the sub-clinical nature of most of the reading and attentional impairments found in participants in this study reflects a low prevalence of deficits in the alerting network, and perhaps therefore the capacity of this network to compensate for an attentional deficit affecting the orienting network.

However, it is also possible that the "alerting" parameter ANT task on the ANT does not have a linear relationship with the efficiency of the alerting network. For example, participants with low baseline levels of arousal may actually benefit more from an alerting cue than those with very high baseline levels. Future studies, using, for example, HRV or skin conductivity indicators of baseline arousal levels, may clarify the relationship between the ANT alerting parameter and baseline arousal. However, on the assumption that ANT "alerting" parameter scores can be taken as a proxy measure for the efficiency of the alerting network, evidence from this study is consistent with a right-lateralized locus for such a network, higher scores on the "alerting" parameter being associated with a more leftward bias on the contralateral TOJ task trials at short SOAs. Alerting scores did not predict temporal order sensitivity.

Orienting scores from the ANT were associated with performance on the TOJ task on trials in which at least one of the stimuli was presented in right hemifield (i.e. contralateral trials and ipsilateral trials presented in right hemifield). Low orienting scores, which were sometimes negative, were associated with poor performance on these trial types, and were also associated with poor phonemic manipulation skills and poor spelling. This suggests the possibility that a left-lateralized deficit that affects orienting scores on the ANT is also associated with the phonological deficit found in dyslexia, and contributes in part to the impoverishment on TOJ task performance found in dyslexic participants in previous studies, particularly impairment on trials in which the target appeared in right hemifield.

If, so, it is pertinent to hypothesize as to what kind of deficit low scores on the orienting parameter on the ANT might reflect. Two findings were made regarding this parameter. The first was a cue-type by flanker-type interaction that indicated that for trials with neutral flankers, the spatial cue was less beneficial than either the central or the double cue, whereas for trials with incongruent flankers it was the only cue-type to give a substantial advantage over the no-cue condition. Fan and colleagues (Fan et al., 2002) found, as was found in Experiment 3, that only spatial cues gave a substantial benefit over no cue for targets with incongruent flankers. However, they did not find, as was found here, that the spatial cue gave less advantage than the alerting cues, particularly the double cue, when targets had neutral flankers. A possible explanation for the second, and possible the first, of these interaction effects may lie in the

phenomenon of Inhibition of Return (IOR). The stimulus onset asynchrony (SOA) in the ANT paradigm reported by Fan and colleagues and used here is 500ms (cue duration=100). This is well within the SOA range in which IOR effects tend to be found (Posner and Cohen, 1984). However, in the ANT, all spatial cues are valid, encouraging exogenous, as well as endogenous, orienting to the cue. Kingstone and Pratt (1999) found that IOR was larger when a target was presented alone than when it was presented with a distractor, and suggest that this reflects greater IOR for exogenous than for endogenous orienting. Conversely, a reduced IOR would be expected for endogenous orienting to targets with distractors, as will tend to be the case in the ANT trials with incongruent flankers, perhaps allowing the orienting response to the spatial cue to surpass the IOR effect, giving a net gain.

The neutral-flanker trials represent the opposite condition. In our particular version of the paradigm, the stimuli were deliberately made easier to distinguish than in the version developed by Fan and colleagues. In the paradigm used here, the target, a large chevron, is so clearly distinguishable from the headless horizontal neutral flankers that it may represents a "pop out" condition, itself inducing exogenous orienting. If so, any endogenous orienting benefit gained from the spatial cue may be confer no additional benefit over its alerting effect, leaving it only with a disadvantageous IOR effect not shared by the spatially neutral cues.

It is possible, therefore, that participants who had low or negative scores on the

"orienting" parameter, did less endogenous orienting, while still experiencing exogenous orienting effects from the spatial cue and thus displaying greater IOR relative to cueing benefit. If so, the finding that low orienting parameter scores were associated with poor performance on TOJ task trial in which at least one stimulus was presented in left hemifield, and also with poor performance on spelling and phonemic manipulation task, may indicate that the deficit underlying poor performance on these three tasks may involve a left-lateralized deficit in that primarily affects endogenous, rather than exogenous, shifts of attention. In an fMRI study, Kim and colleagues found extensive overlap between (Kim et al., 1999) brain areas activated in an exogenous and endogenous orienting task, but found a rightwards asymmetry for the exogenous condition; it is therefore possible that a left lateralized deficit affecting orienting networks may selectively affect endogenous, relative to exogenous efficiency. This would be consistent with the hypothesis that the attenuated peri-saccadic spatial compression effects observed in dyslexic participants in the unilateral, but not in the bilateral spatial compression paradigm may reflect a deficit in endogenous orienting to the anticipated location of an expected salient stimulus.

The second unexpected finding was that those participants who failed to show an "orienting" effect, also tended to gain less from a double cue, relative to central cue. RTs to double cued targets shared variance with RTs to the spatial cue that was not shared with the central cue. This suggests that the double cue may "orient" attention to the two locations at which the target may occur, just as the single spatial cue orients attention to the single spatial location at which the

target will occur. The word "orienting" in such a context becomes somewhat inappropriate; a better model may be that the double and spatial cues both increase the salience of the region or regions of space at which they are presented, as in the model proposed by Godijn and Theeuwes (Godijn and Theeuwes, 2002), and therefore result in faster RTs to targets presented at either of those regions of space. Thus, the finding apparent association between failure to benefit from such a cue, two core symptoms of dyslexia, namely poor spelling and a phonological deficit, and difficulty with TOJ trials in which the initial stimulus was in right hemifield, is consistent with the finding, in Experiment 2b, of an association between factor scores on the TOJ factor postulated to reflect a left-hemisphere deficit with long saccade latencies and slow word recognition.

Lastly, it may be worth pointing out that the inferences drawn by the devisers of the ANT that it taps "the efficiency and independence of attentional networks" (Fan et al., 2002) is predicated on the assumption that the efficiency of the orienting and alerting networks has a monotonic relationship with the reaction time benefit conferred one cue type over another. For a number of reasons this assumption rests on shaky foundations. Firstly, as noted above, the extent to which a consistently valid spatial cue confers an RT benefit over a non-spatially informative cue may depend on the degree to which participants implicitly learn the validity of the cue. Where such learning is impaired, a spatially informative cue presented 500 ms before the task relevant stimulus may elicit "inhibition of return", thus negating, or even reversing, any net benefit. Secondly, as also noted above, the degree to which a participant benefits from an alerting cue may

not solely depend on the "efficiency" of the postulated "alerting" network, but on tonic arousal levels. Where these are low, an alerting cue may result in a large RT benefit; in such circumstance a large RT difference may reflect low tonic, rather than efficient phasic, arousal. Conversely, a participant who is at saturated arousal levels may gain no further net benefit from an alerting cue. For these reasons, therefore, the failure to observe linear correlations between the ANT measures of alerting and orienting, as in this experiment, and as in Fan and Posner's own work (Fan et al., 2002) may not indicate that the two postulated networks are independent.

In a different but related model of attention, Corbetta and Shulman (2002) distinguish "goal-directed" and "stimulus-driven" attentional networks, the first overlapping with Posner's "orienting" network, and the second being analogous to Posner's right-lateralized, noradrenergic "alerting" network, which they describe as acting as a "circuit-breaker" to the first. Kanwisher and Wojciulik (2000) cite evidence to support the case that "top down" or exogenous orienting of attention to objects (and not merely to spatial locations), interacts with incoming information, and modulates both the baseline activity and gain in primary visual cortex. Nobre (2001a) argues that attention may be not only be "oriented" towards locations in space, but to actions or specific points in time, and that the neural systems involved in such attentional shifts include left-lateralized networks that partially overlap with the networks involved in spatial orienting, and with brain areas associated with motor preparation and

anticipation (Nobre, 2001a; Nobre, 2001b).

The association found in this study between failure to benefit from a 100% valid spatial cue, and reading impairment may therefore be more easily interpreted within an attentional model that contrasts endogenous with exogenous attentional processes, rather than "orienting" with "alerting" processes. The apparent association between low benefit from a spatial cue, low sensitivity to abrupt onset stimuli presented to right hemifield, and deficits associated with the core features of dyslexia (poor spelling and phonemic segmentation) may thus reflect a deficit in directing attention that is not limited to spatial locations but includes attention to the kinds of temporal stimuli required for the acquisition of the phonological skills that underpin reading.

5. DYSLEXIA AS AN ATTENTIONAL DEFICIT

5.1 Dyslexia and visual temporal order judgement

A clear finding running through all three of the studies reported in this thesis is that of a strong association between symptoms of dyslexia and impaired accuracy on a lateralized visual TOJ task. This finding remained robust when symptoms of an ADD were controlled for; moreover, not only were there clear differences between non-dyslexic and dyslexic participants, even within dyslexic groups, TOJ task performance was correlated with severity of reading impairment, suggesting a close relationship between reading efficiency and the deficits implicated in impaired TOJ task performance.

The stimuli in all three versions of the paradigm were simple shapes. In the paradigm used in the first study (Chapter 2) not only were the stimuli identical squares, but the response mapping was intuitive – a left key press indicated a stimulus that appeared on the left, a right key-press indicated a stimulus that appeared on the right. Having judged the temporal order of the stimuli, the only level of stimulus discrimination required was spatial location. The task itself, therefore, was unlikely to be dependent on level of reading skill, or even of general intellectual function. In the two later paradigms, in order to render the stimulus-response mapping orthogonal to a variable of interest (lateral bias), a slightly more complex stimulus response mapping was required. In these paradigms, response accuracy was dependent on the ability to discriminate between a circle and a triangle, and to retain temporal order information

while that discrimination was made. Although no time limit was imposed, this may have made more demands on working memory capacity than the first paradigm, and, moreover, may have been more challenging to dyslexic participants with a naming deficit; plausibly, difficulties in labeling the shapes could have increased the cognitive load imposed by the more complex stimulus response mapping. If so, overall performance on this version of the task may have been additionally impaired in dyslexic participants for reasons other than an underlying impairment of temporal or spatial attention; impaired working memory capacity and/or lexical or semantic retrieval deficits may also have contributed to impaired performance.

In the third version of the paradigm, both within-lateral-hemisphere comparisons and between-lateral-hemisphere comparisons were required, allowing the question as to whether impaired overall performance on previous versions of the task was related to poor integration of information across hemifields, or whether within-hemifield performance was also related to severity of reading impairment. While the former might be evidence of either lateral attentional bias, or slow inter-hemispheric transfer, the latter might be a purer indication of poor temporal or spatial integration of stimuli *per se*. However, even on within-hemifield trials, performance on the TOJ task was correlated with both measures of phonological skill and of spelling, both core indicators of reading disorder, findings that remained robust after controlling for reported ADD symptoms.

Thus, even before considering patterns of lateral bias, performance on lateralized

visual TOJ task would seem to be associated with reading disorder *per se*, including the phonological deficits with which it is strongly associated. Given that the TOJ task itself is entirely visual, that it does not involve letter or word stimuli, and that relatively simple stimulus-response mapping is involved, this suggests a robust association between deficits in networks subserving temporal-spatial attention in the visual modality and the core deficits underlying dyslexia.

5.1.1 Left neglect syndrome

Given the finding of Hari and colleagues (2001) that dyslexic participants were not only more inaccurate on a lateralized visual TOJ very similar to the paradigm employed in the study reported in Chapter 1, but also displayed a significant rightwards lateral bias (termed "left mini-neglect" by the authors), it was of note that in none of the studies reported here was there any tendency for a net bias in any one direction to be associated, overall, with reading disorder. Indeed, given the evidence, largely from studies of English-speaking dyslexic participants, of functional disconnectivity in left-hemisphere networks implicated in the mapping of phonology on to orthography, the finding by Hari and colleagues (2001) of an association between dyslexia and a deficit likely to arise from disruption to right-hemisphere networks implicated in attention to left visual hemifield, is, on the face of it, odd.

However, in all three TOJ paradigms reported here, TOJ performance was found to be a function of at least two factors, apparently lateralized. In one, a tendency to report the right hemifield stimulus as having appeared before the left,

particularly at short SOAs, was associated with variance in non-word reading over-and-above variance in non-word reading accounted for by other factors, including irregular word reading (likely to be a measure of print exposure) and phonological skill. If we postulate that this "left mini-neglect" factor is the same as that found in Finnish dyslexic participants by Hari and colleagues (2001), and also by Facoetti and colleagues in Italian speaking dyslexic children with specific difficulties in reading non-words (Facoetti et al., in press), it may be that the failure to find a net rightward bias overall in groups of English speaking participants represents, as initially postulated, a greater prevalence of counteracting left-hemisphere deficits in English speaking dyslexic samples than in groups of participants recruited from speakers of an orthographically transparent language such as Finnish or Italian.

The findings of Facoetti and colleagues (in press) are thus of particular interest, as it would appear that the effect size of the association, found in previous studies by the same team (Facoetti et al., 2000; Facoetti and Turatto, 2000; Facoetti et al., 2001) was enhanced when dyslexic participants were either classified into those with particular difficulty with non-words and those without, or when right-ward bias was correlated with non-word reading measures; of note also is their finding that the association was robust after controlling for a measure of phonological skill.

The evidence from this investigation is thus consistent with the findings of a number of researchers (Auclair and Sieroff, 2002; Eden et al., 2003; Facoetti et

al., 2000; Facoetti and Turatto, 2000; Facoetti et al., 2001; Hari et al., 2001; Sireteanu et al., 2005), of a deficit underlying reading disorder that is manifest in hypo-attention to left hemifield and/or hyper-attention to right hemifield. It is, moreover, is consistent with the findings of Facoetti and colleagues (in press) that this deficit is associated with inaccuracies in non-word reading that are greater than those predicted by a phonological deficit or by age.

The question is therefore raised as to what this deficit might be. One candidate is a deficit in the postulated right-lateralized network proposed by Posner (Posner and Fan, in press; Posner and Petersen, 1990) to subserve the process by which we are alerted to a sudden onset stimulus. The finding of an association between the measure of "alerting" derived from performance of participants on the ANT and a rightward bias on the TOJ is consistent with this hypothesis. Moreover, there is substantial evidence for a "developmental left-neglect" syndrome associated with ADHD (George et al., 2005; Manly et al., 2005; Sheppard et al., 1999) that worsens with time-on-task and improves with stimulant medication, implicating modulation of arousal levels in the disorder. Given the overlap found in this investigation between reading impairment and ADD symptoms, there is evidence to support the conclusion that a developmental left-neglect syndrome may not only be associated with ADHD but also directly with impaired reading, particularly of non-words.

If so, one implication is that non-word reading impairment, often taken as a direct measure of a phonological deficit, may not, in fact, be due to a

phonological deficit in all cases, and that even if a concurrent phonological deficit does lead to selective non-word reading impairment, that additional difficulties with non-words may arise from a right-lateralized deficit in networks governing arousal and attention to left hemifield. In this regard, it is of note that di Pellegrino and colleagues (2001) report that in patients with acquired dyslexia associated with left neglect following right-sided brain damage, non-word reading is more impaired than the reading of real words, and report a case study of a patient with right-sided lesion who was more likely to fixate the contralesional side of a real word than of a pseudo-word. Moreover Arduino and colleagues (Arduino et al., 2003) found that patients with left neglect dyslexia showed impaired reading but unimpaired lexical decision, suggesting that lexical/semantic routes to word recognition were unimpaired, and that sublexical routes, likely to be more strongly implicated in non-word reading than in real word-reading (Coltheart et al., 2001), were affected.

Whitney and Lavidor (Whitney and Lavidor, 2005) propose a model of letter-position encoding (the SERIOL model) in which the left-to-right spatial encoding of the letter sequence in a word is congruent with the acuity gradient in the foveal region projecting to left hemisphere, but runs counter to the acuity gradient in the foveal region projecting to right hemisphere; the acuity gradient of words or portions of words projecting to right hemisphere thus requires to be transformed in order to match the spatial gradient corresponding to the temporal order of the phonemes represented, whereas that of words or portions of words projecting to left hemisphere does not. They propose, and successfully test, a

prediction flowing from the model, namely, that "orthographic neighbourhood effects"[20] ("N" effects), observed during lexical decision tasks when stimuli are presented to left and centre of the visual field, but not when presented to the right, would be eliminated by the artificial manipulation of the "acuity" of letters, achieved by dimming some letters relative to the others, in such a way as to obviate the need for the acuity gradient of letters in the left visual field to be inverted.

If the Whitney-Lavidor model is correct, it suggests that for the left-most portion of words the acuity gradient must be inverted to produce a spatial gradient corresponding to the temporal order of the phonemes represented by each grapheme, while for the right-most portion of words the acuity gradient and the temporal-spatial gradient are already matched. Normal readers tend to fixate words nearer to the beginning than the end (Rayner, 1998), a tendency that might in part be accounted for by the SERIOL model, as this fixation pattern would ensure that for the greater proportion of each word, the acuity gradient is congruent with the temporal-spatial gradient. This, in turn might account for letter-position encoding errors in participants whose attentional bias militates against fixation to the left of centre. Moreover, if such a tendency is partially ameliorated by the lexicality of a word, as suggested by the finding of di

---

[20] A word has a large "orthographic neighbourhood" if there are a large number of words that share all but one letter, in corresponding positions. Thus, the word "LANE" has many neighbours (BANE, CANE, LATE, LACE, LINE, LONE, LAND), while the word "SHED" has very few. High N appears to facilitate lexical decision for words presented in left visual field, but not for words presented in right or central visual field. Whitney C, Lavidor M. Facilitative orthographic neighborhood effects: The SERIOL model account. Cognitive Psychology 2005; 51: 179-213.

Pellegrino and colleagues cited above (di Pellegrino et al., 2001), this might account for the findings reported in this investigation of preferential impairment in non-word reading accuracy encountered by participants with an apparently rightward attentional bias.

A tentative conclusion is therefore that the evidence from the investigation reported in this thesis supports the hypothesis that a right-lateralized deficit in networks subserving alerting and attention to left hemifield may be associated not only with symptoms of ADHD but also directly with reading disorder, in that the deficit may result in disrupted attention to the left-most letters in the absence of lexical recognition, resulting in selective impairment of non-word reading accuracy.

5.1.2 Left hemisphere deficits, dyslexia, and the TOJ task

However, the "left mini-neglect" factor, while it may have contributed to inaccurate performance on the TOJ, did not account for all the variance in TOJ performance associated with reading impairment. In Experiments 1b and 1c, a leftward attentional bias was also associated with reading impairment, particularly with that measured by irregular word accuracy. The possibility was raised therefore, that deficits in left or bilateral networks underlying some aspects of reading impairment were blunting or counteracting, at group, and possibly at individual, level, any rightward attentional biases arising from right hemisphere deficits also prevalent in the dyslexic groups studied.

One question posed concerned the extent to which deficits contributing to

impaired performance on a visual TOJ task lay in networks implicated in supra-modal temporal order judgement, and impacted on reading via their effect on phonological skill, or whether they accounted for visual anomalies that directly impacted on reading. In this respect, the findings of Experiments 2a and 2b are suggestive but remain ambiguous. Experiment 2a tested the hypothesis that if dyslexic participants suffered from a deficit in networks implicated in maintaining spatial constancy across saccades, this might account for both their impaired TOJ performance and for visual anomalies subjectively reported by dyslexic readers, such as letters or words appearing to move on the page. If so, the phenomenon known as "peri-saccadic spatial compression", by which briefly probes presented shortly before saccade onset tend to be mislocalized in the direction of the saccade target, would be likely to be attenuated in dyslexic participants, as the phenomenon has been postulated to arise from the operation of mechanisms implicated in the maintenance of spatial constancy (Lappe et al., 2000; Ross et al., 1997; Ross et al., 2001).

The findings from the experiment supported the hypothesis. Dyslexic participants showed reduced peri-saccadic compression as compared with non-dyslexic control participants, suggesting impairment in networks implicated in the maintenance of spatial constancy. However attempts to correlate attenuation of peri-saccadic spatial compression with TOJ task performance foundered when the spatial compression task was modified in such a way as to make the saccade direction unpredictable. In this modified version of the paradigm, in which saccade targets were presented unpredictably to either left or right of fixation,

differences were only found in a subgroup of participants with ADD symptoms; moreover, the pattern of compression found in both dyslexic and non-dyslexic participants was distinctly different to that observed in the unidirectional paradigm.

In the bi-directional paradigm, rather than compression being confined to immediate peri-saccadic period, compression in both groups was found at all time periods; what changed as saccade onset approached was the focus of the compression, which shifted, in the direction of the saccade, towards the saccade target. It was postulated that the differences in compression patterns between the unidirectional and the bi-directional versions of the paradigm may indicate that effects in the unidirectional paradigm are dependent on the degree to which participants were able to endogenously and covertly attend the visual hemifield in which the saccade target was expected.

It was hypothesized that rather being a specifically "Peri-saccadic" phenomenon, mislocalization of briefly presented probes may represent the operation of a "foveal bias", modulated by the extent to which the probe elicited the representation of a potential saccade on a postulated "saccade map", and that the peri-saccadic compression pattern occurs when the predicted retinotopic vector of the probe is mapped not onto current fixation, but on to the new fixation point, the target of the saccade. If so, it is possible that the differences observed in Experiment 2a between dyslexic and control participants in the degree of peri-saccadic compression elicited were due not to differences in foveal bias, but

differences in the degree to which they were able to shift covert attention, and thus the focus of the compression, to the saccade target in advance of the saccade. In a paradigm in which neither group was able to do this easily, owing to the unpredictability of the saccade target location, group differences may have been reduced.

In the light of this hypothesis, it was of interest to find that saccade latencies observed in Experiment 2b were significantly longer in the dyslexic group, and were also strongly correlated with TOJ task performance. Moreover, when two performance factors were extracted from the TOJ task, one apparently representing a rightward attentional bias at short SOAs, and one representing inaccurate reporting of the temporal order of the stimuli even at long SOAs and associated with a leftward attentional bias, saccade latencies were strongly correlated with the second factor but not with the first. This led to the hypothesis that disruption to networks in left-hemisphere implicated in the endogenous orienting of attention may underlie both poor TOJ task performance at long SOAs, and to slow saccade latencies to unpredictable saccade target locations. As these behavioural measures were also associated with slow reading latencies, and with inaccurate single word reading of both real and non-words, it was postulated that the latent variable may be a deficit in networks implicated in the orienting of attention to both events and locations.

It was thus of particular interest that in the final experiment, Experiment 3, performance on TOJ stimulus pairs in which the first stimulus was presented to

right hemifield, whether or not the second stimulus was presented in the same lateral hemifield or to the opposite hemifield, was correlated not only with a number of core features of dyslexia, including poor spelling and phonological skill, but also with apparently impaired ability to benefit from the spatially informative cue on the ANT. This suggests that disruption to left hemisphere networks that appear to underlie dyslexia, at least in English speaking populations, include networks implicated in the spatial orienting of attention, and the recall of the temporal order of stimuli presented to two different locations in right hemifield.

5.1.3 Executive control of attention

In the final experiment, a third factor underlying impaired TOJ performance in dyslexic participants may have been identified. As measures of ADD symptoms were made on this group of participants, and as the ANT they undertook also provides a measure of the ability to resolve conflicting stimulus – to determine the direction of a target arrow in the presence of incongruent flanker stimuli – it was possible to test the hypothesis that ADD symptoms were associated high costs of incongruency on the flanker task, postulated to measure the efficiency of an attentional network postulated to underlie executive control of attention. This hypothesis was supported, in that degree of ADD symptom reporting was correlated with cost of incongruency on the flanker component of the ANT. Moreover, incongruency costs were also associated with impaired performance on the TOJ task, regardless of lateral presentation of the stimuli. However,

incongruency costs were not correlated with any measure of reading disorder.

Bearing in mind that the participants for this study were recruited because they were from a population likely to have a higher prevalence of reading impairment than the general population (which they did) and that the group also had a higher prevalence of ADD symptoms than would be expected from a sample drawn from the general population, this finding is consistent with the finding from Experiment 2b that ADD symptoms accounted for variance in TOJ performance over and above that accounted for by reading in a dyslexic group. In other words, that impaired TOJ performance in a group recruited because they suffered from dyslexia may be partially due to impairments in executive attentional control networks that underlie overlapping ADHD symptoms.

5.1.4 Models of dyslexia

The findings of this investigation are therefore consistent with models that postulate supramodal attentional deficits as underlying the deficits that contribute to impaired reading. Either a magnocellular deficit (Lovegrove, 1996; Stein and Walsh, 1997) or broader perceptual deficits (Amitay et al., 2002; Ben-Yehudah et al., 2001) might account for the strong association between reading disorder and impaired visual temporal order sensitivity found in these studies, as might automatization deficits, as proposed by Nicolson and Fawcett (Nicolson et al., 2001). The "double deficit" model proposed by Wolf and Bowers (1999), might also be consistent with the findings, particularly with the finding that word recognition latencies were associated with impaired visual temporal order

judgement, suggesting that slow perceptual processing even when no identification is required, may contribute to both naming speed deficits and slow word reading latencies.

However, the findings also suggest that while phonological deficit models (Ramus, 2003) may account for substantial variance in reading impairment, that those phonological deficits may arise from supramodal deficits that are also manifest in performance deficits on purely visual and non-verbal tasks requiring attention to temporal order and spatial location, The finding by Goswami and colleagues (Goswami, 2003; Richardson et al., 2004) that sensitivity to the amplitude rise time of auditory stimuli may be key to the ability to segment onset from rime, and thus underlie the strong relationship between phonological awareness and reading acquisition suggests the possibility that the anomalous performance of dyslexic participants on the visual paradigms used in this investigation may be related to a supramodal blunting of sensitivity to the onsets of sensory stimuli, resulting in a reduction of stimulus salience. However, there is also evidence in the data to support the hypothesis that a developmental left-neglect syndrome (Dobler et al., 2005) may not simply be associated with attentional deficit disorders but may directly impact on reading, specifically of decoding accuracy, possibly via disrupting accurate orthographic perception .

5.1.5 Limitations of the investigation

5.1.5.1 Sample design

Except for Experiment 2b, the studies in this investigation involved adults, recruited from populations that varied from study to study: university students with and without a diagnosis of dyslexia; adults from the general population often without a prior diagnosis and with no history of remediation; and adults recruited because having a dyslexic child made them more likely than adults in the general population to have some degree of reading impairment. Findings unique to each study are therefore of limited generalizability, although findings common to each study may be regarded as more generalizable precisely because of the heterogeneity of the populations studied.

However, the fact that the investigation was largely confined to adults imposes a further limitation in that in adults, degree of print exposure is likely to be less strongly related to the severity of the underlying disorder, and more related to access to remediation, or to the development of compensatory strategies, than in a sample of children. Notably, one participant in Experiment 3, with a high FSIQ and rapid real-word reading latencies was almost unable to read a single non-word correctly, and appeared simply to guess the nearest real word that it resembled, even though he had clearly understood that some of the words "would not be real words at all". Reading skill itself may therefore be, at best, a fairly blunt instrument for measuring the magnitude of the deficit underlying its impairment, and is likely to be blunter in a heterogeneous group of adults with

widely varying uses for literacy skills.

5.1.5.2 Paradigms

All three paradigms present problems with interpretation. The TOJ paradigm was successively modified in order to first to allow a genuine lateral attentional bias to be disambiguated from motor response bias, and then to allow within lateral hemifield effects to be disambiguated from between-hemifield effects. However, with each additional modification, additional brain networks may have been implicated in task performance, and it would seem likely that the two later versions of the task, in which the one-to-one stimulus response mapping was replaced by a response mapping orthogonal to the lateral presentation of the stimuli may have resulted in a larger component of task performance being dependent on the efficiency of attentional networks implicated in conflict resolution, and in stimulus response mapping.

Moreover, it remains ambiguous as to whether performance on the TOJ task taps visual or spatial attention or both. It would have been helpful to have included tasks in which spatial features of the stimuli were manipulated independently from temporal features, in order to ascertain whether performance on a purely temporal task or on a purely spatial one, shared more variance with performance on the TOJ tasks employed.

The spatial compression paradigm used raises interesting questions as to the nature of the phenomenon elicited not only by the unidirectional paradigm employed in Experiment 2a, but also that employed by other researchers, but

it is a limitation of the study that the modifications introduced into the paradigm, largely for practical reasons (to reduce the number of anticipatory saccades and to allow the measurement of saccade latencies) appeared to radically change the nature of the phenomenon it was designed to elicit. Moreover, the strongest finding from the bi-directional paradigm employed in Experiment 2b regarding group differences was the large differences in saccade latencies. In retrospect, a task specifically designed to investigate saccade latencies to targets at varying eccentricities and in different hemifields might have been more informative.

There are also questions that need to be asked regarding the ANT paradigm, perhaps the most fundamental being the extent to which differentials between mean RTs to each condition reflect the efficiency of the network they are intended to assess. While it seems clear that greater incongruency costs are likely to reflect reduced efficiency in networks implicated in resolving conflict between competing stimuli, the meaning of the differentials between cuing conditions is less clearcut. For example, a large differential between mean RT to a cued target and to an uncued target may mean either that a phasic alerting mechanism is particularly efficient – or it may mean that tonic arousal levels are low, which might be the case in a participant whose alerting network was actually inefficient. If so, the relationship between RT differential and the underlying efficiency of the alerting network may not be monotonic. Indeed, this may account for the lack of linear correlation between measures of "alerting" and of "orienting" found in both the Experiment 3 and in other studies that have employed the ANT (Fan et al., 2002), and brings into question the postulated

independence of these two networks. A third potential problem in the ANT paradigm is that the cue-stimulus interval (500ms) falls within the time period in which Inhibition of Return (IoR) is likely to counteract the benefits of valid cueing. Although IoR effects are more likely in paradigms in which a proportion of cues are invalid, possibly because it is attenuated by endogenous attention to the cued location when it is known that the validity of the cue is certain, differentials between RTs to spatially informative cues and RTs to non-spatially informative cues may depend on the extent to which IoR effects are counteracted by endogenous attention. Thus, it is not clear whether the association found in Experiment 3 between attenuated "orienting", impaired TOJ performance, and impaired reading skill may indicates greater IoR or reduced endogenous orienting. A paradigm in which the cue-stimulus interval had been manipulated might have helped disambiguate this finding.

5.2 Future studies

The findings from this investigation indicate at least three potentially fruitful directions for future research.

Firstly, it seems evident that some form of developmental left-neglect is not only associated with ADHD and with dyslexia merely by virtue of the overlap or co-morbidity between these two disorders, but may play a causal role in impairing "word attack" – the reading of unfamiliar, or non-words. If so, future investigations should distinguish between estimates of phonological deficit based solely on performance on auditory tasks, and those inferred from non-word

reading performance, or from discrepancies between non-word reading and real-word reading. When "phonological" and "surface" dyslexics are defined in terms of whether non-word or irregular word reading is more impaired, this may beg the question as to what kinds of deficits underlie non-word reading impairment. What would be of particular interest would be to determine the proportion of variance in non-word reading that is predicted by a measure of phonological skill, and whether further variance is predicted by a measure of the efficiency of networks implicated in arousal and spatial attention. While a number of these studies have been done on Italian readers by Facoetti and colleagues (Facoetti et al., 2003b; Facoetti and Molteni, 2000; Facoetti et al., 2000; Facoetti and Turatto, 2000; Facoetti et al., 2001; Facoetti et al., in press), it would seem important to replicate this work on English readers participants, and to ensure, in such a replication, that measures of reading impairment that are likely to arise from disruption to left-hemisphere networks, including word recognition latencies and phonological skill, are controlled for. A further implication of this finding in this investigation is that rather than screen out participants with ADHD from studies of reading disorder, it may be important to investigate specific reading impairments that may be associated with ADHD, particularly in the absence of an apparent phonological deficit. An investigation into the nature of reading difficulties associated with developmental left-neglect, whether apparently associated with ADHD or not, seems warranted. In particular, it would be of interest to discover whether children with developmental left-neglect tend to fixate words further to the right than un-

impaired, reading age matched children, and, if so, whether this tendency is greater with non-words than real words. This could be further investigated using the manipulation employed by Whitney and Lavidor (Whitney and Lavidor, 2005)

Secondly, the findings of the investigation suggest that a deficit in endogenous spatial orienting may underlie the aspects of reading disorder associated with disconnectivity in left-hemisphere networks implicated in grapheme phoneme mapping. If so, common to both tasks may be the construction of a temporal-spatial representation of future events, whether it is the mapping of temporally ordered phonemes onto spatially ordered text, or mapping a temporally expected target on to a spatially expected location. A possibly informative line of investigation might be the study of fixation patterns and eye movements in dyslexic participants, particularly with dyslexic children who are struggling with the "sounding out" of novel words, and with acquiring fluency in tracking a line of text across a page.

Thirdly, the finding that in a sample of parents of dyslexic children, not only was there a higher than average prevalence of mild reading impairment, but also a higher than average prevalence of symptoms of ADD, suggests a common genetic aetiology. One possibility is that the genotype in question may produce a phenotype that resembles dyslexia or ADHD depending on which of a number of potential, possibly laterally homologous brain networks are most severely compromised. This investigation suggests a number of behavioural measures

that might yield informative linkages with genetic markers for the two conditions, including measures of attentional orienting, measures of both tonic arousal and phasic arousal in response to an alerting stimulus, and measures of executive control of attention. Furthermore, it would seem fruitful to investigate whether the behavioural measures listed here are associated with performance on task likely to tap both magnocellular and cerebellar function, either of which may represent an underlying aetiology that might result in the kinds of deficits reported as being associated with dyslexia in this thesis.

5.3 Summary

This investigation would appear to indicate that at least three partially dissociated deficits are characteristic of people with dyslexia and lead to impaired performance on a lateralized visual temporal order judgment tasks in which participants are required to report the temporal order of two visual stimuli presented at different locations.

Two of these deficits appear to be cerebrally lateralized. One appears to be a left-lateralized deficit implicated in the core symptoms of dyslexia, and associated with difficulty in reporting the temporal order of stimuli presented in right hemifield, in sluggish saccade latencies to uncued stimuli, and in impaired ability to benefit from an informative spatial cue. This may suggests a deficit in attentional orienting – perhaps specifically endogenous attentional orienting – to moments in time and locations in space, and possibly implicating networks that

include association cortex in the inferior parietal lobule.

The second appears to be a possibly homologous right-lateralized deficit implicated in spatial attention and phasic arousal to an alerting stimulus and in sustained attention. It would seem to be associated with ADHD as well as with dyslexia, and to contribute to additional problems in reading unfamiliar words.

The third appears to be a deficit in the executive control of attention and/or working memory, and is associated with co-morbid ADHD symptoms and with overall impairment on the TOJ task, but not with specific deficits in reading.

Taken together, the evidence suggests that dyslexia and ADHD may be dimensions of a common developmental pathology, the phenotype depending on the degree to which each attentional of these three attentional networks is disrupted. It therefore suggests that dyslexia might be usefully viewed as a type of attentional deficit disorder.

References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders: American Psychiatric Association, 1994.

Amitay S, Ben-Yehudah G, Banai K, Ahissar M. Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. Brain 2002; 125: 2272-2285.

Arduino LS, Burani C, Vallar G. Reading aloud and lexical decision in neglect dyslexia patients: a dissociation. Neuropsychologia 2003; 41: 877-885.

Auclair L, Sieroff E. Attentional cueing effect in the identification of words and pseudowords of different length. Quarterly Journal of Experimental Psychology Section a-Human Experimental Psychology 2002; 55: 445-463.

Bakker DJ. Dyslexia and the Ecological Brain. Journal of Clinical and Experimental Neuropsychology 1994; 16: 734-743.

Barkley RA. Issues in the diagnosis of attention-deficit/hyperactivity disorder in children. Brain & Development 2003; 25: 77-83.

Ben-Yehudah G, Banai K, Ahissar M. Patterns of deficit in auditory temporal processing among dyslexic adults. Neuroreport 2004; 15: 627-631.

Ben-Yehudah G, Sackett E, Malchi-Ginzberg L, Ahissar M. Impaired temporal contrast sensitivity in dyslexics is specific to retain-and-compare paradigms. Brain 2001; 124: 1381-1395.

Berger A, Posner MI. Pathologies of brain attentional networks. Neuroscience and Biobehavioral Reviews 2000; 24: 3-5.

Biscaldi M, Fischer B, Hartnegg K. Voluntary saccadic control in dyslexia. Perception 2000; 29: 509-521.

Biscaldi M, Gezeck S, Stuhr V. Poor saccadic control correlates with dyslexia. Neuropsychologia 1998; 36: 1189-1202.

Blakemore SJ, Frith CD, Wolpert DM. The cerebellum is involved in predicting the sensory consequences of action. Neuroreport 2001; 12: 1879-1884.

Booth JR, Burman DD, Meyer JR, Gitelman DF, Parrish TB, Mesulam MM. Relation between brain activation and lexical performance. Human Brain Mapping 2003; 19: 155-169.

Brannan JR, Williams MC. Developmental Versus Sensory Deficit Effects on Perceptual Processing in the Reading Disabled. Perception & Psychophysics 1988; 44: 437-444.

Brown TE. The Brown Attention-Deficit Disorder Scales (Brown ADD Scales) for Adolescents and Adults. San Antonio: The Psychological Corporation: Harcourt Brace, 1996.

Burr DC, Morrone MC, Ross J. Separate visual representations for

perception and action revealed by saccadic eye movements. Current Biology 2001; 11: 798-802.
Cacace AT, McFarland DJ, Ouimet JR, Schrieber EJ, Marro P. Temporal processing deficits in remediation-resistant reading- impaired children. Audiology and Neuro-Otology 2000; 5: 83-97.
Caravolas M, Hulme C, Snowling MJ. The foundations of spelling ability: Evidence from a 3-year longitudinal study. Journal of Memory and Language 2001; 45: 751-774.
Carter CS, Krener P, Chaderjian M, Northcutt C, Wolfe V. Asymmetrical Visual-Spatial Attentional Performance in Adhd - Evidence for a Right Hemispheric Deficit. Biological Psychiatry 1995; 37: 789-797.
Castles A, Coltheart M. Varieties of Developmental Dyslexia. Cognition 1993; 47: 149-180.
Catts HW. Defining dyslexia as a developmental language disorder: An expanded view. Topics in Language Disorders 1996; 16: 14-29.
Cestnick L, Coltheart M. The relationship between language-processing and visual- processing deficits in developmental dyslexia. Cognition 1999; 71: 231-255.
Cohen L, Dehaene S, Naccache L, Lehericy S, Dehaene-Lambertz G, Henaff MA, et al. The visual word form area - Spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. Brain 2000; 123: 291-307.
Colby CL, Duhamel JR, Goldberg ME. Visual, presaccadic, and cognitive activation of single neurons in monkey lateral intraparietal area. Journal of Neurophysiology 1996; 76: 2841-2852.
Coltheart M, Rastle K, Perry C, Langdon R, Ziegler J. DRC: A dual route cascaded model of visual word recognition and reading aloud. Psychological Review 2001; 108: 204-256.
Corbetta M, Akbudak E, Conturo TE, Snyder AZ, Ollinger JM, Drury HA, et al. A common network of functional areas for attention and eye movements. Neuron 1998; 21: 761-773.
Corbetta M, Shulman GL. Control of goal-directed and stimulus-driven attention in the brain. Nature Reviews Neuroscience 2002; 3: 201-215.
Cornelissen PL, Hansen PC, Gilchrist I, Cormack F, Essex J, Frankish C. Coherent motion detection and letter position encoding. Vision Research 1998a; 38: 2181-2191.
Cornelissen PL, Hansen PC, Hutton JL, Evangelinou V, Stein JF. Magnocellular visual function and children's single word reading. Vision Research 1998b; 38: 471-482.
Coull JT, Frith CD, Buchel C, Nobre AC. Orienting attention in time: behavioural and neuroanatomical distinction between exogenous and endogenous shifts. Neuropsychologia 2000; 38: 808-819.
Crawford TJ, Higham S. Dyslexia and the centre-of-gravity effect. Experimental Brain Research 2001; 137: 122-126.
Davis C, Castles A, McAnally K, Gray J. Lapses of concentration and

dyslexic performance on the Ternus task. Cognition 2001a; 81: B21-B31.
Davis CJ, Gayan J, Knopik VS, Smith SD, Cardon LR, Pennington BF, et al. Etiology of reading difficulties and rapid naming: The Colorado twin study of reading disability. Behavior Genetics 2001b; 31: 625-635.
Davis RD, Braun EM. The gift of dyslexia: Why some of the smartest people can't read and how they can learn. New York: Perigee Books, 1997.
De Martino S, Espesser R, Rey V, Habib M. The "temporal processing deficit" hypothesis in dyslexia: New experimental evidence. Brain and Cognition 2001; 46: 104-108.
Denckla MB, Rudel RG. Rapid Automatized Naming (Ran) - Dyslexia Differentiated from Other Learning-Disabilities. Neuropsychologia 1976; 14: 471-479.
di Pellegrino G, Ladavas E, Galletti C. Lexical processes and eye movements in neglect dyslexia. Behavioural Neurology 2001; 13: 61-74.
Dobler VB, Anker S, Gilmore J, Robertson IH, Atkinson J, Manly T. Asymmetric deterioration of spatial awareness with diminishing levels of alertness in normal children and children with ADHD. Journal of Child Psychology and Psychiatry 2005; 46: 1230-1248.
Dougherty RF, Cynader MS, Bjornson BH, Edgell D, Giaschi DE. Dichotic pitch: a new stimulus distinguishes normal and dyslexic auditory function. Neuroreport 1998; 9: 3001-3005.
Eckert MA, Leonard CM, Richards TL, Aylward EH, Thomson J, Berninger VW. Anatomical correlates of dyslexia: frontal and cerebellar findings. Brain 2003; 126: 482-494.
Eden GF, Stein JF, Wood HM, Wood FB. Differences in Eye-Movements and Reading Problems in Dyslexic and Normal-Children. Vision Research 1994; 34: 1345-1358.
Eden GF, Stein JF, Wood MH, Wood FB. Verbal and Visual Problems in Reading-Disability. Journal of Learning Disabilities 1995; 28: 272-290.
Eden GF, Wood FB, Stein JF. Clock drawing in developmental dyslexia. Journal of Learning Disabilities 2003; 36: 216-228.
Epstein JN, Conners CK, Erhardt D, March JS, Swanson JM. Asymmetrical hemispheric control of visual-spatial attention in adults with attention deficit hyperactivity disorder. Neuropsychology 1997; 11: 467-473.
Facoetti A, Lorusso ML, Cattaneo C, Galli R, Molteni M. Visual and auditory attentional capture are both sluggish in children with developmental dyslexia. Acta Neurobiologiae Experimentalis 2005; 65: 61-72.
Facoetti A, Lorusso ML, Paganoni P, Cattaneo C, Galli R, Mascetti GG. The time course of attentional focusing in dyslexic and normally reading children. Brain and Cognition 2003a; 53: 181-184.

Facoetti A, Lorusso ML, Paganoni P, Cattaneo C, Galli R, Umilta C, et al. Auditory and visual automatic attention deficits in developmental dyslexia. Cognitive Brain Research 2003b; 16: 185-191.
Facoetti A, Molteni M. Is attentional focusing an inhibitory process at distracter location? Cognitive Brain Research 2000; 10: 185-188.
Facoetti A, Paganoni P, Lorusso ML. The spatial distribution of visual attention in developmental dyslexia. Experimental Brain Research 2000; 132: 531-538.
Facoetti A, Turatto M. Asymmetrical visual fields distribution of attention in dyslexic children: a neuropsychological study. Neuroscience Letters 2000; 290: 216-218.
Facoetti A, Turatto M, Lorusso ML, Mascetti GG. Orienting of visual attention in dyslexia: evidence for asymmetric hemispheric control of attention. Experimental Brain Research 2001; 138: 46-53.
Facoetti A, Zorzib M, Cestnick L, Lorussoa ML, Moltenia M, Paganoni P, et al. The relationship between visuo-spatial attention and nonword reading in developmental dyslexia. Cognitive Neuropsychology in press.
Fan J, McCandliss BD, Sommer T, Raz A, Posner MI. Testing the efficiency and independence of attentional networks. Journal of Cognitive Neuroscience 2002; 14: 340-347.
Farmer ME, Klein RM. The evidence for a temporal processing deficit linked to dyslexia: A Review. Psychonomic Bulletin & Review 1995; 2: 460-493.
Fawcett AJ, Nicolson RI. The Dyslexia Screening Test (DST). London: The Psychological Corporation, 1996.
Fawcett AJ, Nicolson RI. The Dyslexia Adult Screening Test (DAST). London: The Psychological Corporation, 1998.
Fawcett AJ, Nicolson RI, Maclagan F. Cerebellar tests differentiate between groups of poor readers with and without IQ discrepancy. Journal of Learning Disabilities 2001; 34: 119-135.
Fay MP, Follmann DA. Designing Monte Carlo implementations of permutation or bootstrap hypothesis tests. American Statistician 2002; 56: 63-70.
Feifel D, Farber RH, Clementz BA, Perry W, Anllo-Vento L. Inhibitory deficits in ocular motor behavior in adults with attention-deficit/hyperactivity disorder. Biological Psychiatry 2004; 56: 333-339.
Finch AJ, Nicolson RI, Fawcett AJ. Evidence for a neuroanatomical difference within the olivo-cerebellar pathway of adults with dyslexia. Cortex 2002; 38: 529-539.
Findlay JM, Gilchrist ID. Active Vision. Oxford: Oxford University Press, 2003.
Friedman MC, Chhabildas N, Budhiraja N, Willcutt EG, Pennington BF. Etiology of the comorbidity between RD and ADHD: Exploration of the non-random mating hypothesis. American Journal of Medical Genetics Part B-Neuropsychiatric Genetics 2003; 120B: 109-115.

Frith CD, Frith U. A biological marker for dyslexia. Nature 1996; 382: 19-20.
George M, Dobler V, Nicholls E, Manly T. Spatial awareness, alertness, and ADHD: The re-emergence of unilateral neglect with time-on-task. Brain and Cognition 2005; 57: 264-275.
Godijn R, Theeuwes J. Programming of endogenous and exogenous saccades: Evidence for a competitive integration model. Journal of Experimental Psychology-Human Perception and Performance 2002; 28: 1039-1054.
Goswami U. Why theories about developmental dyslexia require developmental designs. Trends in Cognitive Sciences 2003; 7: 534-540.
Goswami U, Thomson J, Richardson U, Stainthorp R, Hughes D, Rosen S, et al. Amplitude envelope onsets and developmental dyslexia: A new hypothesis. Proceedings of the National Academy of Sciences of the United States of America 2002; 99: 10911-10916.
Grant AC, Zangaladze A, Thiagarajah MC, Sathian K. Tactile perception in developmental dyslexia: a psychophysical study using gratings. Neuropsychologia 1999; 37: 1201-1211.
Greatrex JC, Drasdo N. The Magnocellular Deficit Hypothesis in Dyslexia - a Review of Reported Evidence. Ophthalmic and Physiological Optics 1995; 15: 501-506.
Green DM, Swets JA. Signal Detection Theory and Psychophysics. New York: Wiley, 1966.
Griffiths YM, Snowling MJ. Predictors of exception word and nonword reading in dyslexic children: The severity hypothesis. Journal of Educational Psychology 2002; 94: 34-43.
Habib M. The neurological basis of developmental dyslexia - An overview and working hypothesis. Brain 2000; 123: 2373-2399.
Hari R. Illusory Directional Hearing in Humans. Neuroscience Letters 1995; 189: 29-30.
Hari R, Kiesila P. Deficit of temporal auditory processing in dyslexic adults. Neuroscience Letters 1996; 205: 138-140.
Hari R, Renvall H. Impaired processing of rapid stimulus sequences in dyslexia. Trends in Cognitive Sciences 2001; 5: 525-532.
Hari R, Renvall H, Tanskanen T. Left minineglect in dyslexic adults. Brain 2001; 124: 1373-1380.
Hari R, Valta M, Uutela K. Prolonged attentional dwell time in dyslexic adults. Neuroscience Letters 1999; 271: 202-204.
Horwitz B, Rumsey JM, Donohue BC. Functional connectivity of the angular gyrus in normal reading and dyslexia. Proceedings of the National Academy of Sciences of the United States of America 1998; 95: 8939-8944.
Iles JW, V.; Richardson, A. Visual Search Performance in Dyslexia. Dyslexia 2000; 6: 163-177.
Jaskowski P, Rusiak P. Posterior parietal cortex and developmental dyslexia. Acta Neurobiologiae Experimentalis 2005; 65: 79-94.

Kanwisher N, Wojciulik E. Visual attention: Insights from brain imaging. Nature Reviews Neuroscience 2000; 1: 91-100.
Kaplan BJ, Dewey DM, Crawford SG, Wilson BN. The term comorbidity is of questionable value in reference to developmental disorders: Data and theory. Journal of Learning Disabilities 2001; 34: 555-565.
Keen AG, Lovegrove WJ. Transient deficit hypothesis and dyslexia: examination of whole-parts relationship, retinal sensitivity, and spatial and temporal frequencies. Vision Research 2000; 40: 705-715.
Kerzel D. Memory for the position of stationary objects: disentangling foveal bias and memory averaging. Vision Research 2002; 42: 159-167.
Kim YH, Gitelman DR, Nobre AC, Parrish TB, LaBar KS, Mesulam MM. The large-scale neural network for spatial attention displays multifunctional overlap but differential asymmetry. Neuroimage 1999; 9: 269-277.
Kingstone A, Pratt J. Inhibition of return is composed of attentional and oculomotor processes. Perception & Psychophysics 1999; 61: 1046-1054.
Klein RM, Farmer ME. Dyslexia and a temporal processing deficit - A reply. Psychonomic Bulletin & Review 1995; 2: 515-526.
Kusunoki M, Goldberg ME. The time course of perisaccadic receptive field shifts in the lateral intraparietal area of the monkey. Journal of Neurophysiology 2003; 89: 1519-1527.
Laasonen M, Service E, Virsu V. Crossmodal temporal order and processing acuity in developmentally dyslexic young adults. Brain and Language 2002; 80: 340-354.
Laasonen M, Tomma-Halme J, Lahti-Nuuttila P, Service E, Virsu V. Rate of information segregation in developmentally dyslexic children. Brain and Language 2000; 75: 66-81.
Ladavas E, Shallice T, Zanella MT. Preserved semantic access in neglect dyslexia. Neuropsychologia 1997; 35: 257-270.
Lappe M, Awater H, Krekelberg B. Postsaccadic visual references generate presaccadic compression of space. Nature 2000; 403: 892-895.
Liddle E, Jackson GM, Jackson SR. An evaluation of a visual biofeedback intervention in dyslexic adults. Dyslexia 2005; 11: 61-77.
Livingstone MS, Hubel DH. Psychophysical Evidence for Separate Channels for the Perception of Form, Color, Movement, and Depth. Journal of Neuroscience 1987; 7: 3416-3468.
Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and Anatomical Evidence for a Magnocellular Defect in Developmental Dyslexia. Proceedings of the National Academy of Sciences of the United States of America 1991; 88: 7943-7947.
Lovegrove B. Dyslexia and a transient/magnocellular pathway deficit: The current situation and future directions. Australian Journal of Psychology 1996; 48: 167-171.

MacKeben M, Trauzettel-Klosinski S, Reinhard J, Durrwachter U, Adler M, Klosinski G. Eye movement control during single-word reading in dyslexics. Journal of Vision 2004; 4: 388-402.
Manly T, Cornish K, Grant C, Dobler V, Hollis C. Examining the relationship between rightward visuo-spatial bias and poor attention within the normal child population using a brief screening task. Journal of Child Psychology and Psychiatry 2005; 46: 1337-1344.
Manly T, Robertson IH, Verity C. Developmental unilateral visual neglect: A single case study. Neurocase 1997; 3: 19-29.
Marx P, Weber JM, Schneider W. Dyslexia versus garden-variety poor reading: A comparison of phonological and visual processing skills. Zeitschrift Fur Padagogische Psychologie 2001; 15: 85-98.
Masutto C, Bravar L, Fabbro F. Neurolinguistic Differentiation of Children with Subtypes of Dyslexia. Journal of Learning Disabilities 1994; 27: 520-526.
May JG, Williams MC, Dunlap WP. Temporal-Order Judgements in Good and Poor Readers. Neuropsychologia 1988; 26: 917-924.
McAnally KI, Castles A, Stuart GW. Visual and auditory processing impairments in subtypes of developmental dyslexia: A discussion. Journal of Developmental and Physical Disabilities 2000; 12: 145-156.
McBride-Chang C, Wagner RK, Chang L. Growth modeling of phonological awareness. Journal of Educational Psychology 1997; 89: 621-630.
McDougall S, Hulme C, Ellis A, Monk A. Learning to Read - the Role of Short-Term-Memory and Phonological Skills. Journal of Experimental Child Psychology 1994; 58: 112-133.
Metsala JL, Stanovich KE, Brown GDA. Regularity effects and the phonological deficit model of reading disabilities: A meta-analytic review. Journal of Educational Psychology 1998; 90: 279-293.
Mishkin M, Ungerleider LG, Macko KA. Object Vision and Spatial Vision - 2 Cortical Pathways. Trends in Neurosciences 1983; 6: 414-417.
Moores E, Andrade J. Ability of dyslexic and control teenagers to sustain attention and inhibit responses. European Journal of Cognitive Psychology 2000; 12: 520-540.
Murphy L, Pollatsek A. Developmental Dyslexia - Heterogeneity without Discrete-Subgroups. Annals of Dyslexia 1994; 44: 120-146.
Nicolson RI, Fawcett AJ. Long-term learning in dyslexic children. European Journal of Cognitive Psychology 2000; 12: 357-393.
Nicolson RI, Fawcett AJ, Berry EL, Jenkins IH, Dean P, Brooks DJ. Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. Lancet 1999; 353: 1662-1667.
Nicolson RI, Fawcett AJ, Dean P. Developmental dyslexia: the cerebellar deficit hypothesis. Trends in Neurosciences 2001; 24: 508-511.
Nigg JT. Is ADHD a disinhibitory disorder? Psychological Bulletin 2001; 127: 571-598.
Nobre AC. The attentive homunculus: Now you see it, now you don't.

Neuroscience and Biobehavioral Reviews 2001a; 25: 477-496.
Nobre AC. Orienting attention to instants in time. Neuropsychologia 2001b; 39: 1317-1328.
Oldfield RC. The assesssment and analysis of handedness: The Edinburgh inventory. Neuropsychologia 1971; 9: 97-113.
Patel TK, Licht R. Verbal and affective laterality effects in P-dyslexic, L-dyslexic and normal children. Child Neuropsychology 2000; 6: 157-174.
Paulesu E, Frith U, Snowling M, Gallagher A, Morton J, Frackowiak RSJ, et al. Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. Brain 1996; 119: 143-157.
Posner M, Cohen Y. Components of Visual Orienting. In: Bouma H and Bouwhuis D, editors. Attention and Performance. Vol X. London: Erlbaum, 1984.
Posner MI, Fan J. Attention as an Organ System. In: Pomerantz J, editor. Neurobiology of Perception and Communication: From Synapse to Society the IVth De Lange Conference. Cambridge UK: Cambridge University Press, in press.
Posner MI, Petersen SE. The Attention System of the Human Brain. Annual Review of Neuroscience 1990; 13: 25-42.
Pugh KR, Mencl WE, Jenner AR, Katz L, Frost SJ, Lee JR, et al. Functional neuroimaging studies of reading and reading disability (developmental dyslexia). Mental Retardation and Developmental Disabilities Research Reviews 2000a; 6: 207-213.
Pugh KR, Mencl WE, Jenner AR, Katz L, Frost SJ, Lee JR, et al. Neurobiological studies of reading and reading disability. Journal of Communication Disorders 2001; 34: 479-492.
Pugh KR, Mencl WE, Shaywitz BA, Shaywitz SE, Fulbright RK, Constable RT, et al. The angular gyrus in developmental dyslexia: Task-specific differences in functional connectivity within posterior cortex. Psychological Science 2000b; 11: 51-56.
Rae C, Harasty JA, Dzendrowskyj TE, Talcott JB, Simpson JM, Blamire AM, et al. Cerebellar morphology in developmental dyslexia. Neuropsychologia 2002; 40: 1285-1292.
Ramus F. Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction? Current Opinion in Neurobiology 2003; 13: 212-218.
Ramus F. Neurobiology of dyslexia: a reinterpretation of the data. Trends in Neurosciences 2004; 27: 720-726.
Rayner K. Eye movements in reading and information processing: 20 years of research. Psychological Bulletin 1998; 124: 372-422.
Rey V, De Martino S, Espesser R, Habib M. Temporal processing and phonological impairment in dyslexia: Effect of phoneme lengthening on order judgment of two consonants. Brain and Language 2002; 80: 576-591.
Richardson U, Thomson JM, Scott SK, Goswami U. Auditory processing skills and phonological representation in dyslexic children.

Dyslexia 2004; 10: 215-233.
Ridder WH, Borsting E, Banton T. All developmental dyslexic subtypes display an elevated motion coherence threshold. Optometry and Vision Science 2001; 78: 510-517.
Ridder WH, Borsting E, Cooper M, McNeel B, Huang E. Not all dyslexics are created equal. Optometry and Vision Science 1997; 74: 99-104.
Robertson IH, Mattingley JB, Rorden C, Driver J. Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. Nature 1998; 395: 169-172.
Rorden C, Mattingley JB, Karnath HO, Driver J. Visual extinction and prior entry: Impaired perception of temporal order with intact motion perception after unilateral parietal damage. Neuropsychologia 1997; 35: 421-433.
Rose SA, Feldman JF, Jankowski JJ, Futterweit LR. Visual and auditory temporal processing, cross-modal transfer, and reading. Journal of Learning Disabilities 1999; 32: 256-266.
Ross J, Morrone MC, Burr DC. Compression of visual space before saccades. Nature 1997; 386: 598-601.
Ross J, Morrone MC, Goldberg ME, Burr DC. Changes in visual perception at the time of saccades. Trends in Neurosciences 2001; 24: 113-121.
Rothlind JC, Posner MI, Schaughency EA. Lateralized Control of Eye-Movements in Attention-Deficit Hyperactivity Disorder. Journal of Cognitive Neuroscience 1991; 3: 377-381.
Rueda MR, Fan J, McCandliss BD, Halparin JD, Gruber DB, Lercari LP, et al. Development of attentional networks in childhood. Neuropsychologia 2004; 42: 1029-1040.
Rumsey JM, Nace K, Donohue B, Wise D, Maisog JM, Andreason P. A positron emission tomographic study of impaired word recognition and phonological processing in dyslexic men. Archives of Neurology 1997; 54: 562-573.
Rushworth MFS, Ellison A, Walsh V. Complementary localization and lateralization of orienting and motor attention. Nature Neuroscience 2001; 4: 656-661.
Rushworth MFS, Nixon PD, Renowden S, Wade DT, Passingham RE. The left parietal cortex and motor attention. Neuropsychologia 1997; 35: 1261-1273.
Schulte-Korne G. Annotation: Genetics of reading and spelling disorder. Journal of Child Psychology and Psychiatry and Allied Disciplines 2001; 42: 985-997.
Shaywitz BA, Fletcher JM, Holahan JM, Shneider AE, Marchione KE, Stuebing KK, et al. Interrelationships between reading disability and attention- deficit hyperactivity disorder. Child Neuropsychology 1995; 1: 170-186.
Shaywitz SE, Shaywitz BA, Pugh KR, Fulbright RK, Constable RT, Mencl WE, et al. Functional disruption in the organization of the brain for reading in dyslexia. Proceedings of the National Academy of

Sciences of the United States of America 1998; 95: 2636-2641.
Sheppard DM, Bradshaw JL, Mattingley JB, Lee P. Effects of stimulant medication on the lateralisation of line bisection judgements of children with attention deficit hyperactivity disorder. Journal of Neurology Neurosurgery and Psychiatry 1999; 66: 57-63.
Sieroff E, Pollatsek A, Posner MI. Recognition of Visual Letter Strings Following Injury to the Posterior Visual Spatial Attention System. Cognitive Neuropsychology 1988; 5: 427-449.
Sireteanu R, Goertz R, Bachert I, Wandert T. Children with developmental dyslexia show a left visual "minineglect". Vision Research 2005; 45: 3075-3082.
Slaghuis WL, Ryan JF. Spatio-temporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. Vision Research 1999; 39: 651-668.
Snowling MJ, Goulandris N, Defty N. A longitudinal study of reading development in dyslexic children. Journal of Educational Psychology 1996; 88: 653-669.
Stanovich KE, Siegel LS. Phenotypic Performance Profile of Children with Reading Disabilities - a Regression-Based Test of the Phonological-Core Variable-Difference Model. Journal of Educational Psychology 1994; 86: 24-53.
Stanovich KE, Siegel LS, Gottardo A. Converging evidence for phonological and surface subtypes of reading disability. Journal of Educational Psychology 1997; 89: 114-127.
Stein J. Visual motion sensitivity and reading. Neuropsychologia 2003; 41: 1785-1793.
Stein J, Walsh V. To see but not to read; The magnocellular theory of dyslexia. Trends in Neurosciences 1997; 20: 147-152.
Stein JF, Richardson AJ, Fowler MS. Monocular occlusion can improve binocular control and reading in dyslexics. Brain 2000; 123: 164-170.
Stoodley CJ, Fawcett AJ, Nicolson RI, Stein JF. Impaired balancing ability in dyslexic children. Experimental Brain Research 2005; 167: 370-380.
Tallal P. Auditory temporal perception, phonics and the reading disabilities in children. Brain and Language 1980; 9: 182-198.
Tallal P. Temporal or Phonetic Processing Deficit in Dyslexia - That Is the Question. Applied Psycholinguistics 1984; 5: 167-169.
Tallal P, Piercy M. Defects of Nonverbal Auditory-Perception in Children with Developmental Aphasia. Nature 1973; 241: 468-469.
Temple E. Brain mechanisms in normal and dyslexic readers. Current Opinion in Neurobiology 2002; 12: 178-183.
Temple E, Poldrack RA, Salidis J, Deutsch GK, Tallal P, Merzenich MM, et al. Disrupted neural responses to phonological and orthographic processing in dyslexic children: an fMRI study. Neuroreport 2001; 12: 299-307.
Torgesen JK. Learning Disabilities: An Historical and Conceptual

Overview. In: Wong B, editor. Learning about Learning Disabilities. San Diego: Academic Press, 1998.
Torgesen JK. The prevention of reading difficulties. Journal of School Psychology 2002; 40: 7-26.
Torgesen JK, Wagner RK, Rashotte C. Test of Word Reading Efficiency (TOWRE). Austin, Texas: PRO-ED Inc, 1999.
Townsend JT, Ashby FG. The Stochastic Modelling of Elementary Psychological Processes. Cambridge: CUP, 1983.
Turkeltaub PE, Gareau L, Flowers DL, Zeffiro TA, Eden GF. Development of neural mechanisms for reading. Nature Neuroscience 2003; 6: 767-771.
University of Western Australia: School of Psychology. MRC Psycholinguistic Database, 2003.
Van Ingelghem M, van Wieringen A, Wouters J, Vandenbussche E, Onghena P, Ghesquiere P. Psychophysical evidence for a general temporal processing deficit in children with dyslexia. Neuroreport 2001; 12: 3603-3607.
Vidyasagar TR. A neuronal model of attentional spotlight: parietal guiding the temporal. Brain Research Reviews 1999; 30: 66-76.
Vidyasagar TR, Pammer K. Impaired visual search in dyslexia relates to the role of the magnocellular pathway in attention. Neuroreport 1999; 10: 1283-1287.
Wagner RK, Torgesen JK, Rashotte CA. Development of Reading-Related Phonological Processing Abilities - New Evidence of Bidirectional Causality from a Latent Variable Longitudinal-Study. Developmental Psychology 1994; 30: 73-87.
Waldie KE, Mosley JL. Developmental trends in right hemispheric participation in reading. Neuropsychologia 2000; 38: 462-474.
Walker R, Deubel H, Schneider WX, Findlay JM. Effect of remote distractors on saccade programming: Evidence for an extended fixation zone. Journal of Neurophysiology 1997; 78: 1108-1119.
Waters GS, Caplan D. The reliability and stability of verbal working memory measures. Behavior Research Methods Instruments & Computers 2003; 35: 550-564.
Wechsler D. Wechsler Abbreviated Scale of Intelligence (WASI). San Antonio: The Psychological Corporation: Harcourt Brace, 1999.
Whitney C, Lavidor M. Facilitative orthographic neighborhood effects: The SERIOL model account. Cognitive Psychology 2005; 51: 179-213.
Wilkinson GS. The Wide Range Achievement Test (WRAT). Wilmington, Delaware: Wide Range Inc, 1993.
Willcutt EG, Pennington BF, Olson RK, Chhabildas N, Hulslander J. Neuropsychological analyses of comorbidity between reading disability and attention deficit hyperactivity disorder: In search of the common deficit. Developmental Neuropsychology 2005; 27: 35-78.
Wimmer H, Hutzler F, Wiener C. Children with dyslexia and right parietal

lobe dysfunction: event-related potentials in response to words and pseudowords. Neuroscience Letters 2002; 331: 211-213.
Witton C, Talcott JB, Hansen PC, Richardson AJ, Griffiths TD, Rees A, et al. Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. Current Biology 1998; 8: 791-797.
Wolf M, Bowers PG. The double-deficit hypothesis for the developmental dyslexias. Journal of Educational Psychology 1999; 91: 415-438.