A Behavioural and Electrophysiological exploration of the Working Memory impairment in Developmental Dyslexia

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Memorandum

The research within this thesis was conducted at the School of Psychology, University of Kent, whilst the author was a full-time postgraduate student. The theoretical and empirical work presented within this thesis is the independent work of the author. Intellectual debts are cited within the text and referenced at the end of the thesis. The eight studies presented in this thesis were conducted with limited assistance from others. The author has not been awarded a degree by this, or any other, University for the work included in this thesis.

Abstract

The current thesis provides a behavioural and electrophysiological exploration of Working Memory (WM) processing in developmental dyslexia. This thesis identifies a debate in the literature regarding the extent to which individuals with dyslexia have a specific phonological WM impairment, or a domain general Central Executive (CE) impairment. Predictions from the latter account suggest that dyslexics should show an impairment in visual, and verbal domains of WM. However, findings in the visual domain have been inconsistent, and research has predominantly focused on children. The experimental work in this thesis examines CE processing in dyslexic adults by assessing the behavioural and ERP responses associated with WM, across 8 experiments. Experiments 1-5 present stimuli in the visual domain, while Experiments 6-8 are conducted in the auditory domain. The results indicate that dyslexics are impaired for verbal information specifically, however subtle RT differences emerge during visual-spatial WM, when participants are required to manipulate information. In order to assess why effects are more robust in the phonological domain, Experiment 8 examines the contribution of auditory perceptual problems and phonological WM processing in dyslexia. The Temporal Sampling Theory of Developmental dyslexia (TSTDD; Goswami, 2011) specifies that dyslexics have a difficulty processing tones with long rise-times. In Experiment 8, dyslexic participants show a WM impairment that is specific to tones with long rise-times. The theoretical implications of these findings are discussed, and a new hypothesis regarding the phonological WM impairment in dyslexia is proposed. The original contribution to knowledge of this thesis are threefold. 1) The ERP responses associated with WM processing in developmental dyslexia are examined across modality, using a range of stimuli. 2) A novel task is used to directly investigate CE processing in dyslexia (Experiment 5). 3) The TSTDD is applied in order to investigate phonological WM in dyslexia.

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Thesis Contents

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Chapter 1: Working Memory and methodological approach.

The experiments presented in this thesis examine how Working Memory (WM) is impaired across different modalities of memory in individuals with developmental dyslexia, using behavioural measures (accuracy, and signal detection theory), and Event Related Potentials (ERPs). The current chapter contains an introduction to the WM literature, focusing predominantly on the Baddeley and Hitch WM model, which has facilitated and constrained experiments examining dyslexia and WM. In the latter half of the chapter, a review of relevant task paradigms (N-back, and change detection tasks), and methodologies (ERP) to the experimental work in this thesis is provided.

Thesis Introduction

WM is a system responsible for temporarily maintaining and manipulating information needed in the execution of complex cognitive tasks, such as reasoning, learning, and comprehension (Alloway, 2006). According to Baddeley and Hitch (1974), and Baddeley (2000), WM consists of a domain general central executive store, a phonological loop, a visual spatial sketchpad, and an episodic buffer (see below for a detailed description of these components). For typically developing individuals, WM capacity increases steadily up to the age of 14/15 years, where it reaches maturity (e.g., Alloway, Gathercole, & Pickering, 2006). However, for some individuals, WM shows atypical development, which results in reduced WM capacity, compared to individuals of a similar age (e.g., Westerberg, Hirvokoski, Forssberg, & Klinberg, 2004). Impaired WM is associated with a range of neurodevelopmental disorders (see Alloway, & Gathercole, 2006). Developmental dyslexia is one of the most prevalent neurodevelopment disorders (Cao, Bitan, Chou, Burman, Booth, 2006), present amongst 5-17.5% of the population (Shaywitz et al., 1998). Over thirty years

of research has demonstrated that both children and adults with dyslexia suffer poor phonological WM (Wagner & Torgesen, 1987; Ramus & Szenjovits, 2008).

Understanding WM processing in individuals with neurodevelopmental disorders is essential, as poor WM may lead to a variety of other cognitive difficulties. Impaired WM presents a significant risk factor for poor educational progress (e.g., Gathercole & Alloway, 2008). For example, the contribution of WM for language skills was originally documented by Baddeley, Gathercole, Papagno, and Costanza (1998), who suggest that the phonological WM store is essential for storing novel sounds, while permanent memory records are being constructed. Beneventi et al., (2010) suggest that the development of language skills will depend on WM, as the phonological store is also needed to consciously detect and manipulate speech sounds. It is needed to blend individual phonemes, and the central executive is needed to simultaneously activate grapheme-phoneme conversion rules in LTM. Verbal WM is also a predictor for a range of other complex cognitive functions, such as mathematics (e.g., Bull & Scerif, 2001; Mayringer & Wimmer, 2000; Siegel & Ryan, 1989). Given that WM processing is implicated in a wide range of cognitive tasks, it is essential to understand and conceptualize how WM might be impaired in dyslexia. This importance has been emphasised by the UK Disability Discrimination act, (1995), and the Equality Act, (2010), where it states that there is a need to understand and address all of the difficulties an individual with dyslexia might face.

Despite this need, there is still much debate surrounding the nature of the WM impairment in developmental dyslexia. The majority of research in this area has been largely limited to the phonological domain, and research in the visual domain has been limited. In Chapter 2 of this thesis, it is argued that this research bias has largely been because individuals with dyslexia nearly always show an impairment in the verbal domain, as

opposed to the visual domain (e.g., Vellutino, 1979). This has led some authors to argue that individuals with dyslexia are impaired in phonological WM processing only (e.g., Jeffries & Everatt, 2003, 2004; Kibby, Marks, Jordan & Long, 2004). However, this assumption is currently under debate, with some researchers arguing for a domain general central executive impairments in dyslexia (e.g., Wang & Gathercole, 2013). One way of disiphering between a specific phonological loop impairment in dyslexia, and a domain general central executive impairment, is to assess WM performance for both verbal and visual stimuli. If an impairment in WM is found in both WM domains, then this suggests a domain general impairment in dyslexia. The empirical work in this thesis takes this approach, examining WM processing across modality, in order to conceptualise the nature of WM processing in dyslexia. In Chapter 2, a full outline of the phonological loop vs central executive debate is detailed.

Furthermore, in Chapter 2 it is argued that most of the research examining WM processing in dyslexia has been conducted with children, as noted by Swanson, Zheng, and Jerman (2009). In a meta-analysis, Swanson, Zheng, and Jerman (2009) conclude that experiments examining visual spatial, and central executive processing in dyslexia have predominantly only included 5-18 year olds. One possibility is that central executive dysfunctions are restricted to this group. Indeed, the developmental lag explanation has been proposed in the literature (Case, Kurland, & Goldberg, 1982), and one possibility is that processing speed and efficiency will increase over the developmental trajectory, facilitating WM processing in dyslexia. Thus, the extent to which adults with developmental dyslexia have a domain general central executive impairment is largely unknown, suggesting a pressing need to examine CE processing in this group.

The aims of this thesis are to examine the extent to which adults with developmental dyslexia have a specific phonological loop, or domain general central executive impairment. In order to achieve this goal, two theoretical, 4 empirical, and 1 conclusion chapter are presented. The current chapter continues with an overview of early theoretical accounts of WM, before reviewing the methodological approaches, and task paradigms which form the basis of the empirical work in this thesis. Chapter 2 contains a literature review of research conducted within the area of developmental dyslexia and WM. Chapter 2 completes with a summary of the thesis aims, and an overview of the empirical work conducted. Overall, this thesis makes several predominant original contributions to knowledge:

1) The ERP responses associated with WM processing in developmental dyslexia are examined across modality, using a range of stimuli, in a single task paradigm (predominantly the N-back task). In Chapter 3, stimuli consist of visual-letters, pictorial visual-objects, and Chinese ideograms. These items are static in space. In chapter 4, visual spatial information is manipulated in a WM task, while in Chapter 5 auditory letters and auditory words (manipulated by their Age of Acquisition; AoA) are used. In the final chapter of this thesis, tones (manipulated by the time taken to reach their amplitude peak) are presented during an N-back task. Using a range of stimuli allows researchers identify whether or not individuals with dyslexia have a specific phonological loop impairment, or a domain general CE impairment.

2) A novel task is used in order to directly investigate central executive processing in dyslexia (Experiment 5). The extent to which WM processes are limited to impairments in the phonological loop, or is caused by an additional deficit in central executive functioning is still a major question in the research (Beneventi, Tonnessen, Ersland, and Hughdahl, 2010). Previous research examining central executive processing in children have used complex

span tasks, controlling for simple span task performance. In Chapter 4, a predominant aim is to examine central executive processing in dyslexia using a single task paradigm. The spatial delayed response task (SDRT; Glahn et al., 2002) is used, which allows us to directly compare passive, versus active WM processing. Furthermore, this task allows us to examine the ERP response at encoding and retrieval.

3) The Temporal Sampling Theory of Developmental Dyslexia (TSTDD; Goswami, 2011) is applied in order to investigate phonological WM impairment in dyslexia. It remains unclear whether the WM impairment in developmental dyslexia is just a secondary effect of a low level auditory processing deficit (see Beneventi, Tonnessen, Ersland, & Hughdahl, 2010 for a full review of this argument). Goswami (2011)'s Temporal Sampling Theory of Developmental Dyslexia suggests that a key impairment may lie in oscillatory phase-locking in auditory cortex to sounds with slower temporal modulations. Goswami et al. (2002) have also demonstrated that individuals with dyslexia have a difficulty discriminating amplitude modulated sounds which takes longer to reach their amplitude peak (tones with longer rise-times). In Chapter 6, participants are required to take part in an N-back task with tones which are manipulated by their rise-time. This allows us to examine the relationship between perceptual temporal sampling impairments, and WM.

4) Finally, very few experiments have assessed the electrophysiological correlates of the WM deficit in dyslexia. Research by Beneventi and colleagues (2010), cited earlier in this chapter used fMRI to reveal the anatomical neural correlates of central executive and phonological processing in dyslexia. However research investigating the event related potential (ERP) response during a WM task are not evident in the literature. Measuring ERP responses alongside behavioural measures will provide an additional insight into whether WM processing differences exist between groups, and in which memory domains. ERPs have

excellent temporal resolution, and can thus highlight if any speed of processing deficits might underlie WM impairments in adults with dyslexia, as speculated upon by Swanson, Zheng, and Jerman (2009). The benefits of applying ERPs to examining between group differences in WM processing are described in more detail later in this chapter, and in Chapter 3 and 4. To my knowledge, this is the first time that the electrophysiological correlates WM processing in dyslexia have been assessed, particularly using a wide range of stimuli.

A Theoretical introduction to Short Term Memory (STM) and WM

Hebb (1949) suggested a biological distinction between STM, and Long Term Memory (LTM). The former involved temporary electrical activation, whereas LTM was based upon neuronal growth, and by the mid 1960's, the consensus was that STM and LTM were two distinct systems (Baddeley, 2003). According to the modal model of Murdock (1967), memory can be classified into three levels of storage: sensory stores, STM and LTM. Early attempts to conceptualise STM can be traced to Miller, Galanter, and Pribram (1960), who argued that it is a temporary store for information, in their theoretical account of human planning. The researchers suggested that in order for a plan to be executed, it is likely that important transient information is placed in a 'special' location, where it can be remembered. This special place was likely to be located in the frontal lobes of the brain, however, the specific mechanisms used to store information, were unknown to the authors. Later, Broadbent (1958) proposed a filter model of selective attention. On the basis of dichotomous listening tasks, Broadbent proposed that information enters a sensory buffer where it is held transiently. Transient maintenance was implemented through the recycling of information, before it is selected to enter a limited capacity processing channel. Selection was conceptualized to take place through a filtering mechanism, which prevented the short term

store from becoming overloaded. From the short term store, information could be transferred into a more permanent long term store.

Atkinson and Shiffrin (1968) were the first to propose a serial stage model of STM. All attended information, arrives at a sensory register, before entering a short term store. This information can either be forgotten, or rehearsed and stored within LTM. The latter process was said to involve systematically rehearsing the last few items presented, in a rehearsal buffer. Atkinson and Shiffrin emphasized the importance of this temporary store for human cognition, claiming that it could be equated with consciousness. Furthermore, they suggested that the store could be attributed to the notion of 'WM', as described by Miller, Galanter, and Pribram (1960). Therefore, early work focused on the concept of stores, and the transfer of information among them (Craik & Lockhart, 1972). As well as characterizing the nature of short term storage, early theories predominantly focused on verbal material. Verbal items were thought to be coded in auditory-verbal-linguistic terms (Atkinson and Shiffrin, 1968), in a phonemic fashion (Shulman, 1971). Capacity limits were estimated as 7 plus or minus 2 digits (Miller, 1965).

Baddeley and Hitch (1974) sought to build a model of WM that improved upon some of the limitations that they saw in Atkinson and Shiffrin (1971)'s model. Baddeley (2003) suggests that Atkinson and Shiffrin's account of STM was problematic due to its assumptions it made regarding long term learning. If STM served as a unitary WM, then Baddeley and Hitch theorized that patients with impaired STM should show little capacity for long term learning, or for other cognitive activities. However, patients with STM damage did not show global LTM damage, which would have otherwise been predicted from Atkinson and Shiffrin's model (see Baddeley, 2003). Baddeley and Hitch (1974) used a secondary task to deplete the availability of STM, and found clear, but far from catastrophic impairment upon

LTM functioning. Thus, their 1974 model of WM was proposed. This model has been repeatedly tested and validated in the literature, and is cited consistently in research examining WM impairments in neuro-developmental disorders (for a review, see Alloway & Gathercole, 2006). Thus the model offers a theoretical framework to research examining WM processing in neurodevelopmental disorders, as research has been concerned with defining which aspect of WM processing is impaired in a given disorder e.g., the extent to which the visual-spatial sketchpad is impaired in dyslexia (e.g., Smith-Spark , Fisk, Fawcett, & Nicolson., 2003).

The Working Memory Model (Baddeley & Hitch, 1974; Baddeley, 1986; 2000, 2007)

The tripartite model of Baddeley and Hitch (1974) was the first to propose that WM was not a unitary system. Instead, they proposed that WM consisted of the central executive a control system of limited attentional capacity, and two sub-systems; the Phonological Loop and the Visual-spatial Sketchpad. Later, Baddeley (2000) introduced the Episodic Buffer. All three sub-systems, along with the central executive, are depicted in Figure 1. The Figure displays the interaction between displaying the interaction between the central executive, Visual-spatial sketchpad, Episodic Buffer and Phonological Loop and their relationship to wider cognitive processes.

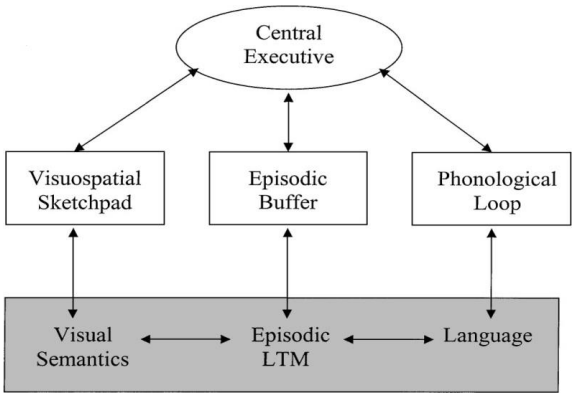


Figure 1: The: revised Working Memory model (Baddeley & Hitch, 1974; Baddeley, 2000 2007).

Initially Baddeley and Hitch (1974) compared the central executive to a convenient homunculus – a little man who sits in the head and in some mysterious way makes the important decisions. The central executive is not equipped with any supplementary storage capacity. Instead, the central executive is responsible for the control of executive processes, including actions, as well as the suppression of irrelevant information. It is also responsible for the coordination of multiple cognitive processes, which require parallel execution. While the earlier work of Baddeley and Hitch (1974) treated the central executive as a pool of general processing capacity, Norman and Shallice's (1980) work provided more specificity. Norman and Shallice (1980)'s model of attentional control was the first attempt to advance understanding of the central executive. The model divided control between two processes: the first relied on the control of behaviour by schemas, while the second comprised the supervisory attentional system (SAS). The SAS acted as an attentionally limited controller that could intervene when routine control was insufficient. For this reason, the central executive is commonly compared with attention, and is responsible for coordinating the three subcomponents of WM described below (The phonological loop, visual spaial sketchpad, and the Episodic Buffer; Baddeley & Hitch, 1974; Baddeley, 2000).

The phonological loop includes a passive phonological store, involved in maintaining auditory information. It stores information in phonological code, for a few seconds, before the code fades. An articulatory rehearsal process, that is comparable to sub-vocal speech, maintains information within the store. This serves to refresh the decaying representations, so they can be re-articulated. Immediate memory has a limited span, because articulation takes place in real time (Baddeley, 2003). The role of articulation is supported by experiments on the word length effect whereby memory span declines as word length increases, due to the slower rehearsal of longer words (e.g., Baddeley, Thomson, &

Buchman, 1975). For a full review of experiments conceptualizing the nature of the Phonological loop, see Baddeley (2003). However, the primary function of the phonological loop is to facilitate speech perception and comprehension (see Baddeley, Gathercole & Papagno, 1998).

The second subsystem, the Visual-spatial sketch pad (VSSP), maintains and manipulates visual, and spatial information (typically about three or four items, Luck & Vogel (1997)). Baddeley (2003) argues for a visual-spatial distinction. This double dissociation has since been supported by research evidence (e.g., Menghini et al., 2011). Logie (1995) terms the visual component, the visual cache, and the spatial component the inner scribe. The visual cache allows humans to form mental images, and the inner scribe allows us to rotate them and navigate mental maps. The VSSP also contains a rehearsal mechanism, which specializes in preserving visual and spatial properties by means of their continual re-imagination (Baddeley & Hitch, 1974; Baddeley, 1986).

In 2000, Baddeley added a third sub-component to the model; the Episodic buffer. Baddeley (2003) claims that the episodic buffer is a crucial feature of the capacity of WM, as it acts as a global work-space, which can be accessed by conscious awareness. The episodic buffer is a limited capacity store, which could be regarded as the storage component of the central executive. The episodic buffer is responsible for the binding of information together to form integrated episodes. Baddeley (2000) suggests the buffer uses multidimensional coding to integrate information from the two other subsystems (i.e., phonological and visual), along with other types of information (e.g., semantic), into an episodic representation. The addition of the episodic buffer also provided an interface between the sub-systems and episodic LTM. Adaptations to the episodic buffer have since been provided by Baddeley, Allen, and Hitch (2010) who suggest that the episodic buffer is a passive store, which stores

bound features. It is not responsible for the binding process, but responsible for making the resulting features accessible to consciousness. Critically, as depicted in Figure 1, the episodic buffer is connected directly to the central executive, as opposed to the phonological loop and visual spatial sketchpad. Thus, the central executive controls access to and from the buffer, and consequently an attentionally demanding concurrent task should have substantial effects on one's capacity to bind information. In a speculative model presented by Baddeley (2012), the episodic buffer is connected both to the central executive and the phonological loop and VSSP.

The distinction between STM and WM

Within the literature, STM and WM are often used to describe distinct cognitive components, but both WM and STM tasks share some common activities (e.g., Swanson, Zheng, & Jerman, 2009; Colom, Flores-Mendoza, Quiroga, & Privado, 2005). Both refer to the ability to temporarily maintain information in mind. As noted by Daneman and Carpenter (1980), STM refers to a passive storage capacity, whereas WM refers to a more active part of human processing. WM includes storage, but also executive processing of the stored products (Baddeley & Hitch, 1974; LaBerge & Samuels, 1974).

STM and WM tasks both involve a controlled process, i.e. rehearsal (e.g., Unsworth & Engle, 2007). For WM tasks, this might involve attention, whereby a memory trace is maintained in the face of interference. This might draw resources from the executive system (Engle, Tuholski, Laughlin, & Conway, 1999). However, for STM tasks, the controlled processes will involve attempts to maintain memory traces above some critical value. Engle, Kane, and Tuholski (1999) state that the controlled processing in STM may utilize perceptual grouping, or chunking skills, phonological coding, and rehearsal speed. As WM is assumed

to have storage as well as active functioning, the phonological loop has been equated with STM (e.g., Baddeley, 1986).

Task paradigms and Analyses

The experimental work in this thesis uses two task paradigms to examine WM processing: the N-back task, which is used in the experiments in chapter 3, 5 and 6, and a spatial delayed response task (SDRT; Glahn et al., 2002), which is used in chapter 4. A review of literature examining WM processing using these two task paradigms will now be provided.

The N- back task

N-back task is a popular task for examining WM processing (Kirchner, 1958). In a typical experimental procedure, participants are presented with a stream of stimuli (auditory or visual) and are required to make a response to every stimulus, regarding whether or not the stimulus matches one presented N items back in the stream. For example, in the 1-back condition, participants respond 'yes' when the current stimulus matches the stimulus presented 1 backwards in the stream. An increase in N typically leads to lower accuracy and longer reaction times (e.g., Jonides, Smith, Koeppe, Awh, Minoshima, Mintun, 1993). The N-back task was originally introduced as a visual spatial WM task, by Kirchner (1958), who aimed to assess the effect of age upon WM. WM load was manipulated at a range of 0-back to 3-back. In the 0-back task, participants were required to respond 'yes' every time the current stimulus matched the first stimulus that had been presented in the stream. In the 1 back task, participants respond 'yes' when the current stimulus matches the one presented one previously, in 2 and 3 back, participants respond 'yes' when the current stimulus matches 2 or 3 back respectively.

Participants were required to observe a row of 12 small lights above a row of 12 associated keys, and to respond using the key where the light had gone out N positions

before. Thus, in the 2-back load, participants were required to compare the current item to the item presented 2-back, and thus maintain, and manipulate 3 items in WM. Once a comparison had been made, participants can drop the first piece of information, in preparation to store the next item. As discussed in chapter 2, there is a debate in the literature regarding the extent to which individuals with developmental dyslexia have a specific WM impairment attributed to the phonological loop (For a full review, see Snowling, 2000; and more recent research by Kibby, Marks, Morgan, & Long, 2004), or a visual spatial sketchpad impairment (e.g., Smith-Spark & Fisk, 2007; Smith-Spark, Fisk, Fawset, Nicolson, 2003; Menghini, Finzi, Carlesimo & Vicari, 2011; Wang & Gathercole, 2014). The authors who uncover a visual spatial sketchpad deficit, argue that the impairment is likely to be due to central executive dysfunction.

However, this claim is still under debate, and the question has been largely neglected in adult populations. Throughout this thesis, the extent to which adults with developmental dyslexia have a specific phonological loop impairment, or a more general central executive difficulty, is investigated by probing both verbal and visual-spatial WM processing. The N-back task provides a relevant context for examining central executive processing in dyslexia. In his seminal work, Kirchner (1958) claimed that there was a central system, similar to the central executive, with limited resources, that organized the interchange of information within the continuous stream. The N-back task was able to capture the workings of this central system. The N-back task has been used to examine the visual spatial sketchpad and phonological loop components of WM, and is thus an excellent task for assessing the interaction between sub-system processing, and the central executive. For example, Mackworth (1959) used the paradigm as a visual letter task, and Awh et al., (1996), and others since (e.g., Braver et al., 1997; Veltman, Rombouts & Dolan, 2003) have used verbal

versions of the task. Furthermore, in Chapter 2, the suitability of this task for examining ERP responses is discussed.

Spatial delayed response tasks (SDRT)

The delayed response tasks, presented in Chapter 4 of this thesis, are modelled on Glahn et al., (2002). In this paradigm participants are presented with a target array (S1), and a probe array (S2), separated by a fixed delay. During S1, an array of circles (with load manipulated as 1, 3, 5, and 7), are positioned pseudorandomly around a fixation point on a computer screen. At S2 a single probe circle is presented, and participants are required to indicate if that circle is in the same position as one of the previously shown target circles. Behvioural performance, reported as percentage correct, shows that as load increases, there is a near-linear decrease in percentage correct responses. Reaction times also increase with increasing memory set size (Glahn et al., 2002a).

The SDRT is similar to change detection tasks (Luck and Vogel, 1997), however it is designed to test the presence or absence of a spatial location in WM, as opposed to the presence or absence of a visual item. During a change detection task, participants are presented with a brief sample array of several stimuli, a short delay, and then a test array which is either identical to the sample array, or different in terms or a feature or objects (e.g., Luck & Vogel, 1997; Schmidt, Vogel, Woodman & Luck, 2002). Subjects are required to report if there was a change between arrays. Glahn et al. (2002b) adapt this paradigm to contrast maintenance and manipulation of spatial information. In the maintenance alone condition, subjects are presented with a fixed number of 3 locations (set size is constant), and are asked to remember each location. In the maintenance plus manipulation conditions, subjects are instructed to flip the maintained representation over a horizontal meridian line, presented explicitly on the screen. Manipulation is therefore operationalized as mental

rotation, as in (Cohen et al., 1996). Critically in this condition, proportion correct decreases and reaction times increase, when participants perform the maintenance plus manipulation condition. Both the N-back task, and the SDRT allow for a thorough examination of WM processing, across modality in developmental dyslexia.

Experiment analysis

The experiments in this thesis assess WM processing in dyslexic and non-dyslexic individuals through an analysis of behavioral measures (Accuracy in terms of hit rate and correct rejects, and Signal Detection Theory parameters). Response accuracy and SDT variables are able to provide information about the presence of a mnemonic representation in WM, and these measures are considered to be the prime dependent variables (DVs) in this thesis. These measures are chosen because the independent variable of interest (WM load) typically modulates the number of correct responses, in both the N-back (e.g. Kirchner, 1958; Mackworth, 1959), and SDRT (Glahn, 2001). Response accuracy is considered informative because the average accuracy of participants in the different experimental conditions provides information about the extent to which an item is held in WM. This allows us to make an important between group comparison. Furthermore, SDT variables were considered to be important dependent variables. SDT is used as an extension of the accuracy DV, to provide information about discriminability, and response bias (see below for a full description of SDT).

While RT analysis was conducted for each experiment, RTs were not considered to be a prime DV of interest for the majority of experiments in this thesis (Experiments 1, 2, 3, 4, 6, and 8). Thus, for these experiments, the RT analysis is reported in a footnote, in each chapter. This is because RTs do not directly provide direct insights into the quality or nature of a mnemonic representation – a WM representation is either present or absent (with some

degree of variability) in WM. Furthermore, the decision to present the RT analysis as a footnote was made because the statistical analysis did not reveal any statistical differences between the groups of interest (dyslexic and non-dyslexic groups). RTs are however presented as a footnote, in order to demonstrate that individuals with dyslexia did not show lower accuracy scores because they were responding quicker than non-dyslexic individuals. However, in Experiments 5 and 7, the experimental question regards how readily available an item is in the retrieval process. In Experiment 5, participants are required to manipulate information, and thus, speed of access to the mental representation is considered an important IV. Similarly, in Experiment 7, the IV of interest is Age of Acquisition, and the hypothesis regards a speed of access advantage for early vs. late learned words. Thus the full RT analysis is reported for Experiments 5 and 7 only.

Alongside these behavioural measures, EEG is recorded, and the P300 (Chapters 3, 4, 5 and 6) and N2 component (Chapter 7) is analysed. These components were chosen because they are electrophysiological markers of WM, which are known to be modulated by difficulty, and WM load (e.g., Watter, Geffen, & Geffen, 2001; Riby & Orme, 2013). The remainder of this chapter focuses on describing these analysis methods, and how they will contribute to the research conducted in this thesis.

Signal Detection Theory

Signal detection theory (Peterson, Birdsall, & Fox, 1954) is used to explain how individuals make binary decisions in the presence of uncertainty. Uncertainty can arise when participants are required to categorise ambiguous stimuli. In the current context, uncertainty arises in the N-back task, when participants are required to respond ‘yes’ or ‘no’ as to whether the stimulus occurred N back (signal plus noise), or not (noise alone). Or, in the SDRT (Glahn et al., 2002), uncertainty arises when the participant must decide if the probe is in one of the

target locations. When the signal was present, i.e. the stimulus did occur N items back, and the subject responds 'yes' then this is considered a hit. If the signal is present, but the subject responds 'no' then this is considered a miss. If the signal is absent and the subject says 'yes', this represents a false alarm, however if the subject selects 'no' when the item is from the noise distribution, then this is a correct reject. Accuracy on this task is therefore the sum of hits and correct rejections.

In the current research, two SDT variables are of interest; the discrimination index (D-prime; d') and the criterion value. The discrimination index represents a participant's ability to discriminate between old and new items. It represents the distance between the means of the signal plus noise, and noise distribution. It is assumed throughout that the standard deviation of the noise plus signal distribution equals the standard deviation of the noise distribution. Therefore d' can be theoretically described as $d' = (\mu n - \mu s) / \sigma$, whereby μn is the mean of the noise distribution, and μs is the mean of the signal plus noise distribution. D-prime and is calculated in Excel as NORMSINV(hits)-NORMSINV(false alarms). The value of d' represents the extent to which the noise, and the signal plus noise distribution overlap. Higher d' scores represent greater separation of these two distributions, and indicate greater task performance.

There are two main components that influence the decision making process: stimulus strength (e.g., how representative the current stimulus is of one that occurred N back), and the criterion. The criterion represents the individuals bias in the decision making process. The criterion is located relative to the intersection of the old and new distributions. The intersection point defines the zero point, and distance from the criterion is measured in z score units. A completely neutral bias has a criterion score of zero. A more positive criterion indicates a more conservative bias, while negative scores indicate a more liberal criteria.

The criterion is computed by the distance of the criterion from the mean of the new distribution, which is set equal to 0, the z score having probability above it equal to the false alarm rate. To change the zero point from the mean of the new distribution to the intersection of the signal plus noise and noise distributions, d'/2 is subtracted from the Z score of FAs (ZFA). Thus, the criterion is calculated with the following formula: C = ZFA – d' / 2 =0.5 (ZFA + ZH), and is calculated in excel using: = – (NORMSINV(hits) + NORMSINV(false alarms)) / 2. The noise, and signal plus noise distribution are represented in Figure 2.

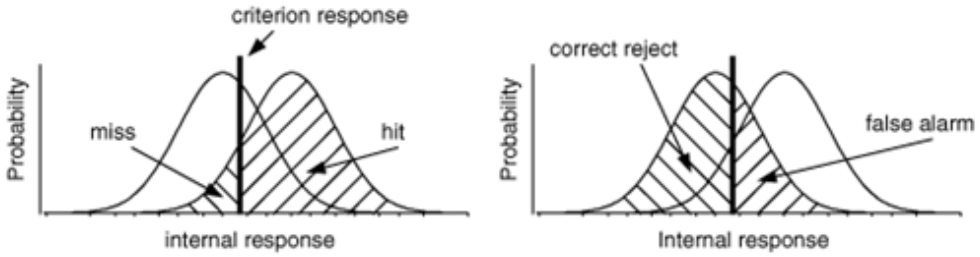


Figure 2: Internal response probability functions for signal and noise distributions. The noise distribution (distribution highlighted in the right panel) and signal plus noise (distribution highlighted in the left panel) distributions are both presented. The black central line represents the criterion, while the overlap of the distributions represents d'. The Figure has been taken from Heeger (1997).

For circumstances where there is a hit rate of 0 or 1, a standard correction method (Snodgrass & Corwin, 1988) is employed. This involves adding 0.5 to the frequency count of each response type, and dividing by N+1. N is the number of trials in that class. This is carried out on all response types (hits, false alarms, correct rejections, misses) regardless of whether they are 0 or 1 for consistency. Therefore, D-prime indicates the strength of the signal relative to noise, while C reflects the response strategy of the participant.

Electrophysiological correlates of WM processing

Electroencephalogram (EEG)

The EEG approach was developed in the nineteenth century, when Caton (1875) found it was possible to record changes in electrical potentials from the scalps of monkeys, cats and rabbits. The EEG signal is generated by the post-synaptic dendrites of pyramidal cells and reflects the summation of postsynaptic potential changes. The signal generated is less than 100 microvolts, so is amplified before recording. The resulting signal is the difference between two electrodes, one of which is a reference electrode.

Cognitive processing requires the integration of many, widely distributed, interacting areas of the brain (e.g., Basar, 2005). Complex cognitive processing could be implemented by the synchronisation of neurons. The synchronised rhythmic activity of large groups of neurons, give rise to oscillations of varying field potentials. Neural synchrony or desynchrony can be assessed by means of EEG recordings, and analysed by assessing EEG power. EEG power reflects the number of neurons that discharge synchronously (Klimesch, 1999), and is often calculated using wavelet or fourier analysis which decomposes the signal into different frequencies. EEG frequencies have been subdivided into frequency bands, including theta (~4-8Hz), alpha (~8-12Hz), beta (~14-30Hz) and gamma (~40Hz) (Krause, Sillanmaki, Koivisto, Saarela, Haggqvist, Laine, & Hamalainen, 2000).

Krause, et al. (2000) presented subjects with a visual letter N-back task, across three different WM load conditions, 0-2 back. The findings were in line with previous observations that EEG theta power increased (e.g., Klimesch et al., 1997), for target trials. Greatest Event Related Synchrony (ERS) in the theta range, peaking at 500ms after stimulus presentation, was observed for target stimuli. The presentation of non-targets elicited a shorter latency, and smaller magnitude theta ERS, than the presentation of targets. This suggests that there is a

unique brain response associated with both target and non-target trials. Specifically, in terms of WM load, the researchers observed effects in the alpha range, with a 6-8 and 8-10Hz ERS. This might reflect an inhibition of frontal cortical brain areas which are no longer involved in task completion. This alpha ERP was observed in the 2-back condition, suggesting it is specific to high WM load conditions. These results were later replicated by Pesonen, Hamalainen, and Krause (2007), with the addition of a 3-back condition. The results demonstrated a long lasting theta ERS, which was of greater magnitude for target stimuli, compared to non-target stimuli. Now, Beta Event Related Desynchrony (ERD) responses were elicited in all load conditions, with a longer ERD with increasing memory load. This is in line with research (e.g., Axmacher et al., 2009) that suggests a modulation of beta in response to load; potentially reflecting increased gamma cycles nesting into beta.

Event related Potentials (ERPs)

The EEG recordings typically refer to a measure of gross global brain activity which is not time locked to a stimulus, or response. Furthermore, in the raw EEG signal, the signal to noise ratio is low. To increase the signal to noise ratio, a standard method has been to average over repeated trials. To calculate ERP components, the EEG is segmented and aligned according to the onset of an external event (e.g., the onset of a stimulus or the onset of the participant's response). The ERP method therefore assumes that an individual evoked response involves a reliable signal as well as uncorrelated noise. Of distinct relevance to the current work is the P300b component. This endogenous ERP component occurs 300-800ms poststimulus, and can be used as an index of processing meaningful, significant, memorised and task related stimuli (Donchin & Coles, 1988; Polich, 2007). It has been associated with consolidation of information into WM (Donchin, 1981; Vogel, Luck, & Shapiro, 1998; Kranczioch, Debener, & Engel, 2003). A depiction of the P300, can be found in Figure 3.

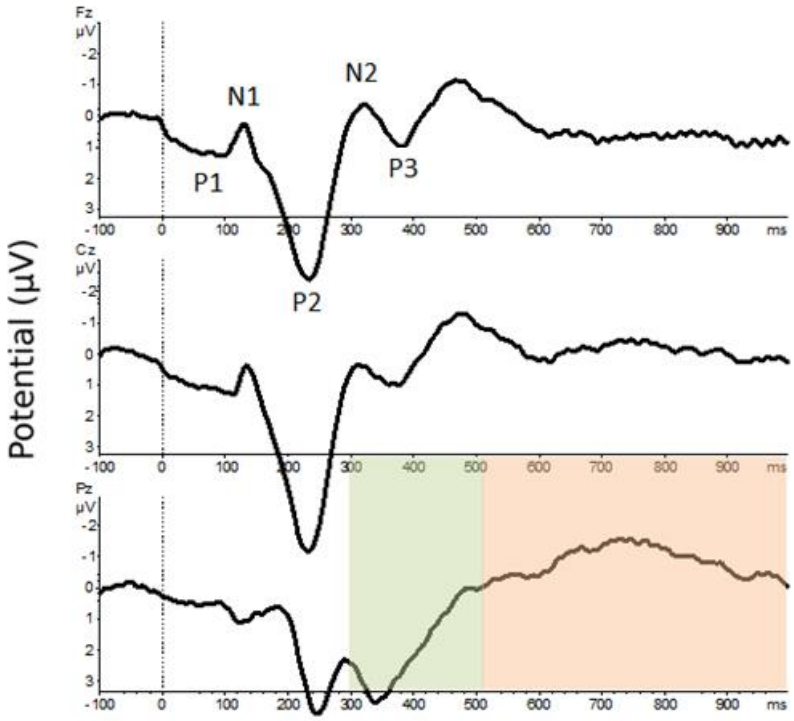


*Figure 3***:** An example of an ERP grand average plot.

In Figure 3, the P300 is the largest positive component, peaking between 300-500ms**.** *Note: Positive is plotted down.* The green region highlights the P300 positive component, while the orange region highlights a negative region, often found to proceed the positive deflection.

To quantify the P300 waveform, researchers typically analyse P300 amplitude and latency. Amplitude (μV) can be defined as the difference between the mean pre-stimulus baseline voltage (approximately 0 μV after baseline correction), and the largest positive-going peak of the ERP waveform within a time window (e.g., 250–500 ms). However, this time window is variable, depending on the stimulus modality, stimulus duration, task demands, etc. Another dependent measure of interest, is the P300 latency (ms). This can be defined as the difference between the time at stimulus onset (0ms) and the point of maximum positive amplitude within the same time window. The P300 scalp distribution is defined as the amplitude change over electrodes (typically Fz, Cz, Pz). The P300 typically increases in

magnitude from the frontal to parietal electrode locations (Johnson, 1993). Thus, one might hypothesise the strongest effect in parietal or posterior electrode locations.

The P300 waveform can be divided into two subcomponents, elicited for novel (P3a) or target items (P3b). When perceptually novel distracters occur in a series of more typical stimuli, the P3a is elicited. This has a frontal central component, with a short peak latency (e.g., Courchesne et al., 1975). This P3a is also known as the "novelty P300" and is thought to relate to prefrontal lobe activity related to the hippocampus (e.g., Knight, 1996). The P3b component has a more posterior-parietal scalp distribution, and a longer latency than the P3a. The P3a can therefore be thought to relate to stimulus evaluation, and the engagement of focal attention, which might facilitate context maintenance (P3b). The P3b is regarded as an electrophysiological manifestation of memory processing, which is evoked by stimulus evaluation in a given task. The P3b may occur when the attentional resource activations promote memory operations in temporal-parietal areas (Squire and Kandel, 1999). This evaluation typically requires some form of action such as on overt or covert response (Donchin, Kramer & Wickens, 1986). The current research in this thesis consequently refers only to the classical P3b component, which is measured in response to increased WM load.

Context updating, and resource allocation theories of the P300b.

In ERP research, it is often necessary to specify a singular overarching explanation for the neuroelectric component. However, because the P300 is observed in many different task contexts, it is a psychological event that is associated with many different aspects of processing. One theory of the P300 is that it indexes inhibition. The generation of a neuroelectric event which is linked to attention and memory related operations, might cause the brain to inhibit extraneous brain activation. This might aid the transmission of stimulus information from frontal to temporal parietal locations (from P3a to P3b).

In the context updating account of the P300, the component is said to index brain activity that underlies the revision of the mental representation. This revision is induced by incoming stimuli (Donchin, 1981), after which, an attention driven process compares the new representation with the previous representation in WM. If a stimulus change is not detected then the current mental schema is maintained, and only sensory evoked potentials are modulated (N100, P200, and N200). However, if change to the representation currently in WM is detected, attentional processes are engaged in order to update the stimulus representation. This updating gives rise to the P300. The context updating hypothesis has received a wealth of support, and has resisted refutation since its proposal in 1981. The context updating account may reflect relatively strong initial target stimulus processing, more related to P3a, which diminishes as the repeated target stimuli occur to produce the P3b (Kok, 2001).

An alternative theory is the resource allocation theory. As well as being sensitive to stimulus probability (as the context updating account would suggest), the P300 is also sensitive to attentional resources engaged during task performance. In a dual task paradigm (such as the N-back task), a primary task with varying cognitive demands is performed, while the subject is also engaged in a secondary task. In the N-back task, the primary task is the matching task, while the secondary task is maintaining varying amounts of information (manipulated via N). As primary task difficulty increases, P300 amplitude degreases (e.g., Isreal et al., 1980). This account suggests that the processing system is affected by arousal level, which governs the amount of attention available for task performance (Kahneman, 1973). Therefore, tasks that require greater amounts of attentional resources, such as those taxing WM processing, will result in a smaller P300 amplitude, and a longer peak latency,

because resources are being utilised for task performance (e.g., Kok, 2001). Figure 4 presents both the Context Updating Theory, and Resource Allocation Theory.

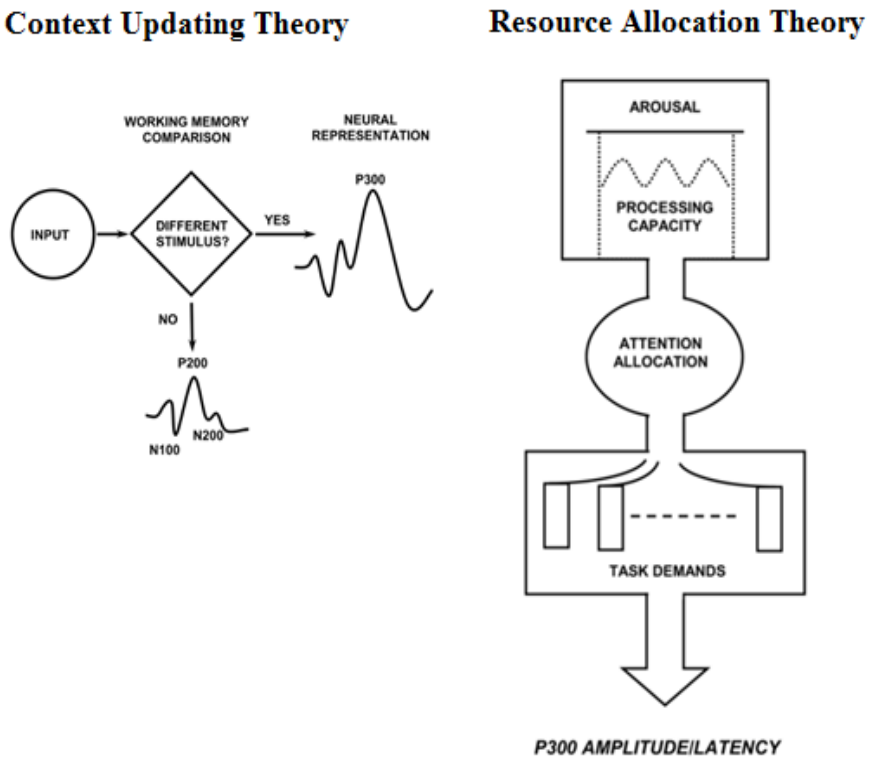


Figure 4: Context Updating, and Resource Allocation accounts of the P300.

In the Context Updating account, after initial sensory processing, an attention-driven comparison process is engaged. This facilitates the comparison of the item's representation in WM. Therefore, the P300 indexes brain activations underlying revision of the mental representation induced by incoming stimuli (Donchin, 1981). In the Resource Allocation account, the conceptual relationship between attentional resource allocation and P300 outcomes is explained by the amount of attentional resources available, whereby more resources give rise to a shorter latency, and larger amplitude.

P300 and WM load

Donchin et al., (1986) and Kramer & Spinks (1991) have suggested that P300 amplitude reflects the dependency on perceptual-central resources, in line with the Resource Allocation account. This has been supported by researchers using dual task studies (e.g., Israel, Chesney, Wickens, & Donchin, 1980) who conclude that P300 amplitude is diagnostic for perceptual-central as opposed to response related processing. Therefore, examination of the P300 amplitude is an insightful tool for understanding processing effort. Processing effort is assumed to be controlled by manipulating task demands, such as the complexity of memory related tasks. Task difficulty manipulations are thought to tap into capacity, as task difficulty determines the intensity or extent to which resource demands are taxed (Wickens, 1987). In a WM task, processing demands are created by the requirement to encode and store information (e.g., Friedman & Johnson, 2000), and also the active manipulation of information (e.g., Watter, Geffen & Geffen, 2001). Along with examining amplitude, researchers also consider P300 latency, which refers to the onset time of the component. P300 latency is often used to indicate stimulus evaluation. P300 latency increases when categorisation of the stimulus becomes difficult (e.g., Coles, Smid, Scheffers, Otten, 1995).

N-back and P300 response

Often, in single task experiments (e.g., Sternberg task), processing capacity is not fully occupied, and therefore, participants can waste resources on irrelevant stimuli. However, during dual-task studies participants must perform two qualitatively different tasks simultaneously: a primary and a secondary task. An increase in difficulty of the primary task will lead to a decrease of resources available for the secondary task. Similar to previous studies that manipulated cognitive load, experimenters introducing a secondary task found a decrease in P300 amplitude (e.g., Isreal et al., 1980; Kramer et al., 1983; 1987). Watter,

Geffen and Geffen (2001) argue that the N-back task is a dual task. The primary task involves comparing a currently presented stimulus, with one which is already active in WM. The secondary task is WM processing, where the individual has to encode, manipulate, search and select information in WM, in order to complete the primary task.

In order to support the dual nature of the N-back task, Watter, Geffen and Geffen (2001) employed a visual N-back paradigm, with four different memory loads (0, 1, 2 and 3). Their predictions were that if participants select the N-back position in WM in preparation for upcoming trials, then P300 latency should not differ across N-back trials. This would confirm the similarity of the matching subtask across all N-back conditions. However, amplitude should decrease with increasing WM conditions, in accordance with the Resource Allocation account of the P300. The authors argue that in a unitary task, such as the Sternberg task (Sternberg, 1966), latency will increase but amplitude will decrease, as WM load increases. This is because participants view the probe, and then engage in a stimulus evaluation process. As WM load increases, this task becomes more demanding, leading to an increase in P300 latency. Furthermore, amplitude of the P300 decreases, due to increased task demands.

The results provided strong evidence for the dual task nature of the N-back paradigm. P300 latency was consistent in the 1, 2 and 3 back tasks, indicating that the cognitive requirements for stimulus evaluation (primary task) were equivalent for the different N-back conditions. However, as the difficulty of the secondary task has increased due to increased WM load resources are allocated away from the primary task. Thus, Watter, Geffen and Geffen (2001) demonstrated that the P300 amplitude reduces as N increased. This amplitude modulation was consistent with previous findings, with both the N-back tasks (Gevins et al.,

1996; McEvoy, Smith & Gevins, 1998). Thus, the authors suggest that there is a reallocation of attention and processing capacity away from the primary task.

Furthermore, the authors found larger P300 amplitudes in the target trial condition. In these conditions, the stimulus matches the stimulus presented N items back. Different electrophysiological activity between target and non-target N-back trials, is in line with the aforementioned oscillatory analysis conducted by Krause et al., (2000), and Pesonen, Hämäläinen, and Krause (2007). Therefore, the electrophysiological analysis conducted in this thesis will focus on target trials only, where the P300 is maximal. Further discussion of the literature, examining the P300 and the N-back task will also be provided in each chapter of this thesis. In this thesis, both a dual task (N-back), and a unitary task (spatial delayed response task) are employed. This is because participants either have to reallocate resources, or actively manipulate items in WM (Chapter 4). Thus, the electrophysiological results will be discussed in relation to the Resource Allocation, and Context Updating account of the P300.

Statistical analysis of the P300

Within the literature, the P300 has typically been analysed by taking the most positive point in a specific time window. For analysis of the P300 during the N-back task, Watter, Geffen and Geffen (2001) define this window from 300-500ms. In order to control type-1 error rate, the 300-500ms window is used for all ERP experiments in this thesis. This time window was an a-priori choice. Once a time window is defined, the P300 can be analysed by searching for the most positive voltage within this region, or by taking a peak to peak measure. Typically, researchers have analysed the most maximal positive point within this window. However, given that ERPs are a signal to noise ratio, taking a single point within a window risks the analysis of a spurious positivity. A way to avoid this, is to take an average greatest activity

occurring within a time window. For example, one can take the greatest average 50ms activity that occurs within 300-500ms. In addition, researchers have also used a peak- peak measure. After the P300 has peaked, there is a rebound of the component, which results in a negative-going deflection. This allows researchers to analyse the P300 using a peak-peak measure. This peak-peak measure is highlighted in Figure 3. The positive value is the largest 50ms average within the green window (300-500ms), and the negative value is the smallest value in the orange window (500-1000ms). The peak-peak value is the difference between these two points.

Typically, the peak-peak analysis is a difference measure between the maximum and minimum waveform of the P300 peak. Soskins, Rosenfeld and Niendam (2000) provide a convincing argument for analyzing the P300 using a peak-peak measure. The authors compared a base-peak, with the peak-peak measure for classifying trials in an oddball task as oddball or frequent. The researchers suggest that although the negative waveform is probably not a real component, the peak-peak method using a high pass filter that is greater than 0.1 Hz, but no more than 0.3 Hz, will yield on average 20% superioriority to base-peak method detection. In their paper, Soskins, Rosenfeld and Niendam compared effects of 0.3Hz with 0.01Hz settings of the high pass amplifier filter, and baseline-to-peak, and peak-to-peak measurements of the P300. The key dependent variable was the intraindividual rate of accuracy in discrimination of oddball vs. frequent evoked P300 responses. The authors argue that the combination of the 0.3 Hz filter setting, and the peak-peak measurement of the P300 correctly diagnosed oddball vs. frequent in 100% of cases. Thus, the work in the current thesis uses a 0.3Hz high pass filter, and adopts a peak-peak measurement. In terms of p-value type-1 error rate, in group ANOVAs, the peak-peak measure yields virtually identical results

with the base-peak measures. There is a correlation of 0.93 between the base-peak and the peak – peak methods (Ellwanger, 1987).

In this thesis, all P300 analysis is conducted relative to the onset of a target trial stimulus. After pre-processing, the peak-peak analysis involves finding the most positive 50ms average voltage between 300-500ms, and the most negative 50ms average between 500- 1000ms. While 300-500ms was taken from Watter, Geffen and Geffen (2001), the negative window was defined by the trial length. In experiments 1 and 2, 1000ms marked the end of the trial. Thus, the negative window involves any point after the positive value has been calculated (500ms), to the end of the trial (1000ms). To make fair comparisons between all ERP experiments in this thesis, the same time windows were maintained.

The N-back task experiments conducted in this thesis are analysed using the peak-peak measure of the P300. However, when analyzing the SDRT, the P300 analysis involves the positive region of the P300 only. This is because the SDRT involves analyzing the P300 during stimulus presentation (encoding; S1), and at the probe (retrieval; S2). Previous research has demonstrated a distinct component, called the negative slow wave (NSW), which is present during delayed response paradigms. This component is the result of maintaining information in visual spatial WM. The NSW appears after S1 presentation, and is strongest during the retention periods. Riby and Orme (2013) examine the NSW from 500-1000 during the target presentation, a time window which overlaps with the P300 negative rebound period. Thus, at S1, it is difficult to distinguish the NSW from the P300 recovery phase. This issue is not as apparent during S2 (retrieval) or in the N-back task, where trials are often analysed to represent matching to the N-back stimuli. Therefore, analysis of the SDRT involves investigating the positive time window only.

Overall, the experiments conducted in this thesis were designed to compare WM processing in adult dyslexic and non-dyslexic participants, across different sensory modalities of WM. In Chapter 3, an overview of research examining WM processing in developmental dyslexia is provided, whereby it is argued that there is debate in the literature regarding the extent to which dyslexic individuals are solely impaired in phonological loop processing, or VSSP processing also. The application of behavioral and ERP techniques for helping to resolve this debate is detailed in Chapter 2, and each experimental chapter in this thesis.

Chapter Summary

Chapter 2 provided a theoretical background to WM, introducing the WM model of Baddeley and Hitch (1974; 1986; Baddeley, 2000), which forms the basis of empirical testing in this thesis. This model has been repeatedly tested and validated in the literature, and is cited consistently in research examining WM impairments in neuro-developmental disorders (see Alloway & Gathercole, 2006 for a full review). The latter half of the chapter included a review of task paradigms and analysis methods, which underlie the empirical work conducted in this thesis. Thus, the chapter ended with a review of research investigating WM and the P300. The rationale for applying these techniques to research examining WM and developmental dyslexia is developed in chapter 3, along with a full description of the experiments conducted in each empirical chapter. The application of ERP techniques allows for a careful and temporally accurate consideration of WM processing in developmental dyslexia. Critically, as described in chapter 2, research debates the extent to which individuals with dyslexia have a phonological loop impairment, or a general central executive impairment. Assessing the latency and amplitude of the P300, in different task contexts, allow us to examine resource allocation, as well as updating (central executive processes), in dyslexia. Unlike pure behavioural measures, the temporal precision of ERP analysis, can be

applied at encoding (where no behavioural response is generally necessary) and retrieval, in order to identify the stage at which individuals with dyslexia are impaired. Thus, these measures are critical for examining the nature of the WM impairment in developmental dyslexia.

Chapter 2: Dyslexia and WM processing

Chapter 1 consisted of a literature review of relevant research in the broad field of WM, along with an outline of the task paradigms and analysis procedures that underlie the experimental work in this thesis. Chapter 2 introduces developmental dyslexia (definitions, and current theories of developmental dyslexia), before outlining traditional and contemporary research on dyslexia and WM processing that has influenced the experimental work in this thesis. Primarily, this includes research which has highlighted a specific phonological WM deficit, before reviewing research which also suggests impaired visual WM processing in dyslexia. The aims of the chapter are therefore to highlight research which has debated the extent to which individuals with developmental dyslexia have a specific phonological loop deficit, or a broader difficulty with central executive functions. To conclude this chapter, the aims of this thesis are described, along with an outline of each Experiment. Finally, the original contributions of this work is highlighted.

An introduction to dyslexia

Definition and accounts of dyslexia.

According to the international dyslexia association (Lyon, Shaywitz & Shaywitz, 2003), dyslexia is a specific learning disability, with unexpected difficulties in accurate or fluent word recognition, decoding and spelling. Vellutino, Fletcher, Snowling and Scanlon (2004), suggest that problems manifest in acquiring basic reading sub skills, such as word identification, and letter sound phonological decoding. These deficits occur despite average intelligence, and are independent of socioeconomic status, or other extraneous factors (e.g., sensory acuity deficits). Developmental dyslexia is diagnosed with an individual's reading achievement, measured by individually administered standardized tests of reading accuracy or comprehension, which is substantially below that expected of their chronological age

(DSM-IV). It is widely accepted that dyslexia is a genetically inherited and neurologically determined disorder, and is the most prevalent neurodevelopment disorder amongst children (Cao, Bitan, Chou, Burman, Booth, 2006), ranging in prevalence from 5-17.5% of the population (Shaywitz et al., 1998).

Multiple explanations of developmental dyslexia have been proposed, however, there is controversy regarding the cognitive impairments that underlie dyslexia (Menghini, Finzi, Carlesimo, & Vicari, 2011). It has been proposed that the disorder is caused by a phonological impairment; the "phonological core deficit hypothesis," (Snowling, 1981; White et al., 2006). This hypothesis suggests that poor readers have impaired knowledge of the speech sounds and their relationship to letters, i.e., poor grapheme-phoneme knowledge. These deficits are associated with difficulty decoding unfamiliar or nonsense words, and a reduced ability to manipulate phonological representations (Muter, Hulme, Snowling & Taylor, 1998). It has also been demonstrated that individuals with dyslexia have a reduced understanding of the phonological structure of words (Gathercole, Willis, Baddeley, & Emslie, 1994). It has been suggested that auditory perceptual difficulties impair the development of high quality phonological representations. Research has demonstrated impaired temporal processing of auditory stimuli (e.g., Tallal, 1980; Tallal, Allard, Miller, & Curtiss, 1997). Specifically Goswami (2011), in the Temporal Sampling Framework for Developmental Dyslexia, proposes a specific deficit in processing low amplitude modulations of the speech signal, whereby individuals with dyslexia show reduced phase locking in the theta range (Goswami, 2011) to these stimuli. In association with this (Richardson, Thompson, Scott, & Goswami, 2004) has demonstrated that individuals with dyslexia have a higher threshold for discriminating between tones of similar rise- times; that is, tones that take a similar amount of time to reach their amplitude peak.

Alternatively, dyslexia has been attributed to cerebellar dysfunction (e.g., Vicari, Finzi, Menghini, Marotta, Baldi, Petrosini, 2005; Nicolson & Fawcett, 1999), which implies that dyslexic individuals have difficulty with skills that require automatic processing (e.g., Nicolson, Fawcett, & Dean, 2001). This idea stems from the automatization deficit hypothesis (Nicolson & Fawcett, 1999). Under this hypothesis, reading depends upon the automatization of sub-skills. Reading becomes a fluent process, when maximum reading speed, with minimum cognitive load is achieved. Vicari, Marotta, Menghini, Molinari, and Petrosini, (2003) have therefore suggested that deficits in automatizing skills, such as reduced implicit learning of visual spatial sequences is observed in developmental dyslexia. Dyslexia has also been described as a magnocellular pathway disorder. The Magnocellular theory (e.g., Stein & Walsh, 1997) suggests impairment in the magnocellular pathway, with intact functioning in the parvocellular pathway (Stein & Talcott, 1999). The magnocellular pathway is responsible for transmitting coarse grain information about location, movement, and depth, whereas parvovells are responsible for transmitting fine grained detailed visual information about colour and shape. Dyslexia has also been described as a multifaceted disorder (e.g., Kinsbourne, Rufo, Gamzu, Palmer & Berliner, 1991), with more than one possible core contribution. This has been exemplified in the double deficit hypothesis (Wolf, Bowers, & Biddle, 2000), which implicates both phonological deficits, and naming speed problems. Under this hypothesis, these two factors can contribute independently or simultaneously.

STM and WM as causal factors of dyslexia

Over thirty years of research has demonstrated that both children and adults with dyslexia suffer poor phonological WM (Wagner & Torgesen, 1987; Ramus & Szenjovits, 2008). Alongside poor phonological awareness, and low lexical retrieval, individuals with dyslexia also have poor verbal STM. Literature investigating STM performance in dyslexia, has

consistently demonstrated reduced digit span, or poor non-word repetition (e.g., Avons & Hanna, 1995; Brady, Shankweiler, & Mann, 1983; Kramer, Knee, & Delis, 2000; Pickering, 2006; Snowling, Goulandris, & Defty, 1996; Wagner & Muse, 2006). This robust finding has led some authors to propose that impaired STM may underlie the reading deficit found in dyslexic individuals.

One contemporary example of this comes from Beneventi, Tonnessen, Ersland and Hugdahl, (2010), who have suggested a causal link between WM and dyslexia. Reading development is a complex process which is likely to involve a number of complex cognitive processes; amongst them is WM. Beneventi, and colleagues claim that the development of phonological awareness, will depend on WM for consciously detecting and manipulating speech sounds. The phonological loop is also needed to allow individual phonemes to be blended, whereas the central executive is needed to simultaneously activate grapheme-phoneme conversion rules in LTM, and to facilitate switching between phonological and orthographic processing strategies (Palmer, 2000). Finally, reading comprehension depends on WM, where words in a sentence need to be maintained for a sufficient time, for meaning to be extracted, which might require multiple parsings of the sentence (e.g., Gathercole & Baddeley, 1998). However, research examining WM processing as a causal factor, has been mixed.

The link between poor STM and reading is, though, deep routed in the literature. Early models of reading performance argue that maintaining phonological information in STM is important for reading (Jorm, 1983). Torgesen and Houck (1980) suggest that tasks measuring STM, such as digit or word span, were important for differentiating readers with and without reading difficulties. However, other research has demonstrated that digit and word span measures are not always successful in differentiating reading ability (Felton &

Brown, 1991). Furthermore, several experiments suggested that processes involved in STM, such as rehearsal and chunking, do not distinguish between students with and without reading problems (e.g., Cohen, 1981). However, Swanson, Saez, and Gerber (2004) suggests that performance on WM tasks may appear to be more promising in differentiating between good and bad readers, particularly for good versus impaired reading comprehension.

However, despite early enthusiasm for memory deficits as a cause of dyslexia, memory problems have taken a back seat to other types of research examining the phonological processing deficit in dyslexia (Wagner & Muse, 2006). Wagner and Muse (2006) suggest another reason for this: phonological awareness ties in more closely to reading than do measures of phonological memory. Evidence for this can be taken from Wagner et al., (1999), who used Structural Equation Modeling (SEM) in order to model causal relations for the time periods from kindergarten to fourth grade. The structure coefficients described here are regression coefficients, with higher numbers representing a stronger causal relationship between a given variable, and reading. For phonological awareness, the structure coefficient is .37 between the ages of kindergarten and 2nd grade, .29 between 1st-3rd grade, and .27 from 2nd – 4th grade. The structure coefficients for phonological memory are .12, -.03 and .07 respectively. This suggested that at all three time periods, individual differences in phonological awareness had a causal influence on subsequent individual differences in word reading. However, no independent causal influence was observed for phonological memory in the same time period.

However, Wagner and Muse (2006) suggest that these structure coefficients might not give a complete picture of the relationship between phonological WM and reading ability. They present an example using multiple linear regressions. If there are two highly correlated predictors and a dependent variable, the independent variable that has a stronger relationship

with the dependent variable will obtain a larger regression coefficient. The other independent variable will have a coefficient near zero, even if it is only negligibly less related to the dependent variable. A strong correlation between phonological awareness and phonological memory has been extensively reported in the literature, with correlations of .88 (e.g., Wagner et al., 1999). Thus Wagner and Muse (2006) suggest that the demise of interest in phonological memory as an origin of developmental dyslexia was perhaps premature. In recent years, WM as a *cause* of dyslexia has received an increasing amount of attention (e.g., Ahissar, 2007; Szmalec, Loncke, Page, & Duyck, 2011; Martinez Perez, Majerus, Poncelet, 2012; Gathercole, Alloway, Willis, & Adams, 2005).

Theories regarding the core deficit in developmental dyslexia are varied. To some extent, this variation reflects the discipline background of the researcher (McLean, Stuart, Colthart, & Castles, 2011), but it also reflects heterogeneity in the dyslexic population. Dyslexia is not a unitary syndrome, and it is unlikely to have a unitary cause (Boder, 1973; Castles & Colthart, 1993). While some researchers (e.g., Ahissar, 2007) examine how WM might *cause* impairments in reading, a somewhat separate line of research has aimed to assess the nature of the WM impairment in developmental dyslexia (e.g., is the visual spatial sketchpad impaired as well as the phonological loop), and has said very little about the contribution of WM functioning to the disorder. For example, Smith-Spark and Fisk (2007) and Menghini, Finzi, Carlesimo, and Vicari, (2011) have examined WM processing in dyslexia across different domains of memory. A thorough examination of the symptoms of dyslexia is an important research question primarily because there is need to understand and address all of the difficulties an individual with dyslexia might face (e.g., The UK Disability Discrimination act, 1995, and the Equality Act, 2010). Given that WM processing is implicated in a wide range of cognitive tasks, it is essential to understand and conceptualize

how WM might be impaired in dyslexia. Thus, the experimental work conducted presently, aims to examine the extent to which WM processing is impaired across different modalities of memory (e.g., verbal; visual-spatial), and aims to examine the behavioural and electrophysiological correlates associated with this.

Characterising WM processing in dyslexia

A specific phonological loop impairment

Vellutino (1979) suggested that individuals with dyslexia have a systematic difficulty on tasks which incorporate a verbal component, however, they perform at the same level as non-dyslexics on tasks without a verbal component. This led to the conclusion that the impairments associated with dyslexia are restricted to the verbal domain (Tijms, 2004). A large body of research suggests that dyslexia stems from an underlying deficit in the phonological processing system (e.g., Beitchman & Young, 1997; Lyon, 1995). Verbal WM deficits in developmental dyslexia have been demonstrated extensively in the phonological domain of memory. In Baddeley and Hitch's (1986) model of WM, the phonological loop is specialized for retaining verbal information over short time periods. The loop has a store, and a rehearsal process for the maintenance of representations. A substantial number of experiments have suggested that dyslexic individuals suffer deficits in phonological processing, which might impair their ability to retrieve information from STM. The phonological loop component of WM has been compared to verbal STM.

In the 1960s, researchers linked poor performance on tasks that examine phonological STM to individuals with dyslexia (Jorm, 1983). In a meta-analysis, Stanley, Kaplan and Poole (1975) using the Auditory Sequential Memory subtests of the Illinois Test of Psycholinguistic Abilities (ITPA), concluded that phonological STM was consistently impaired in dyslexia. Similarly, Spring (1976) employed a digit span task with the direct aim

of comparing performance in people with reading deficits, to controls. His findings displayed a significant deficit in the memory span of those with impaired reading, compared to controls, suggesting a phonological loop impairment. When reviewing studies comparing impaired and control readers, Jorm (1983) noted that in 6 out of 11 instances, impaired readers also showed impairments on the digit span subtest of the Wescler Intelligence Scales for Children (WISC; Rugel, 1974). Moreover, these digit span deficits persist regardless of presentation, (e.g., Rizzo, 1939) and response mode (Koppitz, 1964). To examine phonological WM, over 1200 individuals aged between 6 to 49 were presented with tasks assessing word recognition, pseudo-word decoding, reading comprehension, WM, and STM (Stanovich & Siegel, 1994). The results demonstrated that although WM skills increased from ages 6-19 in all individuals, individuals with dyslexia performed significantly lower than non-dyslexic individuals, on verbal WM and STM tasks, across all age groups. Thus, the deficits in verbal memory tasks persisted into adulthood.

Numerous reasons why individuals with dyslexia have impaired phonological WM have been proposed. Done and Miles (1978) demonstrated that rehearsal suppression gives rise to similar STM performance between individuals with and without dyslexia. Early theories also suggested a lack of motivation to use rehearsal strategies in dyslexia. Torgesen and Houck (1980) compared a group of impaired readers who showed a digit span deficit with a matched group of impaired readers who did not show a deficit. Providing incentives to do well did not affect the digit span of children with a digit span deficit. It has also been suggested that individuals with dyslexia have faulty rehearsal mechanisms, which prevents phonological information from being maintained in the phonological loop. However, with very fast rates of presentation, normal children continued to perform better than those with a digit span deficit (Torgesen & Houck, 1980). Finally, when the experimenter introduced

chunking by presenting the digits in grouped form, the individuals with reading impairments still performed much worse than control individuals, thus, their deficit could not be attributed to a deficit in chunking strategies.

A second proposed cause of these deficits is the inefficiency in the ability of the phonological loop component of WM to store information. This has been attributed to subtle deficits in articulatory processes. According to Baddeley (1986), storage of phonological information is accomplished by replenishing a quickly decaying phonological store by covert articulation or rehearsal. However, this explanation is somewhat limited (Wagner & Muse, 2006) as covert articulation is difficult to quantify. To maintain activation of phonological codes during a STM task, this articulation must be present. However, Cohen (1982) observed that when stimuli are presented so rapidly that articulation isn't possible, memory differences still exist between groups. This claim was later supported by Torgesen, (1996), who demonstrated that STM differences persisted between groups when verbal material was presented visually or auditorially. Thus, Torgesen concluded an inability or unwillingness to articulate code in STM was not a sufficient explanation.

A third account for the origin of impaired phonological STM in dyslexic readers is that they make use of phonological codes that are somehow degraded or less well developed. This may occur as a result of subtle problems in speech perception (e.g., Brady, 1991). For example, Kramer et al., (2000) suggested that inaccurate encoding of the phonological characteristics of the stimulus are the cause of verbal memory impairments in dyslexia, and more general phonological deficits. Indeed, this is an explanation that still persists in the literature (e.g., Tijms, 2004), who suggests that verbal material can be held in a phonetic buffer for only a short period of time. The memory trace will decay, unless it is maintained via a rehearsal mechanism, or regeneration mechanism for reconstructing decaying memory

traces. These mechanisms act on the basis of the phonological representation. If the decaying memory trace is to be held active in a system of inaccurate or unstable phonological representations, the chances of altering, or losing the trace is enhanced. This hypothesis has been supported by early research.

For example, it was proposed that impaired readers, have less efficient coding processing in STM. The effects of phonological confusability have been investigated on recall by using strings of rhyming and non-rhyming items, in good and poor readers. Baddeley (1966) and Conrad (1973) have both suggested that if good readers are better able to form phonetic representations, then they should be able to recall more than poor readers on phonetically distinct, non-rhyming items. However, when the items can be confused due to rhyming, good readers with superior phonetic skills should be impaired, resulting in more similar performance for the reading groups. This prediction has been supported by several experiments (e.g., Mann, Liberman, & Shankweiler, 1980; Brady, Shankweiler, & Mann, 1983). The phonetic similarity effect persists regardless of stimulus modality e.g. visual or auditory presentation (Shankweiler, Liberman, Mark, Fowler, & Fischer, 1979). This suggests that poor readers have a general problem with phonetic code. Furthermore, adult's memory span can be predicted on the basis of the number of words the individual can read in approximately two seconds (Baddeley et al., 1975). Hoosain, (1982) suggests there is a negative correlation between naming speed and digit span. Thus, the phonetic processes involved in encoding and articulating responses are statistically related to memory span performance. Specifically, perception and memory processing in STM may share limited resources (Brady, 1986).

Later, Torgesen (1996) also investigated the link between phonological skills, and phonological STM. Torgesen reported results from three new samples of children with

learning disabilities, whose performance on STM tasks were impaired, or not. There was also a control group without learning disabilities. The three groups were given a span task for three different types of stimuli, which differed in familiarity: digits, words and syllables consisting of pronounceable non-words. Digits were considered to be high in familiarity, words were less familiar, and finally non-words were the least familiar stimuli. The results showed that differences between individuals with STM impairments and those without, were largest for digits, the most familiar kind of stimuli. Smaller differences were found for words, and finally virtually no differences were found for non-words. This suggested that the largest advantage for the most frequent kinds of stimuli resulted from the fact that the children with STM impairments had not developed as substantial unitary phonological codes for digit names.

The neural underpinnings of verbal deficits have also been examined. Emerging evidence has suggested that impaired auditory sampling might underlie the verbal deficit in dyslexia, which could also potentially affect verbal item representation in STM. Lehongre, Ramus, Billermet, Schwartz and Giraud, (2011) examined low-Gamma sampling in the auditory cortex. Magnetoencephalography (MEG) was used to examine the auditory steady state responses (ASSR) which were induced using amplitude modulated white noise. Dyslexic subjects showed reduced left hemisphere bias for phonemic processing, and enhanced cortical entrainment for modulation rates above 40Hz. This reflected phonemic oversampling in the left auditory cortex. The researchers found a negative correlation between ASSR responses and digit span measures across frequencies 45-65Hz. This could indirectly influence phonological/ verbal memory, by resulting in a greater amount of subphonemic perceptual chunks per time unit which are integrated into theta-based processes that underlie the auditory buffer capacity, and syllabic integration. Speculatively, it was

argued that excessively detailed spectrotemporal information was delivered to higher-level processing stages, at the cost of delayed abstraction (the time point when the system encodes the stimulus with a symbolic type of representation).

While the previous theories suggest impairment in the phonetic integrity of the item, research has also suggested that impaired phonological representations are not the only contributing factor to verbal STM and WM impairments in dyslexia. Trecy, Steve and Martine (2013) investigated whether verbal STM deficits in dyslexia were associated with item, or order impairments. STM for item information has been shown to depend on the quality of the phonological representation, whereas STM for order information is considered to reflect core STM processes, which are independent from language processing. The authors demonstrated both item (failure to recall a specific item), and order (recall in the wrong order) impairments in dyslexia, demonstrating that dyslexia presented as a deficit in core verbal STM processes, which cannot be accounted for by language processing difficulties alone.

Ramus and Szenkovits (2008) provide an alternative insight into why verbal WM is impaired in dyslexia. Instead of the phonological representations themselves being degraded, such representations are in fact themselves intact, and all phonetic features are correctly encoded. Ramus and Szenkovits (2008) claim instead that the STM processes themselves are limited, and propose a conscious access deficit. The phonological access hypothesis describes the process by which lexical and sub-lexical phonological representations are accessed for external computations. Verbal STM requires access to phonological representations for the purpose of copying them into buffers, then access to phonological buffers for retrieval. The phonological loop involves conscious access to input representations, which are then copied into output representations. Conscious access to

output representations is then required to recycle the phonological representation to an input representation. Conscious access to phonological representations may place special demands on executive mechanisms, which control access to phonological representations. Conscious access to representation requires central executive processing. Ramus and Szenkovits argue that dysfunction of the central executive need not be domain general, and might only persist in combination with phonological information.

Beneventi, Tonnessen, Ersland, and Hughdahl, (2010) investigated the extent to which domain general executive impairments contributed towards phonological, or verbal WM processing, in an fMRI study. They implemented a modified WM N-back task (0-back and 2-back), where participants remembered the first or last phoneme from the names of common objects, which were shown as pictures. Dyslexic readers had impaired accuracy in both the 0-back and 2-back tasks. Impairment in the 0-back condition was said to reflect phonological processing deficits. Group analysis of the fMRI data showed that dyslexic deficits in the 0-back task were associated with differences in left temporo-parietal areas, which are involved in phonological processing. However, there was also reduced activity in the right inferior frontal gyrus (IFG), and the medial frontal gyrus (MFG), and bilateral activity in the superior parietal lobule (SPL), between dyslexia and control individuals. These fronto-pariental areas are associated with executive processing (e.g., Owen, McMillan, Laird, & Bullmore, 2005). These results might imply that although there are no overt memory demands, the 0-back task requires conscious access, attention to, and manipulation of phonological representations, and thus still involves executive processes to some degree. However, in the 0-back task there were no significant activation differences found in the posterior left areas associated with phonological processing.

For the 2-back task, the results revealed neuronal correlates that could be attributed to executive, rather than phonological processes. There was significantly increased brain activation in the right IFG, and the left SPL for the control group. The results suggested that the processing requirements on the phoneme identification task in the 0-back condition may have exceeded the capacities of the dyslexic readers, leaving no additional resources for the more demanding 2 back task. This might result in a reduction of central executive capacity. Alternatively, less effective phonological processing might demand more central executive processing, which causes a relative reduction in WM capacity. Both explanations offered by the authors suggest a specific WM deficit in dyslexia, which is somewhat separate from a phonological core deficit. These results have also been replicated by the same laboratory, in a letters version of the n-back task. The results showed reduced activation amongst dyslexic participants bilaterally in the posterior MFG, SPL, and left cerebellum. This indicates that these areas were less sensitive to increasing WM demands in the dyslexic groups. The MFG and SPL have been associated with general WM processes such as continuous memory updating, and the temporal order of memory (Wagner & Smith, 2003). The current results suggest that the WM impairments in developmental dyslexia, cannot be explained in whole by faulty phonological processes, because the dyslexic group did not show an increase in activation in neural areas associated executive processes, when task demands required this (2-back task). However, the authors claim their results should be treated with caution (particularly in the 2-back condition), where the dyslexic group performed at chance. Thus, the extent to which they were engaging in WM processes is questionable.

Evidence for central executive impairments

Swanson, Zheng, and Jerman (2009) claim that in the general literature, there is no consensus as to whether individual differences in reading and memory reflect a domain specific or a

common central executive system impairment. Nevertheless, Swanson, Zheng and Jerman (2009) claimed it is unclear whether WM and STM deficits are ubiquitous across verbal and visual-spatial domains, as they are in the phonological domain. Moreover, some researchers have failed to uncover any visual-spatial WM deficits in dyslexia (e.g., Jeffries & Everatt, 2003, 2004; Kibby, Marks, Jordan & Long, 2004). Therefore, the predominant conclusion became that VSSP deficits were absent in dyslexia, and studies which found a deficit only did so because their VSSP measures were not 'process pure', and required processing of verbal information (e.g., Gould & Glencross, 1990).

In fact, some experiments have demonstrated normal visual spatial WM in children with reading difficulties (Swanson, Ashbaker, & Lee, 1996, Experiment 1), while demonstrating effects in later experiments (Swanson et al., 1996, Experiment 2), when they partialed out verbal WM performance. Jeffries and Everatt (2003) tested adults with dyslexia or dyspraxia and compared their performance on measures assessing the function of the phonological loop and VSSP with control adults. Individuals with dyslexia showed deficits in recall tasks involving the phonological loop, whereas dyspraxia individuals showed deficits in tasks involving the VSSP. This suggests that each experimental subgroup showed normal retention in the subsystem of WM that was unrelated to their disability (i.e., normal VSSP in dyslexia).

However, some research has demonstrated a visual WM deficit in dyslexia. De Jong (1998) compared performance of a group of reading disabled children against a control group on a measure of verbal WM, verbal STM, and processing speed. The verbal WM deficits of the group with reading difficulties could not be explained by storage problems, and central executive processes were said to underlie WM deficiencies in dyslexia. Wang and Gathercole (2013) suggest that given the domain general nature of the central executive

(Baddeley & Hitch, 1974; Alloway, Gathercole, & Pickering., 2006), a key prediction of the impaired central executive hypothesis is that children with reading difficulties should extend to complex span tasks involving non-verbal as well as verbal material. The central executive coordinates various slave systems, integrating their storage capacities and making available attentional resources for online processing. Failure of the cchentral executive to perform these tasks, would account for impairments on both verbal and VSSP tasks.

However, findings regarding visual-spatial WM processing in dyslexia are mixed. Some early research demonstrated a central executive dysfunction in dyslexia. Swanson (1994) investigated how central executive function related to literacy in children and adults. Participants in the study were given measures of verbal and visuospatial STM, and measures of central executive function. Swanson demonstrated that the tasks that involved both storage and processing (central executive tasks), made a greater contribution to reading ability. This suggests memory deficits in poor readers go beyond STM (storage only). Later, Swanson and Ashbaker (2000) explored the role that articulation rate played in the relationship between executive processing and reading. It was found that STM and central executive performance of poor readers was worse than that of age matched controls, even after the contribution of articulation rate had been removed from the analysis. These findings suggested that poor readers have a deficit in central executive components of WM, independent of any deficits that could be attributed to the phonological loop. Thus, the results suggest a domain general deficit contributed to impaired WM in reading disabled children.

Furthermore, Reiter, Tucha, and Lange (2005), as part of a wider investigation examining executive functioning in dyslexia, administered verbal STM and verbal and visual WM tasks to participants with and without dyslexia. Verbal WM was measured by backwards digit span, and verbal STM was assessed via forward digits. To assess visual WM,

participants were required to count the number of corners of 15 different rectangular figures immediately after each of them were presented on a computer screen for 200ms. The number of corners varied between 6 and 12, and an individual's visual WM capacity was calculated. Their results suggested that children with dyslexia showed impairment on both verbal and visual WM. Reiter, Tucha, and Lange (2005) interpreted the current results to suggest individuals with dyslexia had figural (object) visual WM impairments. The researchers therefore proposed that mistakes made by dyslexic children on copying tasks, might reflect a reduced ability to store visual information for a short period of time. This suggestion is backed up by Klicpera (1983), who demonstrated that dyslexic children drew fewer details than non-dyslexic children in the Rey Osterrieth complex figure text. Visual WM plays an important role in this complex figure copying task.

Despite a growing body of evidence for impaired visual-spatial WM spans in dyslexic children, Swanson, Zheng, and Jerman (2009) argue that in the general literature, there is still no consensus as to whether individual differences in memory reflect a domain specific phonological impairment, or a common central executive impairment. One reason as to why conflicting results might arise is that different task paradigms have been used to assess visual-spatial WM processing, across experiments. More complex span tasks might be more successful for highlighting deficits in dyslexia. In support of this view, Smith-Spark and Fisk (2007) examined the STM of dyslexic and control children in visual processing and a STM task using simple and complex patterns, across verbal and visual-spatial information. In the verbal simple span tasks, the verbal digit span was recorded. To assess complex verbal span, participants were asked to read either an arithmetic problem or a sentence. From these digits or sentences, they were asked to remember the last digit, or word of the sentence. Depending on the stimulus type (arithmetic or sentence) participants were then required to solve the

arithmetic problem or, answer a comprehension question, while retaining the last digit or word in memory.

To assess visual-spatial simple span, a Corsi block span was administered, while to assess complex span, participants were presented with an array of cells, five of which were highlighted with Cs and one with Os (as in Fisk, 2004). The participants were requested to indicate whether there were more cells highlighted above or below a centrally placed dividing line on the screen. In addition, they were required to remember the position of the cell highlighted with Os. The findings revealed that WM deficits in adult dyslexics were not confined to the phonological domain, but instead extended into the visual-spatial domain. Group differences were found on both simple and complex span tasks, but also on the updating measures. Effect size analysis indicated that dyslexic WM problems were greatest on the complex span measures. Moreover, the results suggested that WM problems are not solely limited to the maintenance of information, supporting the presence of a central executive impairment in dyslexia.

Menghini, Finzi, Carlesimo and Vicari (2011) suggest that Smith-Spark and Fisk's (2007) results are surprising, given past research which suggests phonological WM impairments only. Thus, Menghini and colleagues aimed to clarify the nature of the WM deficits in dyslexia, across verbal, visual-object, and visual-spatial memory spans. The materials for the verbal span task consisted of a list of eight, two syllable low frequency words. In the first block the experimenter read aloud two words at a rate of one item per second and the participants were required to repeat the two words in the same other. Then four additional strings of two words were presented. If an individual is successful in at least three of the five sequences, a sequence one word longer was presented. If the individual fails, the task was discontinued. In the visual-spatial task, the material consisted of a

nonverbal geometric shape, depicted in high contrast colours that appeared for two seconds in one of seven positions. After a one second ISI, the same geometric shape appeared in a second position and then disappeared after two seconds. After 500ms, two empty cells were presented in the same spatial positions as before and the individual was required to indicate the order in which the stimuli appeared. If the child was successful in at least three of five, two position sequences, a sequence one block longer was presented.

Finally, a similar procedure was adopted to assess the visual-object span. The experimental material consisted of seven complex geometric figures, depicted in high contrast colours. At the onset of the task, two figures were presented, one at a time, for two seconds at the center of the computer screen with a one second ISI. 500ms after the second figure disappeared, the two stimulus figures were presented aligned in the center in a random position. The participant was required to indicate the order in which they appeared. If the individual was successful in at least three of the five trials, a sequence one figure longer is presented. Further to these measures, attention and visual perceptual tasks were administered. Results demonstrated lower scores for the dyslexic children compared with normal readers, for verbal information as well as short term retention of sequences of abstract figures and spatial positions. The results therefore support the proposal that dyslexic children do not have an isolated verbal WM deficit, but are impaired on both the phonological loop and VSSP sketchpad components of WM, implicating the central executive. Thus, VSSP span deficits only occurred in dyslexic individuals when the task called for high memory updating.

Wang and Gathercole, (2013) investigated the cause of the reported problems in WM in children with reading difficulties, hypothesizing a domain general deficit. Verbal and visual spatial simple and complex span tasks were administered to children with single word

reading difficulties, and a control group of typically developing children. Children with reading difficulties showed greater impairments in the ability to combine two different cognitively demanding tasks. Difficulties with dual task coordination could be attributed to the limited resources of the central executive. Furthermore, children in this study were also impaired for tasks requiring serial recall (STM) of visual spatial and verbal material. Wang and Gathercole suggest that working memory difficulties in children with dyslexia do not occur due to problems processing verbal material. Instead their results demonstrated central executive dysfunction, in that the children with reading difficulties performed more poorly than typically developing children on memory tasks that involved both verbal and visual spatial material. Deficits on complex span tasks were associated with central executive processing, and this effect was significant even when performance on simple span tests of STM were taken into account. Wang and Gathercole (2013) claim that while these results could be explained in terms of spatial attention differences, this was unlikely given that Menghini and colleagues (2011) had demonstrated that visual spatial WM differences persist when the effect of spatial attention is partialed out of the analysis.

Therefore, researchers, including Smith-Spark and Fisk (2007), Menghini, Finzi, Carlesimo and Vicari (2011), and Wang and Gathercole (2013), have used evidence of impairments in non-phonological tasks, as evidence in favour of a more general central executive deficit, or a domain general WM deficit in dyslexia. Yet the findings remain controversial. In a meta-analysis, Swanson, Zheng and Jerman (2009) found moderate effect sizes for visual-spatial deficits in dyslexia, supporting research which suggests the WM deficits also span outside phonological domains of memory. However, Hierarchical Linear Modeling (HLM) showed no effect of visual-spatial information, suggesting that overall memory problems were primarily moderated by deficiencies related to the accessing of

speech based information, and/or the monitoring of attention processes for verbal material. However, this meta-analysis included literature from 1963-2006, and thus the more recent research reviewed here, by Smith-spark and Fisk (2007), Menghini et al (2011), and Wang and Gathercole (2013), which have demonstrated impaired visual WM deficits, are not included in the review. Thus, the extent to which WM processes are limited to impairments in the phonological loop, or is caused by an additional deficit in central executive functioning is still a major question in the research (Beneventi, Tonnessen, Ersland, and Hughdahl, 2010).

Thesis aims and rationale

In summary, research investigating the extent to which visual WM is impaired in dyslexia has been mixed, with more recent research suggesting that visual WM difficulties might be present in dyslexia, particularly for complex span tasks (e.g., Wang & Gathercole, 2013). In this context, the research presented in this thesis tackles two questions. It first considers the extent to which adults with developmental dyslexia have an impaired central executive. In order to achieve this aim, the N-back task is used across modalities of memory (Chapter 3, and 5). N-back is an excellent task for assessing executive functioning, as it requires on-line monitoring, continuous updating, and temporal order of remembered information (Beneventi, Tonnessen, Ersland, and Hughdahl, 2010). It therefore places a greater demand upon executive functioning than span tasks. Furthermore, in order to directly compare passive maintenance and the active manipulation of information in WM, the Spatial Delayed Response Task (Glahn, 2002) is employed (Chapter 4). Thus, in this thesis, an extensive assessment of phonological WM and central executive processing in dyslexia is conducted. Furthermore, in the final empirical chapter of this thesis, a second question is addressed. This regards the extent to which difficulties with auditory processing might contribute towards the phonological WM impairment in dyslexia (Chapter 6). Here, a theoretical framework from

Usha Goswami's Temporal Sampling Theory of Developmental Dyslexia is adopted, in order to assess how lower level difficulties in processing speech, might contribute towards WM difficulties in dyslexia.

Importantly, these questions are addressed in dyslexic adults. Swanson, Zheng, and Jerman (2009) claim that the majority of research has been conducted on children at primary school grades, thus one possibility is that central executive dysfunctions are restricted to this group. Indeed, the developmental lag explanation has been proposed in the literature, and developmental increases in complex memory performance reflect improvements in processing speed and efficiency that enable additional resources to support storage (Case, Kurland, & Goldberg, 1982). In a meta-analysis, Swanson, Zheng, and Jerman (2009) conclude that experiments examining visual spatial, and central executive processing in dyslexia have predominantly only included 5-18 year olds, reflecting the bias that research has had to focus on individuals younger than 18. Therefore, there have been very few experiments investigating central executive processing in dyslexic adults. The current work examines WM processing in adult dyslexic participants, in order to assess if central executive difficulties in dyslexia still persist into adulthood.

Furthermore, very few experiments have assessed the neural basis of the WM deficit in dyslexia. Research by Beneventi and colleagues (2010), cited earlier in this chapter used fMRI to reveal the anatomical neural correlates of central executive and phonological processing in dyslexia. However research investigating the ERP response during a WM task are not evident in the literature. Measuring ERP responses alongside behavioural measures will provide an additional insight into whether WM processing differences exist between groups, and in which memory domains. ERPs have excellent temporal resolution, and can thus highlight if any speed of processing deficits might underlie WM impairments in adults

with dyslexia, as speculated upon by Swanson, Zheng, and Jerman (2009). For example, the P300 latency has been used to assess stimulus evaluation time. A speed of processing deficit, would result in increased P3b latencies for adults with dyslexia, regardless of processing load.

ERPs can also reveal any resource allocation difficulties that may exist between groups. For tasks that require a greater amount of attentional and WM resources, the P300 amplitude is smaller, and the peak latency is longer (Kok, 2001; Polich, 1987). As demonstrated in Chapter 1, Watter, Geffen and Geffen (2001) employed an N-back task and measured the P300. Watter, Geffen and Geffen interpret a reduced P300 in later N-back conditions as being due to a reallocation of attention and resource capacity away from the matching task, in response to increased WM demands. Therefore, assessment of the P300 can be used as a neural correlate of processing capacity. Recording ERPs during the N-back task will allow us to assess contexts where individuals with dyslexia are impaired versus unimpaired, and will provide a valuable insight into the extent to which individuals with dyslexia have an impaired central executive. In Chapters 3 and 5, EEG recordings during the N-back task are conducted in the visual modality, using visual-objects (which cannot be phonological recoded), visual-letters, and auditory stimuli. In this context, the P300 analysis is interpreted according to the resource model of the P300 (See Chapter 1 for a review).

In Chapter 4, EEG is also recorded during the SDRT (Glahn et al., 2002), in order to dissociate passive maintenance and active manipulation components of WM, and explore passive WM processing, and active central executive processing further. This task allows us to examine the behavioural and electrophysiological responses associated with visual WM functioning, more closely. Furthermore, if individuals with dyslexia are impaired in visual spatial WM, the question still remains as to which stage they are impaired because the

encoding stage is difficult to investigate with traditional behavioural measures. Thus, in Chapter 4, ERPs are examined at encoding and retrieval. Overall, Chapters 3-5, which include experiments 1-7, are concerned with cross modal investigations of WM processing in dyslexia, in order to assess exactly which components of the Baddeley and Hitch (1964; Baddeley, 2000) model are impaired in adult dyslexics. Finally, in chapter 6, a further experiment (Experiment 8) is conducted in order to investigate how lower level auditory impairments might influence the phonological WM deficit in developmental dyslexia.

Experiment Summary

In this thesis, stimuli are presented in the visual, and the auditory domain. During visual presentation, verbal, visual- object, and visual spatial WM processing are assessed. In the auditory domain, phonological WM is assessed using letters, words and tones as stimuli. This thesis includes eight experiments, described below in Table 1.

Table 1 *Summary of Empirical Work*

Chapter	Experiment Number	Presentation	Aims	Paradigm	Independent measures	Dependent measures	Notes
Chapter 3: Visual Presentation	1	Visual	To demonstrate a phonological WM impairment in dyslexia, and assess the ERP responses associated with this impairment.	N-back: visual letters	N (1-4), Trial (target, non-target), and Group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency. N2 amplitude.	In Experiment 1 (and Experiment 2), there is a 2:1 ratio of non-target: target trials.
	2	Visual	To examine visual-object WM in dyslexia, and the associated ERP responses.	N-back: Pictorial visual-objects	N (1-4), Trial (target, non-target), and Group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency. N2 amplitude.	Experiment 2 used a 2: 1 ratio of non-target: target trials. This experiment used pictorial objects as stimuli, which could contain semantic information, and have the potential to be subvocalized.
	3	Visual	To assess visual-object WM processing, by	N-back: visual-objects	N (1-4), Trial (target, non-target), and	Accuracy, d', criterion.	Experiment 3 adopts a 1: 1 non-

			overcoming some of the limitations present in Experiment 2.	(Chinese ideograms)	Group (dyslexic, non-dyslexic).		target: target trial ratio.
Chapter 4: Visual Spatial Working Memory	4	Visual	To assess visual-spatial WM processing in dyslexia, and the associated ERP responses.	SDRT (Glahn, 2002a). In this task participants must state if a probe occurred in the same location as a target stimuli.	WM load (1, 3, 5 and 7), Trial (target, non-target), and Group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency. N2 amplitude.	
	5	Visual	To assess central executive WM processing, by comparing passive versus active processing of stimuli in visual spatial WM.	SDRT (Glahn, 2002a). Load is kept constant. Participants must maintain or manipulate three dots, across a horizontal line.	Task (Maintenance, maintenance plus manipulation), Trial (target, non-target), and Group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency. N2 amplitude.	

Chapter 5: Phonological WM in dyslexia	6	Auditory	To assess phonological WM processing for letters, and the associated ERP components.	N-back task with auditory letters.	N (0-5), Trial (target, non-target), and Group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency.	Here a 1: 1 ratio of non-target: target stimuli are used.
	7	Auditory	To assess phonological WM processing for words, which are manipulated by their Age of Acquisition (AoA).	N-back task with auditory words (words are manipulated by AoA).	N (0-5), AoA (early, late) trial (target, non-target), and group (dyslexic, non-dyslexic).	Accuracy, d', criterion. P300 amplitude and latency.	
Chapter 6: The Contribution of rise-time perception to phonological WM in dyslexia.	8	Auditory	To examine the extent to which the phonological WM impairment in dyslexia might be related to lower level auditory temporal sampling impairments.	N-back task with auditory tones (tones are manipulated by their rise-time (the time taken for the tone to reach its amplitude peak).	N (1-3), Rise-time (short, long) trial (target, non-target), and group (dyslexic, non-dyslexic).	Accuracy, d', criterion.	

Chapter Summary

Chapter 2 provided a theoretical background to the WM deficit in developmental dyslexia. Research in this chapter (e.g., Torgesen, 1996; Rugel, 1974; Gathercole, 1995; Adams & Gathercole, 1996; Alloway, Gathercole, Adams & Willis, 2005) has highlighted a specific phonological working memory deficit in developmental dyslexia. Furthermore, this chapter involved a review of research which has argued against a visual working memory deficit in dyslexia, followed by a discussion of emerging research (Menghini, Finzi, Carlesimo, Vicari, 2011; Wang & Gathercole, 2013; Smith-Spark, Fisk, Fawcett & Nicolson, 2003; Smith-Spark & Fisk, 2007), which suggests the deficit might extent to the visual domain, and therefore be a difficulty with the central executive. The research in this thesis applies behavioural (accuracy, and signal detection theory) and sensitive electrophysiological (ERPs) techniques, in order to characterize WM functioning in dyslexic individuals. Finally, this chapter concluded by presenting an overview of the experimental work which will be conducted in this thesis.

Chapter 3: Visual presentation

Chapter 2 consisted of a literature review of relevant research investigating WM processing in developmental dyslexia. It outlined a debate in the literature, which regards the extent to which individuals with dyslexia have impaired central executive WM processing. In order to investigate central executive impairments in dyslexic adults, three experiments are presented in this chapter, all of which use the N-back task outlined in Chapter 1. In Experiment 1, letters were presented, while in Experiments 2 and 3, visual-objects were used. In Experiments 2 and 3, behavioural responses, and ERP responses (specifically the P300b amplitude and latency) are analysed. The aims of the chapter are therefore to examine the extent to which individuals with dyslexia are impaired for visual, as well as verbal WM processing. If impairments are found in both domains, then there is evidence for a domain general, central executive impairment in dyslexia.

Rationale and hypothesis

It has been well documented that individuals with dyslexia have a significant difficulty with WM tasks, compared to controls (e.g., Siegel & Ryan, 1989; Swanson, 1994; 1999; Ackerman & Dykman, 1993; Cohen, Netley, & Clarke, 1984; Gould & Glencross 1990). This effect is robust in the phonological domain, but the evidence suggesting a visual WM impairment is mixed (see Chapter 3). However, authors such as Smith-spark and Fisk, (2007) and Wang and Gathercole (2013) rationalize that if impairments are found on non-phonological tasks, it is a convincing argument in favor of central executive problems in dyslexia. The current set of experiments aims to assess the extent to which individuals with dyslexia are impaired in verbal and visual WM. Specifically, this chapter includes experiments assessing visual-object WM; visual-spatial WM processing is addressed in Chapter 5.

The experiments in this chapter aim to determine if adults with developmental dyslexia have central executive impairments as suggested by previous work (e.g., Menghini, Finzi, Carlesimo, Vicari, 2011; Wang & Gathercole, 2013; Smith-Spark, Fisk, Fawcett & Nicolson, 2003; Smith-Spark & Fisk, 2007), by examining verbal and visual WM processing. One possibility for the inconsistent findings within the literature, regarding the extent to which individuals with dyslexia have visual WM impairments, is that different task paradigms are used across modalities, each posing a different demand upon executive function. If a task from a specific modality taxes the central executive more specifically than a task examining a different modality, then this might result in incorrect conclusions being drawn for that particular modality. Therefore the three experiments presented in this Chapter use one task paradigm, the N-back task, across WM modalities where individuals with dyslexia are traditionally known to be impaired (verbal WM), or where the effect is less robust (visual-object WM). In addition ERPs are measured in order to provide temporally sensitive information about WM processing between groups.

Here, the P300 is examined, where amplitude during the N-back task is said to reflect resource allocation (Watter, Geffen, & Geffen, 2001). Consequently, researchers have used the P300 in order to index processing capacity, during WM tasks (e.g., Evans, Selinger, & Pollak, 2011), while latency of the P300 reflects stimulus evaluation time (Johnson & Donchin, 1980). P300 latency can also be used to examine strategic differences between groups. If individuals are adequately maintaining information, in order to compare it to the upcoming stimuli, then one would not expect to see latency differences as N increased. However, if the WM representation of the N-back stimuli is weak, then one would expect to see a latency difference as N increased. A more detailed explanation of this effect can be found in Chapter 1, where Watter, Geffen and Geffen's (2001) experiment is discussed.

Furthermore, the experiments in this chapter aim to apply Signal Detection Theory (SDT) to the N-back paradigm. As described in Chapter 2, SDT controls for response bias during a task, and gives a pure measure of signal strength, when individuals are required to make a binary decision. To date, SDT has not been applied to assess performance on WM tasks in individuals with dyslexia. Its application is particularly important because it allows researchers to quantify and control for response bias. Evans, Selingar and Pollak (2011) considered SDT during the N back task, whilst assessing visual WM processing in individuals with Specific Language Impairment (SLI). The results demonstrated that all individuals showed reduced d' values, and increased criterion values as WM load increased. These effects were accentuated in individuals with SLI, whose d' values were lower, and criterion values were higher than controls. Lower d' values suggest a difficulty discriminating signal from noise (a target from a non-target stimulus), and thus reflect an intrinsic difficulty with WM processing at higher N-back levels. An increased criterion suggests individuals with SLI were becoming more conservative in response to increased WM demands, and thus responding more strategically.

However, one limitation of Evens, Selingar, and Pollak's (2011) methodology involves different ratios of target-non-target trials per N-back condition. In the 1-back condition, there were 30% target trials (160 total trials), however in the 2-back condition there were 24% target trials (250 total trials). Furthermore, to maintain a somewhat similar ratio in the 2 back conditions, there were 90 more trials. It is possible that subtle differences between the ratio of non-target: target trials, and number of trials in each condition, influence participant's response decisions between WM load conditions. Performance in a block, where the probability of a non-target trial is more likely, might influence individuals into responding more conservatively. Thus, the response biases in each condition are confounded by this

contextual information, as opposed to reflecting changes in response to WM demands. The studies in the current chapter adopt equal trial ratios per N-back condition, while maintaining a fixed numbers of trials per block.

Thus, the predominant aim of the experiments in Chapter 3 is to establish the extent to which the WM deficit in developmental dyslexia is apparent in the phonological (Experiment 1) or visual-object (Experiment 2 & 3) domain. All experiments in this chapter use visual presentation, rather than auditory stimuli. Beneventi, Tonnessen, Ersland, and Hughdahl, (2010) suggests that this makes it possible to investigate WM processing without the confounding effects of co-existing auditory problems in dyslexia. Additionally, ERP analysis is implemented in experiments 1 and 2 in order to examine the electrophysiological differences between dyslexic and non-dyslexic adults during a WM task.

The following work presents 3 experiments, using the N-back task. In Experiment 1, visual letters are used as stimuli, and in Experiment 2, visual- objects are used (pictures). In both experiments, a 2: 1 ratio of non-target: target trials are used. In Experiment 3, Chinese ideograms are used, as these stimuli cannot be reverbalised, and do not contain any semantic information. In Experiment 3, a 1: 1 ratio of non-target: target trials are used to ensure that participants cannot use probabilistic information to increase their accuracy during a visual WM task. Therefore, any influence of probabilistic context, i.e. a higher probability of the current trial being a non-target trial, upon response is removed. Furthermore, Experiment 3 aims to remove the confound of imaginability, and semantics, which are present in Experiment 2. If behavioural and electrophysiological between group differences are seen when letters, as opposed to visual-objects are used as stimuli, this will provides evidence in favour of a specific phonological loop deficit in dyslexia. This will facilitate an examination

of the electrophysiological correlates of WM processing in situations where individuals with dyslexia are impaired, versus unimpaired.

Overall hypotheses

1. If, as suggested by a wealth of previous research (e.g., Ackerman & Dykman, 1993; Cohen, Netley, & Clarke, 1984; Gould & Glencross 1990, Smith-Spark, Fisk, Fawcell, & Nicolson, 2003; Avons & Hanna, 1995; Wagner & Muse, 2006), WM impairments in dyslexia are unique to the phonological loop, then we would expect to see impaired behavioural responses (reduced accuracy, smaller d' values, and higher than optimal criterion values) amongst dyslexic participants in Experiment 1 only, since verbal letters should recruit the phonological loop.
2. In this context, we would expect reduced P300 amplitude in the dyslexic group compared to non-dyslexics for letters only (Experiment 1).
3. Alternatively, as suggested by Smith-Spark and Fisk (2007), Menghini, Finzi, Carlesimo, and Vicari, (2011), and Wang and Gathercole (2013), if WM impairments in dyslexia are a function of central executive demands, then these behavioural response differences should also emerge when visual-objects are used as stimuli. Thus, behavioural impairments would be seen in all experiments here.
4. In this context, we would expect reduced P300 amplitude in the dyslexic group compared to non-dyslexics for visual-objects, and letters (i.e. in all experiments in this Chapter).
5. All participants will suffer impairments to behavioural responses, and P300 amplitude as WM load increases.

Experiment 1

In Experiment 1, the behavioural and electrophysiological correlates of verbal WM processing are assessed. Letters are used in an N-back task, across 4 levels of N (1-4).

Method

Participants

Participants were 35 adults (18 female, 17 males). Originally 40 participants were tested on the experiment; however, 5 were removed from the analysis. These participants did not have 10 correct target trials to contribute to their average, for the ERP analysis. Out of these 5 participants, 3 had a score of 0 on one or more N-back conditions as they failed to press the response button. Therefore, in the final sample, 18 non-dyslexic, and 17 dyslexic adult participants were analysed. All participants were living in Kent, UK at the time of the testing and had English as their native language. No other language, neurological disorders or visual impairments were reported by the participants. All participants had normal, or corrected to normal vision.

Dyslexic participants were recruited through the dyslexia and disability service at the University of Kent and all had a confirmed diagnosis of dyslexia by an Educational Psychologist, prior to recruitment. In addition to this diagnosis, all participants took part in an extensive dyslexia and IQ assessment. All participants were treated in accordance with the ethical guidelines outlined by the University of Kent, and the BPS standard 3.3 (The British Psychological Society, 2009; see Appendix A). Due to the sensitive nature of this research, confidentiality was maintained using anonymised coding to avoid the inclusion of personal identifiers. Data was only made accessible to the researchers.

Assessment Measures

Measures of Dyslexia and IQ: The following measures are taken from the York Dyslexia Assessment (Hatcher, Snowling, & Griffiths, 2002). Assessment time was approximately 1.5 hours, and participants were offered a break if necessary.

Literacy Skills: Single word reading ability was assessed by the Wide Range Achievement Test - III Reading (WRAT, Tan version; Jastak & Wilkenson, 1993). In the reading test, 42 words were presented in a list and participants were required to read them out loud. The amount of time taken to read the list was recorded, along with the number of correctly read words.

Spelling: To assess single word spelling ability, each participant completed the WRAT Tan spelling test (Jastak & Wilkenson, 1993). In the spelling test, items such as "mnemonic" were read out loud by the experimenter, repeated in a sentence, and then repeated for a third time. Participants were then required to write the word on their response sheet. No participants took longer than 15s to write each word.

Decoding ability: Nonsense passage reading was used to assess decoding skills. Participants were required to read two passages, taken from Gross-Glenn et al., (1990). The first passage contained 17 non-words embedded in a paragraph containing 52 words e.g., "Once upon a time a tawndy Rapsig named Gub found a tix of pertolic ascueese." The second passage was 44 words long and contained 13 non-words. For example, "The traphestal difference between the bafister jacepot and the torquil wexid lies in the function of the digton." Participants were required to read both passages out loud, while the experimenter recorded their accuracy and reading time.

Proof reading: To assess the participants' ability to identify written errors in text, they were required to complete a proof reading task. This consisted of a passage with 13 errors, including spelling, punctuation, grammar and word repetition. Incorrect responses on this task included an error of omission, or incorrectly identifying an error.

Phonological skills: Spoonerisms task. Based upon Perin (1983), participants' ability to segment and manipulate phonemes was examined by asking them to exchange the beginning

sounds of two words. The words were well known names (e.g., "Walt Disney", which becomes "Dalt Wisney"). Participants were asked to practice on the name Michael Kane. If participants needed further practice, they were asked to practice with the name "Neil Kinnock" and then their own name. Accuracy and response times were recorded for 12 Spoonerism trials.

Writing tasks: Writing speed: Participants were presented with a written 13 word sentence, containing words of varying length from 2-11 letters, e.g., "Atmospheric dust includes small particles including snow and ice" Participants were required to write this out as many times as possible in 2 minutes. Words per minute were calculated.

Timed Précis: Students were asked to read an article to themselves taken from the Independent newspaper (April, 1999) and to be prepared to write a timed Précis of that article. The reading time was recorded to give a measure of silent reading rate. The students were then asked to write a Précis of the article, in three and a half minutes. They were allowed to refer back to the article if they wished to do so. Accuracy was scored for content, structure and legibility.

Cognitive processing skills: To assess speed of processing, participants completed the WAIS-III Digit symbol subtest (Wechsler, 1997). In this test, each digit had an associated symbol, and participants were required to write symbols below each number. The score is the number of symbols copied in a minute. Verbal short term memory: To assess verbal short term memory, the digit span subtest was used from the WAIS-III (Wechsler, 1997). Digits were recited by the experimenter at a rate of 1 per second.

In order to ensure Dyslexic and control individuals did not differ in terms of general intelligence, the Silverstein (1976) subtest of the WAIS III was administered to individuals in both groups. This involved measuring verbal and non-verbal IQ.

Verbal IQ was measured using Vocabulary and Arithmetic subtests. The vocabulary test of the WAIS III was given in order to measure expressive word knowledge. Participants were asked to describe the meaning of 35 words, e.g., Ominous. Arithmetic was examined in order to assess distractibility and numerical reasoning. Participants were asked to answer 14 mental arithmetic questions. They were not allowed to use paper, or to ask for the question to be repeated. However, it should be noted that this subtest comes under the short term memory label, as participants are required to store and manipulate the information online before generating an answer.

Performance IQ was measured using Block design and Picture arrangement. In block design, the block design test of the WAIS III was administered in order to measure spatial reasoning. Participants were asked to use 4-8 blocks to form a design. On the first instance, the experimenter constructed the design, and the participant was asked to replicate this. Thereafter, the participant was required to make the design shown in picture format. Picture arrangement: The picture arrangement test of the WAIS III was given in order to assess logical sequencing abilities. 10 sets of small pictures were given out of sequence and the participant is required to arrange them into a sensible order. A one-way ANOVA was used to test the difference between the dyslexic and non-dyslexic group for age, and these assessment measures. The results of this analysis are presented in Table 2[1].

[1] Some participants had missing data for certain behavioural measures. Thus, the df differs slightly between behavioural assessment tests.

Table 2*: Assessment test results for Experiment 1.*

	Control	Dyslexic	Difference
Dyslexia Assessment			
Decoding ability: Nonsense passage reading errors: passage 1	1.13 (1.36)	5.07 (4.18)	$F(1, 28)=12.79^{***}$
Nonsense passage reading errors: passage 2	0.69 (.95)	4.00 (2.90)	$F(1, 28)=12.79^{***}$
Spoonerisms accuracy	23.00 (1.57)	20.82 (4.75);	$F(1, 33)=18.58^{+}$
Spoonerisms centile	33.61 (20.06)	19.94 (16.20)	$F(1, 33)=12.79^{*}$
Writing speed: Words/ seconds	29. 67 (5.19);	24. 67 (5.87)	$F(1, 33)=7.14^{**}$
Writing speed: centile	25.00 (35.02)	8.52 (22.06)	$F(1, 33)=2.73$
Timed Précis: Reading speed	110.55 (40.52)	121.48 (27.47)	$F(1, 30)=.80$
Timed Précis: Reading centile	40.31 (31.59)	19.70 (16.00)	$F(1, 31)=5.56^{*}$
Timed Précis: Content score	11.77 (1.92)	9.88 (2.91)	$F(1, 33)=5.21^{*}$
Timed Précis: Content centile	38.61 (28.84)	23.82 (24.33)	$F(1, 33)=2.67$
Proof reading: Number of errors	3.44 (2.38)	4.29 (2.54)	$F(1, 33)=1.04$
Proof reading: response time / seconds	60.31 (19.81)	83.01 (26.22)	$F(1, 32)=8.12^{**}$
WRAT-III Spelling (raw score)	45.58 (2.98)	39.18 (5.83)	$F(1, 32)=16.28^{***}$
WRAT-III Reading (raw score)	50.87 (5.77)	46.88 (5.78)	$F(1, 29)=3.70^{+}$
Processing speed: Digit symbol coding items/minute	82.64 (11.57)	62.67 (22.72)	$F(1, 30)=10.18^{**}$
Digits Forward	11.12 (2.39)	9.37 (1.70)	$F(1, 30)= 5.67$*
Digits Backward	7.31 (1.74)	6.68 (1.85)	$F(1, 30)= .97$
Verbal IQ (WAIS-III)			
Vocabulary	44.59 (6.41)	41.06 (6.48)	$F(1, 33)= 2.47$
Arithmetic	13.33 (2.61)	13.75 (1.91)	$F(1, 33)= .27$
Non- Verbal IQ (WAIS-III)			
Block Design	48.11 (11.89)	44.65 (13.47)	$F(1, 33)= .65$
Picture Arrangement	15.00 (3.99)	14.50 (3.84)	$F(1, 33)= .14$

Note: + $p<.10$; *$p<.05$; ** $p<.01$; *** $p<.001$. For centile and accuracy scores, higher scores suggest higher performance. For time based scores, lower scores reflect higher performance.

Materials

A visual letters version of the N-back working memory task was administered to all participants (e.g., Awh et al., 1996; Braver et al., 1997; Veltman, Rombouts & Dolan, 2003; Bemevemto et al., 2010). Ten letters were used in total, all of which were physically dissimilar (e.g., G was not used in case it was confused with C). Letters were displayed in the centre of the screen individually in upper case format. All letters were presented in black, in size 40 on a white background. In the N= 1, 2, 3 and 4 back conditions, a hit occurred when the participant correctly identified that the stimulus had occurred N items back. A correct reject occurred when the participant correctly identified that the stimulus did not occur N-back.

Design

For the behavioural analysis, a 2 x (2 x 4) mixed design was used. The between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target) and N (1 vs. 2 vs. 3 vs. 4). The behaviour dependent variables were accuracy at each level of N, for target trials (hit rate), and non-target trials (correct reject rate). When signal detection theory was implemented a 2 x (4) design was used with group and N as independent variables and d-prime and Criterion as dependent variables. In the main experiment there were 4 blocks with 150 experimental trials in each block. Out of the 150 trials, 100 were non target trials, and 50 were target trials, creating a 2: 1 ratio of non-target: target trials. WM load was manipulated between blocks. The first block represented the N=1 condition, the second represented the N=2, the third represented the N=3 and the fourth represented the N=4 condition. Within each block, no more than 2 targets were allowed in a row, furthermore, the same letter was not presented more than 3 times in a row. Response keys were counterbalanced so that half of the participants responded ‘yes’ with the

letter m, while the other with letter z. The order of N was not counterbalanced between participants[2]. There was a short stimulus presentation time (500ms), and ISI (500ms), giving the participant a total of 1000ms to make a response for each trial. Given these high task demands, the 3-back and 4-back tasks were considered to be too demanding to start the experiment with.

Procedure

Participants read the information sheets and signed informed consent forms. Each participant read instructions on a computer screen which informed them they would be presented with stimuli visually, and would have to respond yes or no, depending on whether or not the current letter occurred N back. They were given a preview screen demonstrating an example sequence for each N back task and the subsequent associated correct responses. The experimenter also verbally described the experimental procedure to each participant. The experiment was controlled using E-prime software. Each trial began with the presentation of a letter, which was on-screen for 500ms. This was followed by a blank screen of 500ms. The next experimental trial was initiated immediately after the blank screen. Participants were asked to respond as quickly and accurately as possible (as described above). After the experiment was completed, participants took part in the Dyslexia and IQ measures.

[2] The implications of this potential confound are discussed later in this chapter, and in Chapter 7.

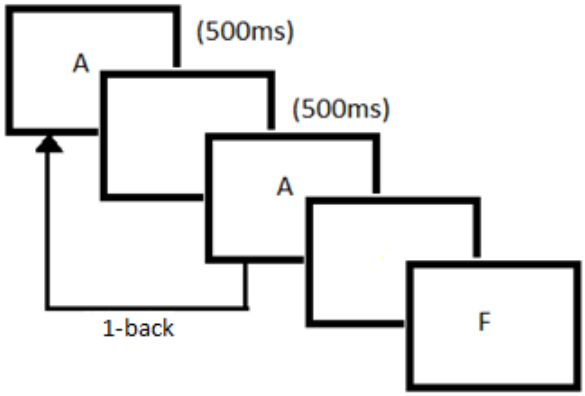


Figure 5: Illustration of the experimental procedure for N=1.

There is a presentation rate of 500ms, and an ISI of 500ms.

EEG Recording

EEG was continuously recorded with an average reference from 19Ag-AgCl electrodes. These electrodes included, Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, Pz, P4, T6, O1 and O2. An electrode map can be seen in Figure 6. Each was mounted in an Easy Cap. Furthermore, two electrodes (A1 and A2) were used to record activity from the left and right mastoids for off-line re-referencing. Two bipolar Ag-AgCl electrodes were used to monitor eye movements, placed to the outer side of each eye (HEOG), and to the top and bottom of the right eye (VEOG). An abrasive electrolyte gel was used to gently remove dead skin cells and to aid impedance. Impedance values were typically below 10kΩ and never exceeded 20 kΩ. The EEG signal was amplified using a Quickamp 72 amplifier, and recorded using Brain Vision Recording software (version 1.02). The data was continuously recorded at a sample rate of 500Hz.

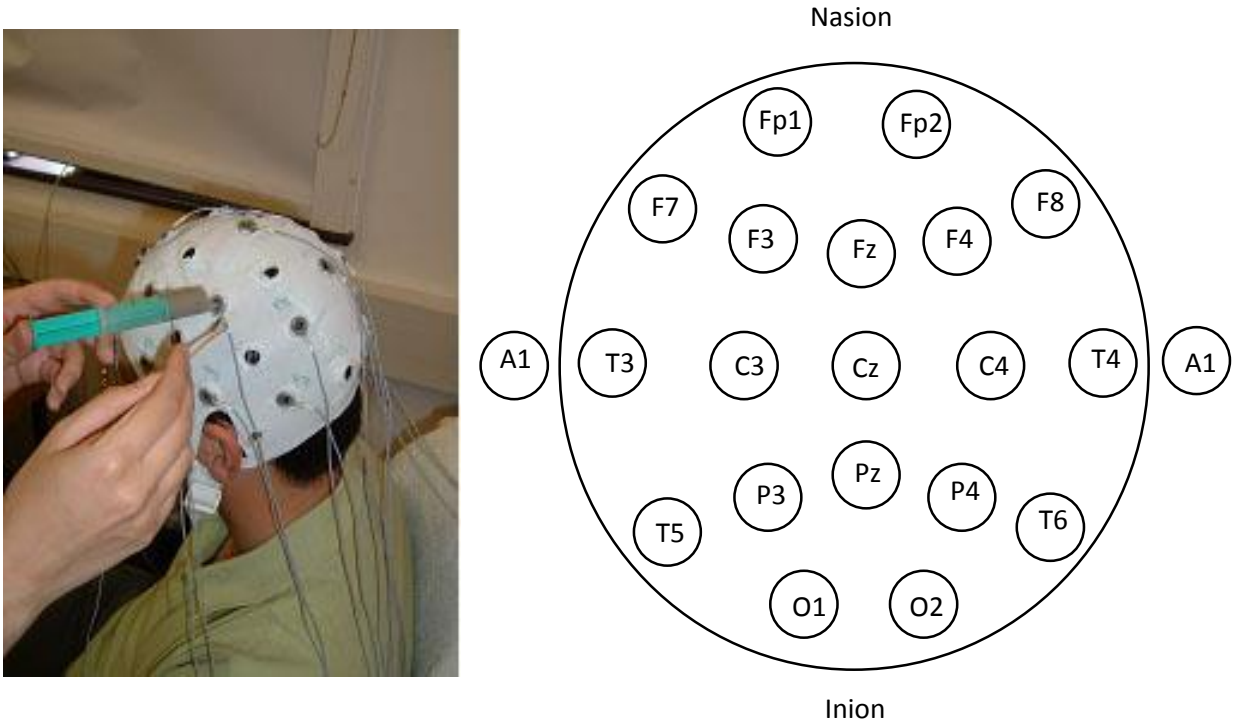


Figure 6: Electrode placement for Experiments 1 and 2.

Illustration of the participant and cap placement during the EEG setup phase (left). An illustration of the 19 EEG electrode positions (right).

Results

Behavioural effects

Trials where participants did not respond, or where they responded in less than 150ms were not included in the analysis. This cutoff was placed because it was considered unrealistic that the brain could extract information from visual stimuli at a rate quicker than a couple of hundred milliseconds. Research suggests that the inferior occipital-temporal area shows electrophysiological dissociations between words and non-words at around 180 ms, so it was deemed unlikely that the participant would be able to give meaningful responses between 150-200ms. Therefore, very quick responses were likely to be very late responses to the previous trial, or guess responses. To determine a precise cutoff, which could then be used as an a-priori cutoff for subsequent experiments, the distribution of reaction times was plotted,

as in Figure 7. The cut-of point was determined under the assumption that RT distributions are positively skewed, because the maximum possible reaction time is theoretically infinite. To determine the cutoff, the distribution was visually inspected for the point at which the assumed distribution was likely to reach zero. To maintain consistency across all experiments in this thesis, this cutoff was maintained across all experiments.

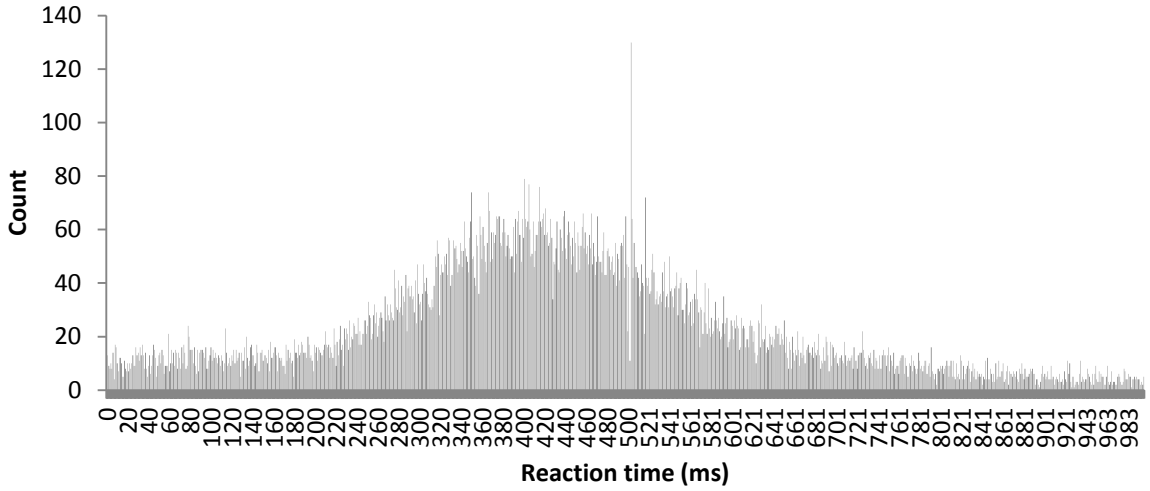


Figure 7: Reaction time distributions in Experiment 1. Assuming a positive skew, a cut-off of 150ms was employed.

Mean accuracy scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each level of N. The results are summarised in Table 3.

Table 3: *Mean hit and correct reject scores for Experiment 1.*

Trial	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
Target	Non-dyslexic	.82 (.08)	.53 (.13)	.41 (.12)	.38 (.15)
	Dyslexic	.72 (.14)	.45 (.18)	.32 (.15)	.30 (.14)
Non-target	Non-dyslexic	.94 (.06)	.85 (.12)	.85 (.11)	.82 (.15)
	Dyslexic	.93 (.04)	.90 (.08)	.88 (.07)	.86 (.12)

Note: Standard deviations are reported in parentheses.

Results were analysed using a three way GLM analysis with N (N = 1- 4), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the

between subjects variable. When Mauchly's (1940) test suggested heterogeneity of variance between conditions, the Huynh-Feldt adjustment was employed (Huynh & Feldt, 1970). Therefore, the adjusted degrees of freedom are reported for within subject effects, and interaction terms involving the within subject variable. The results revealed a main effect of N, $F(3, 99) = 129.23$, $p < .001$, $\eta p^2 = .80$ and a main effect of trial, $F(1, 33) = 266.30$, $p < .001$ $\eta p^2 = .89$, with higher accuracy for non-target trials. There was a significant interaction between trial * group, $F(1, 33) = 5.50$, $p = .025$, $\eta p^2 = .14$. Dyslexic participants' accuracy scores were lower in comparison to non-dyslexic participants for target trials, however, their accuracy was comparable for non-target trials. This interaction can be seen in Figure 8, where accuracy on target and non-target trials is plotted separately for dyslexic and non-dyslexic participants. Furthermore, there was a significant interaction between trial * N, $F = (2.47, 81.58) = 53.26$, $p < .001$, $\eta p^2 = .62$. In order to examine this interaction further, response accuracy data was analysed separately for target (hits) and non-target trials (correct rejects).

For the hit rate, a mixed GLM analysis was conducted, revealing a main effect of N on performance across all participants, $F(3, 99) = 136.55$, $p < .001$, $\eta p^2 = .80$. Furthermore, there was a main effect of group, $F(1, 33) = 5.19$, $p = .029$, $\eta p^2 = .14$. The interaction term for N * group was not significant, $F(3, 99) = 0.02$, $p = 1.00$, $\eta p^2 = .001$, suggesting that the difference between dyslexic and non-dyslexic participants accuracy was consistent across all levels of N. To analyse correct rejects, another 2x3 mixed GLM analysis was conducted, revealing a main effect of N, $F(2.44, 80.47) = 10.37$, $p < .001$, $\eta p^2 = .24$, where increasing N was associated with decreased correct-rejects. Neither the main effect of group, or the interaction between N * group were significant [all Fs <.97] suggesting no difference between dyslexic and controls' correct reject accuracy, even as working memory demands

increased. Thus, the two way group * trial interaction is driven by decreased hit rate in dyslexic participants, but comparable correct reject. The between group effect was not significant, F= (1, 33) = 21.91, p =.17, ηp^2 = .06, and neither was the interaction between N * group and trial * N * group were not significant [All Fs <.48][3]. Figure 8 displays accuracy for target and non-target trials seperately.

Figure 8: Response accuracy for dyslexic and non-dyslexic individuals on target and non-target trials.

Signal detection theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants, at each level of N (1, 2, 3 and 4). The results are presented in Table 4.

[3] Reaction times were analysed using a 2 (trial) x 4 (N=1-4) and 2 (dyslexic, control) repeated measures ANOVA, with median RTs as the dependent measure. For experiment 1, all effects were non-significant [Fs < 1.60].

Table 4: *Signal Detection Theory analysis for Experiment 1.*

Parameter	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
d'	Non-dyslexic	2.51 (.58)	1.28 (.69)	.87 (.42)	.70 (.61)
	Dyslexic	2.11 (.62)	1.27 (.74)	.75 (.50)	.63 (.55)
Criterion	Non-dyslexic	.41 (.20)	.55 (.26)	.67 (.32)	.66 (.37)
	Dyslexic	.43 (.20)	.76 (.34)	.87 (.34)	.86 (.39)

Note: Standard deviations are reported in parentheses.

A 2(group) x 4(N) mixed GLM analysis was carried out to examine the between condition differences associated with d-prime values. This analysis revealed a main effect of N, $F(2.81, 92.74) = 97.84$, $p < .001$, $\eta p^2 = .75$, with decreasing d-prime values as N increases. The effect of group, and interaction between N * group were not significant [$F=1.32$]. A mixed GLM analysis was carried out to examine the between condition differences associated with criterion values. This showed a main effect of N, $F(2.67, 88.22) = 19.66$, $p < .001$, $\eta p^2 = .37$, whereby the criterion increases as N increases. There was also a marginally significant main effect of group upon the criterion value $F(1, 33) = 3.56$, $p=.068$, with dyslexic participants placing an overall higher criterion. The interaction term N * group was not significant [$F=1.51$].

Electrophysiological Analysis

Off-line, recordings were re-referenced to the averaged left and right mastoid electrodes, and were passed through a bandpass filter of .30-30Hz. EEG data was corrected for vertical and horizontal eye movements using the BrainVision Analyzer 2 semi-automatic ocular ICA for correction. EEG recordings were then segmented into epochs of 1100ms, time-locked to stimulus onset, including 100ms baseline. Each epoch was screened for artifacts (e.g., remaining eye movements) using semiautomatic artifact rejection methods and aligned to the 100ms pre-stimulus baseline. The P300 amplitude (peak-peak) and latency was examined for target trials only, between dyslexic and non-dyslexic participants, at Fz, Cz, and Pz, as a

function of WM load. Due to low accuracy in higher N-back conditions, conditions 1 and 2 back were combined into a low load condition, and conditions 3 and 4 were combined into a high load condition. This ensured that at least 10 trials contributed to the subject specific mean for each condition. Data was then imported into EEGlab and the P300 was analysed using the peak-peak method. Traditionally peak-peak analysis takes the difference between the positive waveform from the negative recovery phase. Thus for each participant the most positive average 50ms period between 300-500ms was identified, along with the most negative 50 ms period during recovery phase. This negative recovery phase was analysed from the point at which the most positive voltage was recorded, until the end of the trial (1000ms). The peak-peak amplitude values and P300 peak latency were subjected to a multivariate ANOVA. Grand average ERP waveforms for each condition and group are presented in Figure 9.

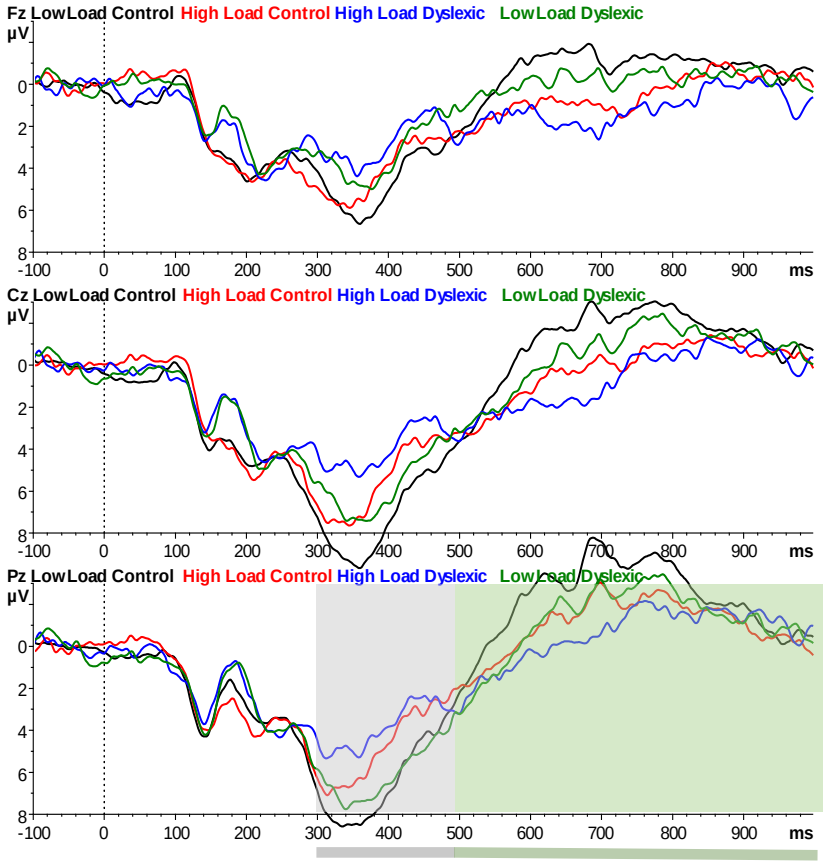


*Figure 9***:** ERP waveforms at retrieval for Experiment 1.

ERPs are plotted for **low non-dyslexic**, high non-dyslexic, low dyslexic, high non-dyslexic conditions, where low and high refer to the WM load conditions. The grey window donates the P300 time window for the positive region. The green region reflects the time window for the P300 rebound.

In order to examine these differences, a three way GLM analysis was conducted with electrode (Fz, Cz, Pz) and N-back low (N = 1 & 2), and high (N = 3 & 4) as within subjects variables and group (dyslexic, non-dyslexic) as the between subjects variable. Results revealed a significant main effect of electrode, $F(1.66, 54.89) = 35.12$, $p < .001$, $\eta p^2 = .52$, whereby the mean peak-peak amplitude at each electrode were: 9.37 uV at Fz, 12.02 uV at Cz, and 12.43 uV at Pz. This topography is indicative of a P3b component, which typically occurs maximally in posterior scalp regions. Furthermore, the results revealed a significant

main effect of N, $F(1, 33) = 5.07$, $p<.05$ $\eta p^2 = .13$, whereby the P300 amplitude reduces as N increases. The main effect of group upon Peak – Peak distance was marginally significant $F(1, 33) = 3.43$, $p=.073$ $\eta p^2 = .09$. Furthermore, there was a significant interaction between electrode * N, $F(1.43, 47.24) = 9.51$, $p=.001$, $\eta p^2 = .22$. Planned [4]post-hoc paired sample t-tests were carried out for Fz, Cz and Pz to examine which electrodes revealed significant differences between low and high WM load conditions. The statistical results suggest a significant difference between WM load at Cz and Pz; see Table 5). The interactions between N * group, and N * electrode were not significant [All *F*s <.36].

Table 5: *Paired t-test results for Experiment 1.*

Electrode	df	t value	*p*-value
Fz	34	.98	.33
Cz	34	2.54	.02*
Pz	34	2.87	.007**

Note: * $p<.05$, $p<.01$**; *t-test are conducted at electrode sites Fz, Cz and Pz, comparing low versus high WM load.*

P300 latency was calculated as the middle time point of the 50ms average, for the positive peak window only (between 300 – 500ms). To examine the effects on latency, a three way GLM analysis was conducted with electrode (Fz, Cz, Pz) and N-back (low (N = 1 & 2), and high (N = 3 & 4) as within subjects variables and group (dyslexic, non-dyslexic) as the between subjects variable. All effects were non-significant [*F*s<2.25][5].

[4] The post-hoc tests were carried out at a-priori defined electrodes (where the P300 occurs maximally), and thus, the p-values are not corrected for multiple comparisons.

[5] Inspection of the grand average also revealed between group differences in a negative component, between 150-250ms. This component was interpreted to be the N2, despite occurring slightly earlier than the traditional N2 component, which commonly peaks between 200-300ms (See Folstein & Petten, 2008 for a review), but can occur earlier (e.g., Ham, Strien, Oleksiak, Wezel & Postma, 2010). A three way GLM analysis was conducted with electrode (Fz, Cz, Pz) and N-back (combined 1 & 2, and combined 3 & 4) as within subjects variables and group (dyslexic, non-dyslexic) as the between subjects variable. Due to the exploratory nature of this analysis, the boneferroni correction was used, with a significance threshold of $p<.02$. However, the group effect was not significant $F(1, 33) = 2.68$, $p=.11$. All other effects were not significant [*F*s<2.23]. This analysis was conducted for experiment 1 only.

In summary, the behavioural results showed an interaction between group and trial type. When target trials are examined, dyslexic participants have reduced accuracy compared to the non-dyslexic group. However, their performance on non-target trials is comparable to non-dyslexic participants. This may have been the result of a higher criterion placement, as suggested by a marginally significant between group effect. Amplitude analysis of the P300b demonstrated that dyslexic individuals have a smaller P300b response to target trials, however, this effect only reached marginal significance. Overall, the behavioural and electrophysiological results of Experiment 1 suggest individuals with developmental dyslexia have a phonological WM impairment. Experiment 2 and 3 examine the extent to which these differences are also found in the non-verbal domain.

Experiment 2

The aims of the following experiment are to examine the extent to which dyslexic individuals are impaired in their performance on the N-back task for visual-object information. In Experiment 1, a phonological WM impairment in dyslexia was identified. Experiment 2 eliminates phonological activation, and focuses more specifically on visual-object information. In the same way as Experiment 1, the N-back procedure included a load of N= 1-4. If, as suggested by recent evidence from Smith-Spark and Fisk (2007), Menghini, Finzi, Carlesimo, & Vicari, (2011), and Wang and Gathercole (2013), that WM impairments in dyslexia are a function of central executive demands, then these between group differences should also emerge when visual-objects are used as stimuli.

Method

Participants

Participants were 35 adults (20 female, 15 male), 19 of whom had normal reading ability, and 16 had a confirmed diagnoses of dyslexia. Dyslexic participants were age matched to non-

dyslexic participants, with a mean age of 22.63 in the non-dyslexic group, and 21.06 in the dyslexic group, $F(1, 33) = 1.0$, $p = .30$. Dyslexia and IQ assessment and inclusion criteria were maintained from Experiment 1. Results are presented in Table 6.

Table 6: *Behavioural Assessment results for Experiment 2.*

	Control	Dyslexic	Difference
Dyslexia Assessment			
Decoding ability: Nonsense passage reading errors: passage 1	1.12 (1.32);	5.84 (4.37)	$F(1, 28)=17.91^{***}$
Nonsense passage reading errors: passage 2	0.76 (.97)	4.61 (2.81)	$F(1,29)=27.77^{***}$
Spoonerisms accuracy	22.90 (1.59)	20.37 (4.85)	$F(1, 33)=4.55^{*}$
Spoonerisms centile	15.93 (17.05)	15.94 (17.05)	$F(1, 33)=5.91^{*}$
Writing speed: Words/ seconds	29.61 (5.05)	24.27 (4.45)	$F(1, 34)=10.77^{**}$
Writing speed: centile	24.21 (34.20)	3.43 (7.00)	$F(1, 34)=5.67^{*}$
Timed Précis: Reading speed	109.79 (39.36);	128.59 (45.87)	$F(1, 30)=1.56$
Timed Précis: Reading speed centile	40.88 (30.68)	20.00 (16.93)	$F(1, 31)=4.83^{*}$
Timed Précis: Content score	11.68 (1.92)	10.18 (2.54)	$F(1, 33)=3.95^{+}$
Timed Précis: Content centile	37.11 (28.78)	23.75 (23.42)	$F(1, 33)=2.21$
Proof reading: Number of errors	3.32 (2.38)	4.81(3.23)	$F(1, 33)=2.48$
Proof reading: Number of errors; response time / seconds	66.30 (31.88)	87.20 (32.27)	$F(1, 32)=3.59^{+}$
WRAT-III Spelling (raw score)	45.55 (2.89)	38.50 (5.91)	$F(1, 32)=20.25^{***}$
WRAT-III Reading (raw score)	51.13 (5.67)	46.33 (5.77)	$F(1, 29)=5.44^{*}$
Processing speed: Digit symbol coding items/minute	82.33 (11.30)	65.85 (22.72)	$F(1, 30)=7.22^{*}$
Digits Forward	11.05 (2.33)	9.20 (1.69)	$F(1, 30)= 6.49^{*}$
Digits Backward	7.24 (1.71)	6.80 (2.04)	$F(1, 30)= .43$
Verbal IQ (WAIS-III)			
Vocabulary	44.39 (6.28)	40.00 (7.08)	$F(1, 31)= 3.56^{+}$
Arithmetic	13.26 (2.56)	12.80 (2.24)	$F(1, 32)= .31$

Non- Verbal IQ (WAIS-III)			
Block Design	48.42 (11.63)	46.19 (13.70)	$F(1, 33)= .27$
Picture Arrangement	15.00 (3.87)	14.73 (3.82)	$F(1, 32)= .04$

Note: $^{+}p<.10$; $^{*}p<.05$; $^{**}p<.01$; $^{***}p<.001$. *Standard deviations are reported in parentheses.*

Measures

A picture version of the N-back working memory task was administered to all participants. 10 pictures were used in total, all of which were coloured and presented on a white background. Each picture was sized at 120 * 120 pixels and displayed in the centre of a 1024 * 768 resolution screen. An example of the stimuli can be displayed in Figure 10.

Figure 10: Example of pictorial visual-object stimulus.

Design and Procedure

A 2 x (2 x 4) mixed design was employed. The between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target) and N (1 vs. 2 vs. 3 vs. 4). The mixed design enabled the research to differentiate between dyslexic and non-dyslexic participants' working memory ability in the same task. The behaviour dependent variables were accuracy at each level of N. As in Experiment 1, a hit occurred when a participant answered 'yes' and the current item had occurred N items back. A correct reject was recorded when the participant correctly identified (responded 'no'), that the stimulus did not occur N-back. When signal detection theory was implemented, a 2 x (4) design was analysed with group and N as independent variables, and

d-prime and Criterion as dependent variables. Trial presentation timings and target: non-target ratio were fixed as in Experiment 1. Thus, there were 4 blocks with 150 experimental trials in each. Out of the 150 trials, 100 were non target trials, and 50 were target trials. The same contingencies, and counterbalancing procedures were implemented as in Experiment 1. The procedure was identical to that described in Experiment 1, except that the images described above replaced the letter stimuli. EEG was recorded as in Experiment 1.

Results

Behavioural effects

Mean accuracy scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each level of N. The results are summarised in Table 7.

Table 7: *Mean hit and correct reject scores for Experiment 2.*

Parameter	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
Target	Non-dyslexic	.74 (.10)	.50 (.16)	.33 (.10)	.32 (.15)
	Dyslexic	.73 (.13)	.47 (.16)	.33 (.19)	.30 (.13)
Non-target	Non-dyslexic	.96 (.04)	.91 (.06)	.87 (.10)	.84 (.10)
	Dyslexic	.95 (.03)	.90 (.06)	.86 (.07)	.86 (.10)

Note: Standard deviations are reported in parentheses.

Results were analysed using a three way GLM analysis, with N (N= 1- 4), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The results revealed a main effect of N, $F(2.48, 81.96) = 120.17$, $p<.001$, $\eta p^2 = .78$, with decreasing hit rates and correct-rejects as N increased, and a main effect of trial, $F(1,33)= 298.47$, $p<.001$, $\eta p^2 = .90$, with higher accuracy (hits and correct rejects) for non-target trials. There was a significant interaction between trial * N, $F(2.43, 80.14)= 46.19$, $p<.001$, $\eta p^2 = .58$, whereby participants had a larger decrease in hit rate as N increased, but a slower decrease in correct rejects. This interaction is later explored in the

criterion score, as it is likely to be the result of strategic responses in relation to increased WM load. The effects of group, trial * group, N * group and trial * N * group were not significant [All *F*s <.35]. Unlike experiment 1, dyslexic individuals did not perform differently to controls depending on trial type[6].

Signal detection theory

Mean d' and criterion values and standard deviations are presented in Table **8**.

Table 8: *Mean scores for Signal Detection Theory parameters for Experiment 2.*

Parameter	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
d'	Non-dyslexic	2.52 (.96)	1.46 (.71)	.80 (.57)	.60 (.55)
	Dyslexic	2.31 (.65)	1.32 (.49)	.65 (.44)	.60 (.46)
Criterion	Non-dyslexic	.54 (.30)	.72 (.27)	.84 (.31)	.79 (.35)
	Dyslexic	.49 (.19)	.72 (.37)	.78 (.38)	.87 (.37)

Note: Standard deviations are reported in parentheses.

A mixed GLM analysis was carried out to examine the between condition differences associated with d-prime values. This analysis revealed a main effect of N, $F(2.34, 77.04) = 96.25$, $p < .001$, $\eta p^2 = .75$, with decreasing d-prime values as N increases. The effect of group, and the interaction between N * group were not significant [*Fs*<.43]. For the criterion, a mixed GLM analysis was carried out, and revealed a significant main effect of N, $F(2.63, 86.90) = 17.52$, $p < .001$, $\eta p^2 = .34$, whereby the criterion increases as N increases. The effect of group and the interaction between N * group was not significant [*F*<.85].

[6] For experiment 2, RT analysis was conducted using the same independent variables as Experiment 1 There was a main effect of trial $F(1, 33)=17.65$, $p<.001$, $\eta p^2 = .35$ with quicker RTs for non-target trials, and N, $F(2.11, 69.70) = 2.76$, $p=.02$, $\eta p^2 = .10$, with decreasing RTs as N increases. All other effects were not significant [*F*s<1.95].

Electrophysiological Analysis

Off-line ERP preprocessing was consistent with Experiment 1. Data was imported into EEGlab and the P300 was analysed using the peak-peak method. The peak-peak amplitude values and P3 peak latency were subjected to a multivariate ANOVA. Grand average ERP waveforms are presented in Figure 11.

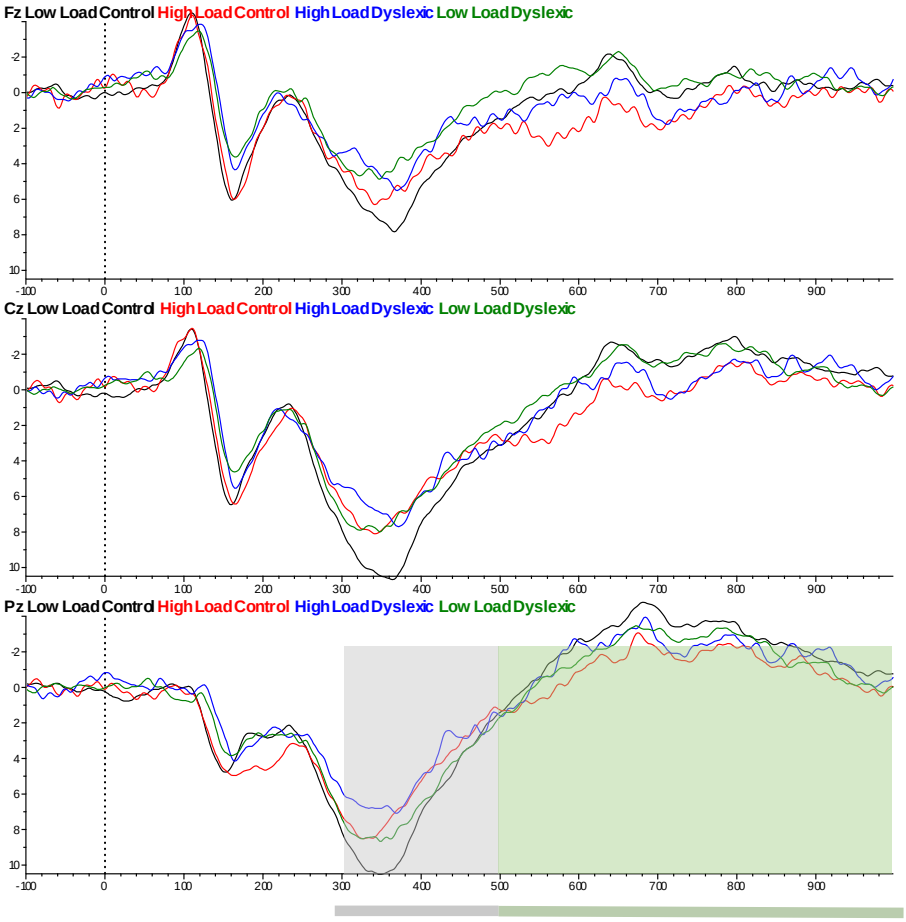


Figure 11: Grand average ERPs for Experiment 2 at electrodes sites Fz, Cz and Pz. ERPs are plotted for **low non-dyslexic**, high non-dyslexic, low dyslexic, high non-dyslexic conditions, where low and high refer to the WM load conditions.

In order to examine these differences, a three way GLM analysis was conducted with electrode (Fz, Cz, Pz) and N-back low load (N= 1 & 2) and high load (N = 3 & 4) as within subjects variables and group (dyslexic, non-dyslexic) as the between subjects variable. Results revealed a significant main effect of electrode, $F(1.51, 49.75) = 29.99$, $p < .001$, $\eta p^2 =$

.48, whereby the mean peak-peak values at each electrode were: 10.38 uV at Fz, 13.45 uV at Cz, and 14.29 uV at Pz. Again, this topography indicates a P300b component, which typically occurs maximally in posterior regions. Furthermore, the results revealed a significant main effect of N, $F(1, 33) = 10.28$, $p<.003$ $\eta p^2 = .24$ where N decreases P300 amplitude. This interacted with electrode, $F(1.41, 46.41) = 6.51$, $p=.007$, $\eta p^2 = .17$. Thus, planned post-hoc paired sample t-tests were carried out for Fz, Cz and Pz to examine at which levels of N these waveforms significantly differed. The statistical results suggest a significant difference between WM load at Cz and Pz, whereby high WM load results in a smaller P300 amplitude compared to low WM load. The results are presented in Table 9. All other effects were not significant [All *F*s <2.57].

Table 9: *Paired t-test results for Experiment 2*

Electrode	df	t value	p-value
Fz	34	1.68	$.10^{+}$
Cz	34	3.47	$.001^{**}$
Pz	34	3.85	$.000^{***}$

Note: $+p<.10$, $p<.05^{*}$, $p<.01^{**}$, $p<.001^{***}$; t-tests are conducted *at electrode sites Fz Cz and Pz, comparing low versus high WM load.*

To examine the effects of P300 latency, a three way GLM analysis was conducted with electrode (Fz, Cz, Pz) and N-back (low WM load: N= 1 & 2, and high WM load: N= 3 & 4) as within subject variables, and group (dyslexic, non-dyslexic) as the between subjects variable. For visual objects, there was a main effect of electrode, $F(1.35, 44.67) = 9.25$, $p=.02$, $\eta p^2 = .21$, with larger latencies occurring at Cz and Pz. All effects were non-significant [*F*s<1.51].

Overall Experiment 2 suggests that dyslexic participants are not impaired in visual-objects WM processing. WM load increased the P300 amplitude, but had no effect on P300

latency. Unlike Experiment 1, the interaction term between trial * group was not significant, suggesting that dyslexic and control participants' accuracy on both target and non-target trials was comparable. No differences were revealed in the SDT or P300b analysis. The results are indicative of unimpaired visual object WM processing in dyslexic adults, which taken together with Experiment 1 might suggest a specific phonological loop impairment, as opposed to a domain general central executive impairment. However, one limitation of Experiment 2, is that the visual-objects were pictures, containing semantic information. Riby and Orme (2013) have recently demonstrated that visual objects containing semantic information aid visual WM capacity, resulting in a larger P300 response. To ensure these null effects translate in a setting where there is no semantic information available, Experiment 3 is a replication of the visual-object WM task used in Experiment 2, using Chinese ideograms as stimuli.

Experiment 3

In Experiment 3, the visual stimuli were replaced with Chinese ideograms. Chinese ideograms have been used previously in the literature to assess visual-object WM processing (e.g., Klauer & Zhao, 2004). The benefit of these stimuli is that they cannot be sub-vocalised by Native English speakers, and do not contain semantic information to assist or scaffold visual WM processing. Furthermore, this experiment adopts a 1: 1 ratio between non-target: target trials to ensure that participants cannot use the probabilistic context to influence their responses during this task, which was the case in Experiment 1. Therefore, the aim of the current experiment was to ensure that group differences would not emerge when visual stimuli without semantic content were used and when balanced target/non-target bial numbers were used. Therefore, EEG was not recorded during this experiment, since

behavioural measures (i.e. accuracy and SDT) were deemed the most valuable measures to address this question.

Method

Participants

36 participants were originally run on the experiment, however, two withdrew half way through. An additional participant was removed, as they failed to make any behavioural responses during two blocks of the experiment. Therefore, participants were 33 adults (21 female, and 14 males). Of whom, 18 had normal reading skills. Of the 33, 16 had a confirmed diagnosis of dyslexia from an Educational Psychologist. Participants were matched for age across both groups $F(1, 32)=1.21$, $p= .28$, with a mean age of 21 ($SD = 2.86$) in the non-dyslexic group and 20 ($SD = 1.45$) in the dyslexic group. Participants took part for course credits, or a small monetary payment. All participants were Native English speakers, and reported no other neurological or neurodevelopmental disorder.

Assessment Measures

Participants took part in a comprehensive dyslexia assessment and assessment of verbal and non-verbal IQ. The dyslexia assessment was updated to the York Adult dyslexia assessment revised (YAA-R; Warmington, Stothard & Snowling, 2013), and thus a full description of these assessment measures is provided here, as the assessment measures in Experiment 3 differ slightly to those described in experiment 2. The assessment battery consists of tests of reading, spelling, writing, and phonological skills. To assess verbal (vocabulary) and non-verbal IQ (picture arrangement, mental arithmetic, and block design) the Silverstein subtest selection (1976) of the WAIS III was administered from the WAIS-III (Wechsler, 1997), which were described in Experiment 1.

The following measures are taken from the York Adult Assessment Revised (YAA-R; Warmington, Stothard & Snowling, 2012) and the WRAT-III. Assessment time took approximately 2 hours, and participants were offered a break if necessary.

Reading: Participants completed the Reading comprehension test, from a passage entitled "The history of Chocolate" to assess reading comprehension, reading errors, reading time and reading rate. The passage contains a non-fictional piece of written text, comprised of 492 words, and 15 comprehension questions to assess reading knowledge (7 items), vocabulary (4 items) and inference making (4 items). Reading time as words/min and reading accuracy (number of errors), are recorded. Comprehension questions are scored as correct or incorrect, out of a total of 15. Each participant also completed the WRAT-III Tan reading test.

Summarisation skills: Participants were tested on their ability to summarise what they had read from "The history of Chocolate" after completing the comprehension questions. This provides a measure of summarisation skills and writing under time pressure, without the opportunity to refer back to the text. Participants were scored on the number of content points, which were scored as correct regardless of spelling errors, except for the distinction between cacao beans and cocoa butter. Writing rate was expressed as words per minute.

Spelling: To assess spelling, a spelling rate score was computed from the written précis test, which included the number of errors divided by the number of written words. To assess single word spelling ability, each participant completed the WRAT Tan spelling test (Jastak & Wilkenson, 1993). In the spelling test, items such as "mnemonic" were read out loud by the experimenter, repeated in a sentence, and then repeated for the third time. Participants were then required to write the word. No participants took longer than 15s to write each word.

Phonological skills: Spoonerisms task. Based upon Perin (1983), participants' ability to segment and manipulate phonemes was examined by asking them to exchange the beginning sounds of two words. The words were well known names (e.g., "Wayne Rooney", which becomes "Rayne Wooney"). The test contained 12 items, and total accuracy was out of 24 (2 words per item). The Spoonerism rate was calculated for correct items only (scores of 2), and was expressed as seconds per item. To assess RAN of digits and objects, participants named an array of 50 items from left to right, as quickly and accurately as possible. Both the digits and objects version began with a practice trial. Naming time, and rate as words per second were expressed. Verbal short term memory: To assess verbal short term memory, the digit span subtest (digits forward and digits backward) were used from the WAIS-III (Wechsler, 1994). Digits were recited by the experimenter at a rate of 1 per second.

Writing tasks: Writing speed: Participants were presented with a written 12 word sentence, containing words in varying length from 2-11 letters "Erosion is a gravity driven process that moves solids in the environment" Participants were required to write this out as many times as possible in 2 minutes. Words per minute were calculated.

Cognitive processing skills: To assess speed of processing, participants completed the WAIS-III Digit symbol subtest (Wechsler, 1997). In this test, each digit had an associated symbol, and participants were required to write symbols below each number. The score is the number of symbols copied in a minute.

Results of all dyslexia assessment and WAIS III (Silverstein subtest selection, 1982) measures are presented in Table 10.

Table 10: *Behavioural Assessment test results for Experiment 3.*

	Control	Dyslexic	Difference
Dyslexia Assessment			

Passage reading errors	4.05 (2.94)	11.00 (7.54)	$F(1, 32)=12.39^{***}$
Reading comprehension	7.58 (1.22)	8.50 (2.09)	$F(1, 32)=2.36$
Timed Précis: Content score	11.29 (2.80)	10.13 (3.57)	$F(1, 32)=1.10$
Timed Précis: words per minute	22.24 (4.22)	18.21 (4.94)	$F(1, 32)=6.36^{**}$
Timed Précis: Spelling errors	1.94 (2.92)	5.43 (3.59)	$F(1, 32)=9.44^{**}$
Writing speed	32.23 (3.76)	28.10 (4.16)	$F(1, 32)=8.71^{**}$
Spoonerisms Accuracy	22.23 (3.45.)	15.00 (6.73)	$F(1, 32)=15.35^{***}$
Spoonerisms correct seconds/item	1.87 (1.95)	3.46 (2.40)	$F(1, 32)=4.40$*
RAN Digits Total time	15.37 (2.54)	26.07 (16.25)	$F(1, 32)=7.18^{**}$
RAN Digits items/ sec	3.25 (.79)	2.36 (.83)	$F(1, 32)=9.74^{**}$
RAN Objects Total time	25.97 (5.62)	36.41 (16.46)	$F(1, 32)=6.09^{**}$
RAN objects items/ sec	1.98 (.33)	1.54 (.44)	$F(1, 32)=10.52^{**}$
WRAT-III Spelling (raw score)	52.70 (3.06)	47.12 (3.72)	$F(1, 32)=22.27^{***}$
WRAT-III Reading (raw score)	44.40 (2.61)	39.06 (3.66)	$F(1, 32)=21.57^{***}$
Processing speed: Digit symbol coding items/minute	40.85 (7.12)	38.20 (7.89)	$F(1, 32)=1.02$
Digits Forward	11.94 (1.95)	9.87 (2.52)	$F(1, 32)=6.96^{**}$
Digits Backward	7.76 (2.75)	5.38 (1.92)	$F(1, 32)=8.25^{**}$
Verbal IQ (WAIS-III)			
Vocabulary	40.47 (7.64)	36.75 (6.87)	$F(1, 32)=2.15$
Arithmetic	14.17 (3.48)	12.18 (3.98)	$F(1, 32)=2.39$
Non- Verbal IQ (WAIS-III)			
Block Design	52.82 (10.03)	52.13 (11.44)	$F(1, 32)=.04$
Picture Arrangement	12.00 (3.20)	15.50 (3.16)	$F(1, 32)=9.96^{**}$

Note: $^{+}p<.10$; $^{*}p<.05$; $^{**}p<.01$; *** p<.001. *Standard deviations are reported in parentheses.*

Measures

An object version of the N-back working memory task was administered to all participants. 10 Chinese characters were used in total, all of which were black, presented on a white background. Each picture was sized at 120 * 120 pixels and displayed in the centre of a 1024 * 768 resolution screen. An example of the stimuli used is displayed in Figure 12.

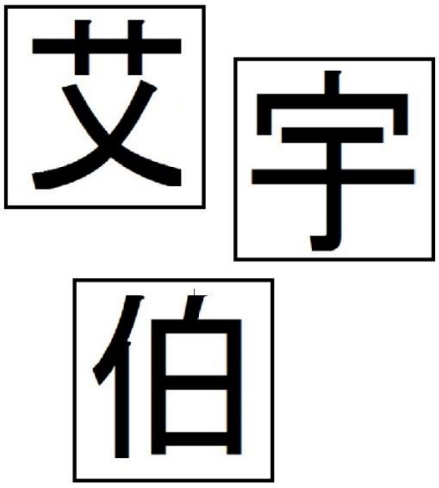


Figure 12: Example of visual-object stimuli for Experiment 3.

Design and Procedure

Participants took part in a visual-object version of the N-back task, with Chinese ideograms. In order to replicate Experiment 1 and 2, a 2 x (2 x 4) mixed design was utilized. The between subjects variable was Group (dyslexic, non-dyslexic), while the within subjects variables were trial (target, non-target) and N (1, 2, 3, 4). When signal detection theory was implemented a, 2 x (4) design was implemented with group and N as independent variables and d-prime and Criterion as dependent variables. In the main experiment there were 4 blocks with 200 experimental trials in each block. In contrast to Experiment 1, a 1: 1 ratio of non-target: target trials was used. Therefore, out of the 200 trials, 100 were non target trials, and 100 were target trials. Response keys were counterbalanced, as in Experiment 1 and 2. The procedure was a direct replication of Experiment 1 and 2.

Results

Behavioural effects

Mean accuracy scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each level of N. The results are summarised in Table 11.

Table 11: *Mean hit and correct reject scores for Experiment 3*

Parameter	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
Target	Non-dyslexic	.75 (.14)	.55 (.15)	.42 (.13)	.41 (.17)
	Dyslexic	.72 (.18)	.53 (.16)	.39 (.17)	.39 (.18)
Non-target	Non-dyslexic	.79 (.16)	.67 (.19)	.64 (.18)	.66 (.19)
	Dyslexic	.77 (.10)	.73 (.16)	.72 (.16)	.69 (.16)

Note: Standard deviations are reported in parentheses.

Results were analysed using a three way GLM analysis, with N (N = 1- 4), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The results revealed a main effect of N, $F(3, 93) = 63.97$, $p<.001$, $\eta p^2 = .67$, with decreasing accuracy scores as N increased, and a main effect of trial, $F(1, 31) = 25.29$, $p<.001$ $\eta p^2 = .45$, with higher accuracy for non-target trials. The interaction between trial * N was also significant $F(3, 2.92) = 13.62$, $p<.001$, $\eta p^2 = .31$. All other effects, including the effect of group, and interaction between group * N, did not reach significance [*F*s <.88].

Signal detection theory

Mean scores and standard deviations were calculated for both d-prime and criterion. The results are presented in Table 12.

Table 12: *Signal Detection Theory parameters for Experiment 3.*

Parameter	**Group**	**N-back**			
		N=1	N=2	N=3	N=4
d’	Non-dyslexic	1.60 (.86)	. 22 (.58)	.22 (.38)	.22 (.30)
	Dyslexic	1.40 (.76)	.33 (.73)	.33 .(73)	.24 (.50)
Criterion	Non-dyslexic	.09 (.25)	.18 (.39)	.29 (.38)	.33 (.47)
	Dyslexic	.08 (.28)	.31 (.40)	.47 (.33)	.44 (.46)

Note: Standard deviations are reported in parentheses.

A mixed GLM analysis was carried out to examine the between group differences associated with d-prime values. This analysis revealed a main effect of N, $F(3, 93) = 54.6$, $p < .001$, $\eta p^2 = .64$, with decreasing d-prime values as N increased. The effect of group, and interaction between N * group were not significant [$Fs < .47$]. For the criterion, a mixed GLM analysis was carried out, and revealed a main effect of N, $F(3, 93) = 11.80$, $p < .001$, $\eta p^2 = .28$, whereby the criterion increased as N increased. The effect of group and interaction between N * group was not significant [$F < 1.00$][7].

General discussion

The aims of the experiments conducted in this chapter were to examine WM processing during a visual N-back task in developmental dyslexia. Previous research has demonstrated poor WM processing in developmental dyslexia within the verbal domain. However, the effects in the visual domain are mixed. Research that has demonstrated effects in the visual WM has been conducted predominantly with children, and between group differences in visual WM contexts has been interpreted to indicate central executive dysfunction in dyslexia. The experiments in this chapter extended the literature in three key ways. Firstly, adults were used to determine the extent to which central executive dysfunction is prevalent in adults with dyslexia. In order to achieve this goal, difference stimuli were used. In Experiment 1, letters were used in order to access the phonological loop, while in Experiments 2 and 3, visual-object stimuli were implemented. Experiment 3 allowed us to assess visual WM processing without the influence of semantics. SDT was also implemented

[7] RTs were analysed for experiment 3, there was a main effect of trial $F(1, 31) = 10.66$, $p < .001$, $\eta p^2 = .23$ and N, $F(3, 93) = 13.06$, $p = .02$, $\eta p^2 = .31$, with decreasing reaction times as N increases. There was also an interaction between trial * N, $F(3, 93) = 7.46$, $p < .001$, $\eta p^2 = .19$, whereby RTs for target trials increased with increasing N for target trials, until 2 back where they decreased. RTs decreased for non-target trials. All other effects were not significant [$Fs < 1.95$].

which allows researchers to examine discriminability, while controlling for response bias, and it allowed us to examine response biases between groups. This was particularly useful, given the 2:1 ratio of non-target: target trials used in Experiments 1 and 2, which gave rise to a probabilistic bias that a given trial would be a non-target. Finally ERPs were measured. ERP analysis has excellent temporal resolution, and can reveal underlying amplitude or latency differences which might help distinguish between contexts where dyslexic individuals are impaired, or not.

The behavioural results of the first experiment, that used visual letters as stimuli, showed that dyslexic participants are impaired for target trials, and were less likely to indicate accurately that a letter has occurred N items back. However, for non-target trials, dyslexic participants show comparable accuracy to non-dyslexic participants. However, when pictorial objects were used in Experiments 2 and 3, group did not interact with trial type, and dyslexic and control participants showed comparable accuracy for identifying targets and non-targets. Furthermore, in Experiment 1, there was a marginal between group difference in the criterion, whereby the trend suggested that dyslexic participants responded more conservatively, and were more likely to make a 'No' response. Critically, in Experiment 1 and 2, there was a 2:1 ratio of non-target: target trials. This means the probability of the current trial being a non-target was twice as much as it being a target.

Sensitivity to this imbalance might have driven the significant interaction between group * trial type. Thus, one possibility is the between group effect for target trials is a consequence of the dyslexic group being more sensitive to this probabilistic bias, and therefore responding more conservatively, as opposed to an intrinsic difficulty with phonological WM processing. In accordance with this, the d-prime measure was not significant between groups, which implies they were not impaired in visual-object

processing. However, given comparable criterion scores in experiment 2 and 3 it is likely that the phonological stimuli used in Experiment 1 resulted in dyslexic participants having a specific difficulty with phonological WM processing, and so were more inclined to rely on strategy. Consequently, accuracy for target identification was lower for this group in Experiment 1. This view is supported by the marginally significant between group difference for P300 amplitude in Experiment 1.

The P300 peak to peak analysis in both Experiments 1 and 2 demonstrated a main effect of WM load, as predicted. P300 peak-peak distance decreased progressively as WM load increased. This finding is consistent with previous experiments (e.g., Watter, Geffen & Geffen, 2001; Gevins, Smith, McEvoy, Yu, 1996; McEvoy, Smith, & Gevin, 1998). Watter, Geffen and Geffen (2001) interpret these findings as reflecting a reallocation of attention and processing capacity away from the process relative to which the P300 is generated. Thus, in the N-back task setting, a reduced P300 is taken to suggest a reallocation of attention and processing capacity away from matching evaluation of a new stimulus, to increased WM requirements. For experiment 1, there was a marginal between group difference in the P300 response, which might suggest that dyslexic individuals have a greater reallocation of attention and processing capacity away from the task, in response to increased difficulty with processing WM load. However, there was no N * group interaction, which might be because the task demands were already difficult in the 1-back condition, especially because of the fast stimulus presentation rate.

P300 latency analysis was also conducted for Experiment 1 and 2, revealing no effect of WM load upon latency. This might seem surprising given that P300 latency is typically affected by perceptual complexity and cognitive processing demands of a given task (Watter, Geffen, & Geffen, 2001). However, Watter, Geffen and Geffen (2001) suggest that an

absence of a latency effect is indicative of the dual task nature of the N-back paradigm. In an N-back task, participants must be able to search the contents of WM for a candidate N-back stimulus, and be able to perform a matching decision while maintaining and updating the content of WM. Watter, Geffen and Geffen (2001) argue that participants select the N-back position in WM in preparation for the upcoming trials, and therefore P300 latency should not differ across N-back tasks. However, if participants had been waiting for the presentation of the current stimulus li to begin their search and selection of the N-back stimulus, then latency should increase as WM load increases. Absence of latency effects is taken to support the former theory, and suggests the similarity of the matching subtask across WM loads. Moreover, latency effects did not differ between groups, suggesting that both dyslexic and non-dyslexic participants held the N-back stimuli online and in WM, in preparation for a comparison at the current trial. However, in Experiment 1, when letters were used as stimuli, more resource allocation was directed away from this matching task as WM load increased for dyslexic participants, as indexed by the reduced P300 in the dyslexic group.

Experiment limitations and future modifications

Despite providing valuable preliminary data on the nature of WM deficits in dyslexia, the experiments in this chapter bring to light a series of limitations, which are overcome in subsequent chapters of this thesis. In Experiments 1 and 2 there was a 2:1 ratio of non-target: target trials. Dyslexic individuals appeared to maximise their correct rejects (choose no), but, at a cost to the hit rate. In the signal detection framework, a larger noise distribution provides a greater probabilistic context for the current trial to be a non-target. Thus, if task demands are high (due to increasing N, or a general WM deficit), it makes strategic sense to rely on this probability. In the current context, increasing ones criterion is sensible, as it allows for the correct reject rate to be maximised. Thus, the probabilistic context might

scaffold WM functioning, and allow participants to rely on strategy. In the current experiments, as N increased, participants became more conservative. To ensure this probabilistic context did not affect visual object WM performance, Experiment 3 adopted a 1: 1 ratio between trial types, and did not find any effects of group on the criterion. Further experiments in this thesis adopt a non-target to target ratio of 1: 1, as an unbiased measure.

Furthermore, the reaction time analysis in experiments 2 and 3 demonstrated decreasing RTs with increasing N. The results from SDT for all these 3 experiments demonstrated that for the criterion analysis, there was a main effect of N, suggesting participants were more likely to respond conservatively as N increased. Relying on strategy could have promoted quicker response times for all participants. For further experiments we increase the display time and ISI. More relaxed task demands are less likely to encourage participants to be strategic. A short response time of 1s, of which 500ms was stimulus display, ensured the task was demanding enough to reveal any between group effects for visual-object WM. However, this also had implications for the ERP analysis, because accuracy was overall quite low. ERP analysis required correct responses only, which limited the number of trials in each individual subjects' ERP when accuracy was low. To help increase the signal to noise ratio, we used an average voltage around the peak, and averaged effects of N across 1 and 2-back (low WM load), and 3 and 4-back (high WM load). This increases the probability that the value analysed comes from signal, as opposed to a spike in the data. However, future experiments in this thesis that use the N-back task, present participants with at least 100 target trials (as opposed to 50 in the current chapter), and also use longer presentation times, and ISIs to increase overall accuracy, thus retaining higher numbers of trials for analysis.

While the current experiments suggest a specific phonological loop deficit in adults with developmental dyslexia, visual WM processing was examined for visual-object processing only. Experiments presented in chapter 4 examine visual spatial WM to assess the effect of WM load (Experiment 4) and manipulation during visual spatial WM (Experiment 5).

Chapter Summary

Chapter 3 presents 3 experiments, using the N-back task, in order to examine the extent to which individuals with dyslexia are impaired for verbal (Experiment 1), or visual-objects (Experiment 2 and 3) WM. It was hypothesized that if individuals with dyslexia have an impaired central executive, then they should show an impairment in both visual, as well as verbal material. All experiments applied behavioural (accuracy, and signal detection theory) and sensitive electrophysiological (ERPs) techniques, in order to assess WM functioning. The results of Experiment 1 showed an impaired hit rate for individuals with dyslexia, and a trend towards a reduced P300 response. However, this impairment was not seen in Experiments 2 and 3, when visual-object information was used. The findings indicate intact visual-object WM processing in dyslexia.

Chapter 4: Visual Spatial working memory

Chapter 2 outlined the controversy in the literature regarding the extent to which the WM impairment in developmental dyslexia is a specific phonological loop deficit, or an impairment in the central executive. To examine these effects in adult participants, the experiments presented in Chapter 3, compared WM processing for visual letters and visual-objects. These experiments used visual-object information that is static in space, but changing across time. However, the visual WM system can be dissociated into processes associated with visual-object, and visual-spatial processing. Thus, the experiments presented in the current chapter examine visual spatial WM processing, and the associated ERP components. Conclusions regarding impaired central executive dysfunction in dyslexia has been based on research demonstrating an impairment on complex WM tasks when controlling for simple visual span tasks (Smith-Spark & Fisk 2007; Wang & Gathercole, 2013) under the assumption that once simple span performance is controlled for, central executive deficits can be isolated. In the current chapter, the benefits of an experimental design which directly compares passive maintenance versus active processing is highlighted. To investigate central executive functioning in dyslexia further, the experiments in this chapter manipulate WM load (Experiment 4), and compare passive maintenance versus active manipulation processes (Experiment 5) within the same task paradigm.

Background: Examining visual spatial and central executive processing in developmental dyslexia

As stated in Chapter 2, previous research has debated the extent to which the WM deficit in developmental dyslexia is the result of a specific phonological loop deficit, or a domain general central executive impairment. Swanson and Ashbaker (2000) claimed that poor performance on verbal complex span tasks of children with reading impairments might reflect

deficits in the central executive. Given the domain general nature of the central executive, (Alloway, Gathercole & Pickering, (2006), Wang & Gathercole (2013) argue that a key prediction of this hypothesis is that children's memory difficulties should extend to non-verbal complex span tasks. Chapter 2 provided an overview of experiments (Smith Spark, Fisk, Fawcett, and Nicolson, 2003; Smith-Spark & Fisk, 2007; Menghini, Finzi, Carlesimo & Vicari, 2011; Wang & Gathercole, 2013), which have demonstrated a visual spatial WM deficit in dyslexia.

Smith-Spark and Fisk (2007), suggest that in contrast to the passive storage requirement of simple span tasks, complex WM span procedures involve simultaneous processing. These dynamic processing demands will draw upon the central executive, as well as the relevant slave system. To examine central executive processing, Smith-Spark and Fisk (2007) suggest controlling for simple span capacities when analyzing performance on complex visual spatial WM tasks. Thus, in Smith-Spark and Fisk (2007) an ANCOVA was used to assess verbal and visual-spatial WM, whereby the scores of a simple span task were entered as a covariate. The results indicated a significant group difference on verbal WM span measures when controlling for simple memory span. For spatial WM, significant group differences remained after controlling for Corsi block span. Furthermore, Wang and Gathercole (2013) adopted a similar analysis procedure, whereby STM performance (measured by simple span) was covaried out of verbal WM and visuospatial WM tasks, and found significant between group effects. In the current experiments, passive capacity processes and active central executive processes are manipulated within one task design, to directly compare processing demands created by each task in individuals with and without dyslexia.

Neuroimaging experiments have dissociated maintenance only from central executive (manipulation) components of WM processing. These studies (e.g., Rowe, Toni, Josephs, Frackowiak, Passingham, 2000) have shown that the ventrolateral prefrontal cortex (VLPFC) is necessary for maintaining information, while the dorsolateral prefrontal cortex (DLPFC) supports the executive processes involved in using these stored representations to accomplish a goal (i.e., manipulation in WM). The association between the DLPFC and executive processing has also been demonstrated in the verbal domain (D'Esposito, Postle, Ballard, & Lease, 1999).

Postle, Stern, Rosen, and Corkin (2000) demonstrated increased activity in the DLPFC during spatial temporal manipulation, compared to spatial temporal maintenance. In order to investigate this dissociation further, Glahn et al. (2002) conducted two experiments to assess areas active for passive versus executive functioning. In their first experiment, a spatial delayed response task (SDRT) was used, whereby participants were presented with a target array (encoding) of 1, 3, 5 or 7 items, followed by a probe array (retrieval) composed of a single item. Participants are required to state if the item occurred in the same location as in the target array. In their experiment 2, maintenance and manipulation in WM were compared, keeping WM load constant. In the maintenance alone condition, subjects were presented with a fixed (3) number of locations, and were asked to maintain these locations. In the maintenance plus manipulation condition, subjects were asked to flip the maintained representation over a horizontal line. The authors demonstrated the VLPFC was active for both maintenance and maintenance plus manipulation, while the Superior Frontal Sulcus (SFS) activity was associated with passive maintenance only. The DLPFC was active for the task involving manipulation, but also activated in experiment 1, for conditions with higher WM loads (3 & 7 spatial locations). The activation of this region was interpreted as higher loads requiring greater organizational strategies. Thus, it was argued that the DLPFC was

activated to support strategic or selection processing. The behavior results demonstrated that accuracy decreased as load increased, while in experiment 2, accuracy decreased and reaction times increased for the maintenance plus manipulation condition, reflecting the need for greater central executive involvement. Thus the researchers argued for a double dissociation in the prefrontal cortex, whereby the DLPFC is involved in central executive, or active processes of WM.

Application of these two task paradigms to the current context allows central executive processing to be dissociated from passive maintenance within the same experimental design. The application of this task paradigm to the current question, offers one main advantage over the N-back task, which has been used in chapter 3. In N-back tasks, WM load is parametrically increased, and central executive and maintenance components of WM are confounded with manipulation. As WM load increases, maintenance increases, as well as increased complexity of central executive processing. As set size increases, temporal order becomes more important, as does WM updating. However, the SDRT allows for passive versus more active processing in WM to be directly compared. Similar tasks have been applied to individuals with Schizophrenia, in order to examine the extent to which such individuals' WM difficulties can be explained by central executive dysfunction (E.g., Kim, Glahn, Nuechterlein, and Cannon, 2004).

In the WM literature, maintenance processes, and manipulation, or central executive related processes are difficult to dissociate (Glahn et al., 2002). As stated previously, in the neurodevelopmental literature examining WM and dyslexia, the influence of central executive functioning has been examined by comparing performance on complex versus simple span tasks, under the assumption that the former places more demands on central executive processing (Wang & Gathercole, 2013; Smith Spark et al, 2003; Smith-Spark & Fisk, 2007). Analysis of covariance does not easily allow for central executive versus passive

maintenance processes to be dissociated. Work in the current chapter uses, Glahn et al. (2002)'s visual spatial delay paradigms to more readily dissociate passive versus active central executive processes.

In this chapter, two experiments are presented. Experiment 4 manipulates WM load, and WM capacity is compared between participants with and without dyslexia. If dyslexic participants are impaired in central executive processing, one might expect a load * group interaction in accuracy (hit rate, and d'), and ERP measures, whereby higher loads which require greater organizational strategies, are more difficult for individuals with dyslexia. This should result in a reduced P300, if individuals with dyslexia have reduced resources to deal with more difficult WM demands. Furthermore, Experiment 5 allows for passive maintenance and central executive processes to be dissociated in one experiment. If dyslexic individuals are impaired in central executive function, then one would expect a task * group interaction, whereby individuals with dyslexia show a greater impairment when required to manipulate visual spatial information. This should give rise to impaired accuracy (hit rate, and d'), alongside a reduced P300. This reduced P300 might arise due to lack of resources, or impaired updating in WM.

Visual-spatial WM and ERP responses

Early ERP studies of WM processing focused on the P300 response, which reduces as WM load increases (e.g., Watter, Geffen & Geffen, 2001; Polish, 2007). This effect was also demonstrated in Chapter 3, Experiments 1 and 2. Research in the WM domain has demonstrated that the P300 amplitude indexes the updating of WM (e.g., Courchesne, Hillyard, & Galambos, 1975). In the N-back task, the P300 amplitude has been shown to be modulated by the availability of resources available for a given task (e.g., Watter, Geffen and Geffen, 2001). In addition, the latency of the P300 reflects stimulus evaluation time (Johnson

& Donchin, 1980). Further research examining the P300, in the context of WM was provided in Chapters 1 and 3.

The SDRT reported here has a similar structure to the Sternberg task, where stimuli are encoded, maintained, and then at retrieval a probe is displayed which requires participants to decide whether or not an item was present at encoding. ERP versions of the Sternberg task (Sternberg, 1966), have typically investigated the P300. ERP responses to the probe, which is presented at the retrieval phase, reveal a large sustained parietal positivity, which increases in latency as memory set size increases (Pelosi, Holly, Slade, Hayward, Barrett, & Blumhardt, 1992; Pelosi, Hayward, & Blumhardt, 1995), reflecting longer stimulus evaluation time (Johnson & Donchin, 1980; Verleger, 1997) in response to increased WM demands. At the retrieval phase, the P300b amplitude is said to index the updating of WM, (e.g., Courchesne, Hillyard, & Galambos, 1975; Friedman, Cycowicz, & Gaeta, 2001; Goldstein, Spencer, & Donchin, 2002; Squires, Squires, & Hillyard, 1975), but has also been used as a measure of resource allocation (e.g., Watter, Geffen, & Geffen, 2001). Thus, analysis of the P300 during the probe, provides detailed information about the time-course of WM updating in individuals with dyslexia.

Furthermore, the P300 was also found to be evoked during the 'study phase' where items are encoding into WM. The amplitude of the P300 at the study phase has been associated with successful retrieval (e.g., Chao & Knight, 1996; Kotchoubey et al., 1996), which is congruent with the suggestion that the P300 reflects rich memory representations at encoding (see Yonelinas, 2002 for a review). These results demonstrate that measuring the ERP components associated with the SDRT allow us to interrogate WM efficiency at two important stages of processing: encoding and retrieval.

Therefore, the P300 (amplitude and latency) are examined in non-dyslexic and dyslexic individuals. The P300 is used as a measure of processing and capacity limits available to the matching task. Further, the latency of the P300 reflects stimulus evaluation time during the matching process. These effects are examined at the midline electrodes (Fz, Cz and Pz), as in experiments 1 and 2. Thus, from previous literature demonstrating that the P300 occurs maximally at Cz and Pz (e.g., Watters, Geffen, & Geffen, 2001) and Experiment 1, we have an a-priori prediction that group differences in the P300 should be found in more posterior electrodes, such as Cz or Pz. Furthermore, the effect of hemisphere is also examined, which is based upon research which suggests the P300 occurs maximally in the right hemisphere during visual spatial WM tasks (e.g., Van der Lubbe, Schölvinck, Kenemans, & Postma, 2006; Van der Ham, Strien, Oleksiak, Weze, & Postma, 2010). Thus, in the current experiment, the N200 and P300 are examined in four regions (anterior left and right, and posterior left and right), as indexes of early visual attention, and resource allocation respectively.

Therefore, the aims of the experiments in this chapter are to examine visual spatial WM processing in dyslexia. EEG is recorded at both encoding, and retrieval, in order to assess the underlying electrophysiological components associated with maintenance versus manipulation processes in visual spatial WM. To my knowledge, this is the first time that the electrophysiological correlates of a visual spatial WM task have been assessed in dyslexia. Critically, in an ERP setting, there has been no attempt to dissociate the ERP components associated with passive maintenance and active manipulation in dyslexic and non-dyslexic individuals. Measuring ERPs is also especially insightful because it allows us to examine WM processing at encoding and retrieval. As previous research examining visual spatial WM processing in dyslexia has not used ERP measures, very little is known about visual-spatial

WM encoding in dyslexia. Thus, ERP measures allow us to interrogate any between group effects which might emerge at either processing stage. The aims and rationale of the experiments conducted in this chapter, are reiterated and summarized below.

Rationale

1) The aims of the current chapter are to distinguish the extent to which individuals with developmental dyslexia are impaired in visual-spatial WM processing, therefore suggesting a central executive impairment in developmental dyslexia. Findings within this domain have been largely inconsistent to date, and the majority of work has focused on children with dyslexia, with very little known about visual-spatial, and central executive processing in adults with dyslexia. Thus, a predominant aim of this work is to examine visual spatial WM processing in adults.

2) Typically, central executive processing in dyslexia has been examined by controlling for simple span measures during complex span tasks. Here, two experiments are presented, which examine central executive processing within the same task design. In Experiment 4, load is manipulated, with the intention that higher WM load conditions place greater demands on central executive processes. This assumption is based upon the findings of Ghan et al., (2002) who has demonstrated significant DLPFC activity for tasks involving the maintaining of higher WM loads (3 & 7 spatial locations). Furthermore, central executive processing is measured more directly in Experiment 5, whereby conditions involving WM maintenance versus manipulation are directly compared.

3) The third aim of this work is to examine the ERP components associated with visual spatial WM tasks. EEG has excellent temporal resolution and is able to provide information about which cognitive processes might be impaired, or delayed in time. Recording EEG during the SDRT allows WM processing to be examined during encoding of

the stimulus set, and retrieval. Examining WM processing at encoding and retrieval has not yet been explored in the literature examining WM and dyslexia.

Finally, the ERP components associated with maintenance versus manipulation have not been explored in the research, in either non-dyslexic or dyslexic participants.

Experiment 4

For Experiment 4, the effect of increasing visual spatial WM load is examined using a simple short-term memory span task, using the SDRT (Ghan et al., 2002).

Hypothesis

1. Increasing WM load will reduce accuracy and d' values for all participants. The P300b peak-peak amplitude will reduce as WM load increases.
2. If, as suggested by Smith-Spark and Fisk (2007), Menghini, Finzi, Carlesimo, and Vicari, (2011), and Wang and Gathercole (2013), WM impairments in dyslexia are a function of central executive demands, then lower accuracy and d' scores would be expected for dyslexic participants, compared to non-dyslexic participants as load increases. This will manifest in a reduced P300 amplitude, and a longer P300 latency.
3. However, if dyslexic adults do not suffer a central executive deficit, then between group differences will not emerge, and behavioural and ERP differences will not be found[8].

[8] Note: Analysis of the N2 are reported in a footnote. Based upon previous research (e.g., Riby & Orme, 2013, it was hypothesised that the N2 would become more negative as WM load increased.

Method

Participants

35 participants were originally tested, however, one was removed from the analysis as they withdrew half way through the EEG recording procedure. Two were not analysed because they pressed the same response key throughout the experiment. Therefore, participants were 32 adults, of whom, 16 had normal reading skills, and 16 had a confirmed diagnosis of dyslexia from an Educational Psychologist. 18 participants were female, and 14 were male. Participants were matched on age, $F(1, 30) = .50$, $p = .48$, with a mean age of 20.68 years for non-dyslexics, and 20.12 years for dyslexics. Participants took part for course credits, or a small monetary payment. Alongside a confirmed diagnosis, each participant took part in the York Adult dyslexia assessment revised (YAA-R; Warmington, Stothard & Snowling, 2013). The assessment battery consists of tests of reading, spelling, writing, and phonological skills. To assess verbal (vocabulary) and non-verbal IQ (picture arrangement, mental arithmetic, and block design) assessments were administered from the WAIS-III (Wechsler, 1994). For a full description of the assessment measures, see Chapter 3, Experiment 3. Results from this assessment are documented in Table 13.

Table 13: *Behavioural Assessment results for Experiment 4.*

	Non-dyslexic	Dyslexic	Difference
Dyslexia Assessment			
Passage reading errors	3.88 (2.84)	9.19 (7.61)	$F(1, 30)=5.70^{*}$
Reading rate (words/min)	21.68 (4.09)	17.99 (4.09)	$F(1, 30)=6.69^{*}$
Reading comprehension	7.56 (1.20)	8.69 (1.99)	$F(1, 30)=3.73^{+}$
Timed Précis: Content score	11.81 (3.01)	10.81 (3.31)	$F(1, 30)=.79$
Timed Précis: Spelling errors	1.50 (2.75)	4.87 (3.69)	$F(1, 30)=8.60^{**}$
Writing speed	31.90 (4.02)	27.36 (3.91)	$F(1, 30)=9.79^{**}$
Spoonerisms Accuracy	22.50 (3.54)	16.25 (7.00)	$F(1, 30)=10.16^{**}$
Spoonerisms correct seconds/item	1.83 (1.97)	3.16 (2.40)	$F(1, 30)=1.49$
RAN Digits Total time (sec)	14.69 (2.60)	25.98 (16.39)	$F(1, 30)=6.94^{**}$
RAN Digits items/ sec	3.51 (.65)	2.40 (.90)	$F(1,30)= 5.14^{***}$
RAN Objects Total time	26.71 (5.86)	36.48 (16.45)	$F(1, 30)=4.72^{*}$
RAN Objects items/ sec	1.93 (.35)	1.54 (.43)	$F(1, 30)=7.78^{**}$
WRAT-III Spelling (raw score)	52.75 (2.93)	48.31 (3.42)	$F(1,30)=15.52^{***}$
WRAT-III Reading (raw score)	44.53 (2.69)	39.87 (3.93)	$F(1,29)=15.06^{**}$
Processing speed: Digit symbol coding items/minute	39.52 (6.75)	37.39 (8.65)	$F(1, 30)=.60$
Digits Forward	12.06 (2.00)	10.31 (2.39)	$F(1,30)=5.24^{*}$
Digits Backward	7.88 (2.72)	6.00 (1.89)	$F(1, 30)=5.09^{*}$
Verbal IQ (WAIS-III)			
Vocabulary	41.31 (7.96)	39.00 (6.64)	$F(1, 30)=.77$
Arithmetic	14.62 (3.46)	13.56 (4.18)	$F(1, 30)=.67$
Non- Verbal IQ (WAIS-III)			
Block Design	53.43 (9.78)	53.43 (11.93)	$F(1, 30)=.0001$
Picture Arrangement	12.87 (2.62)	16.19 (3.06)	$F(1, 30)=10.78^{**}$

Note: $+ p<.10$; $*p<.05$; $** p<.01$; $*** p<.001$. *Standard deviations are reported in parentheses.*

Design

For the behavioural analysis, a 2 x (2 x 4) mixed design was used. The between subjects

variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial type (target vs. non-target) and WM load (1 vs. 3 vs. 5 vs. 7). The behavioural dependent variables were accuracy and reaction times at each level of N. When signal detection theory was implemented a 2 x (4) design was used with group and WM load as independent variables and d-prime and Criterion as dependent variables.

Materials and Procedure.

Participants sat approximately 80cm away from the screen. The procedure and materials were a direct replication of the spatial delayed response task (SDRT; as in Glahn et al., 2002). The task consisted of 3 stages: encoding, memorisation and retrieval. Each trial started with a fixation cross, which remained on screen for 500ms. For encoding, each trial involved the presentation of a target array, composed of 1, 3, 4 or 7 blue circle(s) which remained on screen for 2000ms. These were positioned pseudo-randomly around a fixation cross. After the target presentation, there was a fixed delay, marking the memorisation stage. This lasted for 3000ms. During the retrieval stage, they were shown a single yellow circle (probe), and are required to respond 'yes' if the probe appeared in the same position as one of the target blue dots, or 'no' if it occurred in a different location. The probe was on screen for 3000ms, or until the participants made a response. This participant's response (yes or no), and reaction times are recorded for each trial. Participants were asked to fixate on the fixation cross during stimulus presentation, and were encouraged to make as few eye blinks, and movements as possible during the experiment, to maintain the quality of the EEG recording. A detailed portrayal of the experiment procedure can be seen in Figure 13.

In the main experiment there were 4 blocks with 80 experimental trials in each block. Out of the 80 trials, 40 were target trials, and 40 were non-target trials, creating a 1: 1 ratio of non-target: target trials. WM load was manipulated between blocks, whereby the first block

represented the lowest WM load condition, and the final block represents the highest WM load condition (as in Glahn et al., 2002). Total testing time took approximately 50 minutes per participant.

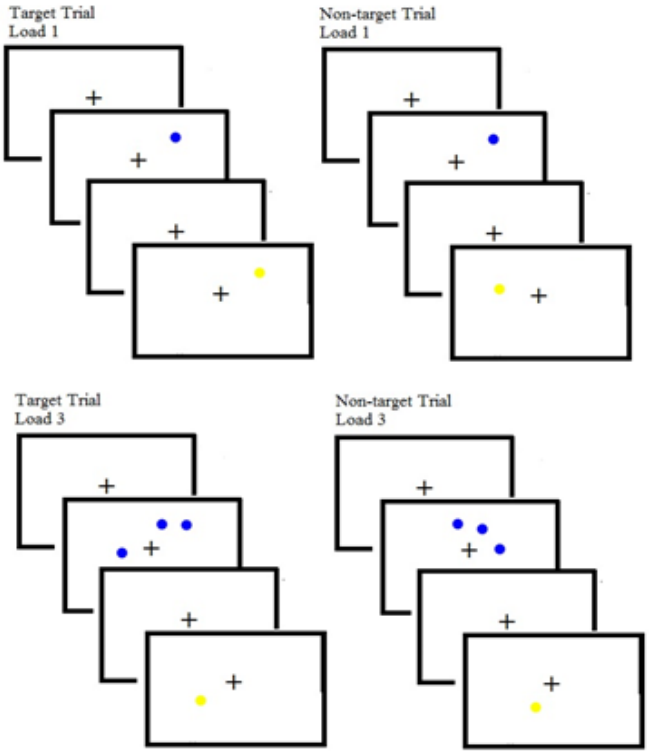


Figure 13: Experimental procedure for Experiment 4.

Depicted is a target and non-target (top) trial with a WM load of 1 and a target and non-target (bottom) trial with a WM load of 3.

EEG recording and pre-processing

EEG was continuously recorded with an average reference from 64 Ag-AgCl electrodes. A visual display of the electrodes used can be found in Figure 14. Furthermore, TP9 and TP1 were used to record activity from the mastoids for off-line re-referencing. Two electrodes were used to monitor eye movements, one placed beside the left eye (HEOG), and another under the right eye (VEOG). An electrolyte gel was used at each electrode site to decrease impedance. Electrode impedances did not exceed 25 kΩ.

The EEG signal was amplified using a Quickamp 72 amplifier, and recorded using Brain Vision Recording software (version 2). The data was continuously recorded at a sample rate of 1000Hz, and later down sampled to 500Hz. A high pass filter of 0.1 Hz and a low pass filter of 35Hz was used, along with a notch filter of 50Hz. Off-line, recordings were re-referenced to the linked mastoid electrodes. EEG data was corrected for vertical and horizontal eye movements using the BrainVision Analyzer 2 software which implemented the ocular correlation with ICA method for artifact rejection method. EEG recordings were then segmented into epochs of 1000ms according to stimulus onset, aligned to a 100ms baseline. Finally each epoch was screened for artifacts using a semiautomatic artifact rejection method. EEG recordings were time-locked to the onset of the initial presentation screen (target array), and the response screen (probe).

Next, date was exported into Matlab, where EEGlab was used to generate average event related potentials for each participant and condition at electrodes sites Fz, Cz and Pz. Furthermore, to examine hemisphere effects, electrodes were pooled into 4 regions: Left anterior, left posterior, right anterior, and right posterior (see Figure 14). In order to statistically analyse these waveforms, a window was defined between 300-500ms, and the greatest average 50ms of activity was taken. This method was used, instead of the peak-peak, because a Negative Slow Wave (NSW) has been demonstrated in the encoding phase after 500ms (Riby & Orme, 2013) and thus it becomes unclear the extent to which the negative component is part of the P300, NSW, or a combination of the two. However, given previous research has identified that statistical results are identical between the peak, and peak-peak method, this allowed us to compare the current results to ERP studies, in this thesis which use the peak-peak method.

Results

Behavioural effects

Mean accuracy scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each WM load condition. The results are summarised in Table 14.

Table 14: *Mean hit and correct reject scores for Experiment 4*

Trial	**Group**	**WM load**			
		1	3	5	7
Target	Non-dyslexic	.92 (.05)	.82 (.10)	.85(.11)	.78 (.16)
	Dyslexic	.90 (.08)	.80 (.14)	.80 (.11)	.69 (.21)
Non-target	Non-dyslexic	.97 (.04)	.92 (.06)	.85 (.13)	.84 (.12)
	Dyslexic	.96 (.96)	.90 (.08)	.83 (.12)	.85 (.07)

Note: Standard deviations are reported in parentheses.

Results were analysed using a mixed three way GLM analysis, with WM load (1, 3, 5, and 7), and trial type (target and non-target) as within subjects variables, and group (dyslexic and non-dyslexic) as the between subjects variable. The Huynh-Feldt adjustment (Huynh & Feldt, 1970) was employed as appropriate. Results revealed a main effect of WM load, $F(2.31, 69.23) = 30.98$, $p<.001$, $\eta p^2 = .51$, where accuracy reduced as WM load increased, and a main effect of trial, $F(1, 30) = 14.68$, $p<.001$ $\eta p^2 = .33$, where there was higher accuracy for non-target trials (correct rejects). There was a significant interaction between trial * load, $F(2.68, 80.40) = 5.22$, $p=.003$, $\eta p^2 = .15$, in that there is a significant difference between conditions 5 and 7 for target trials $t(31) = p$ 3.93, p<.001, but not for non-target trials $t(31) = -.53$, $p=.60$. All other effects were not significant [Fs<1.40][9].

[9] To assess RTs, a three way mixed ANOVA was conducted, maintaining the same independent variables from the accuracy analysis. The analysis revealed a main effect of load, $F(2.25, 75.80)= 5.51$, $p=.003$, $\eta p2 = .05$, whereby load increased reaction times from 705ms, 758ms, 786ms, to 797ms. There was a significant trial * load interaction, whereby RTs increase for target trials more substantially between the 1 and 3 WM load conditions. The mean reaction times for target trials were

Signal Detection Theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants, at each level of WM load (1, 3, 5 and 7). The results are presented in Table 15.

Table 15: *Mean Signal Detection Theory parameters for Experiment 4.*

Parameter	**Group**	**N-back**			
		1	3	5	7
d'	Non-dyslexic	3.32 (.67)	2.41 (.77)	2.29(1.00)	1.96 (.97)
	Dyslexic	3.07 (.42)	2.23 (.52)	1.94 (.88)	1.68 (.73)
Criterion	Non-dyslexic	.24 (.22)	.25 (.22)	.04 (.18)	.13 (.27)
	Dyslexic	.20 (.36)	.24 (.38)	.06 (.28)	.23 (.39)

Note: Standard deviations are reported in parentheses.

A mixed GLM analysis was carried out on d-prime values, with load and group as the independent variables. This analysis revealed a main effect of WM load, $F(2.47, 74.37) = 37.96$, $p < .001$, $\eta p^2 = .56$, with decreasing d-prime values as N increases. The effect of group, and interaction between N * group were not significant, [F=1.47]. A mixed GLM analysis was carried out on criterion scores. This revealed a main effect of load $F(3, 90) = .57$, p=.005, $\eta p^2 = .13$, whereby the criterion increased as N increased, from a load of 3 onwards (there was no significant difference between the WM load conditions 1 and 3, $t(31) = -.41$, p=.68). The effect of group, and interaction between WM load and group were not significant [Fs<.57].

Electrophysiological Analysis

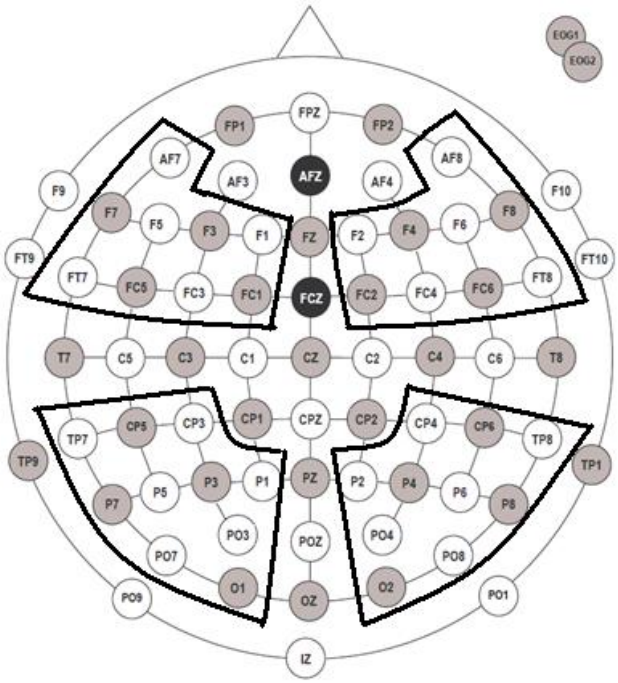
To remain consistent with previous work in this thesis, the P300 analysis of the positive peak was defined for each individual as the maximum 50ms average to occur between 300-500ms,

693.19ms, 803ms, 791ms, 788ms. For Non-target trials the RTs were 718ms, 713ms, 781ms, and 806ms.

while for the N2 a window of 160-260ms (as in Riby & Orme, 2013) was defined, and the largest negative 30ms average was calculated. To allow for the analysis of both hemisphere (left and right), and region (anterior and posterior), four distinct regions were created: right anterior, right posterior, left anterior, and left posterior. The electrodes included within each region are displayed in Figure 14.

Left Anterior

Right Anterior



Left Posterior

Right Posterior

Figure 14: Electrode placement in the 64 electrode Acticap system, displaying electrode areas of interest (AOI).

In accordance with previous experiments in this thesis, electrodes in the midline were examined (Fz, Cz, and Pz). Separate analyses were conducted at encoding (Target array) and retrieval (Probe array). For lateral electrodes, a 4 way GLM analysis was conducted, with Hemisphere (right, left), Region (anterior, posterior), load (1, 3, 5, 7), and group (dyslexic, non-dyslexic) as independent variables. When the analysis was conducted at the midline, a 3

way GLM analysis was conducted, with electrode (Fz, Cz, and Pz), load (1, 3, 5, 7), and group (dyslexic, non-dyslexic) as independent variables.

Encoding

Lateral electrodes

Grand average plots are displayed in Figure 15, for non-dyslexic and dyslexic participants at each region of analysis. Analysis of the P300 amplitude revealed a significant main effect of hemisphere, $F(1, 30) = 11.38, =.002, \eta p^2 = .26$, with greater positivity in the right hemisphere (2.27 uV) compared to the left hemisphere (1.83 uV). There was also a main effect of region, $F(1, 30) = 31.08\ p<.001, \eta p^2 = .51$, whereby the P300 occurred maximally in posterior regions (2.74 uV, compared to 1.36 uV in anterior regions). Hemisphere and region also interacted $F(1, 30) = 72.80, p<.001, \eta p^2 = .71$, whereby the P300 occurred maximally in posterior regions on the right. All other effects did not reach significance, Fs<2.48.

The latency of the P300 component was also analysed. The analysis revealed a significant main effect of region, $F(1, 30) = 22.86, p<.001, \eta p^2 = .43$, whereby the P300 occurred earlier in posterior (359 ms) compared to the anterior regions (386 ms). There was a significant interaction between hemisphere * WM load, $F(1, 30) = 4.62\ p=.005, \eta p^2 = .13$, whereby the P300 occurred later in the left hemisphere as WM load increased. However, in the right hemisphere, mean latencies did not differ as WM load increased. There was also a significant region * WM load interaction, $F(2.45, 73.56) = 72.80, p=.001, \eta p^2 = .19$, whereby the P300 showed a clear effect of being modulated by WM load (i.e. it occurred later as load increased) in the anterior region (with latency values changing from 371ms to 286ms, 289ms, and 398ms as N increased). However, in posterior regions WM load did not show the typical effect of increasing the P300 latency, with values changing from 373ms, 359ms, 355ms, and 351ms. All other effects did not reach significance [Fs<1.81].

Midline Electrodes

Analysis of the P300 amplitude at midline electrodes revealed that the P300 occurred maximally at Pz $F(1.55, 46.57)= .56.86$ $p=.004$, $\eta p^2 = .03$, with the P300 amplitude increasing from a mean of 1.95 uV, 2.07 uV and 4.36 uV from Fz, Cz to Pz, demonstrating a posterior P3. All other effects did not reach significance, Fs<1.70.

Analysis of the P300 latency revealed a main effect of electrode, $F(1.55, 46.57) = 11.62$, $p<.001$, $\eta p^2 = .28$, whereby the P300 peaked earlier at Pz, then Cz and Fz. The analysis also revealed a marginally significant main effect of WM load, $F(2.41, 72.39) = 2.71$, $p=.05$, $\eta p^2 = .08$, whereby WM load increased the P300 latency. There was also an electrode by WM load interaction, $F(1.55, 46.57) = 11.62$, $p<.001$, $\eta p^2 = .28$, whereby WM load increased the latency of the P300 at Fz and at Cz, however at Pz, WM load did not increase the latency of the P300. Interestingly, the effect of WM load interacted with group, whereby non-dyslexics showed a marginal main effect of WM load, load, $F(3, 45) =2.33$, $p=.86$, $\eta p^2 = .14$, but did not show a typical pattern of increased latency as WM load increased. Their values change from 365, 385, 375, to 369ms. However, for dyslexic individuals, there was a significant effect of WM load upon the P300 latency, $F(3, 45) =3.66$, $p=.02$, $\eta p^2 = .20$, whereby, latency increased as load increased, 366ms, 356ms, 374ms, to 390ms. All other effects were not significant, Fs<.2.04. ERP plots for midline electrodes can be found in Appendix D.

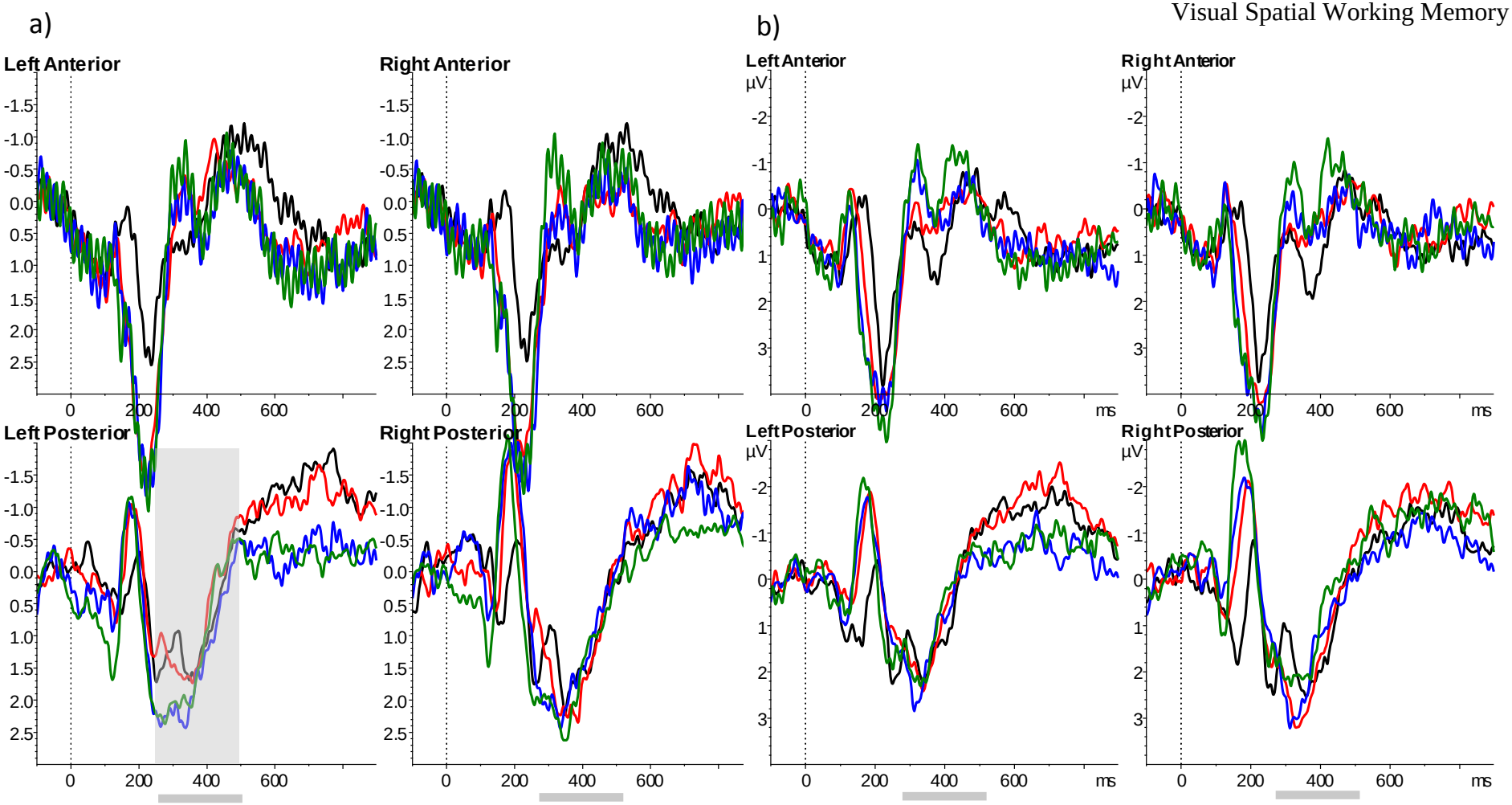


Figure 15: Grand average ERP waves for Experiment 4, at encoding.

a) Non-dyslexic individuals, at WM load conditions of **1 (black),** 3 (red), 5 (blue) and 7 (green). b) Dyslexic individuals, at WM load conditions of **1 (black),** 3 (red), 5 (blue) and 7 (green). The grey highlighted region donates the P300 time window.

Retrieval

Lateral Electrodes

Grand average plots are displayed in Figure 16. For the P300 amplitude, the results revealed a significant main effect of hemisphere, $F(1, 30) = 8.86$, $=.006$, $\eta p^2 = .23$, with greater positivity in the right hemisphere (2.51 uV) compared to the left hemisphere (2.08 uV). There was also a main effect of region, $F(1, 30) = 62.34$ $p<.001$, $\eta p^2 = .68$, whereby the P300 occurred maximally in posterior regions (3.39, compared to 1.29 in anterior regions). There was also a marginally significant interaction between hemisphere and region, $F(1, 30) = 2.47$, $p=.067$, $\eta p^2 = .08$, whereby the P300 occurred maximally at anterior regions on the right, consistent with the expected topography of Visual Spatial WM processing. The interaction between hemisphere * load * group showed a trend, $F(1, 30) = 2.18$, $p=.096$, $\eta p^2 = .07$, whereby for dyslexic participants in the right hemisphere increasing WM load decreased the P300, whereas non-dyslexic participants in the right hemisphere, did not show a clear pattern.

Analysis of P300 latency revealed a significant main effect of hemisphere, $F(1, 30) = 11.33$, $p=.002$, $\eta p^2 = .27$, with an overall later latency on the right (382ms) compared to the left hemisphere (371ms). This effect also interacted with group, $F(1, 30) = 9.91$, $p=.004$, $\eta p^2 = .25$, whereby the later latency in the right hemisphere vs. left hemisphere was present for non-dyslexic participants (369 ms vs. 390), but not for dyslexic participants (373ms vs. left 373ms). There was a significant main effect of WM load, $F(3, 90) = 5.26$, $p=.002$, $\eta p^2 = .15$, whereby the P300 peaked later as load increased. The load effect also interacted with region, $F(3, 30) = 4.98$, $p=.005$, $\eta p^2 = .14$, whereby the P300 showed the typical effect of occurring later by load, in the posterior regions, but not in anterior regions. There was also a significant hemisphere * region * load interaction, $F(3, 90) = 3.51$, $p=.018$, $\eta p^2 = .11$, whereby the largest effect of load emerged in the right posterior region.

Midline electrodes

Analysis of the P300 amplitude revealed that the component occurred maximally at Pz, $F(1.39, 41.68) = .79.77$ $p<.001$, $\eta p^2 = .73$, with the P300 amplitude increasing from Fz, to Cz to Pz (1.62 uV, 2.70 uV, 4.90 uV). There was also a significant electrode by load interaction, $F(1.39, 41.68) = 79.77$ $p<.001$, $\eta p^2 = .73$, where increasing WM load decreased the amplitude of the P300 at Pz. All other effects did not reach significance, Fs<1.70.

Analysis of the P300 latency showed a marginal main effect of electrode, $F(1.67, 50.10) = .2.64$ $p=.09$, $\eta p^2 = .08$, whereby the P300 latency became longer towards the back of the head. There was also a marginal interaction between electrode * group $F(1.67, 2, 41) = 3.06$ $p=.09$, $\eta p^2 = .01$, whereby, the latency is 372ms, 371ms and 393ms for non-dyslexic from electrodes Fz, Cz and Pz. For dyslexic participants, the corresponding mean latency values are 373ms, 367ms and 369ms. There was also a marginal effect of load, $F(2.41, 72.39) = 2.71$, $p=.06$, $\eta p^2 = .08$, whereby generally the latency increased as WM load increased from 370ms, 369ms, 374ms to 383ms. The effect of load also interacted with electrode, with a significant interaction, $F(6, 180) = .2.72$ $p=.12$, $\eta p^2 = .08$, whereby the mean values at Cz did not change as load increased, while at Pz WM load increased the latency[10]. ERP plots for midline electrodes can be found in Appendix D.

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[10] At encoding the N2 became more negative as WM load increased in the frontal region, $F(3, 90) = 4.80$, $p=.004$, $\eta p^2 = .14$, however, there were no between group effects observed in the N2 component. At retrieval, there were subtle topographic differences between groups. There was a marginally significant region by group interaction, $F(1, 30) = 4.14$, $p=.051$, $\eta p^2 = .1$, whereby the dyslexic group showed a more negative N2 in anterior regions compared to non-dyslexics (with means or -1.32 uV for dyslexic participants versus to -.59 uV for dyslexic participants), and more positive in posterior regions (1.25 uV for dyslexic participants compared to .85 uV for non-dyslexic participants) for non-dyslexics. However, analysis at the anterior, and posterior region separately, did not reveal any between group differences. Thus, these subtle topographic differences that emerge between groups are not interpreted.

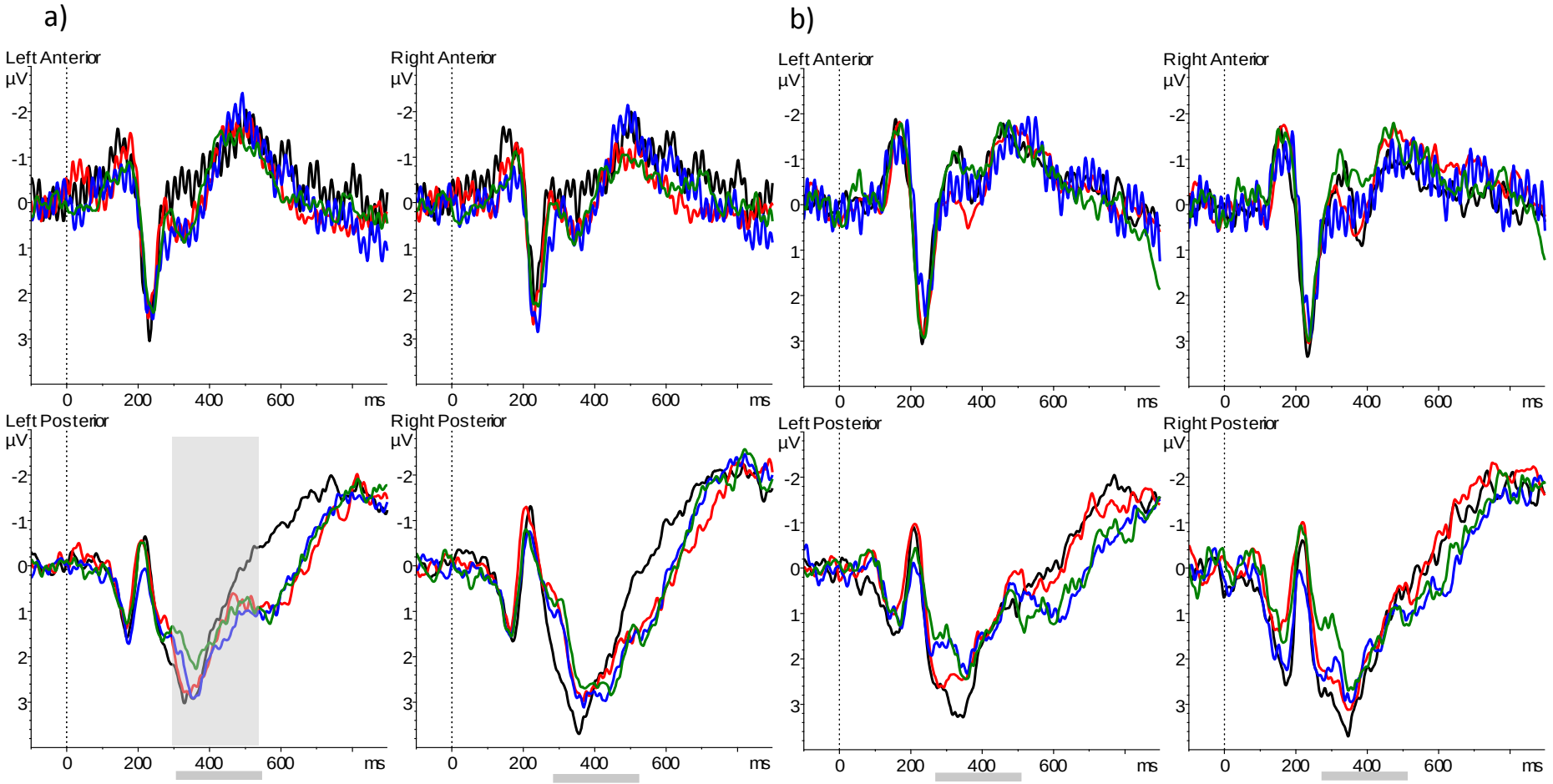

Figure 16: Grand average ERP waves for Experiment 4, at retrieval.

a) Grand average ERP waves at retrieval for non-dyslexic individuals, at WM load conditions of **1 (black)**, 3 (red), 5 (blue) and 7 (green). b) Grand average ERP waves for dyslexic individuals, at WM load conditions of **1 (black)**, 3 (red), 5 (blue) and 7 (green).

Experiment 4 Summary

Overall, the results of Experiment 4 suggest that dyslexic participants are not impaired in visual-spatial WM processing. Behaviourally, dyslexic participants performed at a comparable level to controls at each WM level. This interpretation was also demonstrated in the signal detection theory analysis, whereby dyslexic participants had comparable d-prime scores, suggesting there was no between group differences for distinguishing signal from noise. Furthermore, dyslexic participant's comparable accuracy could not be explained by strategic responses, as suggested by comparable criterion scores. The alternative hypothesis suggested that if individuals with developmental dyslexia suffered central executive impairments, then impairment would be more likely to manifest at higher WM loads, as previous research (Ghan et al., 2002) has suggested that higher WM loads recruit neural resources associated with WM manipulation and executive processing. Furthermore, analysis of the N2 and P300 wave did not reveal any between group differences in terms of amplitude or latency, despite some differences in topography. The results are indicative of unimpaired visual spatial, and central executive WM processing in dyslexic adults.

While the current task allowed us to tap into any interaction effects which might emerge as visual-spatial WM load increased, and the corresponding ERP components associated with this, the task was a passive storage task. It requires participants to hold information online, and then make a matching judgment. The extent to which Experiment 4 is a WM task per se can therefore be questioned. Central executive processing differences might emerge between groups in tasks involving the simultaneous processing of information. In Experiment 5, a WM paradigm is presented which dissociates passive versus active WM processing. This allows us to effectively test the hypothesis that individuals with dyslexia will

be disproportionately impaired in conditions that tax central executive components of WM, as opposed to conditions that require maintenance only.

Experiment 5

Experiment 5 aims to extent experiment 4 by using a task paradigm designed to dissociate passive versus active central executive processing in WM. The following experiment is based upon Glahn et al. (2002b). The authors developed a novel paradigm, to contrast the neurological processes involved with maintenance only versus maintenance plus manipulation of spatial information. In the maintenance alone condition, subjects were presented with three objects in different spatial locations above a horizontal line, and after a delay, the participant was required to state if 3 probe dots occurred in the same position as the target array. In the maintenance plus manipulation condition, participants were required to flip the target presentation across the central horizontal line, and after a fixed delay state whether or not a probe display matched their mentally flipped representation. While simply maintaining information in WM will recruit central executive related processes, conditions requiring manipulation of working memory, tax central executive function to a greater extent. Thus, the paradigm allows us to dissociate neural processes associated with passive versus active WM processing. Here, the paradigm is applied in order to examine central executive processing difficulties in developmental dyslexia. This allows us to effectively test the hypothesis that participants with dyslexia will be disproportionately impaired in the conditions that tax the central executive component of WM, relative to the conditions that tax only the maintenance component of WM (Experiment 4).

Hypotheses

1. The maintenance plus manipulation condition will result in reduced accuracy and d' values for all participants. Following the predictions of the context updating model, manipulating information in WM might result in a larger P300.
2. If, as suggested by Smith-Spark and Fisk (2007), Menghini, Finzi, Carlesimo, & Vicari, (2011), and Wang and Gathercole (2013), WM impairments in dyslexia are a function of central executive demands, then lower accuracy and d' scores would be expected for dyslexic participants in conditions requiring manipulation. Following Experiment 5, it is not expected that individuals with dyslexia will be impaired in the maintenance only condition.
3. If individuals with dyslexia have impaired central executive functioning, this will manifest as a reduced P300 amplitude, and longer P300 latency for dyslexic participants vs. non-dyslexic participants in the manipulation condition[11].
4. However, if dyslexic adults do not suffer a central executive deficit at all, then between group differences will not emerge for either maintenance or manipulation conditions.

Method

Participants

36 participants were recruited for the experiment. 1 participant withdrew, and 1 participant was excluded as they only made occasional responses during the task. Participants were therefore 33 adults, where 16 had normal reading skills (6 male, 10 female), and 17 (5 male, 12 female) had a confirmed diagnosis of dyslexia from an Educational Psychologist.

[11] Note: Although the N2 analysis is reported in a footnote in this chapter, it was predicted that the N2 component would become larger as WM load increased, in line with Riby and Orme (2013).

Participants were matched on age, with a mean age of 20.69 years for non-dyslexic participants, and a mean age of 20.12 years for dyslexic participants, $F(1, 31)=0.43$, $p=.51$. The same assessment measures were maintained from experiment 5. Results from this assessment are documented in Table 16.

Table 16: *Behavioural assessment results for Experiment 5.*

Assessment	**Control**	**Dyslexic**	**Difference**
Dyslexia Assessment			
Reading rate (words/min)	21.69 (3.99)	17.84 (4.78)	$F(1, 31)=7.59^{**}$
Reading comprehension	7.56 (1.21)	8.53 (1.97)	$F(1, 31)=2.70$
Timed Précis: Content score	11.81 (3.02)	10.47 (3.50)	$F(1, 31)=1.38$
Timed Précis: Spelling errors	1.50 (2.75)	4.82 (3.57)	$F(1, 31)=8.86^{**}$
Writing speed	31.91 (4.03)	27.30 (4.32)	$F(1, 30)=10.77^{**}$
Spoonerisms Accuracy	22.50 (3.54)	3.54 (2.47)	$F(1, 31)=8.68^{**}$
Spoonerisms correct seconds/item	1.96 (2.04)	3.40 (2.52)	$F(1, 31)=3.23^{+}$
RAN Digits Total time	14.69 (2.61)	25.53 (15.98)	$F(1, 31)=6.73^{*}$
RAN Digits items/ sec	3.51 (.65)	2.42 (.87)	$F(1, 31)=15.50^{***}$
RAN Objects Total time	26.71 (5.88)	36.31 (15.94)	$F(1, 31)=4.84^{*}$
RAN objects items/ sec	1.94 (.38)	1.54 (.41)	$F(1, 31)=8.43^{**}$
WRAT-III Spelling (raw score)	52.75 (2.93)	48.17 (3.36)	$F(1, 31)=17.27^{***}$
WRAT-III Reading (raw score)	44.53 (2.56)	40..00 (3.84)	$F(1, 31)=14.99^{***}$
Processing speed: Digit symbol coding items/minute	39.53 (6.76)	37.78 (8.53)	$F(1, 31)=.41$
Digits Forward	12.06 (1.91)	10.29 (2.31)	$F(1, 31)=5.68^{*}$
Digits Backward	7.88 (2.73)	6.06 (1.85)	$F(1, 31)=5.06^{*}$
Verbal IQ (WAIS-III)			
Vocabulary	41.31 (8.19)	38.17 (7.27)	$F(1, 31)=1.47$
Arithmetic	14.63 (3.05)	13.35 (4.13)	$F(1, 31)=1.00$
Non- Verbal IQ (WAIS-III)			
Block Design	53.44 (9.78)	53.00 (11.48)	$F(1, 31)=.01$
Picture Arrangement	12.88 (2.63)	16.17 (2.96)	$F(1, 31)=11.40^{**}$

Note: + p<.10; *p<.05; ** p<.01; *** p<.001. *Standard deviations are reported in parentheses.*

Design

A 2 x (2 x 2 x 2) mixed design was employed. The between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target) and Task (maintenance vs. manipulation). The mixed design enabled the research to differentiate between dyslexic and non-dyslexic participants' working memory ability in the same task. The behavioural dependent variables were accuracy and reaction times at each level of WM load. When signal detection theory was implemented a 2 x (4) design was used with Group and Task as independent variables, and d-prime and Criterion as dependent variables.

Materials and procedure

A spatial delayed response task (SDRT; as in Glahn et al., 2002(b)) was administered to all participants. The task includes two trial types: maintenance only, or maintenance and manipulation. Participants remained at a fixed viewing distance of 80ms. They were required to fixate at the center of the horizontal line, which was marked with a small vertical line. Participants were instructed to make as few eye-movements and blinks as possible. Each trial initiates with a 500-ms blank screen. This was followed by a pre-cue screen, consisting of a horizontal meridian line, presented in the middle of the computer screen, with the word 'flip' or 'same' written above. This indicated whether the trial required maintenance, or maintenance plus manipulation respectively. The pre-cue remained on the screen for 1000 ms. Next, a target array of three blue circles appeared, positioned pseudo-randomly above the horizontal line, for 1500ms. After a fixed delay of 6000ms seconds, a probe array appeared. This consisted of three yellow dots, which remained on screen for 3000ms, or until the participant made a response. In the maintenance only condition, participants were

required to indicate if the probe circles were in the same position as the target array. In the manipulation and maintenance condition, participants had to flip the target representation during the delay period. Thus, in the maintenance plus manipulation condition, participants had to state whether or not three yellow circles match their mental representation. A detailed illustration of the experiment procedure can be seen in Figure 17.

Trial presentation and target: non-target ratio are set to 50: 50, to ensure individuals could not rely on strategic responses to perform this task. Thus there were 4 blocks with 20 experimental trials in each block. The order of stimulus presentation was randomized between participants. Half of the participants responded ‘yes’ with the letter m, while the other with letter z.

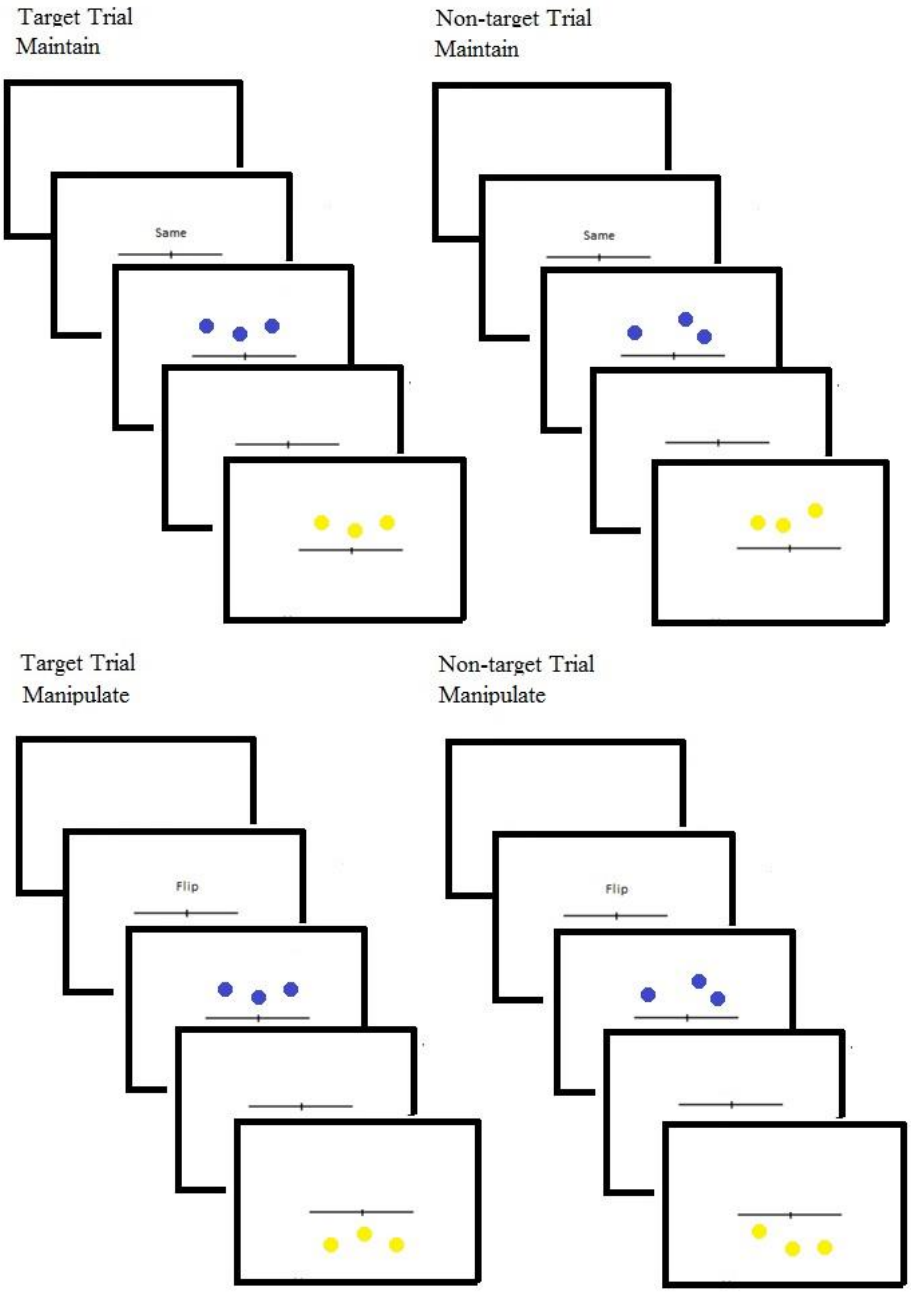

Figure 17: Depicting the experimental procedure for the maintenance condition. Displaying a target and non-target trial for the maintain condition (top) and the maintenance plus manipulation condition (bottom).

Results

Behavioural effects

Mean scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials for both the *Maintain* and *Maintenance plus manipulation* conditions. The results are summarised in Table 17.

Table 17: *Mean hit and correct reject scores for Experiment 5*

Trial	**Group**	**Task**	
		Maintain	Manipulate
Target	Non-dyslexic	.91 (.09)	.84(.14)
	Dyslexic	.89 (.08)	.78 (.15)
Non-target	Non-dyslexic	.95 (.05)	.88 (.15)
	Dyslexic	.94 (.07)	.88 (.10)

Note: Standard deviations are reported in parentheses.

Results were analysed using a mixed three way GLM analysis with Task (maintain and manipulate), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The results revealed a main effect of task, $F(1, 31) = 44.71$, $p<.001$, $\eta p^2 = .59$, with higher accuracy in the maintenance only condition, and a main effect of trial, $F(1, 31) = 6.34$, $p=.017$ $\eta p^2 = .17$, with higher accuracy for non-target trials, compared to target trials. All other effects were not significant, $Fs<1.07$. Given the RT analysis highlighted some important between group differences, the full analysis is reported here.

Reaction Times

Median reaction times were calculated for dyslexic and non-dyslexic participants for target and non-target trials for both maintain, and maintain plus manipulation conditions. Mean scores were then calculated for each condition, in Table 18.

Table 18: *Mean RT (ms) for Experiment 5.*

Trial	**Group**	**Task**	
		Maintain	Manipulate
Target	Non-dyslexic	866.38 (267.87)	929.93 (293.41)
	Dyslexic	853.38 (238.53)	1049.10 (259.54)
Non-target	Non-dyslexic	799.22 (219.32)	938.34 (230.82)
	Dyslexic	834.03 (169.60)	914.26 (164.15)

Note: Standard deviations are reported in parentheses. Mean scores are calculated for target and non-target trials

The results were analysed using a mixed three way GLM analysis, with Task (maintain and manipulate), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The results revealed a main effect of task, $F(1, 31) = 36.20$, $p < .001$, $\eta p2 = .54$ with longer RTs in the manipulate condition, and a significant three way interaction between task * trial * group $F(1, 31) = 5.28$, $p = .028$ $\eta p^2 = .15$. To analyse the significant three way interaction further, a separate GLM analysis was conducted for target and non-target trials, maintaining task and group and independent variables. The analysis of target trials displayed a main effect of task, $F(1, 31) = 13.73$, $p = .001$ $\eta p^2 = .31$, whereby there were longer RTs in the manipulation condition, compared to maintenance only. The task * group interaction revealed a trend, $F(1, 31) = 3.56$, $p = .07$ $\eta p2 = .10$, whereby dyslexic participants were slower for manipulation conditions than were non-dyslexic participants. The between subjects effect was not significant, $F = .39$, suggesting that individuals with and without dyslexia had comparable reaction times across all conditions for target trials. For non-target trials, the analysis revealed a main effect of task, $F(1, 31) = 27.94$ $p < .001$ $\eta p^2 = .47$, where all individuals were slower for the manipulation condition. All other effects were not significant [Fs<2.53].

Signal detection theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants for d' and criterion scores, at each task condition (maintain, and manipulate). The results are presented in Table 19.

Table 19: *Mean Signal Detection Theory scores for Experiment 5.*

Parameter	**Group**	**N-back**	
		Maintain	Manipulate
d'	Non-dyslexic	3.32 (.67)	2.41 (.77)
	Dyslexic	3.07 (.42)	2.23 (.52)
Criterion	Non-dyslexic	.24 (.22)	.25 (.22)
	Dyslexic	.20 (.36)	.24 (.38)

Note: Standard deviations are reported in parentheses.

A mixed GLM analysis was carried out on d-prime values, with load and group as the independent variables. This analysis revealed a main effect of task, $F(1, 31) = 57.59$, $p < .001$, $\eta p^2 = .65$, with smaller d' values for the manipulation condition. The effect of group, and interaction between task * group were not significant [F=1.74]. A mixed GLM analysis was carried out to examine the effect of load upon the criterion. No effects reached significance [Fs<.37].

Electrophysiological Analysis

Electrophysiological preprocessing replicated that described in Experiment 4. Like experiment 4, the analysis was conducted with 4 regions of interest (right anterior, right posterior, left anterior, and left posterior), and at the midline (Fz, Cz, and Pz). Analysis was conducted for N2 amplitude, P300 amplitude and latency. For lateral electrodes, a 4 way GLM analysis was conducted, with Hemisphere (right, left), Region (anterior, posterior), Task (maintenance only, maintenance plus manipulation), and group (dyslexic, non-dyslexic) as independent variables. When the analysis was conducted at the midline, a 3 way GLM

analysis was conducted, with electrode (Fz, Cz, and Pz), Task (maintenance only, maintenance plus manipulation), and group (dyslexic, non-dyslexic) as independent variables.

Encoding

Lateral Electrodes

For the P300 amplitude, the analysis revealed a marginally significant main effect of hemisphere, $F(1, 31) = 3.84$, $p=.059$, $\eta p^2 = .11$, with greater positivity in the right hemisphere (1.00 uV) compared to the left hemisphere (1.31 uV). There was a main effect of region, $F(1, 31) = 72.80$, $p<.001$, $\eta p^2 = .71$, whereby the P300 occurred maximally at posterior regions (3.00 uV, compared to -.69 uV in anterior regions). Hemisphere and region also interacted $F(1, 31) = 72.80$, $p<.001$, $\eta p^2 = .71$, whereby the P300 occurred maximally at anterior regions, in the right hemisphere. All other effects did not reach significance, [Fs<2.48].

Analysis of P300 latency revealed a main effect of region, $F(1, 31) = 7.13$, $p=.012$, $\eta p^2 = .19$, where the P300 occurred later in anterior regions (390.11 ms) compared to posterior regions (368.43 ms). Furthermore there was a significant hemisphere by region interaction, $F(1, 31) = 7.13$, $p=.12$, $\eta p^2 = .19$, whereby the P300 peak latency was later over the right anterior electrodes. All other effects were not significant, [Fs<.2.42]. ERP plots can be found in Figure 18.

Midline electrodes

Analysis of P300 amplitude revealed a marginal effect of electrode, $F(1.16, 35.79) = 65.22$, p<.001, ηp2 = .68, whereby the P300 occurred maximally at Pz (3.73 uV) compared to Fz (-1.13 uV) and Cz (.14 uV). All other effects failed to reach significance [Fs<1.22]. Analysis of P300 latency revealed a marginal effect of electrode upon latency, $F (2, 62) =3.03$, $p=.055$, $\eta p^2 = .089$, whereby the P300 peaked earlier at Pz (373 ms), then Cz (386

ms), and then Fz (390 ms). All other effects did not reach significance [*F*s<.2.18]. ERP plots for midline electrodes can be found in Appendix E.

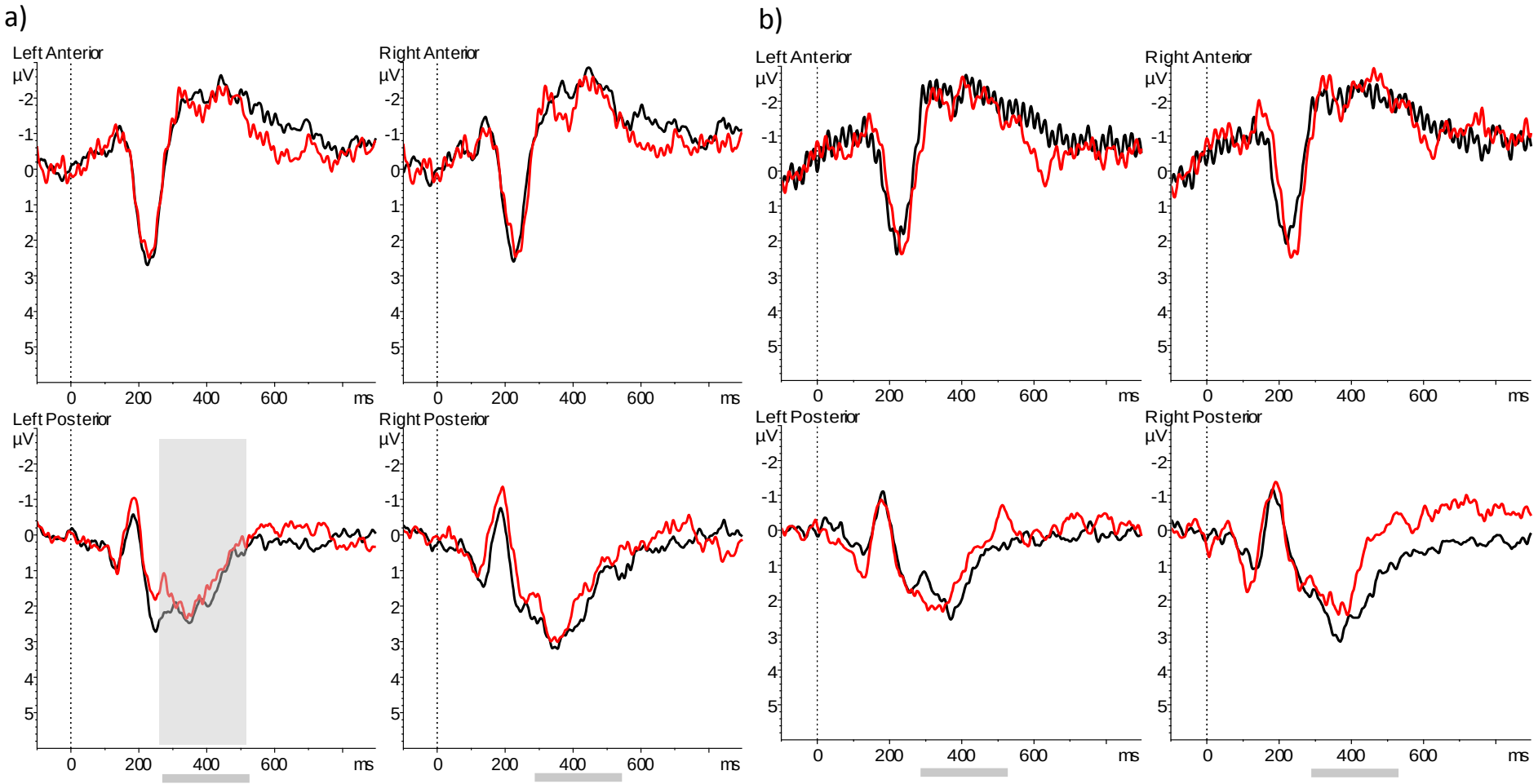

Figure 18: Grand average ERP waves for Experiment 5, at encoding.

a) **Non-dyslexic (black)** and dyslexic (red) individuals in the maintenance condition. b) N**on-dyslexic (black)** and dyslexic (red) individuals in the maintenance plus manipulation condition.

Retrieval

Lateral electrodes

Grand average ERPs are plotted in Figure 19. For the P300 amplitude, a 3 way GLM analysis was conducted, with the P300 as the dependent variable. The grand average ERP is plotted Figure 19. Results revealed a significant main effect of hemisphere, F (1, 31) =6.03, p=.02, ηp2 = .16, with greater positivity in the right hemisphere (2.84 uV) compared to the left hemisphere (2.39 uV). There was a main effect of region as the P300 also occurred maximally in posterior regions (3.80 uV, compared to 1.43 uV in anterior regions). Furthermore, the results revealed a significant main effect of task, $F(1, 31) = 7.85$, $p=.009$ $\eta p^2 = .20$, with a more positive P300 response for the manipulation condition (2.90 uV) compared to the maintain condition (2.34 uV). All other effects did not reach significance, [Fs<2.10].

Analysis of the P300 latency revealed a main effect of hemisphere, with the P300 peaking earlier in the right hemisphere (M= 374 ms) compared to the left (M= 363 ms), F (1, 31) = 12.69, $p<.001$, $\eta p^2 = .29$. There was also an interaction between hemisphere and group, $F(1, 31) = 7.05$, $p<.001$, $\eta p^2 = .19$, whereby the P300 peaked later in the right hemisphere for non-dyslexic participants (364 ms in the left compared to 382 ms in the right) but there was no difference between hemispheres for dyslexic participants (M= 353 in the left compared to 365 in the right). Group also interacted with region $F(1, 31) = 40.27$, $p=.047$, $\eta p^2 = .12$, where in non-dyslexic participants there was a main effect of hemisphere F (1, 15) = 18.10, $p=.001$, $\eta p^2 = .55$, which was not significant for dyslexic participants F (1, 16) = .43, $p=.53$, $\eta p^2 = .03$. All other effects were not significant [Fs <.3.50]. ERPs are plotted in Figure 19.

Midline electrodes

Analysis of the P300 amplitude showed a main effect of electrode, $F(1.16, 35.79) = 65.22$, $p<.001$, $\eta p2 = .68$, whereby the P300 occurred maximally at Pz (3.73 uV) compared to Fz (-1.13 uV) and Cz (.14 uV). All other effects failed to reach significance [*F*s<1.22]. Analysis of the P300 latency revealed a main marginal main effect of electrode upon latency, $F (2, 62) =3.03$, $p=.055$, $\eta p^2 = .089$, whereby the P300 peaked earlier at Pz (373 ms), then Cz (386 ms), and then Fz (390 ms). All other effects did not reach significance [*F*s<.2.18][12]. ERP plots for midline electrodes can be found in Appendix E.

[12] Besides topographical differences, analysis of the N2 did not reveal any significant between group effects [*F*s<.1.02]. At encoding, there was a marginal effect of task, $F(1, 31) =3.96$, $p=.055$, $\eta p^2 = .11$, whereby the manipulate condition gave rise to a larger N2 component (-2.14 uV, compared to -1.55 uV). There was also a marginal effect of task at retrieval, $F (1, 31) =3.96$, $p=.055$, $\eta p^2 = .11$, whereby the manipulate task elicited a larger N2 component (-2.14 uV), compared to the maintenance task (-1.55 uV).

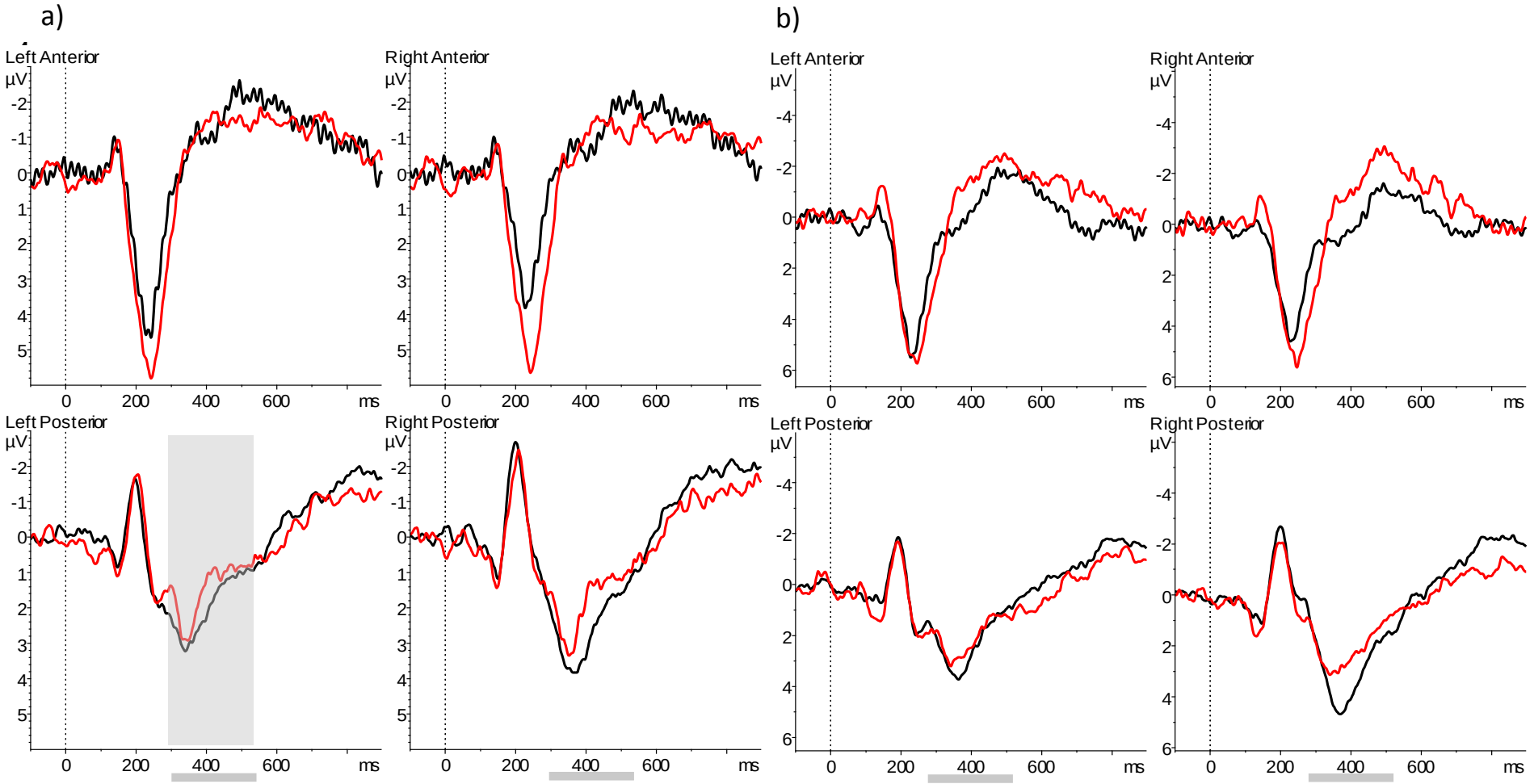

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Figure 19: Grand average ERP waves for Experiment 5, at retrieval.

a) **Non-dyslexic (black)** and dyslexic (red) individuals in the maintenance condition. b) N**on-dyslexic (black)** and dyslexic (red) individuals in the maintenance plus manipulation condition.

Discussion

The aims of Chapter 4 were to examine visual spatial WM processing in adults with developmental dyslexia. Research in this area has debated the extent to which individuals with dyslexia are impaired in visual spatial WM processing, however, emerging research (e.g., Wang & Gathercole, 2013), has demonstrated between group differences for complex span tasks, when controlling for simple span performance. This finding has been interpreted as a central executive impairment in dyslexia. Research with adult participants is sparse; only two known published studies have demonstrated a WM impairment in adults with dyslexia (Smith-Spark & Fisk, 2007; Smith-Spark et al., 2003). Furthermore, experiments investigating these effects in adults did not investigate the electrophysiological correlates of WM processing. Here, the P300 was analysed in order to tap into specific electrophysiological differences at different stages of cognitive processing. During the task, behavioural responses were also analysed.

Previous research has interpreted the amplitude of the P300 to reflect the efficiency of neural generators underlying WM (Kok, 2001). P300 amplitude occurs proportionately to processing capacity (e.g., Kramer & Strayer, 1988; Parasuraman, 1990). Equivalent P300 amplitude between individuals with and without dyslexia suggests that the allocation of processing capacity was equal between groups. Research has demonstrated that P300 latency is affected by perceptual complexity, and the cognitive demands of a given task (e.g., Wickens, Braune, & Stokes 1987). The P300 latency in Sternberg (1969) paradigms have demonstrated increased P300 latency with increased memory load (e.g., Ford, Roth, Mohs, Hopkins, & Kopell, 1979). Strayer, Wickens, and Braune, (1987) interpreted these effects as memory search, and stimulus category decision processes. To examine the electrophysiological components associated with increasing WM load, the P300 amplitude

and latency were examined. When interpreting the results of the current experiments, only effects that reach a reporting threshold ($p<.05$), and are fully significant, are interpreted. For Experiment 4, load was manipulated (1, 3, 5 and 7 targets). Based upon the work of Glahn et al (2002a), that if individuals with developmental dyslexia had a central executive impairment, then we would see a behavioural and electrophysiological impairment for higher WM load conditions.

The behavioural analysis demonstrated that increasing the set size of WM decreased all individuals' average accuracy, d' and criterion scores, while increasing RTs. This suggests that increasing WM load was associated with a behavioural cost. An increase in the criterion suggests that all individuals became more conservative as they were required to store more information in WM. However, there were no group differences in these dependent variables. ERPs were also analysed during encoding and retrieval.

During the memorization stage, participants were presented with a target array which they passively viewed, before responding to a probe (retrieval). EEG was recorded, and the amplitude of the P300, and N2 were analysed, along with the latency of the P300. Analysis of the N2 was conducted, as it is a component typically analysed in spatial WM experiments (e.g., Riby & Orme, 2013). However, analysis of this component did not reveal any between group differences, at encoding or retrieval. Thus, the remainder of the ERP discussion presented in this chapter focuses on the P300, specifically because this component was analysed in other chapters of this thesis, and has shown differences between groups (See Chapter 1, Experiment 1).

Further P300 analysis at encoding demonstrated that increasing WM load, decreased the P300 amplitude. The results demonstrated a larger P300 at posterior sites on the right. Moreover, analyses demonstrated that individuals with dyslexia did not differ from controls

in terms of the P300 amplitude, suggesting that resource allocation and processing demands did not differ between groups. The latency of the P300 demonstrated that the P300 peaked later as load increased in anterior regions. The midline electrode analysis demonstrated an interaction between load * group, whereby non-dyslexic individuals showed no effect of WM load upon latency, however, dyslexic individuals showed an effect of WM load upon the P300 latency, where increasing WM load resulted in the P300 peaking later. This might reflect early attempts to rehearse visual information online, and earlier engagement of the memory matching process.

At retrieval, the P300 amplitude is a marker of stimulus classification during the matching process. Analysis of the P300 demonstrated that the effect was predominantly right lateralised, and occurred maximally in posterior regions. In posterior regions, where the P300 is known to occur maximally, the P300 reduced as WM load increased. Analysis of P300 latency demonstrated that WM load resulted in the P300 peaking later. There were some topographical differences that emerged between groups in terms of the P300 latency, whereby the P300 occurred later in the right hemisphere than in the left hemisphere, however this effect was only there for individuals with dyslexia. However, overall the P300 analysis did not reveal any significant group differences (e.g., P300 amplitude differences).

Overall, the results of Experiment 4 suggest that individuals with developmental dyslexia do not have impairments in maintaining visual spatial information online, even as WM load is increased. Subtle topographic differences in terms of the P300 latency were unraveled at encoding, which might suggest that individuals with dyslexia engaged in stimulus classification processes earlier than non-dyslexic individuals. However, this did not produce any observable behavioural impairment, or an impairment in P300 responses at retrieval.

Overall, the behavioural and ERP analysis presented in Experiment 4 argue against a visual-spatial WM impairment in dyslexia, even in higher WM load conditions. Dyslexic individuals performed comparably to non-dyslexic participants during the SDRT (Glahn et al., 2002a). If as suggested by Smith-Spark et al., (2003) and Smith-Spark and Fisk (2007), that visual spatial WM impairments can be explained by faulty central executive function, then lower d-prime values, and increased RTs would have been expected, particularly in higher WM loads. The results of experiment 4 suggest that individuals with developmental dyslexia are not impaired at visual spatial WM processing.

These results are therefore discrepant with Smith-Spark and Fisk (2007), who demonstrated between group differences on a simple task in adult dyslexic participants. Smith-Spark and Fisk (2007) acknowledge that one explanation for their results could be due to co-morbid AD(H)D symptoms. However, they argue that this is unlikely as participants in their sample did not mention any co-morbidity, and the behavior of the participants demonstrated in the experiment was not consistent with the presence of AD(H)D. Furthermore, their participants performed comparably to their control group on the picture completion subtest of the WAIS-III (Wechsler, 1998) a measure sensitive to the presence of AD(H)D. However, this was not directly controlled for by the authors, as it is in the current context. For the current experiment, individuals were recruited on the basis that they had no other neurodevelopmental disorder (e.g., AD(H)D). Thus, one possibility, is that the participants in Smith-Spark and Fisk (2007) experiment had impairments in attentional and central executive difficulties which impaired their WM task performance (e.g., Barkley, 1997). Furthermore, in the Smith-Spark and Fisk (2007) experiment, only 32% of the sample showed a significant deficit on the spatial WM task (Defined by having a -1 effect size from the control mean). Furthermore, the frequency verbal WM measures was determined to be

much higher than visual WM measures, suggesting that effects were more robust in the phonological domain.

Another important difference is that in Smith-Spark and Fisk (2007), recalling the order of presentation was particularly important, a factor absent from this experiment, whereby participants were required to state only if a single dot had occurred in a previous location. Thus it can be argued that the current task is a spatial span task, whereby Smith Spark and Fisk (2007), measure item span, and sequence memory. In their simple span task, participants were required to write down the position of each cell that an X appeared in, and in the order in which the X's appeared. Thus, one possibility is that the item representation was intact in dyslexia, but order information was impaired. This was also the case in their complex span task, where a Corsi type arrangement of cells was implemented and participants were required to maintain the order of presentation. Thus, one possibility is that individuals with dyslexia have specific impairments with order memory, in the spatial domain.

Evidence suggests that adults with dyslexia have impaired short-term memory for order. Recent evidence in the verbal domain has suggested that individuals with dyslexia have impaired item, and order memory (e.g., Martinez Perez, Majerus, & Poncelet, 2013; Martinez Perez, Majerus, Mahot, & Poncelet, 2012). The researchers demonstrated that STM for item information depended on the quality of the underlying representation, and was impaired in dyslexia. Order information, was also impaired, and was considered to reflect core STM processing. However, this research has examined verbal STM only, so it is unclear as to whether these effects would transpire in visual spatial WM tasks.

Research by Szmalec, Loncke, Page and Duyck (2011) has also demonstrated that individuals with dyslexia are impaired on learning serial-order information, transpiring as

impaired Hebb repetition learning, demonstrating an inability to manage complex sequential structures. It has been argued that the central executive is recruited when re-sequencing items during recitation (Wildgruber, Kischka, Ackermann, Klose, & Grodd, 1999). However, this explanation for the discrepant findings alone, is unlikely. In experiments 2 and 3, presented in chapter 3 of this thesis, dyslexic participants performed comparatively to controls on visual-object N-back tasks. The N-back task requires both WM for item representations, and order. Participants must respond as to whether the current item matched an item N-back. Thus the sequence of presentation must be prioritized in visual WM. It is unlikely that sequence disorder in WM is the only factor impairing performance in dyslexic individuals.

While Experiment 4 allowed for an examination of the between group differences which might emerge as load increased, the task is a passive storage task. As suggested by Smith-Spark et al., (2003) Smith-Spark and Fisk (2007), and Wang and Gathercole (2013), any impairment individuals with dyslexia show on spatial WM tasks, are likely to be due to problems with central executive processing. Therefore, central executive processing differences might emerge between groups in tasks involving the simultaneous processing of information, as in Gathercole and Wang (2013). To test this hypothesis further, Experiment 5 administered the SDRT (Ghan et al., 2002b) to examine WM processing in contexts where participants had to perform a matching task under passive maintenance, versus a condition where they were required to maintain and simultaneously manipulate that information online. Participants were given a pre-cue, which instructed them to maintain the representation, or to flip it across a horizontal meridian line and maintain this mental representation. Participants then responded to a probe, to indicate if the three dots matched their maintained representation.

Results from Experiment 5 showed that, in both the conditions (maintain, and maintain plus manipulate), participants with dyslexia were not impaired in their accuracy, compared to non-dyslexic individuals. Further signal detection theory analysis demonstrated statistically equivalent d' and criterion values. Thus, when controlling for response bias, participant's with dyslexia demonstrated equivalent behavioural performance, to the non-dyslexic participants. Furthermore, their criterion scores were equivalent to non-dyslexic participants. However, some evidence of impaired performance was revealed in the reaction time analysis: there was a statistically significant interaction between trial type, task and group, whereby dyslexic individuals were slower for target trials when they were required to actively manipulate the stimuli. However, a simple main effects analysis on target-manipulation trials, only revealed a marginal between group difference. Therefore, there is a trend indicating that individuals with dyslexia needed more time to compute the manipulation, before the matching task could be performed, or took longer to access their mental representation and match it with the probe. Alternatively, the result may indicate that individuals with dyslexia were being more conservative when performing the matching task. This trend in RTs in the maintenance plus manipulation condition supports the work of Smith-Spark et al., (2003), Smith-Spark and Fisk (2007), and Wang and Gathercole (2013), who suggest the visual spatial WM impairment is a result of impaired central executive processing, but we show that this only occurs in conditions requiring active manipulation.

Again ERPs were analysed to assess the electrophysiological components associated with maintenance and manipulation in visual-spatial WM. At encoding and retrieval, analysis of the N2 demonstrated that the N2 was again more negative when individuals were required to manipulate information in WM, which at encoding might represent top down attentional control associated with preparing, or early attempts to manipulate the stimuli

before the interval period. This is likely given that participants were warned that they would have to 'flip' the next stimulus, and could thus exert some top-down control. At retrieval, the more negative N2 for manipulate conditions might represent top down cognitive control associated with manipulating information in WM. There were no between group differences in the amplitude of the N2 component. For the P300, the amplitude was greatest in posterior regions, particularly in the right hemisphere. However, even in conditions requiring active manipulation, dyslexic participants had a comparable P300 in terms of amplitude and latency.

Analysis at retrieval demonstrated that the P300 component peaked maximally in the right hemisphere. The P300 was also largest in posterior regions. The P300 was larger for tasks requiring manipulation. This effect was somewhat surprising, given the P300 typically reduces with increased demand. Within the WM literature, differences in P300 amplitude have been considered to reflect the allocation of attention, and processing capacity (Watter, Geffen & Geffen, 2001). However, the results are predictable under the context updating hypothesis of the P300 (Donchin, 1981). Donchin (1981) suggests that an attention driven comparison process is required to evaluate the representation of the previous event in WM, and if the representation is updated, a larger P300 occurs. One possibility is that the manipulation condition requires greater context updating. For example, in the manipulation condition, the mental representation may be weaker than in the 'maintenance only' condition. Therefore, in the manipulation condition, the evaluation process might require greater updating of the WM representation, in order to match it to the probe.

While the P300 was larger for conditions requiring manipulation, the P300 also occurred later. Watter, Geffen and Geffen (2001) interpret latency differences in the P300 as evidence of a matching process, which occurs at the retrieval stage (probe). In the N-back task, the authors argue that the participant already has the stimuli in mind, ready to match to

the upcoming stimuli, which means they do not have to 'look back' to the target stimuli in order to determine a match. However, the authors argue that in Sternberg like tasks, individuals do engage in a matching process at the probe, so the P300 occurs later as WM load increases. Here, an increased P300 latency might occur due to interference or competition between the original target stimuli, and the new mental representation. Thus, the amount of time taken to perform the matching task was increased.

Future research

One possibility for future research would be to reduce the target presentation time. Here, the procedure was a direct replication of Glahn et al (2002), and thus replicated their timings in the trial sequence. This was done in order to ensure the same neural substrates were recruited, and ensure that participants would perform the manipulation task adequately. However, one possibility to extend on the work in this thesis, would be to reduce the stimulus presentation time to under 1000ms (e.g., 250ms), to increase task demands. Furthermore, task demands would be increased in Experiment 5, by making the trial type (e.g., maintenance only, or maintenance plus manipulation) unpredictable. In experiment 5, participants were pre-cued in order to instruct them to maintain or manipulate the item in visual spatial working memory. Thus, participants had a chance to exert top-down control. Evidence suggests that top-down control from the prefrontal cortex can contribute to WM accuracy (e.g., Edin, Klingberg, Johansson, McNab, Tegner, & Compte, 2009). Future research could therefore investigate central executive processing in dyslexia, when participants have or do not have the opportunity to anticipate task demands. For example, the task (maintenance only, or maintenance plus manipulation) would be manipulated with a retro cue. The remainder of the work in this thesis focuses on phonological WM processing in dyslexia, and the associated electrophysiological correlates. This is because Experiments in Chapter 3 also indicated

comparable performance on visual WM tasks in dyslexia, using the N-back task. The N-back task is executively demanding, and thus, if adults with developmental dyslexia had a global central executive deficit, we would expect impaired performance in dyslexic individuals for these tasks also.

Chapter conclusions

Research with children has suggested that individuals with dyslexia are impaired in tasks which require central executive processing (e.g., Wang & Gathercole, 2013). The extent to which this applies in adulthood was examined in this chapter. Here, we examine whether individuals with dyslexia are impaired in maintaining items in visual spatial WM, and how accuracy might be affected as load increases (Experiment 4), or if there is a specific impairment in central executive functioning (Experiment 5). Analysis of the behavoural data suggested no difference in accuracy between groups. Furthermore signal detection theory analysis demonstrated that individuals with dyslexia, in both experiments, were equally able to discriminate signal from noise within the task. Furthermore, they were no more conservative than non-dyslexic individuals (as with Experiment 1). Although there was a significant interaction in the RT analysis in Experiment 5, between trial * task * group, when examining the simple main effects, the between group difference for target manipulation trials was not significantly different between groups. Furthermore, between group differences did not manifest in the ERP analysis.

Furthermore, even previous research which has demonstrated a visual-spatial deficit in developmental dyslexia has only demonstrated an effect for a small proportion of participants (e.g., Smith-Spark & Fisk, 2007). Furthermore, the Experiments in this chapter (Experiments 4 and 5), along with the results of Experiments 2 and 3 in Chapter 3, suggest

that it is unlikely that adults with developmental dyslexia have a domain general central executive impairment underlying their WM deficit.

Chapter 5: Phonological WM in dyslexia

A finding of impaired WM processing in dyslexia in the phonological domain has been reported extensively in the literature. For example, many authors have demonstrated that the phonological loop's storage capacity is impaired (e.g., Ackerman & Dykman, 1993; Cohen, Netley, & Clarke, 1984; Gould & Glen-cross 1990). Here, the electrophysiological correlates of phonological WM processing are investigated for auditory verbal stimuli. Two experiments are presented. For Experiment 6, letters are presented in the auditory domain during the N-back task. In Experiment 7, words are presented in the auditory domain, which are manipulated by their Age if Acquisition (AoA).

Experiment 6

In Experiment 1, Chapter 1, letters were presented visually, and participants were required to respond as to whether the current stimuli matched the one presented N items back. Similar investigations have been conducted in the neuroimaging literature, where Beneventi, Tonnessen, Ersland, and Hugdahl (2010) presented letters visually to dyslexic and control children, during an N-back task. The researchers demonstrated that as WM load increased, dyslexic individuals showed reduced activation in areas associated with WM load, suggesting that individuals with dyslexia are impaired in verbal WM processing. Furthermore, while research has shown a smaller P300 in dyslexia for tasks that are not directly designed to assess WM (e.g., target discrimination tasks; Neville, Coffey, Holcomb, and Tallal, 1993), the ERP responses associated with WM processing in dyslexia have not been examined. In Chapter 3, the electrophysiological responses associated with WM processing in dyslexia were examined for verbal, and visual-object stimuli, however, the work in the current chapter aims to explore these effects further, by overcoming some of the limitations that were present in Experiment 1.

For example, in Chapter 1 Experiment 1, we did not observe an N * group interaction, in the bahavioural or the P300 amplitude analysis. Beneventi, Tonnessen, Ersland, and Hugdal (2010) suggest that it is still unclear whether the phonological WM deficit observed in individuals with dyslexia, is caused by phonological loop problems or a more basic deficit in phonological processing, such as an impairment in phonological sensitivity (a difficulty detecting, blending and manipulating individual sounds). In Experiment 1, individuals with dyslexia were impaired for all WM load conditions, so it is difficult to conclude if this was due to a WM impairment, or a lower level deficit in phonological processing. One likely possibility is that individuals with dyslexia were impaired at each level of N because Experiment 1 imposed very high task demands with a quick presentation rate. Thus, to ensure the differences between groups become larger as N increases, here N is manipulated between 0-5 back, and the stimuli were presented for 800ms, with a 1600ms ISI.

Furthermore, in Experiment 1, individuals had a behavioual hit rate impairment that could be explained by a strategic response, as opposed to a signal detection impairment. Their d' scores were comparable to controls. This is likely to be due to the 2: 1 non-target: target ratio adopted in Experiment 1. Dyslexic individuals appeared to maximise their correct rejects (choose no), but, at a cost to the hit rate. In the signal detection framework, a biased ratio between non-target: target trials, weighted towards non-target trials, provides a greater probabilistic context for the current trial to be noise. Thus, the probabilistic context might scaffold WM functioning, and allow participants to rely on strategy. In order to ensure that individuals with dyslexia have a WM impairment, as opposed to a general difficulty with phonology, one should see a difference in d', which should also become greater as N increases.

Establishing behavioural and ERP effects in the phonological and verbal domain is essential for examining effects in the visual domain. If null results in the visual domain (as in Experiments, 2, 3, 4 and 5) are going to be meaningful, or interpretable, it is essential to ensure the effects are present in a context where they are specifically expected.

Rationale and hypothesis

1) In Experiment 1 of Chapter 1, short presentation times were given, and the ISI was 500ms. Given such difficult task demands, individuals with dyslexia were impaired at each level of N in terms of their accuracy. Thus, it is unclear if the impairment demonstrated is a result of impaired WM processing per se. Here, N is increased from 0-5 back. Furthermore, the ISI is increased in order to provoke a group * load interaction in accuracy, d' and the P300 amplitude analysis. Impairment in higher WM loads in dyslexia is suggestive of a WM impairment, as opposed to a difficulty with processing phonological information, particularly at quick processing rates. Furthermore, a 0-back control condition is added, where participants with dyslexia should not show an impairment.

2) Furthermore in Experiment 1, individuals with dyslexia showed an increased criterion compared to non-dyslexic participants. One possibility is that this response bias was a result of increased sensitivity to the 2: 1 ratio of non-target: target stimuli. A higher probability that the current stimulus is a non-target might have allowed individuals with dyslexia to increase their false alarm rate. Here a 1: 1 ratio or non-target: target stimuli is used (as was the case in Experiments 3, 4 and 5) to ensure probabilistic information cannot influence a response bias.

3) The aims of the current experiment are to examine auditory WM processing in dyslexia. Findings within this domain have demonstrated strong robust effects (e.g., Ackerman & Dykman, 1993; Cohen, Netley, & Clarke, 1984; Beneventi, Tonnessen, Erland,

& Hugdal, 2010), and consequently impaired phonological WM is one of the three main factors associated with the phonological impairment in dyslexia (e.g., Ramus & Szenkovits, 2008). Thus, in this chapter the behavioural and ERP responses associated with this deficit are considered. EEG has excellent temporal resolution and is able to provide information about what cognitive process might be impaired, or delayed in time. Demonstrating ERP differences in the phonological domain, but not in the visual domain (as in Experiments 2, 6 and 7 of this thesis) would provide a convincing argument for a specific phonological loop hypothesis in dyslexia. Furthermore, examining the latency of the P300, using Watter, Geffen and Geffen (2001)'s approach can provide us with insights into whether individuals with dyslexia apply the same effortful strategy of holding the N-back item in memory for comparison with the upcoming stimulus.

4.2 Overall hypotheses

1. There will be an impaired behavioural response (reduced accuracy, smaller d' values) in individuals with dyslexia. There will be an N * group interaction in both the hit rate, and d'. Now that there is a 1: 1 ratio of non-target: target trials, we should not see a criterion difference between groups, as probabilistic information has been removed.
2. There will be a reduced P300 amplitude in the dyslexic group compared to non-dyslexics. However, this might only be for conditions of higher N.

Method

Participants

37 participants were initially tested in this experiment. Data from four participants were not analysed because they failed to take part in both the behavioural dyslexia assessments, and the EEG study which were run on two separate days. A further 1 participant withdrew during the EEG procedure. Therefore 32 participants were included in the analysis, from which, the

non-dyslexic participants were 17 adults with normal reading skills. All participants were female, one of whom was left handed, and 16 were right handed. The dyslexic group were 15 adults, recruited from the Dyslexia and Disability service at the University of Kent. Two of whom were left handed; all were female. Participants were matched in age, with a mean age of 20 in the non-dyslexic group, and a mean age of 22 in the dyslexic group, $F(1, 30) = 2.18$, $p=.15$. All participants were living in Kent, UK at the time of the testing and had English as their native language. No other language, neurological disorders (including neuro-developmental disorders) or visual impairments were reported by the participants. All individuals with dyslexia had a diagnosis of dyslexia by an Educational Psychologist. In addition to this diagnosis, all participants took part in an extensive dyslexia assessment to ensure the groups differed on measures sensitive to dyslexia, but were equivalent in IQ. The results are presented in Table 20.

Table 20: *Mean scores for Behavioural Assessments for Experiment 6.*

	Non-dyslexic	Dyslexic	Difference
Dyslexia Assessment			
Decoding ability: Nonsense passage reading errors: Passage 1	29.26 (8.63)	37.84 (8.02)	$F(1, 29)=31.24^{***}$
Nonsense passage reading errors: Passage 2	23.64 (5.82)	35.46 (9.63)	$F(1, 30)=20.26^{***}$
Spoonerisms accuracy	23.59 (28.60)	20.57 (5.70)	$F(1, 29)=2.34^{+}$
Spoonerisms centile	39.71 (27.18)	12.14 (!3.54)	$F(1, 29)=22.02^{***}$
Writing Speed words/sec	31.66 (3.79)	26.40 (4.47)	$F(1, 30)=12.94^{**}$
Writing Speed Centile	30.59 (28.71)	26.40 (4.47)	$F(1, 30)=8.13^{***}$
Timed Précis: Reading speed	89.26 (19.84)	113.85 (39.59)	$F(1, 29)=16.59^{***}$
Timed Précis: Reading centile.	38.82 (24.34)	12.50 (14.37)	$F(1, 29)=12.68^{***}$
Timed Précis: Content score	11.70 (2.31)	11.00 (1.51)	$F(1, 30)=1.01$
Timed Précis Content centile	39.71 (25.95)	24.66 (15.97)	$F(1, 30)=3.77+$
Proof reading: Accuracy	7.64 (3.33)	2.93 (4.34)	$F(1, 29)=11.72**$
Proof reading / seconds	61.59 (18.56)	67.72 (17.83)	$F(1, 29)=.87^{**}$
WRAT-III Spelling (raw score)	45.56 (2.87)	40.13 (2.17)	$F(1, 29)=34.89^{***}$
WRAT-III Reading (raw score)	43.31 (2.44)	39.27 (3.75)	$F(1, 29)=12.83^{***}$
Processing speed: Digit symbol coding items/minute	90.18 (15.70)	77.07 (16.28)	$F(1, 28)=5.03^{**}$
Digits Forward	11.35 (2.71)	10.73 (1.79)	$F(1, 30)=.56^{***}$
Digits Backward	7.17 (2.81)	5.00 (2.00)	$F(1, 29)=6.20**$
Verbal IQ (WAIS-III)			
Vocabulary	43.93 (9.88)	39.33 (5.89)	$F(1, 29)=2.44$
Arithmetic	10.50 (3.22)	9.40 (2.84)	$F(1, 29)=1.07^{***}$
Non- Verbal IQ (WAIS-III)			
Block Design	42.47 (10.64)	39.85 (8.26)	$F(1, 29)=.56^{***}$
Picture Arrangement	13.29 (4.66)	12.60 (3.50)	$F(1, 30)=.22^{***}$

*Note: + $p<.10$; *$p<.05$; ** $p<.01$; *** $p<.001$. Standard deviations are reported in parentheses.*

Materials

An auditory letters version of the N-back WM task was administered to all participants (e.g., Awh et al., 1996; Braver et al., 1997; Veltman, Rombouts & Dolan, 2003; Bemevemto et al., 2010). Ten letters were used in total, all of which were presented in the auditory domain. Each letter could be easily discriminated, and phonologically similar letters (e.g., C and G) were not used. A constant volume was maintained across letters, and participants. In the N= 0, 1, 2, 3, 4 and 5 back conditions, a hit occurred when the participant answered yes on trials where the stimuli had occurred 0, 1, 2, 3, 4 and 5 back respectively. A correct reject was awarded when the participant correctly identified (responded no), that the stimuli did not occur N-back.

Design

For the behavioural analysis, a 2 x (2 x 6) mixed design was used. The between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target) and N (0, 1, 2, 3, 4, & 5). The behavioural dependent variables were accuracy and reaction times in each condition. When signal detection theory was implemented a 2 x (5) design was implemented, given that trial (target, non-target) were no longer present, with N (within) and group (non-dyslexic, dyslexic) as independent variables and D-prime and Criterion as dependent variables. In the main experiment, there were 4 blocks with 100 experimental trials in each block. Out of the 100 trials, 50 were non target trials, and 50 were target trials. The ratio of target to non-target trials was consistent across blocks in order to reduce the impact of trial ratio upon strategy changes (criterion). WM load was consistent within blocks, and increased per block. The first block included the N=0 condition, while the final sixth block included N = 5. The hand used for target responses was

counterbalanced so that half of the participants responded ‘yes’ with the letter m, while the other with letter z.

Procedure

Participants read the information sheets and signed informed consent forms. Consenting participants listened to the instructions verbally, and then read instructions on a computer screen which informed them they would hear different letters, and would have to respond yes or no to each stimulus, depending on whether or not the current letter occurred N back. They were given an example scenario for each N-back block and were verbally probed for their response to a hypothetical N-back scenario, to check their task understanding. Furthermore, participants participated in 40 practice trials before each block. The experiment was controlled using E-prime software. The experiment began with a fixation cross, which remained onscreen for the duration of the experiment to minimize eye movements during the EEG. There was a 1600ms ISI between trials (as in Evans, Selingar & Polack, 2011). Participants were asked to respond as quickly and accurately as possible. The experimental procedure is depicted in Figure 20. Overall, the experiment took approximately 45 minutes, with self-paced breaks between blocks.

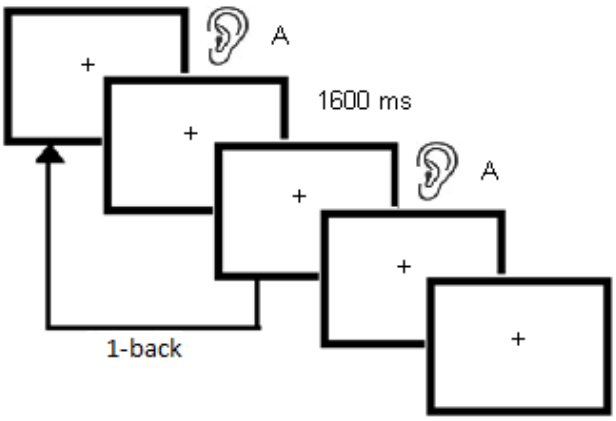


Figure 20: Illustration of the experimental procedure for Experiment 6.

The N back procedure depicts the N=1 condition, with a presentation rate of 600ms, and an ISI of 1600ms.

Participants returned to the lab within two weeks to complete the dyslexia and IQ assessments. Testing time for part 2 (assessment measures) took approximately 1.5-2 hours, and participants were given the opportunity for a break within the session.

EEG Recording and Pre-processing

EEG was continuously recorded with an average reference from active 64 Ag-AgCl electrodes. All preprocessing stages were identical to those described in Experiments 4 and 5. EEG recordings were time-locked to the onset of the letter, with a -100ms baseline. Data was then exported into Matlab, where EEGlab was used to generate average event related potentials for each participant and condition at electrodes sites Fz, Cz and Pz. Furthermore, to examine the hemispheric effects, electrodes were pooled into 4 regions: Left anterior, left posterior, right anterior, and right posterior. In order to statistically analyse these waveforms the peak-peak measure was used (as in Experiment 1, and 2). While load was manipulated from 0-6 back to examine load * group interactions in the behavioural results, analysis of ERPs was collapsed into low, medium and high task demand conditions, by collapsing across 0 and 1 back, 2 and 3 back, and 4 and 5 back. This allowed the ERP waves to be more easily interpreted and ensured the participant specific average was based on at least 30 correct artifact-free EEG trials[13]. Here, a region analysis was conducted, for consistency were other experiments in this

[13] Collapsing across conditions is common in ERP research, to minimise the amount of waveforms compared, while still maintaining relevant factors (Luck, 2013; ERP boot camp). This reduces the number of factorial levels, results in fewer simple main effect comparisons, fewer spurious interactions, and smaller experiment wise error. This was essential for Experiment 7, where AoA (early and late) is also a factor, and N is still manipulated from 0-5 back. To avoid comparing dyslexic and non-dyslexic participants across there 12 conditions, N was collapsed into 3 levels: Low (0 & 1 back), Medium (2 and 3 back) and High (4 & 5 back). Thus to remain consistent across the experiments in this chapter, this collapsing contingency was carried out in Experiment 6 also. Although the 0-back condition is technically the baseline condition, like 1-back there are low

thesis. However, the a-priori expectation was that the P300 would occur maximally in posterior regions, or at Pz. This was based upon a wealth of research (see Polich, 2007 for a full review).

Results

Behavioural Effects

Accuracy

Mean scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each WM load condition. The results are summarised in Table 21.

Table 21: *Mean hit and correct reject scores for Experiment 6*

Trial	**Group**	**N-back**					
		0	1	2	3	4	5
Target	Non-dyslexic	.98 (.03)	.94 (.04)	.89 (.80)	.78 (.19)	.70 (.18)	.61 (.19)
	Dyslexic	.97 (.03)	.91 (.13)	.90 (.18)	.64 (.16)	.56 (.17)	.55 (.17)
Non-target	Non-dyslexic	.99 (.01)	.98 (.02)	.95 (.05)	.95 (.03)	.91 (.07)	.89 (.05)
	Dyslexic	.98 (.03)	.98 (.03)	.95 (.05)	.89 (.10)	.86 (.06)	.80 (.08)

Note: Standard deviations are reported in parentheses.

Results were analysed using a mixed three way GLM analysis, with N (0-5), and trial type (target and non-target) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The Huynh-Feldt adjustment (Huynh & Feldt, 1970) was employed as appropriate. The results revealed a main effect of N, $F(3.33, 99.95) = 87.01$, $p < .001$, $\eta p^2 = .74$, whereby accuracy decreased with WM load. There was a main effect of trial, $F(1, 30) = 56.22$, $p < .001$ $\eta p^2 = .65$, where accuracy has higher for non-target trials. There was a

WM demands. The amplitude and latency of the 0-back is also the same as the 1-back (e.g., Watter, Geffen, & Geffen, 2001), and the behavioural analysis here did not reveal a significant difference between 0 and 1 back conditions, $t(31) = 1.35$, $p = .19$ (paired t-test).

significant interaction between trial * N, F (3.28, 98.49) = 32.83, p<.001, ηp^2 = .15, in that the accuracy the WM load of 5 condition increased for target trials, but decreased for non-target trials. There was a significant main effect of group, F (1, 30) = 7.69, p=.009. There was a significant interaction between N * group, F (3.28, 98.49) = 32.83, p<.001, ηp^2 = .15, whereby there was no significant difference between groups for lower WM load conditions (ts<.88). However, from a WM load of 2 back there was a marginally significant difference between groups t(30)=1.72, p=.096. There was also a significant difference (3 t(30) =2.25, p=.035), 4 (t(30) =2.08, p=.046), and 5- back (t(30) =2.08, p=.046) conditions. All other effects were not significant [Fs<1.89][14].

Signal detection theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants, at each level of N. The results are presented in Table 22.

[14] Analysis of RTs revealed a significant effect of trial F(1, 30) =26.81, p<.001 ηp^2 = .47, whereby individuals had longer RTs for non-target trials. There was also a significant main Analysis of RTs revealed a main effect of N, F(3.99, 119.56) =17.92, p<.001 ηp^2 = .37, whereby higher N increased the RTs. There was a significant trial * N interaction, F(3.28, 98.43) =16.08, p<.001 ηp^2 = .34, whereby the RTs were initially quicker in the target condition, however as N increased to 5-back they became longer than in the non-target condition. All other effects did not react significance [Fs<.99].

Table 22: *Mean scores for Signal Detection Theory parameters in Experiment 6.*

Parameter	**Group**	**N-back**					
		0	1	2	3	4	5
d'	Non-dyslexic	4.20 (.55)	3.59 (.56)	3.06 (.92)	2.55 (.93)	2.06 (1.04)	1.74 (.61)
	Dyslexic	3.85 (.63)	3.58 (.88)	2.55 (.77)	1.65 (.76)	1.23 (.62)	1.00 (.51)
Criterion	Non-dyslexic	.07 (.17)	.14 (.19)	.14 (.19)	.30 (.34)	.39 (.31)	.38 (.31)
	Dyslexic	.04 (.18)	.22 (.28)	.34 (.27)	.44 (.24)	.46 (.21	.37 (.28)

Note: Standard deviations are reported in parentheses.

A mixed GLM analysis was carried out on d-prime values, with N and group as the independent variables. This analysis revealed a main effect of N, $F($3.76, 112.88) = 109.21, $p < .001$, $\eta p^2 = .78$, with decreasing d-prime values as N increased. Furthermore, there was a main effect of group, F (1, 30) =8.45, $p=.007$, $\eta p^2 = .220$, with dyslexic individuals showing overall lower d' values. There was a significant interaction between group and N, $F($3.76, 112.88) = 2.72, $p=.036$, $\eta p^2 = .083$, whereby individuals with dyslexia are not significantly different from non-dyslexic individuals at 0-back t(30) =1.66, p=.11, 1-back t(20) =0.05, p=.97. At 2-back there is a trend towards a significant difference, t(30) =1.17, p=.09. However, there is a significant difference between groups at 3-back, t(30) =3.18, p=.006, 4-back t(30) =2.69, p=.01 and 5-back t(30) =3.65, p<.001.

A mixed GLM analysis was carried out to examine the effect of N and group upon the criterion. The analysis revealed a main effect of N, $F($5, 150) = .15.39, $p<.001$, $\eta p^2 = .34$ only, whereby the criterion increased as N increased. The effect of group, and interaction between N * group were not significant [Fs<1.54].

Electrophysiological analysis

All preprocessing and analysis stages were identical to that described in Experiments 4, and 5 in Chapter 4. ERPs were time-locked to the onset of each letter, with a -100ms baseline. Only

correct target trials were considered in the analysis. As stated previously, effect of WM load was examined by averaging N back conditions 0 and 1 into low load, 2 and 3 into medium load, and 4 and 5 into high load. This gave rise to the independent variable WM load, with three levels. To remain consistent with previous work in this thesis, the P300 analysed the positive peak, as defined for each individual as the maximum 50ms average to occur between 300-500ms. The peak to peak method was used to analyse the P300. To remain consistent with Experiments 4 and 5, the analysis was conducted for midline electrodes (Fz, Cz, and Pz), and for lateral electrodes by averaging electrodes into pools, according to both hemisphere (left and right), and region (anterior and posterior), four distinct regions were created: right anterior, right posterior, left anterior, and left posterior. For lateral electrodes, a 4 way GLM analysis was conducted, with Hemisphere (right, left), Region (anterior, posterior), load (low, medium, high), and group (dyslexic, non-dyslexic) as independent variables. When the analysis was conducted at the midline, a 3 way GLM analysis was conducted, with electrode (Fz, Cz, and Pz), load (low, medium, high), and group (dyslexic, non-dyslexic) as independent variables. Grand average plots are displayed in Figure 21non-dyslexic and dyslexic participants at each region of analysis.

Lateral electrodes

The results revealed a significant main effect of region, $F(1, 30) = 6.45, =.017, \eta p^2 = .18$, whereby the P300 was larger in the posterior region. There was also a significant region * group interaction, $F(1, 30) = 6.45$ $p=.014$, $\eta p^2 = .19$, where the posterior P300 was smaller for individuals with dyslexia, $F(1, 30) = 7.64$, $p=.010$, $\eta p^2 = .20$, with mean values of 4.06 uV for non-dyslexic participants, and 2.62 uV for dyslexic participants (mean value across all WM load conditions). There between group effect was not significant in anterior regions, $F(1, 30) = 0.13$, $p=.12$, $\eta p^2 = .004$. Hemisphere and region also interacted $F(1, 30) = 72.80$,

p<*.001*, ηp^2 = .71, whereby the P300 occurred maximally in posterior regions on the right. All other effects did not reach significance [*F*s<2.48]. Analysis of latency did not reveal any significant effects [*F*s<1.89].

Midline electrodes

The analysis revealed a significant main effect of WM load, *F(*1.92, 57.73) = 5.45, *p*=.007, ηp^2 = .15, where the mean P300 peak to peak distance decreased as WM load increased. Furthermore, there was a marginal interaction between electrode and group, *F(*1.42, 1.92) = 2.67, *p*=.077, ηp^2 = .08, whereby the mean peak to peak value at Pz for non-dyslexic participants was 5.82 uV, but 4.15 uV for dyslexic participants. All other effects were not significant [*F*s<1.42]. Further analysis examined the effects at Pz only, because the P300 is known to occur maximally in posterior regions. However, at Pz, the main effect of load was only marginally significant, *F(*1.66, 49.8) = 2.71, *p*=.085, ηp^2 = .083, whereby load decreased the P300. There was also a marginal effect of group, *F(*1, 30) = 3.03, *p*=.092, ηp^2 = .09, whereby non-dyslexic individuals had a mean P300 peak to peak value of 5.82 uV, compared to 4.15 uV in the dyslexic group. Analysis of latency did not reveal any significant effects [Fs<1.94]. ERPs for midline electrodes can be found in Appendix F.

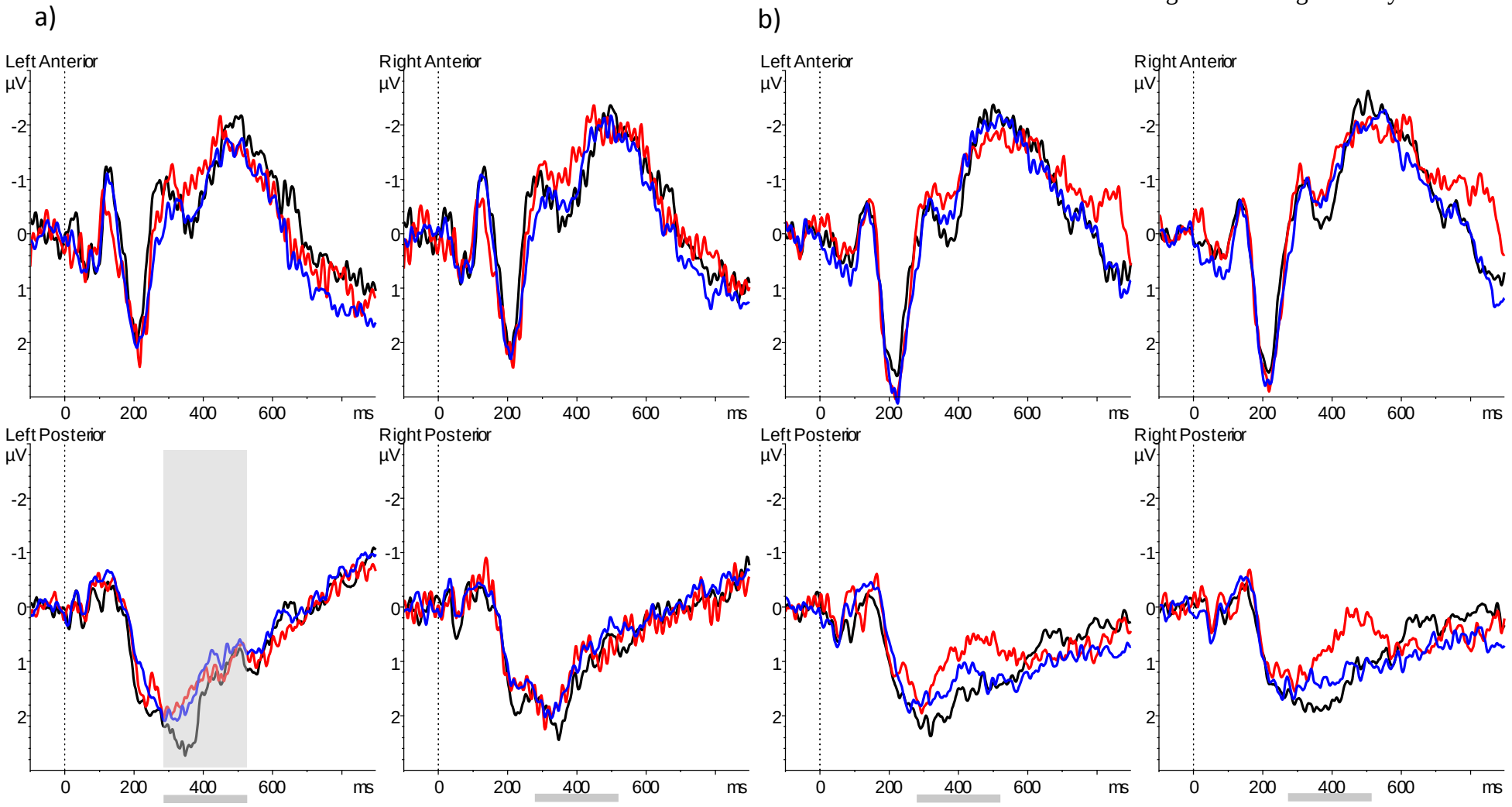


Figure 21: The grand average event related potentials (ERPs) for Experiment 6.

a) ERP plot for non-dyslexic, and b) dyslexic individuals. For **low (black)**, medium (blue), high (red) WM load conditions.

Discussion

The aim of Experiment 6 was to examine phonological WM processing in dyslexia, using behavioural and ERP methods. Research indicating a phonological loop impairment in developmental dyslexia are longstanding, and thus the behavioural evidence here support previous research which has demonstrated that individuals with dyslexia are impaired in phonological STM storage (e.g., Ackerman & Dykman, 1993; Mann, Liberman, & Shankweiler, 1980; Roodenrys & Stokes, 2001). Here, we extended previous research by examining phonological processing in terms of the P300 amplitude and latency. According to Watter, Geffen and Geffen (2001), analysis of the P300 provides insight into the allocation of resources during a WM N-back task, while analysis of latency provides information about the strategies individuals use to perform the task. Furthermore, another predominant aim of this experiment was to examine whether a group * N interaction would emerge, now the N-back conditions ranged from 0-5 back, and an ISI of 1600ms was used. A second aim was to ensure the between group differences in the criterion observed in Experiment 1, which occurred without a d' effect, could be explained in terms of the 2:1 non-target: target ratio. Here the non-target: target trials to 1: 1.

Experiment 4 demonstrates an interaction between N-back and group, whereby significant between group differences begin to emerge from 2-back onwards. This is also reflected in the signal detection theory analysis, where from 2-back onwards, individuals with dyslexia show reduced d' values. Thus, when the signal to noise distributions are equal, participants with dyslexia were unable to use probabilistic information to perform the task, and consequently an impairment was demonstrated. These behavioural results indicate the importance of considering findings from a signal detection framework, which might reveal different strategies adopted between groups. They also indicate the importance of keeping the

ratio of non-target: target trials equal within the same experiment, which has previously not been the case (e.g., Evans, Selinger & Pollack, 2012). Because these accuracy measures interact with N, we can be more confident that difficulties with phonological sensitivity cannot completely explain the WM impairment found in Experiment 1.

Experiment 6: P300 analysis

Using a high density electrode system, we examined the electrophysiological correlates (specifically the P300), during an auditory phonological N-back task, which to my knowledge has not been examined before in the literature. Previous research has demonstrated that the P300 amplitude increases proportionally as greater processing capacity is allocated (e.g., Kramer, & Strayer, 1988). In a dual task setting, the P300 produced by a secondary task, decreases in amplitude when the difficulty of the primary task (e.g., WM load) is increased. This reflects a reallocation of processing capacity away from the secondary task, to the primary task (Kramer, Strayer, & Buckley, 1991; Watter, Geffen & Geffen, 2001). Along with examining the P300 amplitude, a latency analysis was also conducted. The latency of the P300 is assumed to reflect higher order cognitive processes involved in stimulus evaluation and categorization (e.g., Polich, 1987; Polich & Heine, 1996). Examining the latency of the P300 can serve as a temporal measure of neural activity underlying the speed of attention allocation, and immediate memory operations (Cohen & Polich, 1997). Critically, latency differences were not found between groups. The P300 analysis demonstrates that individuals with dyslexia had a reduced P300 in all WM load conditions, although this only translated to a behavioural d' difference from 2-back onwards (at 2-back there was a marginal effect), which became significant from 3-back onwards. The amplitude analysis therefore suggests a processing capacity difference between groups, which becomes more impactful as WM demands increase. However, latency differences were not observed

between groups, even at higher N-back levels. This suggests that individuals with dyslexia were still engaging in the same effortful strategy as non-dyslexic participants, holding the current item on-line, ready to match to the upcoming stimuli. While the current experiment has helped establish the phonological WM effects in dyslexia, which were also observed in Experiment 1, it can only tell us about central executive processing in developmental dyslexia when the results are compared to Experiments using visual stimuli (e.g., Experiments 2, 3, 4 and 5).

While the research conducted so far in this thesis supports a phonological WM impairment, the experiments have been limited to abstract stimuli, i.e., letters, which do not carry semantic meaning. However, Experiment 7 investigates WM processing with concrete word stimuli, presented in the audio domain. According to Clark et al. (2000), there are a number of reasons to hypothesise that WM processing of concrete words should be different from that of phonological WM processing for letters and numbers. All stimulus types are symbolic objects, which facilitate analytical processing of the world, however, concrete words denote natural, physical objects within the world. As such, concrete words include activation in regions of the brain concerned with memory for objects, specifically bilateral activation in BA40 (e.g., Tulving et al., 1994; Andreasen et al., 1995). Similarly, neuroimaging experiments have demonstrated that WM storage for visual material also activates the BA40 bilaterally (E.g., Kosslyn et al., 1994).

Experiment 7

Experiment 6 demonstrated a clear difference between dyslexic and non-dyslexic participants, for phonological WM processing. Here, we examine phonological WM processing for words, a very different type of phonological WM. Baddeley (2003) in the "crystallized" system of WM, suggests that language knowledge facilitates WM tasks. Non-

words of any given length that resemble English words are more easily repeated by English speakers than words that do not resemble English words (Gathercole, 1995). Furthermore, in the Just and Carpenter (1992) framework, individual differences in verbal WM spans may be due to differences in total capacity, or efficiency in which long term linguistic knowledge is processed. Thus, it is of little surprise that research has demonstrated that memory span for words are better than memory span for non-words (e.g., Roodenrys, Hulme, & Brown, 1993).

The advantage of words over non-words in immediate recall tasks is attributed to the availability of lexical phonological representations for words, over non words. Here, we examine WM processing, and the electrophysiological correlates of such processing using words, which are manipulated by their Age of Acquisition (AoA), whereby words learnt earlier in life are more readily available, and have lexical retrieval advantages compared to words learnt later in life. The current study aims to test AoA effects upon WM processing for auditory words, in individuals with and without developmental dyslexia. The current experiment again uses the N-back procedure, maintaining a 1: 1 non-target: target trial ratio, presentation time, and ISIs from experiment 7. Thus, the current research relies on the assumption that words learnt earlier in life have an advantage in terms of their lexical access. In the following literature review, research exploring this assumption, along with the relationship between LTM (in terms of AoA) and STM will be discussed.

Age of Acquisition (AoA) and lexical retrieval advantages

Hernandex and Li (2007) describe AoA as the age in which a skill is acquired. More specifically, in the linguistic domain, it refers to the age at which a lexical item is acquired in monolinguals, or, the age at which L2 learning begins in multilingual learners. Research investigating AoA, has aimed to understand how early versus late learning affects the way such items are subsequently processed. Carroll and White (1973) were amongst the first to

demonstrate that early learned words were processed differently than late learned words. Researchers have demonstrated that the age of word acquisition significantly affects the speed and accuracy in which a word is accessed, thus giving them a processing advantage (e.g., Barry, Morrison & Ellis, 1997). Consequently, early learned words elicit faster response times than late learned words, in auditory and visual lexical decision tasks, reading, picture naming and face recognition. Various theoretical accounts have been outlined in order to explain this processing advantage. The next section will describe the theoretical literature which has aimed to understand the exact mechanisms underlying these AoA effects.

Brown and Watson (1987)'s phonological completeness hypothesis suggests that early learned words are stored in a holistic representation, while late learned words are stored in a fragmented fashion. Late learned words therefore require reconstruction before the whole phonological shape can be produced. This extra processing step is not required for early learned words, which are therefore pronounced more quickly than late learned words. However, Hernandez and Li (2007) have presented a number of studies that have not supported this account. The phonological completeness hypothesis has difficulty accounting for AoA effects that do not involve overt naming, such as face recognition (Valentine & Moore, 1998; Moore & Valentine, 1999) and object processing (Moore, Smith-Spark, & Valentine, 2004).

An alternative account, the semantic loci hypothesis claims that early learned words have a semantic advantage over late learned words because they enter the representational network first, and affect the semantic representations of later learned words (Brysbaert, Van Wijnendaele & Deyne, 2000). Crystaert et al., (2000) found that participants generated associations faster to early learned words, than to late learned words. Morrison and Gibbon (2006) found that AoA effects in living versus non-living semantic categorization tasks but

only for living items. The semantic locus hypothesis, suggests that early learned words are more conceptually enriched than late learned words, and these representations affect later learning.

Relatively few researchers have examined the neural basis of AoA effects. Fiebach, Friederici, Müllervon, Cramon, Hernandez (2003) examined AoA using functional magnetic resonance imaging (fMRI). Participants were asked to make visual and auditory lexical decisions to words and pronounceable pseudo-words. Results in both the visual and auditory modalities revealed increased activity in the left inferior prefrontal cortex for late relative to early learned words, which extended to the lateral orbitofrontal cortex. The prenuneus was more activated for early learned words, relative to those learned later in life. In addition, increased activity in the region of the left temporal operculum, near Heschls gyrus was observed for early relative to late learned words, in the visual modality. Due to the finding that the auditory association cortices were activated, the researchers concluded that participants automatically co activated auditory representations when making lexical decisions to early learned words that were visually presented. The increase in inferior frontal activity during processing of late learned words is compatible with theories suggesting that the IPFC is involved in semantic processing. The left IPFC appears to be critical in the effortful or strategic activation of information from semantic knowledge (e.g., Fiez, 1997).

Thus the processing of late learned words, is likely to involve complex retrieval, or selection processes instantiated by inferior frontal brain areas. Hernandez and Fiebach (2006) have demonstrated that when participants read single words during fMRI scanning, increased activity to late learned words was found in the left planum temporale (the posterior superior temporal gyrus), and in the right globus pallidus and the middle frontal gyrus and the inferior frontal gyrus. This suggests that late learned words engage brain areas in the left

hemisphere, that are involved in mapping phonological word representations and areas in the right hemisphere that aid articulatory and motor processing.

These results implicate neuro-anatomical substrates that may be associated with plasticity. In all of these studies, processing of late learned words involved brain areas thought to be involved in effortful retrieval, including effortful semantic, and phonological retrieval. The results are consistent with the notion that neural substrates of early learned words is at a basic level, late learned words build on these representations and require additional processing during lexical tasks. Since words with a lower Age of Acquisition (AoA) are accessed more quickly, and with higher accuracy than words learned later in life, thus giving them a processing advantage (e.g., Barry, Morrison & Ellis, 1997), we aim to assess whether this quick lexical retrieval advantage will translate in a higher level WM task.

AoA and WM

The speed and accuracy advantage often demonstrated for words learned earlier in life over words encountered later in life has been demonstrated across a number of tasks, such as word writing, reading, naming, object processing as well as lexical decision tasks (e.g., Barry, Johnston, & Wood, 2006; Bonin, Chalard, Méot, & Fayol, 2002). However, the relationship between AoA and WM processing is underexplored, and unclear. As stated previously, there is evidence to suggest that linguistic knowledge affects verbal WM capacity (e.g., Mainela-Arnold & Evans, 2005). Early work has demonstrated that there is a direct relationship between language abilities and individual efficiency and accuracy on STM immediate recall tasks (e.g., Engle, Nations, & Cantor, 1990; Gathercole, 1995). Indeed, connectionist models by McDonald and Christiansen (2002), and Seidenberg & McDonald (1999) have supported this view. Processing capacity emerges from an interaction between features inherent in the language input (e.g., frequency and regularity of patterns in language) and innate neurological structural factors of the individual speaker. Thus, individual differences in

language processing abilities may be the result of representational strength of long term linguistic knowledge being manipulated, as opposed to WM capacity.

Language based models of STM have been proposed by several authors (e.g., Martin, Shelton, & Yaffee, 1994; Gupta, 2009; Majerus, 2009). All of these models consider that item information temporarily activates corresponding phonological lexical and semantic representations. The richer and easier to access the underlying representation in the language network, the more robust their temporary activation is in STM. This leads to an advantage in recall in STM tasks. In support of this, many studies have shown that the linguistic properties of the word, such as the lexical status, lexical frequency, phonetic frequency, word imaginability, have a direct impact on the recall probability in a STM task (Gathercole, Frankish, Pickering, & Peaker, 1999; Roodenrys, Hulme, Alban, & Ellis, 1994; Walker & Hulme, 1999). Critically, the lexicality effect has the most prototypical effect upon STM performance, with familiar words, as opposed to a list of non-words, showing higher recall (Gatercole, et al., 1999). The effects of long term language knowledge (e.g., word frequency, lexicality, neighborhood density, and phonotactic probability) upon STM have been extensively researched in the language processing literature (e.g., Gathercole, Frankish, Pickering, & Peaker, 1999).

Critically, a number of experiments have shown an effect of word frequency upon STM tasks (e.g., Watkins, 1977). Watkins (1977) demonstrated that memory span scores were higher when the first half of the list comprised high frequency words, and the second half contained low-frequency words. Frequent words were recalled from long-term memory, whereas low frequency words were retrieved from STM. Wright (1979) demonstrated that low-frequency words took longer to articulate than high frequency words, even when they were equated for number of letters. Thus, Wright (1979) argued that low-frequency words which were also had a longer spoken duration, resulted in greater decay, or fewer rehearsals

before recall. Thus, frequency might have an effect on how quickly words can be rehearsed in the articulatory loop. Overall, this research suggests that verbal STM is not a self-sufficient cognitive function, but instead substantially recruits linguistic knowledge bases (Najerys & D'Argembeau, 2011).

Thus, we might expect STM span to be effected by AoA, a construct which is highly correlated with word frequency (Roodenrys, Hulme, Albam, Ellis, & Brown, 1994). Gilhooly and Watson (1981) suggest that AoA might influence STM recall because, earlier words have increased lexical access (Roodenrys, Hulme, Albam, Ellis, & Brown, 1994). Speed of access to representations stored in a speech output lexicon will increase the speed at which those representations can be converted into speech motor codes and executed as articulations. Furthermore, AoA should affect how well the information can be remembered (e.g., Baddeley & Hitch, 1974). Roodenrys, Hulme, Schweickert, and Brown (1994) claim that retrieval of partially decayed traces from a STM phonological store is facilitated by the availability of information about the phonological form of the word stored in LTM, and early learned words should be more readily available due to their quicker speed of access. Therefore, it is likely that these representations will have a WM advantage (Majerus and D'Argembeau, 2011).

Previous research has aimed to demonstrate this. Roodenrys, Hulme, Alban, Ellis, and Brown (1994) aimed to assess the relationship between AoA, word frequency, speech rate, and STM span. STM span was measured for a set of words which were manipulated by word frequency and AoA. The authors demonstrated that AoA had a small but significant effect upon speech rate differences, but these did not translate into STM span differences; likely because the effect size between AoA and speech rate is small. However, WM tasks such as the N-back task rely more heavily upon quick access to phonological representations. The N-

back task involves repeated updating of information in WM, and thus speed of access to lexical representations might be more crucial than in a simple span task. In Experiment 7, the effect of AoA is investigated at increased WM load conditions.

Rationale: The influence of dyslexia

Multiple accounts exist that aim to explain why words learned earlier in life have quicker lexical access; these have been described earlier. As stated by Ramus and Szenkovits (2008), one of the three main dimensions to the phonological deficit in developmental dyslexia (Wagner & Torgesen, 1987) is slow lexical retrieval. This has been exemplified in Rapid Automatic Naming (RAN) tasks (e.g., Torgesen et al., 1997; Wagner et al., 1993; see Vellutino, Fletcher & Snowling, 2004 for a full review), where individuals with dyslexia are impaired. Impaired RAN performance reflects an impairment in retrieving the phonological codes from memory, i.e., impaired lexical access (Chiappe, Stringer, Siegel, & Stanovich, 2002; Schatschneider, Carlson, Francis, Foorman, & Fletcher, 2002).

Ramus and Szenkovits (2008) argue that the phonological representations of people with dyslexia are normal, however the nature of the phonological deficit lies in conscious access to these phonological representations. In their phonological access proposal, the means by which lexical and sublexical phonological representations are accessed for external computations is impaired in dyslexia. Verbal STM requires access to phonological representations for the purpose of coping them into buffers, and then access to phonological buffers for retrieval. Phonological access is also required for access to input representations to copy them into output representations, and access to output representations to recycle them into input representations (i.e. the phonological loop, Baddeley & Hitch, 1974; Baddeley, 1984).

Thus while we might expect words with quicker lexical access (i.e. early learned words) to have an advantage in STM tasks in individuals who adequately access phonological representations, the same AoA effects might not be demonstrated in individuals with dyslexia, who suffer from slow access to phonological constructs. In this instance, we might predict shorter RTs, and even increased accuracy for words learned earlier in life in the N-back task for early words, only for non-dyslexic individuals. Dyslexic individuals would not demonstrate this advantage, or would be even slower than non-dyslexic participants for words learned later in life. Then, in the ERP analysis we might expect an AoA effect (a larger P300 for early learned words compared to late learned words), for non-dyslexic participants only. However, an alternative hypothesis could be that individuals with dyslexia show more dramatic AoA effects, in that words learned later in life are accessed with even more difficulty. In this instance we would expect individuals with dyslexia to show slower RTs for early learned words, compared to non-dyslexic participants, and even longer RT differences for words learned later in life: a group * AoA interaction. The current experiment is thus somewhat exploratory, and tests these two possible scenarios.

Hypothesis

For Experiment 7, the following hypotheses are proposed. Hypothesis 1 and 2 are maintained from Experiment 6.

3) Individuals will show higher accuracy and shorter RTs for items learned earlier in life.

4) The P300 will be reduced for words learnt later in life, compared to words learned earlier in life.

5) If individuals with dyslexia have impaired access to lexical and phonological constructs for early as well as late learned words, then we might not expect the same advantage for words learnt earlier in life. Alternatively, individuals with dyslexia might show a greater

disadvantage for words learnt later in life, and thus AoA differences will increase. Thus, the null hypothesis is that non-dyslexic and dyslexic group both show the same AoA effect.

Method

Participants

One participant withdrew from the experiment during the EEG procedure. Furthermore, 4 participants did not take part in both the behavioural assessment, and EEG session. This left 32 participants who were included in the analysis. The non-dyslexic group were 16 adults with normal reading skills, (all subjects were female, and 1 participant was left handed while 15 were right handed). The dyslexic group were compromised of 16 adults, two were left handed, and 14 right handed. All participants were female. The dyslexic group had a confirmed diagnosis of dyslexia from an educational psychologist at the University of Kent. The non-dyslexic and dyslexic participants were matched in age, with a mean age of 20 in the non-dyslexic group, and a mean age of 21 in the dyslexic group, $F(1, 30) = 1.18$, $p = .29$. Dyslexic participants were recruited through the dyslexia and disability service at the University of Kent. All participants were living in Kent, UK at the time of the testing and had English as their native language. None of the participants had been diagnosed with any other neurological or neurodevelopmental disorder, such as ADHD or Autism. All participants took part in an extensive dyslexia and IQ assessment, the results of which are presented in Table 23.

Table 23: *Behavioural Assessment results for Assessment measures for Experiment 7.*

Dyslexia Assessment	**Non-dyslexic**	**Dyslexic**	**Difference**
Decoding ability: Nonsense passage reading errors:			
Passage 1	1.50 (1.71)	4.26(2.15)	$F(1, 29)=15.77^{***}$
Passage 2	1.06 (1.23)	4.00 (2.30)	$F(1, 30)=20.11^{***}$
Spoonerisms accuracy	23.43 (.96)	20.00 (5.83)	$F(1, 29)=5.19^{*}$
Spoonerisms centile	41.87 (20.95)	20.33 (18.36)	$F(1, 29)=9.20^{**}$
Writing Speed words/sec	31.55 (4.22)	26.96 (3.72)	$F(1, 30)=10.60^{**}$
Writing Speed Centile	31.88 (30.54)	9.37 (7.50)	$F(1, 30)=8.18^{**}$
Timed Précis: Reading speed	92.29 (21.82)	135.21 (48.96)	$F(1, 29)=10.16^{**}$
Timed Précis: Reading centile.	36.56 (26.56)	15.00 (16.90)	$F(1, 29)=7.16^{**}$
Timed Précis: Content score	11.75 (2.32)	11.00 (1.59)	$F(1, 30)=.29$
Timed Précis Content centile	41.25 (27.17)	24.06 (16.25)	$F(1, 30)=1.13^{*}$
Proof reading: Number of errors	7.50(3.42)	4.40 (3.96)	$F(1, 29)=5.45^{*}$
Proof reading errors / seconds	61.42 (18.68)	67.34 (19.80)	$F(1, 29)=.73$
WRAT-III Spelling (raw score)	45.44 (2.89)	39.87 (2.44)	$F(1, 30)=34.42^{***}$
WRAT-III Reading (raw score)	43.13 (8.54)	39.13 (6.46)	$F(1, 29)=9.82^{**}$
Processing speed: Digit symbol coding items/minute	86.93 (15.72)	80.27 (13.97)	$F(1, 25)=.42$
Digits Forward	11.69 (2.57)	10.25(2.17)	$F(1, 30)=2.91^{+}$
Digits Backward	7.00 (2.80)	5.18 (1.93)	$F(1, 30)=4.52^{*}$
Verbal IQ (WAIS-III)			
Vocabulary	44.19 (8.54)	39.13 (6.46)	$F(1, 30)=3.57^{+}$
Arithmetic	10.44 (2.87)	10.00 (3.01)	$F(1, 30)=.18$
Non- Verbal IQ (WAIS-III)			
Block Design	42.25 (9.62)	42.87 (10.14)	$F(1, 29)=.03^{*}$
Picture Arrangement	14.75 (3.94)	13.87 (3.38)	$F(1, 30)=.45$

*Note: + $p<.10$; *$p<.05$; ** $p<.01$; *** $p<.001$. Standard deviations are reported in parentheses.*

Materials

The auditory N-back WM task (e.g., Awh et al., 1996; Braver et al., 1997; Veltman, Rombouts & Dolan, 2003) was administered to all participants, using auditory words as stimuli. A constant volume was maintained across words, and participants. In the N= 0, 1, 2, 3, 4 and 5 back conditions, a hit occurred when the participant answered 'yes' on trials where the stimuli had occurred 0, 1, 2, 3, 4 and 5 back respectively. A correct reject was awarded when the participant correctly identified (responded no), that the stimuli did not occur N-back.

Age of Acquisition: Words were selected from the MRC psycholinguistic database (1981; 1987). The AoA norms are based upon Gilhooly and Logie (1980), multiplied by 100 to produce a range from 100 to 700. The early category consisted of words learnt between the ages of 2-4, while late consisted of words learned between 8-10 years old. Therefore, the Early category included AoA scores between 242– 389, while the late category included scores between 500 – 586 (see Table 24 for mean AoA scores). Early and late words differed significantly in terms of their AoA rating $F(1, 38) = 296.415$, $p < .001$. Words were controlled across conditions for familiarity (FAM), $F(1, 38) = 2.01$, $p = .16$, imaginability (IMG), $F(1, 38) = 1.33$, $p = .26$, Ketura and Francis Frequency (KFFRQ; Katura & Francis, 1967), $F(1, 38) = 0.02$, $p = .90$, Log Frequency (LOGFREQ Hal; Balota et al. (2007)), $F(1, 38) = 0.423$, $p = .51$, number of letters (NLET), $F(1, 38) = 0.98$, $p = .33$, number of syllables (NSYL), $F(1, 38) = 1.27$, $p = .26$. (NPHN). Mean scores for each of these variables can be found in Table 24. Forty words were used in total (20 in the Early group, and 20 in the late group), all of which were presented in the auditory domain via speakers. A full list of the experiment stimuli, and raw scores for FAM, IMG, KFFRQ, LOGFREQ Hal, NLET, NSYL can be found in Appendix C.

Table 24: *Mean AoA and counterbalancing scores for early and late word categories.*

Category	AoA	FAM	IMG	KFFRQ	HAL	NLET	NSYL
Early	322.9	427.85	466.15	13.83	7.71	4.20	1.15
Late	533.85	402.15	441.5	15.38	7.40	4.45	1.30

Design

For the behavioural analysis, a 2 x (2 x 2 x 6) mixed design was utilised. The between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target), AoA (early vs. late) and N (0 vs. 1 vs. 2 vs. 3 vs. 4 vs. 5). The behavioral dependent variables were accuracy and reaction times at each level of N. When signal detection theory was implemented a 2 x (2 x 6) design was implemented with group and N as the independent variables and d-prime and Criterion as the dependent variables.

N was manipulated across blocks, and for each level of N there were 2 blocks, corresponding to early and late words. Thus, there were 8 blocks in total. Each block consisted of 100 experimental trials, with a 1: 1 ratio of non-target: target trials. The order in which participants saw early versus late words was counterbalanced within and between participants (by alternating early and late blocks between N-back blocks). This can be demonstrated in Table 25. Furthermore, version 1 and 2 of the experiment were counterbalanced between participants. Half of the participants responded ‘yes’ with the letter m, while the other did with letter z.

Table 25: Counterbalancing procedure for Experiment 7.

N-back	**0**	**1**	**2**	**3**	**4**	**5**
Version 1	early, late	late, early	early, late	late, early	early, late	late, early
Version 2	late, early	early, late	late, early	early, late	late, early	early, late

Procedure

The same experimental procedure was maintained from Experiment 6. EEG recordings and pre-processing methods were kept identical to those in Experiment 6. Once again, in order to statistically analyse these waveforms, the peak-peak method was employed in EEGlab. Participants took part in the EEG experiment at part 1, and the dyslexia and IQ assessment within two weeks as part 2.

Results

Behavioural Effects

Mean scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each WM load condition. The results are summarised in Table 26.

Table 26: *Mean hit and correct reject scores for Experiment 7.*

AoA	Trial	Group	N 0	1	2	3	4	5
Early	Target	Non-dyslexic	.91 (.16)	.93 (9.07)	.91 (.10)	.76 (.18)	.69 (.16)	.68 (.20)
		Dyslexic	.92 (.16)	.88 (.16)	.84 (.14)	.71 (.15)	.60 (.19)	.55 (.19)
	Non-target	Non-dyslexic	.97 (.03)	.98 (.02)	.97 (.02)	.91 (.08)	.89 (.12)	.84 (.12)
		Dyslexic	.98 (.01)	.98 (.03)	.94 (.05)	.87 (.07)	.83 (.09)	.81 (.16)
Late	Target	Non-dyslexic	.90 (.20)	.94 (.06)	.91 (.11)	.73 (.19)	.72 (.19)	.69 (.18)
		Dyslexic	.92 (.17)	.91 (.12)	.84 (.14)	.67 (.18)	.63 (.19)	.63 (.19)
	Non-target	Non-dyslexic	.99 (.02)	.99 (.01)	.97 (.03)	.94 (.05)	.89 (.08)	.84 (.10)
		Dyslexic	.98 (.02)	.98 (.01)	.93 (.05)	.89 (.07)	.83 (.09)	.79 (.11)

Note: Standard deviations are reported in parentheses.

The results were analysed using a mixed four way GLM analysis conducted with trial (non-target, target), N (0, 1, 2, 3, 4, 5), and AoA (early, late) as within subjects variables, and group (dyslexic and control) as the between subjects variable. The Huynh-Feldt adjustment (Huynh & Feldt, 1970) was employed as appropriate. The results revealed a main effect of WM load, $F(2.31, 69.23) = 30.98$, $p<.001$, $\eta p^2 = .51$, with accuracy decreasing as WM load increases, and a main effect of trial, $F(1, 30) = 14.68$, $p<.001$ $\eta p^2 = .33$, with higher accuracy for non-target trials. There was a significant interaction between trial * load, $F(2.68, 80.40) = 5.22$, $p=.003$, $\eta p^2 = .15$, in that the accuracy at a WM load of 5 condition increases for target trials, but decreases for non-target trials. All other effects were not significant [Fs<1.40].

Reaction Times

Median reaction times on correct trials were calculated for each participant, in each condition of analysis. From this, group means for dyslexic and non-dyslexic participants were calculated for each condition. The results are described in Table 27, and the AoA effect is also plotted in Figure 22.

Table 27: *Mean hit and correct reject scores for Experiment 7*

AoA	Trial	Group	N 0	1	2	3	4	5
Early	Target	Non-dyslexic	609.18 (128.01)	670.40 (110.31)	764.56 (164.01)	864.21 (198.370	789.25 (165.57)	770.00 (183.03)
		Dyslexic	631.65 (124.70)	643.56 (132.600	747.65 (198.57)	755.34 155.90)	724.50 (187.32)	712.62 (226.62)
	Non-target	Non-dyslexic	706.28 (104.23)	756.43 (110.06)	860.50 (131.53)	833.69 (114.25)	805.25 (94.83)	814.09 (180.48)
		Dyslexic	718.18 (103.82)	777.00 (133.18)	823.28 (189.82)	799.46 (172.29)	743.97 (113.79)	722.34 (164.14)
Late	Target	Non-dyslexic	622.63 (180.15)	665.84 (134.01)	811.53 (152.80)	862.72 (227.09)	798.34 (148.88)	851.00 (214.25)
		Dyslexic	623.41 (132.36)	671.72 (130.28)	786.59 (201.31)	866.09 (223.34)	771.53 175.33)	737.65 (219.38)
	Non-target	Non-dyslexic	681.91 (172.23)	707.46 (167.23)	876.81 (105.41)	870.09 (114.09)	746.50 (214.52)	843.37 (152.59)
		Dyslexic	706.47 (129.37)	792.21 (124.98)	805.17 (160.61)	816.53 (202.59)	768.03 (138.20)	761.88 (136.48)

Note: Standard deviations are reported in parentheses.

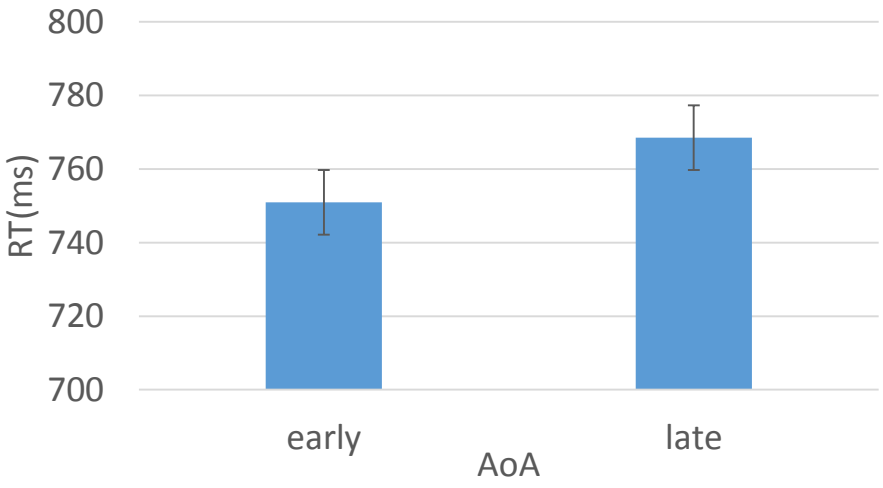


Figure 22: RT graphs for Experiment 7.

Depicts the mean RT (ms) collapsed across WM load conditions, and across trial type.

A 4 way GLM analysis was conducted with AoA (early, late) trial (non-target, target), and N (0-5) as within subject variables, and group (non-dyslexic and dyslexic) as the between subjects variable. The analysis revealed a main effect of AoA, $F(1, 30) = 5.55$, $p=.025$, $\eta p^2 = .16$, whereby words that were learned earlier in life had quicker RTs than words learned later in life. There was also a significant main effect of trial, $F(1, 30) = 5.55$, $p=.025$, $\eta p^2 = .16$, whereby non-target words elicited longer RTs. All other effects were not significant [Fs<1.77].

Signal detection theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants, at each level of N (0, 1, 2, 3, 4, 5), and each Early and Late AoA condition, for d' and the criterion. The results are presented in Table 28.

Table 28: *Mean Signal Detection Theory parameters for Experiment 7.*

AoA	Parameter	Group	N 0	1	2	3	4	5
Early	d'	Non-dyslexic	3.68 (.84)	3.48 (.76)	3.08 (1.03)	2.08 (.93)	1.64 (.58)	1.40 (.87)
		Dyslexic	3.81 (.71)	3.33 (.80)	2.78 (.88)	1.71 (.61)	1.28 (.65)	1.03 (.71)
Late		Non-dyslexic	3.84 (1.12)	3.65 (.52)	3.36 (.79)	2.43 (.93)	2.60 (.92)	1.60 (.66)
		Dyslexic	3.80 (.89)	3.18 (.76)	2.32 (.80)	1.79 (.70)	1.39 (.70)	1.03 (.70)
Early	Criterion	Non-dyslexic	.28 (.49)	.31 (.33)	.21 (.32)	.37 (.28)	.45 (.39)	.37 (.44)
		Dyslexic	.18 (.38)	.29 (.34)	.23 (.30)	.27 (.28)	.35 (.25)	.37 (.30)
Late		Non-dyslexic	.19 (.38)	.26 (.22)	.16 (.33)	.38 (.34)	.31 (.34)	.28 (.35)
		Dyslexic	.14 (.36)	.23 (.22)	.21 (.12)	.35 (.30)	.30 (.28)	.31 (.33)

Note: Standard deviations are reported in parentheses.

A GLM analysis, using the same independent variables, upon d', revealed a significant main effect of N only $F(3.08, 92.55) = 125.47$, $p<.001$, $\eta p^2 = .80$, whereby d' becomes smaller with increasing N, demonstrating an impaired ability to discriminate signal as WM load

increases. There was also a marginal effect of group, $F(1, 30) = 3.86$, $p = .059$, $\eta p^2 = .11$, whereby individuals with dyslexia had lower d' scores. Furthermore, there was a marginal effect of AoA, however this was in the opposite direction to our prediction, $F(1, 30) = 2.97$, $p = .095$, $\eta p^2 = .09$, with late learned words having a slightly larger d'. With the criterion as the dependent variable, the only main effect was that of N, $F(3.7, 110.97) = 3.83$, $p = .007$, $\eta p^2 = .113$, where the criterion increases for higher values of N.

Electrophysiological Analysis

Off-line, recordings were re-referenced to the mastoid electrodes, and were passed through a bandpass filter of .30-30Hz. EEG data was corrected for vertical and horizontal eye movements using the BrainVision Analyzer 2 semi-automatic ICA for artifact removal. EEG recordings were then segmented into epochs of 1000ms according to stimulus, realigned to a 100ms baseline. Finally, each epoch was screened for artifacts (e.g., remaining eye movements) using semiautomatic artifact rejection methods. To remain consistent with previous work in this thesis, the positive peak in the P300 analysis was defined for each individual as the maximum 50ms average to occur between 300-500ms.

All preprocessing and analysis stages were identical to that of Experiments 6 in this chapter. ERPs were time-locked to the onset of each word, with a -100ms baseline. Only correct target trials were considered in the analysis. The effect of WM load was examined by averaging N back conditions 0 and 1 into low load, 2 and 3 into medium load, and 4 and 5 into high load. The window for the P300 was defined in accordance with Experiment 6 and the peak-peak method was employed, as described in previous experiments. To statistically analyse the data at lateral electrode sites, a 5 way mixed GLM analysis was employed, with hemisphere (left, right), Region (anterior, posterior), AoA (early, late), load (easy, medium, high) and group (non-dyslexic, dyslexic) as variables. For the midline, a 4 way mixed GLM

analysis was employed, with electrode (Fz, Cz, Pz), AoA (early, late), load (easy, medium, high) and group (non-dyslexic, dyslexic) as independent variables.

Grand average plots of the ERP brain responses to each word, in each condition are displayed in Figure 23 for non-dyslexic and dyslexic participants at each region of analysis.

Lateral electrodes

There was a marginal effect of load, $F(1, 30) = 2.38$ $p=.10$, $\eta p^2 = .07$, whereby increasing load decreased the P300 amplitude. The results revealed a marginally significant interaction between region * group, $F(1, 30) = 3.58$ $p=.068$, $\eta p^2 = .11$, whereby compared to non-dyslexic participants, dyslexic individuals have smaller mean peak-peak values in posterior regions, but not in anterior regions. There was also a marginally significant interaction between AoA and hemisphere, $F(1, 30) = 3.82$ $p=.06$, $\eta p^2 = .13$, whereby there was a larger difference between early and late words in the right hemisphere, with a mean value of 2.92 uV for early words, and 2.72 uV for late words. However in the left hemisphere, the mean value for early words was 2.83 uV, and 2.78 uV for late learned words. There was a marginally significant interaction between region * AoA, $F(1, 30) = 3.85$ $p=.059$, $\eta p^2 = .14$, whereby in the anterior region, there was a significant main effect of AoA, $F(1, 30) = 7.04$ $p=.01$, $\eta p^2 = .139$. Words learnt later in life had a smaller P300 value of 2.46 uV, compared to words learnt earlier in life which had a mean value of 2.74 uV. All other effects were non-significant [Fs<2.44].

A separate analysis was conducted in the posterior region, where the P300 is known to occur maximally. This showed that there was a significant main effect of WM load, $F(1, 30) = 3.29$ $p=.04$, $\eta p^2 = .09$, whereby load decreased the P300 peak-peak values from 3.21 uV, 3.15 uV to 2.72 uV. There was a significant interaction between hemisphere and AoA, $F(1, 30) = 4.72$ $p=.038$, $\eta p^2 = .13$, whereby in the right hemisphere, words learnt later in life

had a smaller P300 amplitude, compared to the peak-peak values in the left hemisphere. The interaction between load * group was also marginally significant, $F(2, 60) = 2.51$ $p=.09$, $\eta p^2 = .077$, whereby dyslexic individuals had a smaller P300 amplitude in the high WM load condition, compared to non-dyslexic participants. Given an a-priori expectation that the effect of load upon WM performance should be strongest for dyslexic individuals at higher WM load conditions, this interaction was then followed up by a between group analysis in the high WM load condition only. This demonstrated a significant main effect of group, $F(1, 30) = 5.64$ $p=.02$, $\eta p^2 = .16$, with individuals with dyslexia showing a reduced peak-peak P300 value of 2.10 uV compared to the non-dyslexic group, who have a value of 3.35 uV.

Latency

The analysis revealed a main effect of region $F(1, 30)=4.98$, $p=.03$ $\eta p^2= .14$, whereby the P300 occurred earlier in the posterior region, with an average latency of 350ms, compared to 355ms in anterior regions. There was a main effect of WM load, $F(2, 60)= 9.29$, $p=.001$ $\eta p2 = .24$, whereby the mean values were 359 ms in the low, 349 ms in the medium, and 350 ms in the high load condition. Thus, the latency differences are likely to be due to the P300 occurring later in the low load condition. There was also a significant region * AoA * N interaction, $F(1.88, 56.48) =35.75$, $p=.04$, $\eta p2 = .10$, where there is a significant interaction between AoA * N, $F(1.66, 49.77) = 3.44$, $p=.048$, $\eta p^2 = .10$ in the posterior region, but not in the anterior region, $F(2, 60) = 3.44$, $p=.54$, $\eta p^2 = .02$. In the posterior region, the mean latency for early words in the low, medium and high WM load condition was 358 ms, and 350 ms and 345 ms, respectively. There was a significant differences between the low and medium $t(31) =4.10$, $p<.001$, and low and hard WM load conditions, $t(31) =4.08$, $p<.001$. However, for words learned later in life, the equivalent means were 352 ms, 347 ms, and 351 ms, with the only significant difference is between low and medium, $t(31)=2.58$, $p=.02$.

Midline

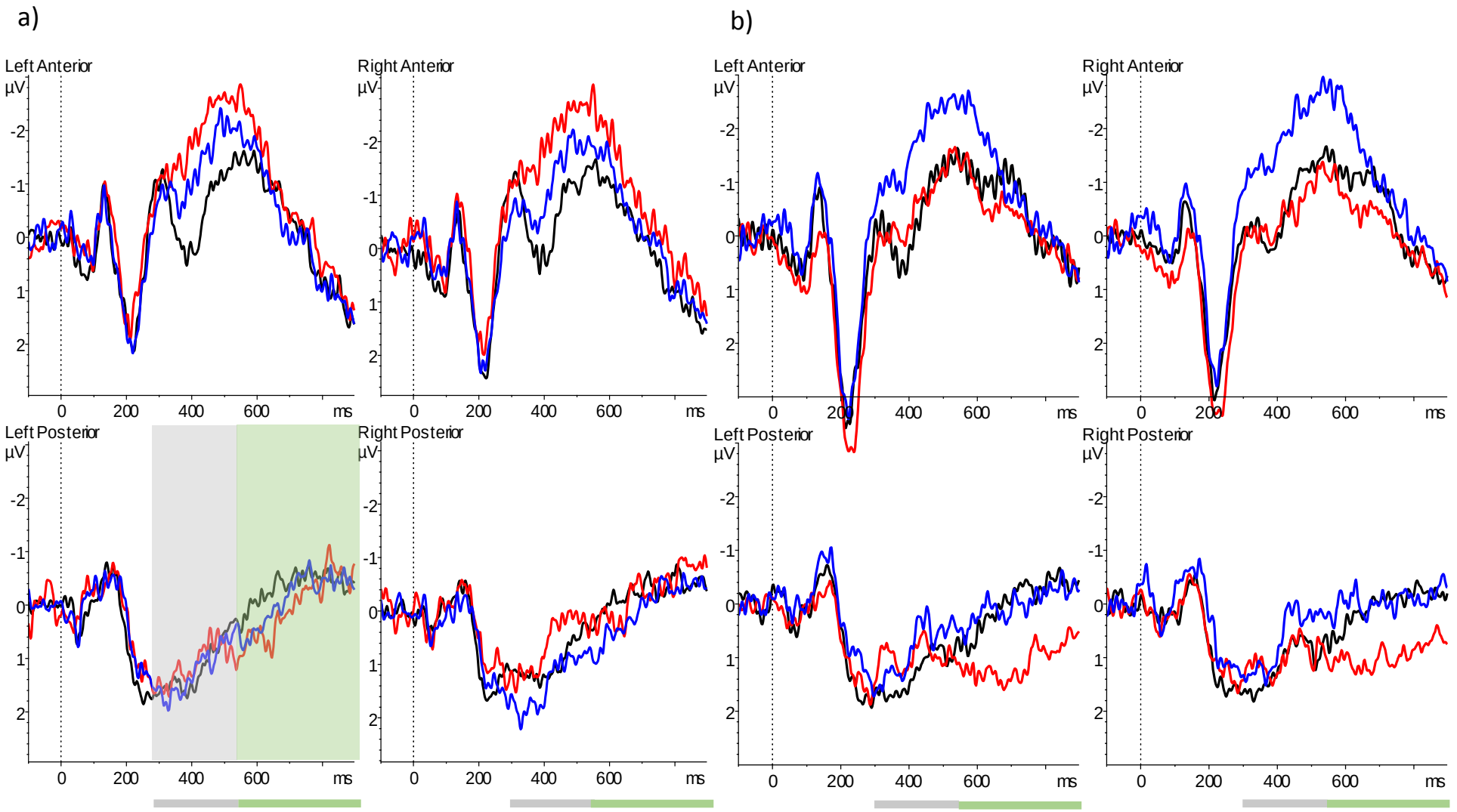
Analysis of the midline electrodes revealed a significant main effect of WM load, *F(*1.75, 53, 57) = 6.40, *p*=.005, ηp^2 = .18, whereby the P300 reduced as WM load increased. There was a significant difference between low and high (*t*(31) =3.78, *p*<.001), and medium and high WM (*t*(31) =3.46, *p*=.002) load conditions. Although there was not a significant effect of electrode, the analysis was conducted at Pz only, where the P300 is known to occur maximally. Again, there was a main effect of WM load, *F(*2, 60) = 8.70 *p*=.001, ηp^2 = .22. There was also a significant interaction between AoA * WM load * group, *F(*2, 60) = 3.37, *p*=.04, ηp^2 = .10, whereby for words learned late in life, there was a significant interaction between group * WM load *F*(2, 60) = 5.06, *p*=.009, ηp^2 = .10, but not for words learned early in life, *F*(2, 60) = .04, *p*=.96, ηp^2 =.001. At a high WM load, for words learned late in life, individuals with dyslexia had a smaller P300 peak-peak value (*t*(30) =2.05, *p*=.049) compared to non-dyslexic participants. However, there was not a between group effect at low, *t*(30) = -1.49, *p*=.88 or medium WM loads *t*(30) =1.30, *p*=.20, for words learned late in life.

Latency

The analysis revealed a main effect of electrode, with the P300 occurring later at Pz, *F(*2, 60) =7.56, *p*=.001 ηp^2 = .20. There was a significant main effect of WM load, *F(*2, 60) =8.06, p=.001 ηp^2 = .21, where WM load increased the P300 latency. These two factors also interacted, with a significant interaction between Electrode * WM load, *F(*1, 29) =35.75, *p*.001 ηp^2 = .55. Significantly, the effect of WM load was larger at Pz, where the mean latencies for early words, at low, medium and high WM load conditions, respectively is 356 ms, 349 ms and 345 ms. However, for words learnt later in life, equivalent mean values are 349 ms, 347 ms, 353 ms. There was also a significant interaction between electrode * AoA *

N, *F(*14, 120) =3.12, p=.018 ηp^2 = .09, whereby at Pz there was a marginally significant interaction between AoA * N, *F(*14, 120) =3.12, p=.018 ηp^2 = .09. There was also a significant 4 way interaction between Electrode * AoA * N * group, *F(*14, 120) =3.29, p=.014 ηp^2 = .10. Whereby, at Pz, there was an interaction between AoA * N * group, *F*(2, 60) =3.85, p=.027 ηp^2 = .11. For words learned earlier in life, at Pz there was a marginally significant interaction between N * group, *F*(2, 60) =2.77, p=.071 ηp^2 = .08, whereby non-dyslexic individuals did not show an effect of N, but individuals with dyslexia did. Specifically, for individuals with dyslexia, the P300 occurs more quickly as N increases, from 363 ms, to 354 ms and 342ms[15]. ERPs for midline electrodes can be found in Appendix G.

[15] To ensure the collapsing of the 0-back condition and the 1-back condition did not drive the significant Electrode * N * AoA * Group interaction, the analysis was conducted again without this condition. Thus N became a 2 level factor, with medium and high load. The 4 way interaction was no longer significant, F(2, 60) =.34, *p*=.70, ηp^2 = .01.


a)
b)
Left Anterior
Right Anterior
Left Posterior
Right Posterior
μV
-2
-1
0
1
2
0
200
400
600
ms

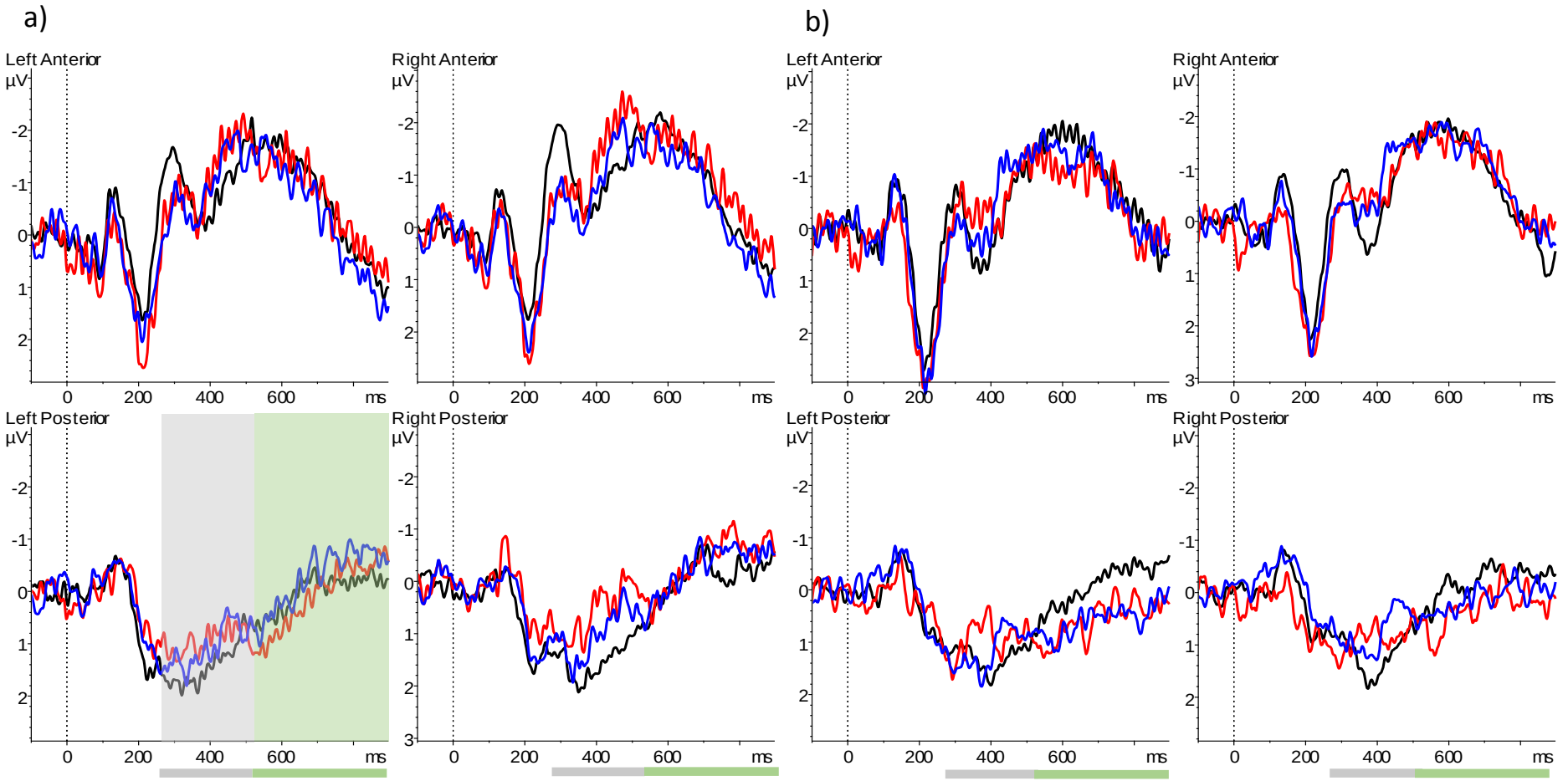


Figure 23: ERP plots for Experiment 7.

The grand average ERPs were plotted for early words, for a) non-dyslexic and b) dyslexic individuals, and for late learned words for c) non-dyslexic and d) dyslexic individuals. ERPs are plotted for **low WM load (black),** medium WM load (blue), high WM load (red) conditions. The ERP plots are presented at each region of analysis (Left anterior, Right Anterior, Left posterior, and Right Posterior).

Discussion

Two experiments were reported in this chapter, Experiment 6 (letters) and 7 (words, manipulated by AoA). In Experiment 6, the stimuli were auditory letters, and N ranged from 0-5 back. There was a 1:1 ratio between non-target: target trials, and thus it was hypothesized that individuals with dyslexia would now show a d' difference, and not a criterion difference. The results indicated that individuals with dyslexia had a reduced d', from N-back of 2 onwards, suggesting a phonological WM impairment. This impairment was associated with a reduced P300 amplitude, although there were no temporal differences in the onset of this component between groups. Thus, this research is consistent with previous results which also suggest a phonological WM impairment in dyslexia. (e.g., Ackerman & Dykman, 1993; Mann, Liberman, & Shankweiler, 1980; Roodenrys & Stokes, 2001). Now that impairments have been established in the phonological domain, we can be more confident that a failure to find a visual WM impairment in Experiments 2, 3, 4 and 5, are not the result of a paradigm flaw, or because the P300 would not be sensitive to any capacity differences between groups.

In Experiment 7 words were used as stimuli, and were manipulated by their AoA. The speed and accuracy advantage often demonstrated for words learned earlier in life over words encountered later in life has been demonstrated across a number of tasks, such as word writing, reading, naming, object processing, as well as lexical decision tasks (e.g., Barry, Johnston, & Wood, 2006; Bonin, Chalard, Meot, & Fayol, 2002), however, the relationship between AoA and WM processing is unclear. It was hypothesized that words which were learned earlier in life would have faster lexical access, and thus would be maintained within the phonological WM system. This could be because these words can be rehearsed at a quicker rate, and/or because reintegration will be quicker. Reintegration refers to the retrieval

of partially decayed traces from LTM, to help maintain the WM representation. The more available a representation is, the more efficient reintegration will be.

The results support an advantage in WM performance for words learned earlier in life. The speed in which the phonological representation is accessed, is evident in RT data, whereby faster responses can be seen for words learned earlier in life. Furthermore, in an a-priori defined ERP window, we see frontal effects of AoA, whereby words learned earlier in life have a larger positive deflection, than words learned later in life. Upon careful examination of the P300 grand average plots, WM conditions apart from those in the low load condition, are negative in polarity. However, this is likely to be due to a frontal N2 component preceding the P300. After this negativity, in the P300 time window, the component deflects in the positive direction. Words learned earlier in life have larger positive deflections, compared to words learned later in life. Furthermore, regardless of region (anterior, posterior), AoA also effects P300 amplitude in the right hemisphere. Whereby words learned later in life have a smaller P300 amplitude. Thus the result is reported, and interpreted as a neurophysiological difference modulated by AoA, reflecting an advantage in processing of early learned words.

This finding is consistent with research from Tainturier, Tamminen, Thierry, (2005), who examined spoken word recognition during an oddball task, with words which were manipulated by their AoA. The research findings demonstrate that the amplitudes of the P300 is higher for early acquired words than it is for late acquired words. The authors interpret their findings as a stronger recognition response for words learned earlier in life. However, Tainturier, Tamminen, and Thierry (2005) observed the effect in the posterior region, and larger in the right hemisphere, or in both hemispheres at in anterior regions. This topographical discrepancy is not easily interpretable, but it is important to note that

Tainturier, Tamminen, and Thierry (2005) used a significantly different paradigm (the oddball paradigm) to elicit a P300[16].

Overall, the findings demonstrate there is an influence of AoA upon WM processing, which has not been demonstrated elsewhere in the literature. Although, it is important to note that this was purely a RT advantage and did not translate into an accuracy advantage, suggesting the effect is specific to speed of access in WM. This is likely to be because an item retrieved or accessed more quickly in WM is not necessarily maintained more precisely. Instead, the benefit of AoA seems to be in the speed of access. The results contrast earlier work by Roodenrys et al., (1994), who demonstrated that AoA did not effect STM recall. The authors argued that their null result regarding AoA and STM was surprising, given their hypothesis that earlier words will have an advantage in the speed of access to their lexical representation. One key difference between Roodenry et al's., (1994) experiment and the current experiment is that they used a simple span task, while the current experiment used the N-back task which requires continuous updating of information on-line. Thus, in the N-back task, speed of access to lexical representations is likely to be crucial.

Another question addressed in this experiment, was whether individuals with dyslexia would show the same AoA advantage. The behavioural analysis did not demonstrate any between group differences in relation to AoA. Although the relationship between AoA and WM processing in dyslexia has not been examined before, Smith-Spark and Moore (2009) examined the relationship between dyslexia, AoA, and the speed at which participants can

[16] Counterbalancing of N was not implemented, because it was deemed too difficult for participants for participants to start the experiment at a higher N back level. Even if floor effects could be avoided at a higher N-back level, it was deemed likely that participants might use different strategies if they started the task in a condition where WM demands were too high. Given one of the dependent measures was criterion differences, the decision was made to compare group responses when N incremented from 0 to 5 back.

name famous faces. The researchers did not find any accuracy differences between groups, however, he did demonstrate a significant group * AoA interaction in the RT data. Individuals without dyslexia showed faster naming for faces they learned earlier in life, but individuals with dyslexia did not show the same AoA advantage. Smith-Spark suggests that this is because access to underlying lexical representation associated with the visual information is impaired in dyslexia, even for items with quicker lexical access. However, dyslexic participants in the current experiment show the same AoA effects as non-dyslexic participants. This is likely to be because the words were presented in the auditory domain, so to some extent they have already been 'accessed'. This gives the words a phonological advantage in the N-back task. Future research could present words in the visual domain, and assess the relationship between AoA, WM, and dyslexia.

Moreover, there were subtle differences in the latency of the P300 component between groups, for words learned early in life. At electrode Pz, the P300 peaked earlier for early learned words at higher N-back conditions in individuals with dyslexia. This might indicate a practice effect, where dyslexic participants were becoming more familiar with the matching task. This practice effect was not present for words learned later in life. One possibility is that dyslexic individuals did not have P300 latency differences for words learned later in life because the late learned words have a more complex lexical retrieval process, and individuals with dyslexia have impaired lexical retrieval (Ramus & Szenkovits, 2008). Overall, this interaction effect is not overly interpreted because it was not hypothesized a-priori, and the effect was no longer significant when the analysis was conducted without the low WM load condition.

Chapter Limitations

One limitation of the current work, is that individuals with dyslexia might differ in the age in which words were acquired. However, given that individuals with dyslexia showed the same

behavioural pattern of results as the non-dyslexic participants (there was no interaction between group * AoA upon RTs or accuracy), this is considered unlikely. Another limitation of this work is that there were a lot of experimental factors for an ERP experiment. This was particularly the case in Experiment 7, where the experiment IVs were N (6 levels), AoA (2 levels), group (2 levels), and specific areas of interest on the scalp. This might have resulted in some spurious interactions. To help resolve this, and increase the number of trials per condition, we collapsed the trials into three WM conditions: low, medium and high. To avoid making false positive claims, only effects which were hypothesized were interpreted.

Chapter conclusions

Overall, Experiments 6 and 7 suggest that individuals with dyslexia have impaired phonological WM processing. One of the aims of Experiments 6 was to determine if making the task easier (with longer presentation times, and the addition of a 0-back condition) would lead to an N * group interaction. The behavioural analysis demonstrates that as N increases, individuals with dyslexia show reduced performance (hit rate, and d') compared to controls. Impaired phonological WM processing in dyslexia is an established effect in the literature, but here the electrophysiological correlates associated with this impairment have been identified. Compared to non-dyslexic individuals, individuals with dyslexia show a reduced P300 amplitude, suggesting reduced processing capacity (Watter, Geffen, & Geffen, 2001). However, there was no latency differences between groups, which suggests the WM impairment is not due to the speed at which dyslexic individuals make a matching decision in the N-back task. Furthermore, under the Wattter, Geffen, and Geffen (2001) framework, this suggests that individuals with dyslexia were using the same effortful strategy of holding a stimulus on-line, ready to compare with an upcoming stimulus. Thus, Experiment 6 served a valuable purpose of examining the ERP differences between groups during a WM task.

In this chapter, the relationship between AoA and WM was also explored, in Experiment 7. It was hypothesized that words which were learned earlier in life would show an advantage in WM processing, because of the speed at which the lexical representation can be accessed (or reaccessed) for phonological rehearsal. While accuracy is not affected by AoA, the RT data suggests words learned earlier in life have an advantage within the N-back task. Furthermore, within an a-priori defined time window, words learned early in life appeared to be associated with a larger P300 amplitude. There were however, no interpretable between group effects in terms of AoA, suggesting that regardless of the nature of the phonological stimuli, individuals with dyslexia still show an impairment.

Establishing this ERP effect between groups in the phonological domain is essential for making comparisons between experiments, if one wants to interpret null effects in the visual domain. Unless a phonological effect had been established, it would be difficult to interpret any null between group effects that were found in visual WM experiments (Experiments, 2, 3, 4 and 5). Thus, Experiment 6 plays an important role in facilitating comparisons between experiments. Critically, it can now be concluded that individuals with dyslexia show a behavioural and ERP difference for phonological items, contradicting previous research (e.g., Wang & Gathercole, 2013) which suggests a domain general WM impairment. In Chapter 6, I examine why individuals with dyslexia might show a more robust WM impairment in the phonological domain.

Chapter 6: The Contribution of rise-time perception to phonological WM in dyslexia.

The predominant finding of the experiments presented in this thesis thus far, have been impaired phonological WM processing in dyslexia, a finding which is also in accordance with previous research (e.g., Ackerman & Dykman, 1993; Cohen, Netley, & Clarke, 1984; Gould & Glen-cross 1990, Smith-Spark, Fisk, Fawcell, & Nicolson, 2003). Previous experiments in this thesis (Experiments 2, 3, 4 and 4) have aimed to demonstrate between group effects in the visual domain. However, the behavioural and electrophysiological results did not demonstrate any WM impairments in dyslexia. In this chapter, one final behavioural experiment is presented, which aims to assess how lower level auditory difficulties, as described in the Temporal Sampling Theory of Developmental Dyslexia (Goswami, 2011) may contribute towards the WM impairment in developmental dyslexia.

Experiment 8

Background and Rationale

Throughout this thesis, the extent to which individuals with dyslexia have a general central executive impairment (Smith Spark et al., 2003; 2007, Wang & Gathercole, 2013), or a specific phonological loop impairment, has been examined. The experiments thus far in this thesis have demonstrated behavioural and electrophysiological differences which appear specific to the phonological domain. According to Smith-Spark and Fisk (2007), there remains debate in the literature over whether the working memory impairments in developmental dyslexia are a result of phonological processing difficulties, or working memory per se. Beneventi, Tonnessen, Ersland, and Hughdahl (2010) also claim that it is unclear whether the WM impairment in dyslexia is caused by phonological loop problems, or a more basic deficit in phonological processing, as it proves difficult to experimentally divorce phonological memory from other phonological processes (Snowling, Chait, &

Hulme, 1991). Here the relative contributions of phonological and memory processes in dyslexia are assessed.

Richardson, Thomson, Scott and Goswami (2004), and Goswami (2003; 2010) suggest that auditory perceptual difficulties may impair the development of high-quality phonological representations in individuals with dyslexia. The predominant auditory perceptual theory was proposed by Tallal (1980) and Tallal, Miller, and Fitch (1993). Tallal and her colleagues suggest that dyslexic children have particular difficulties in processing rapidly changing, or transient acoustic events. When one stimulus rapidly follows another, in both a temporal order judgment paradigm (TOJ) and a same-different discrimination paradigm, impairments were found in 8 out of 20 dyslexic children (Tallal, 1980). Transient information is critical for phoneme perception, and phoneme awareness is necessary for reading. The rapid processing deficit has however received criticism (McArthur, & Bishop, 2001; Rosen, 2003). The findings have been difficult to replicate, and studies have found differences in either TOJ or same-different judgments. Furthermore, the impairments are only present at long ISIs (e.g., Share, Jorm, MacLean, & Matthews, 2002), and fail to account for independent variance in reading and spelling (Farmer & Klein, 1993).

However, developmental research has shown that awareness of syllables in children precedes awareness of onsets and rhymes, which in turn precedes the awareness of phonemes (Ziegler & Goswami, 2005). Individuals with dyslexia show developmental difficulties at each linguistic level (Goswami, 2003). Goswami et al. (2002) proposes that syllable-level information is primary in early language acquisition; a difficulty perceiving speech rhythm – driven by syllable level phonological structure would impair the development of the entire phonological system, as is the case in developmental dyslexia. Therefore, phonological processing in dyslexia is effortful and slow, even in transparent languages where orthography

and phonology are well mapped. Goswami et al. (2002) have developed a non-speech assessment, whereby children judge whether an amplitude modulated sound comprising of one element fluctuating in loudness, or two different elements (a distinct bear, and a background sound) are distinct. The sharper the rise time of the modulation, the more likely it is that two sounds are perceived. Difficulties like this occur because individuals with dyslexia may show an impairment in phase locking to slower amplitude modulations in the theta band (Goswami, 2011). Evidence for this account will now be reviewed.

A temporal sampling framework for the auditory impairments in dyslexia Goswami, 2011)

The multi-time resolution model (MTRM) of speech processing describes how information present in the acoustic speech signal is encoded at different temporal rates in parallel in the cortex, by stimulus-induced modulations. Phase locking of inherent cortical rhythms in delta, theta, and gamma frequency bands is induced. The output of these different oscillatory networks is then bound together into speech perception (Poepell, Idsardi, van Wassenhove, 2008). Poepell et al., (2008) propose a right lateralised theta sampling network, which is driven by oscillations in lower frequencies. These slower temporal rates allow for the encoding of lower modulation frequencies in the speech signal, facilitating temporal integration at the syllabic scale. Morillon et al. (2010) suggest that slower band oscillations become lateralised to the right hemisphere during speech processing. However, higher frequency modulations are encoded by the gamma sampling network, which is bilateral. These higher frequency oscillations allow temporal integration at the phonetic level. Thus, different temporal integration scales characterise the different oscillatory networks and effectively yield varying time windows during which information is extracted, thereby sampling the signal in different linguistic proportions.

Goswami (2011) proposes a key impairment in dyslexia may be in oscillatory phase-locking in auditory cortex to slower temporal modulations, specifically delta and theta (0.5-4, 4-8Hz respectively). The amplitude envelope is the intensity varying waveform that the ear actually receives, namely energy variation over time. The auditory system codes amplitude modulation in both natural sounds across different frequency channels and time scales (Joris, Schreiner, & Rees, 2004). Sensitivity to the envelope structure and dynamics is critical for speech perception. The envelope signal speech rate, carries stress and tonal contrasts, and reflects prosodic information (Giraud & Poeppel, 2012). The syllable structure reflected by the envelope appears to be perceptually critical for phonological development (Jusczyk, 1999).

Rise times are specifically related to amplitude envelope (AE) dynamics. A rise time can be defined as the rate of onset of amplitude fluctuations in the envelope. These are critical events of the AE. They signal the onset of new syllables, and stressed syllables have more marked rise times, facilitating parsing of the acoustic signal into acoustically meaningful units. Luo and Poeppel (2007) used MEG to show that the phase pattern of the theta band tracked the AE of spoken sentences, segmenting the incoming speech signal into syllable-sized packets, and resetting to track speech dynamics. Neural encoding of the AE depends on the phase of oscillatory networks resetting to align with onsets in the signal (Giraud & Poeppel, 2012). Rise times signal syllable onsets; they are likely to play a role in phase resetting (Goswami, 2011). In summary, the perception of rise time has been suggested to impact reading acquisition because it supports the prosodic and syllable segmentation processes important for setting up the phonological lexicon and in the formation of well-specified phonological representations (see Goswami, 2011 for a recent review of the Temporal Sampling Theory of Developmental Dyslexia).

A potential link between amplitude envelope onsets and phonological representatons can be understood in terms of the identity of perceptual centres (P-centres). Rise time detection is critical for identifying P-centres in acoustic signals. P-centres are experienced moments in time at which different speech (Morton, Marcus, & Frankish, 1976) and musical (Gordon, 1987) sounds occur. The P-centre determines the onset of the signal, and is associated with a rapid increase of mid band spectral energy, which typically occurs around the onset of a vowel (Marcus, 1981). P-centres provide a non-speech mechanism for perceptually segmenting syllable onsets and rhymes. Accurate detection of P-centers is important for the quality of phonological representations.

Long versus short rise times

Goswami et al (2002) have proposed that syllable level information is primary in early language acquisition, a difficulty in perceiving the syllable level phonological structure impairs speech rhythm perception. Goswami et al. (2002) propose that this deficit may be the core impairment in developmental dyslexia. To examine this further, Goswami et al., (2002) developed a phonological judgment task, requiring children to judge whether an amplitude modulated sound was comprised of one element fluctuating in loudness, or two different elements. The sharper the rise time of the modulation, the more likely that two sounds would be perceive. Dyslexic children were significantly impaired at this beat detection task. Precocious readers were however superior compared to both dyslexics and normally developing controls. Dyslexic children lost perception of the beat when the rise times were extended, however, they perceived the beats easily when rise times were short. Control children still perceived the beats with extended rise times, however, when they lost perception, precocious readers could still detect a beat.

Furthermore, Witton, Stein, Stoodley, Rosner, and Talcott (2002) showed that dyslexic children need deeper modulations for detection, and as rise times covaried with modulation depth in their study, this was consistent with the idea that dyslexics need sharper rise times to detect the beat accurately. Thus, enhanced ability to integrate temporal information over long time windows is associated with better reading. The ability to perceive the amplitude envelope cues, may give rise to well specified phonological representations in good readers. When rise time perception is impaired, it could lead to problems representing the syllable in terms of the sub-syllabic units of onset and time.

In an experiment by Richardson, Thomson, Scott and Goswami (2004), dyslexic and control children were given a battery of phonological tasks, to assess the relations between deficits in dyslexic performance on a rise time discrimination task and phonological awareness, reading and spelling ability. Specifically related to this proposal is the rise time of amplitude envelope onset (A X B) task (Richardson, Thomson, Scott & Goswami, 2004), where A and B refer to two separate auditory tones. 40 tones were made from a 500Hz sinusoid, with a 0.7Hz amplitude modulation (depth of 50%). The rise time envelope varied logarithmically from 15 to 300ms. The steady state of the stimuli had a duration of 700 ms. The linear fall time was fixed at 50ms. Thus, the overall duration of the stimuli varied from 765 to 1050 ms. Stimuli with the shortest rise time (15ms) are used as the standard. Children were required to choose the sound that sounded different at the beginning. Thus this required them to choose which sound had a ramp with the longest rise times.

Hämäläinen, Fosker, Szücs and Goswami (2011) later examined the neural mechanisms that underlie this rise time perception deficit in developmental dyslexia. The neurophysiological event-related potential (ERP) responses to stimuli with different rise times were investigated during a passive listening task. The T-complex waves presents as a

negative-positive– negative morphology, and has greatest amplitudes over the lateral temporal channels, 140–144 ms after stimulus presentation (Näätänen & Picton, 1987; Wolpaw & Penry, 1975). The Tb wave of the T-complex showed differences between groups, with the amplitudes for Tb becoming less negative with increased rise time for the participants with dyslexia only. The group difference was especially pronounced for stimuli with the shortest 10 ms rise time.

Stefanics, Fosker, Huss, Mean, Szucs, & Goswami, (2011) have demonstrated ERP effects in earlier components also. The researchers demonstrated a slower fronto-central P1 response in the dyslexic children compared to controls. Furthermore, the amplitude of the P1 to tones with slower rise times and lower intensity was smaller, compared to tones with sharper rise times and higher intensity. In the dyslexic group, there was also a decreased N1 amplitude to tones with slower rise times, compared to tones with sharper rise times. Together, these ERP experiments suggest impaired auditory neural processing mechanisms in children with dyslexia, as a function of stimuli rise-time, supporting the Temporal Sampling Theory of developmental Dyslexia (Goswami, 2011).

To my current knowledge, the relationship between rhythmic prosody, in terms of temporal sampling of slow rise-times, has not been examined directly in the WM context, in either individuals with or without dyslexia. Goswami, Thomson, Richardson, Stainthorp, Hughes, Rosen, and Scott (2002) examined amplitude envelope onsets and developmental dyslexia in order to explore the relationship between beat detection and phonological processing, reading and spelling. The researchers demonstrated that individual differences in sensitivity to rise-times account for a quarter of the variance in reading and spelling acquisition. However, this research suggests that rise-time perception contributes to

phonological skills such as reading, however, the influence of rise-time perception has not been examined within a WM task.

Rationale and hypothesis

It remains unclear whether the working memory impairment in developmental dyslexia is just a secondary effect of a low level auditory processing deficit (see Beneventi, Tonnessen, Ersland, & Hughdahl, 2010). Thus, the current experiment aims to determine how an impairment in rise time modulation affects working memory performance, and the extent to which these two processes are independent. In order to investigate this, an N-back task is used, where stimuli consist of 10 sinosoidal tones, either with short or long rise-times. Rise-times increased logarithmically between 15-300ms, and were presented at 3 different N-back levels (1-3); participants had to state if the item occurred N-items back. Tones with faster phase locking properties (short rise times), should show an advantage in the N-back task.

How could latency differences in perceptual phase locking in the auditory cortex result in impaired WM performance?

The temporal correlation hypothesis (Singer & Gray, 1995) has been proposed as the mechanism of neural binding. Recent experiments suggest a powerful role of neural coherence between brain areas for the integration and binding of information (e.g., Steinmetz et al., 2000; Nakatani & van Leeuwen, 2006). Sauseng et al. (2009) have demonstrated cross-frequency phase synchronization between theta and gamma oscillations at parietal regions, which is associated with successful maintenance of information. Although there has been some disagreement on exactly which frequency bands concern which functions of WM; there is convergence in the literature to suggest that cross- frequency coupling and phase coding may serve as an important neural mechanism underlying WM processes (see Fell & Axmacher, 2011).

The MTRM demonstrated that the auditory signal is fractionated in frequency and time, and in the auditory cortex there is spontaneous neural activity at oscillatory frequencies of 3-6Hz, and 28-40Hz, in the theta and gamma ranges respectively. The phase pattern of the Theta bad tracks and discriminates spoken language, segmenting the incoming speech signal into syllable-sized packets, and resetting and sliding to track speech dynamics. This phase resetting mechanism is driven by syllable rise time, and reflects neural coding of the AE. This oscillatory behavior can be thought of as operating at the level of microcircuits, whereby inhibitory interneuron networks impose rhythmic synchronization capable of effectively controlling the gain of the neuronal spiking output (Bartos et al., 2007). If individuals with dyslexia are impaired at phase locking (particularly to slower rise time modulations), then encoding might become impaired due to time errors in the synchronisaton between early sensory areas, and higher order neural areas. This is because the coherent output from local neuronal groups needs to selectively synchronise over long-range connections, with task relevant neuronal groups in more distal brain regions (e.g., Buschman & Miller, 2007), which will be responsible for maintaining the representation. Therefore, impaired phase locking in the theta range might have more widely distributed consequences. Alternatively, a deficit in low level auditory processing may cause disruptions to the phonological system, across the developmental trajectory. Furthermore, a difficulty discriminating tones of longer rise-times would lead to increased competition between stimuli within WM. The current research makes the following specific predictions.

Hypotheses

1) As N increases, both dyslexic and non-dyslexic participants will show reduced accuracy and longer reaction times.

2) Stimuli with shorter rise times will be easier to discriminate and therefore maintain in WM than stimuli with long rise times, given they have faster phase locking properties.

3) There will be an interaction between rise-time length and WM load, whereby tones with short rise times will have an advantage in WM, and will therefore not show the same magnitude decrease in response to WM load as the tones with longer rise times.

4) If individuals with dyslexia have a WM impairment which operates independent of a lower level sensory deficit (in this context: an impairment with long rise-times) then we would expect a main effect of group, whereby individuals with dyslexia are impaired in both short and long rise-times. This impairment might become worse as N increases. However, if individuals with dyslexia do not have a phonological WM difficulty, which is independent of sensory difficulties, we would expect an impairment for the slow rise-time tones only.

Method

Participants

A total of 34 participants were tested, however only 32 were included in the final analysis, as two participants failed to press a response key during the experiment. In the non-dyslexic group, participants were 15 adults with normal reading skills, while the dyslexic group consisted of 17 adults with a diagnostic history of developmental dyslexia. All participants in the non-dyslexic group were female, 14 were right handed, and 1 was left handed. In the dyslexic group, all participants were female and 15 were right handed, and two were left handed. The dyslexic group were recruited from the Dyslexia and Disability service at the University of Kent, all of whom had a diagnosis of dyslexia from an Educational Psychologist. Participants from both groups were matched in age, with a mean age of 20 in the non-dyslexic group, and a mean age of 20 in the dyslexic group, $F(1, 30) = .33$, $p = .57$. All participants were living in Kent, UK at the time of the testing and had English as their native

language. No other language, neurological disorders or visual impairments were reported by the participants and none had any other neurodevelopmental disorder (e.g., ADHD, Autism). All participants took part in an extensive dyslexia assessment to ensure the groups differed on measures sensitive to dyslexia, but were equivalent in IQ. The results are presented in Table 29.

Table 29: *Behavioural Assessment measures for Experiment 8.* .

Dyslexia Assessment	Non-dyslexic	Dyslexic	Difference
Passage reading errors	4.07 (3.05)	10.29 (7.46)	$F(1, 30)=9.06$**
Reading rate (words/min)	21.69 (4.1)	18.52 (4.77)	$F(1, 30)=.3.95$+
Reading comprehension	7.53 (1.25)	8.47 (1.97)	$F(1, 30)=2.50$
Timed Précis: Content score	11.46 (2.92)	10.47 (3.50)	$F(1, 30)=.75$
Timed Précis: Spelling errors	1.67 (2.79)	4.88 (1.15)	$F(1, 30)=7.84$**
Writing speed	32.10 (3.97)	27.86 (4.32)	$F(1, 30)=7.79$**
Spoonerisms Accuracy	22.4 (3.64)	16.82 (6.89)	$F(1, 30)=7.85$**
Spoonerisms Seconds/item	36.83 (23.56)	57.38 (44.57)	$F(1, 30)=3.86$+
RAN Digits Total time (sec)	15.33 (2.72)	25.64 (15.93)	$F(1, 30)=6.11$*
RAN Digits items/ sec	3.26 (.85)	2.40 (.87)	$F(1, 30)=7.79$**
RAN Objects Total time	26.42 (5.86)	36.58 (15.81)	$F(1, 30)=5.50$**
RAN objects items/ sec	1.96 (.34)	1.52 (.41)	$F(1, 30)=10.37$**
WRAT-III Spelling (raw score)	44.57 (2.62)	39.76 (3.83)	$F(1, 30)=15.59$***
WRAT-III Reading (raw score)	52.60 (2.97)	47.70 (3.90)	$F(1, 29)=15.85$***
Processing speed:	40.06 (7.23)	37.72 (8.45)	$F(1, 30)=.69$
Digit symbol coding items/minute			
Digits Forward	11.93 (2.05)	10.23 (2.36)	$F(1,30)=4.66$*
Digits Backward	8.07 (2.74)	6.06 (1.85)	$F(1, 30)=6.03$*
Verbal IQ (WAIS-III)			
Vocabulary	40.27 (8.07)	38.08 (7.24)	$F(1, 30)=.66$
Arithmetic	14.66 (3.03)	12.88 (4.02)	$F(1, 30)=1.95$

Non- Verbal IQ (WAIS-III)	52.07 (10.34)	52.47 (11.36)	$F(1, 30)=.01$
Block Design			
Picture Arrangement	12.06 (2.96)	16.05 (2.95)	$F(1, 30)=14.66$**

+ $p<.10$; *$p<.05$; ** $p<.01$; p *** $p<.001$.

Materials

An auditory version of the N-back WM task was administered to all participants (e.g., Awh et al., 1996; Bemevemto et al., 2010) at a constant volume. Ten tones were used in total, all of which were presented in the auditory domain. The stimuli replicated those used in Richardson, Thomson, Scott and Goswami (2004). Stimuli consisted of 10 sinusoids, varying in rise time logarithmically between 15ms to 300ms. Fall time kept constant. A continuum of 10 stimuli were created from a 500 Hz sinusoid with 0.7 Hz amplitude-modulation (depth of 50%), varying the linear rise time envelope logarithmically from 15 to 300 ms. The steady state of the stimuli had a fixed duration of 700 ms. The linear fall time envelope was fixed to 50 ms (thus, as with Richardson, Thomson, Scott and Goswami (2004), the overall duration of the stimuli varied from 765 to 1050 ms). An example of the stimuli can be found in Figure 24.

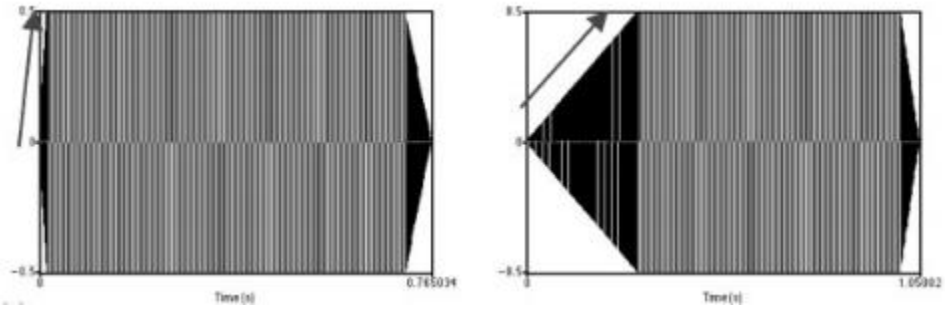


Figure 24: Depictions of the stimulus wave forms for Experiment 6.

This Figure is taken from Richardson et al (2002). The left diagram displays a sinusoid with a 15ms rise time. On the right, the rise time is increased to 300ms.

Design

Due to the difficulty of distinguishing between tones, and the abstract nature of the stimuli, N was not increased beyond 3-back. To conduct a behavioural analysis of the data, the stimuli were categorised into slow vs. long rise-times, which gave rise to a binary variable Rise-time. Although a 0-back condition was run, these results were not analysed because the first stimuli in the stream was either a short, or long rise-time stimuli. Therefore, the between subjects variable was Group (dyslexic vs. non-dyslexic), while the within subjects variables were trial (target vs. non-target), N (1, 2, 3) and Rise-time (2). Thus, for the behavioural analysis, a 2 x (3 x 2) mixed design was used. The behavioural dependent variable was accuracy and reaction times at each level of N. When signal detection theory was implemented a 2 x 2 x (2) design was implemented with N, Rise-time (within) and group (non-dyslexic, dyslexic) as independent variables and D-prime and Criterion as dependent variables. In the main experiment, there were 4 blocks with 100 experimental trials in each block. Out of the 100 trials, 50 were non target trials, and 50 were target trials. The ratio of target to non-target trials was consistent across blocks. WM load was consistent within blocks, and increased per block. The first block included the N=0 condition, while the final fourth block included N =

3. The hand used for target responses was counterbalanced so that half of the participants responded ‘yes’ with the letter m, while the other with letter z.

Procedure

This study took place across two sessions. Part 1 was an extensive dyslexia and IQ assessment, while part two consisted of the experimental procedure detailed above. Participants read the information sheets and signed informed consent forms. Consenting participants listened to the instructions verbally, and then read instructions on a computer screen which informed them they would hear different letters, and would have to respond yes or no to each stimulus, depending on whether or not the current letter occurred N-back. They were given an example scenario for each N-back block, and were verbally probed for their response to a hypothetical N-back scenario, to check that they understood the task instructions.

Participants were told that the stimuli differed at the beginning of the tone. They were told to base their decision upon these subtle differences at the beginning of the tone. Furthermore, participants completed 40 practice trials before each block. The experiment was controlled using E-prime software. The experiment began with a fixation cross, which remained onscreen for the duration of the experiment to minimize eye movements. Each stimuli were 1050 ms in duration, with an inter-stimulus interval of 1600ms. To equalize stimulus presentation between rise-time tones, extra time was added to each trial to equalize the tone length and keep the trial length consistent. This enabled each tone to have equivalent processing advantages. Participants were asked to respond as quickly and accurately as possible.

Results

Behavioural Effects

All results (accuracy, RT, and SDT) was analysed using a mixed three way GLM analysis which was conducted with rise-time (2), N (1-3), and trial type (target and non-target) as within subjects variables, and group (dyslexic and non-dyslexic) as the between subjects variable. The Huynh-Feldt adjustment (Huynh & Feldt, 1970) was employed as appropriate.

Accuracy

Mean scores were calculated for dyslexic and non-dyslexic participants for target and non-target trials at each WM load condition, for short and long rise-times. The results are summarised in Table 30.

Table 30: *Mean hit and correct reject scores for Experiment 8*

Rise time	Trial	Group	N-back		
			1	2	3
short	Target	Non-dyslexic	.78 (.12)	.71 (.18)	.74 (.17)
		Dyslexic	.78 (.19)	.72 (.21)	.64 (.23)
	Non-target	Non-dyslexic	.49 (.22)	.43 (.19)	.43 (.19)
		Dyslexic	.35 (.23)	.40 (.24)	.44 (.22)
long	Target	Non-dyslexic	.78 (.1)	.72 (.14)	.70 (16)
		Dyslexic	.76 (.21)	.64(.18)	.58 (.21)
	Non-target	Non-dyslexic	.55 (.15)	.48 (.16)	.46 (.09)
		Dyslexic	.51 (.27)	.48 (.22)	.42 (.21)

Note: Standard deviations are reported in parentheses.

The results revealed a main effect of N, $F(2, 60) = 9.29$, $p<.001$, $\eta p^2 = .23$, whereby accuracy decreased with increased WM load. There was a main effect of trial, $F(1, 30) =23.86$, $p<.001$ $\eta p^2 = .44$, where accuracy has higher for target trials. There was also a marginally significant interaction between trial * N * group, $F(3.28, 98.49) = 2.46$, $p=.094$, $\eta p^2 = .15$, whereby for

non-dyslexic individuals' in target trials, there is a reduction in accuracy between 1 back and 2 back, but not between 2 back and 3 back. However, for dyslexic individuals in target trials, there is a reduction in accuracy between 1 back and 2 back, and 2 back and 3 back. For non-dyslexic participants at non-target trials, N decreases accuracy, however for dyslexic individuals at non-target trials, there is little effect of N.

There was also a significant N * rise-time interaction, $F(2, 60) = 3.59$, $p<.034$, $\eta p^2 = .11$, where performance decreased for short rise times between 1 and 2 N-back, but not between 2 and 3-back. For tones with long rise-times, accuracy decreased from 1 to 2-back, and 2 to 3-back. Furthermore when N was 1 and 2, rise time increased accuracy. However in the 3 back condition long rise-times have a lower overall accuracy. Furthermore, there was a significant trial * rise-time interaction, N, $F(1, 30) =7.06$, $p=.013$, $\eta p^2 = .19$, where individuals were more accurate for target trials, suggesting a very liberal response bias. The between group effect was also significant, $F(1, 30) =4.45$, $p=.043$, $\eta p^2 = .13$, where individuals with dyslexia had an overall lower accuracy score, across all trials and tones (regardless of rise time). All other effects were non-significant, [Fs<1.71].

Proportion correct was also measured for target trials only, as an assessment of hit rate. Again, there is a main effect of N, $F(2, 60) =9.29$, $p<.001$, $\eta p^2 = .23$, whereby accuracy decreased as N increased. There was also a main effect of rise-time, $F(1, 30) =23.86$, $p=.04$, $\eta p^2 = .13$, whereby short rise-times led to a higher overall hit rate than long rise times. However this effect may be related to group, with a marginally significant interaction between rise-time and group, $F(1, 30) =3.12$, $p=.088$, $\eta p^2 = .094$. Individuals with dyslexia performed poorly

compared to non-dyslexic for tones with long rise times, but not those with short rise times[17]. All other effects failed to reach significance [*F*s<1.66].

Given that the marginally significant interaction term between group * rise-time, the analysis was followed up by a 2-way GLM analysis, for each group separately. For individuals without dyslexia, the effect of rise-time was not significant, $F(1, 14) = 0.97$, $p = .76$, $\eta p^2 = .007$. However, for the group with dyslexia, there was a significant effect of rise-time, $F(1, 16) = 6.54$, $p = .021$, $\eta p^2 = .29$, whereby individuals with dyslexia performed with reduced accuracy for long rise-times (.71 compared to .64 for short compared to long rise times)[18].

Signal detection theory

Means and standard deviations were calculated for non-dyslexic and dyslexic participants, at each level of N and each Rise-time (short v long), for d' and the criterion. The results are presented in Table 31.

[17] Although there was not a significant 3 way interaction between group * N * rise-time, independent samples between group analysis *t*-tests were assessed for the 1-back and 3-back conditions only, for long rise times. This was done in order to determine if the group * rise-time interaction was due to sensory perceptual impairments only (which are independent of WM), or if rise-time difficulties become worse in higher WM load conditions. The behavioural trend suggests that individuals with dyslexia show a greater impairment for rise-times at higher WM loads, compared to non-dyslexic participants (3-back; $p = .07$) but not smaller WM load conditions (1-back, $p = .27$).

[18] RT analysis revealed quicker RTs for short rise-time tones, $F(1, 30) = 26.81$, $p < .001$ $\eta p^2 = .47$. All other effects did not react significance [*F*s<.99].

Table 31: *Mean Signal Detection Theory Parameters for Experiment 8.*

Rise time	Trial	Group	N-back		
			1	2	3
d-prime	Short	Non-dyslexic	.87 (.66)	.60 (.51)	.39 (.43)
		Dyslexic	.40 (.54)	.50 (.41)	.26 (39)
	Long	Non-dyslexic	.92 (.61)	.58 (.63)	.44 (.47)
		Dyslexic	.85 (.59)	.26 (.53)	.15 (.41)
criterion	Short	Non-dyslexic	-45 (.49)	-.43 (.50)	-.44 (.52)
		Dyslexic	-.65 (.62)	-.51 (.64)	-.35 (.61)
	Long	Non-dyslexic	-.31 (.24)	-.34 (.34)	-.31 (.28)
		Dyslexic	-.34 (.68)	-.22 (.50)	-.24 (.55)

Note: Standard deviations are reported in the parenthesis.

A mixed GLM analysis was carried out on d-prime values, with N, rise-time, and group as the independent variables. This analysis revealed a main effect of N, $F(2, 60) = 16.20$, $p < .001$, $\eta p^2 = .35$, with decreasing d-prime values as N increased. Furthermore, there was a marginally significant main effect of group, $F(1, 30) = 3.10$, $p = .08$, $\eta p^2 = .09$. There was a significant interaction between N * rise-time, $F(2, 60) = 4.69$, $p = .013$, $\eta p^2 = .14$, where N decreased d' to a larger extent in the long rise-time condition between 1 and 2 back, than for short rise times. This effect also interacts with group, $F(2, 60) = 4.69$, $p = .013$, $\eta p^2 = .14$, whereby individuals with dyslexia have a lower d' value in the short 1 back condition, but comparable short rise-times in the 2-back and 3 back conditions. Individuals with dyslexia have lowest d' values in the 3-back condition with long rise times.

A mixed GLM analysis was carried out to examine the effect of N and group upon the criterion. Note that values below zero indicate a liberal criterion relative to optimal, whereas values above zero indicate a more conservative response bias relative to optimal. The analysis revealed a main effect of rise time only, $F(1, 30) = .11.85$, $p = .002$, $\eta p^2 = .28$, whereby

individuals had more negative criterion scores for short rise-times. This suggests that in the low rise-time condition, participants were more liberal, and more likely to state the stimuli had occurred N items back. All other effects upon the criterion did not reach significance [*F*s< 2.11].

Discussion

The aims of the current chapter were to examine how impairments at the auditory perceptual level might influence higher level WM processing, specifically in dyslexia. According to the MTRM (See Poeppel, Idsardi, Wassenhove, 2008; Goswami, 2011), auditory input is processed on multiple temporal scales. The auditory signal is fractionated in frequency and time. Amplitude changes over time provide us with crucial information about speech. In the auditory cortex, spontaneous oscillations at theta and gamma frequencies are modulated by the speech stimulus. The inherent cortical rhythms are thus essential for speech analysis.

It is the lateralized theta sampling network that is driven by slower temporal rates. The rise-time of the stimuli forms a crucial part of the amplitude envelope, marking syllabic stress. Theta oscillations, which are modulated by these tones, enable the temporal integration into syllable sized packets. This resets and slides to track the speech dynamics, as the speech rate varies. This resetting mechanism is thought to be driven by the onset of the edge of sounds (syllable rise times), and it reflects the neural encoding of the AE. Applications of this model to dyslexia have been made (See Goswami, 2011), which suggests that impaired phonological processing in dyslexia might be attributed to impaired phase locking to tones that take longer to reach their amplitude peak. Here, the extent to which rise-time affects WM performance is examined, by comparing tones with short versus long rise-times in an N-back task. It was hypothesized that if individuals with dyslexia had a WM deficit independent of their phonological impairment, then we would see a group difference for short rise-times, as well as

long-rise times. While the current data cannot argue against a general WM impairment, it indicates that tones with impaired processing (the long rise time tones), result in a larger impairment during the N-back task, for dyslexic individuals. Thus, it is likely that the phonological impairment amongst individuals with dyslexia confounds their WM performance.

The current results demonstrate that tones with shorter rise-times have an advantage in phonological WM. However, when conducting this analysis for non-dyslexic and dyslexic individuals separately, the results suggest that this effect is only present for individuals with dyslexia. For non-dyslexic individuals the effect of rise-time is not significant. This analysis was conducted based upon a strong a-priori hypothesis we had about rise-time processing in developmental dyslexia, and upon a marginally significant group by rise-time interaction, using a two tailed test. Thus, the current results are consistent with Goswami's (2011) Temporal Sampling Theory of Developmental Dyslexia, given that it suggests that an impairment with slow amplitude modulations contributes towards the phonological impairment in dyslexia. The current results are inconsistent with Tallal and her colleagues (Tallal, 1980 Tallal, Miller, & Fitch, 1993), who have argued that dyslexic individuals have specific difficulties processing rapidly changing, transient acoustic events. The ability to process rapid successive information is fundamental to setting up the phonological system. From this perspective, we might expect short rise-time tones to also show an impairment.

Given that we have demonstrated that tones with long rise-times are impaired more substantially in individuals with dyslexia we might question the extent to which individuals with dyslexia have a general WM impairment. Some previous research (e.g., Jeffries & Everatt, 2003, 2004 Kibby, Marks, Morgan, & Long, 2004), along with research conducted earlier in this thesis, has argued against a visual-spatial deficit, thus the evidence points away from a general central executive impairment. The current results are in accordance with this.

Furthermore, the results can inform previous research which suggests that phonological processes can account for the WM deficit in developmental dyslexia (Avons, & Hanna, 1995 Hulme, Roodenrys, Brown, & Mercer, 1995; McDougall & Donohoe, 2002), where it is suggested that slow articulation rate underlies the phonological WM difficulty. However, these results have been difficult to replicate (Swamspm & Ashbaker, 2000), as reading difficulties on WM and STM measures remain when articulation speed is partialed out from the analysis.

Previous research has also claimed that learning, encoding or using phonological representatons may be impaired in developmental dyslexia (Carroll & Snowling, 2004; Kramer, Knee, & Delis, 2000). Indeed, the current research suggests there may be a difficulty encoding specific stimuli into WM, since the disadvantage in long rise-times occurs at the perceptual level. The current results therefore allow us to revisit the question of whether or not problems with the WM system are merely a secondary effect of deficits in the phonological system.

Indeed the group * rise time, which reached marginal significance, is suggestive of a perceptual influence upon WM processing in dyslexia. However, the N * group * rise-time 3 way interaction was not significant. If individuals with dyslexia had a general WM impairment, then one might expect a between group difference to emerge for short rise-time tones, especially in higher WM load conditions. Furthermore, one might expect that dyslexic individuals should show a greater impairment, compared to non-dyslexic participants, for long rise time tones in higher WM load conditions, as this would suggest a specific contribution of WM. Simple main effects analysis demonstrates a trend in this direction, whereby the difference between dyslexic and non-dyslexic participants, for long rise-times increases as WM load increases. However, from the current data we cannot confirm the exact relationship between rise-time perception and WM in dyslexia, given the absence of this 3-way interaction.

However, we can be somewhat confident that the between group difference is larger in a WM task when the auditory stimuli have longer rise-times. Indeed, this has implications for questions posed by Beneventi et al., (2010), regarding the extent to which individuals with dyslexia have a general WM impairment, or a sensory perceptual impairment which confounds WM performance.

In accordance with this point, it is important to emphasize that WM processing, and sensory perceptual processing are not independent. WM involves holding and manipulating a small amount of information for a limited period of time (e.g., Baddeley & Hitch, 1974). Cowan (1999) suggests that WM can be defined by the collection of mental mechanisms that hold information in a temporarily accessible form, so that they can be used for cognitive tasks. However, the central executive, which selects and processes information, must interact with peripheral elements of memory, which are the buffers for short-term storage of small amounts of information. This auditory, or indeed visual information, is stored at different locations, and needs to be coordinated in order to provide coherent cognition. Thus, the WM system is not just a system of maintenance or manipulation, it is an interactive network, involving the frontal cortex, association cortex, and lower level sensory cortex (e.g. LaBar, Gitelman, Parrish, Mesulam, 1999). Thus, an impairment in lower level sensory processing is indeed an encoding problem, and consequently this may transpose into a maintenance problem. Thus the research question ‘do individuals with developmental dyslexia have a WM problem, or an auditory impairment?’ might be invalid, since the maintenance system, and the sensory perceptual system are not mutually exclusive. However, the current data suggests that perceptual difficulties might have consequences for the WM system overall.

The question as to why auditory entrainment at certain frequencies may lead to a disadvantage for dyslexic individuals in WM is however, one that still remains. The temporal

correlation hypothesis has proposed the mechanism of neural binding. Recent experiments suggest a powerful role of neural coherence between neural areas for the integration and binding of information (e.g., Steinmetz et al., 2000; Nakatani & van Leeuwen, 2006). Experiments on WM have demonstrated that the alpha rhythm in particular sensory regions (parietal or temporal) is associated with memory storage. Furthermore, the theta rhythm in frontal regions is associated with manipulating information in WM (Kawasaki et al., 2010). Along with the study of local oscillatory processes, connections between areas have also been studied. Sauseng et al (2009) has demonstrated cross-frequency phase synchronization between theta and gamma oscillations at parietal regions, which is associated with successful maintenance of information. Although there has been some disagreement on exactly which frequency bands concern which functions of WM, there is convergence in the literature to suggest that cross- frequency coupling and phase coding may serve as an important neural mechanism underlying WM processes (see Fell & Axmacher, 2011).

Individuals with developmental dyslexia have impaired phase locking in the theta range for longer rise-times (See Goswami, 2011). Here we have shown a trend towards individuals with dyslexia having lower performance in the N-back task, especially for tones with long rise times. This oscillatory behavior can be thought of as operating at the level of microcircuits, whereby inhibitory interneuron networks impose rhythmic synchronization capable of effectively controlling the gain of the neuronal spiking output (Bartos et al., 2007). For example, Kaysar and colleagues (2009) examined the temporal pattern of local field potentials (LFP) associated with complex sounds, in non-human primates in the delta, theta and gamma range. Neuronal spiking and LFP phase (entrainment or realignment) below 30Hz carried complementary information, enhancing the information received about the temporal structure of the signal. It has been suggested that the phase of the underlying neuronal oscillations that

generate the LFP may exert temporal control over neuronal excitability and gate spiking activity in such a way that it occurs at the most relevant times (e.g., Lakatos et al., 2005; Schroeder & Lakatos, 2009).

If individuals with dyslexia are impaired at phase locking (particularly for slower rise time modulations), then encoding might become impaired due to timing errors in the synchronisaton between early sensory areas, and higher order neural areas. This is because the coherent output from local neuronal groups needs to selectively synchronize over long-range connections, with task relevant neuronal groups in more distal brain regions (e.g., Buschman & Miller, 2007). The long range connections become responsible for maintaining the representation. Impaired phase locking in the theta range might have more widely distributed consequences. Therefore, impairments in rhythmic synchronization within a neuronal group may not only decrease the impact on postsynaptic target neurons in a feed-forward manner, but may also reduce the areas' ability to communicate with more distal regions.

Future research

Following on from the current work, a variety of future investigations might be proposed. Here, although there was a main effect of group upon WM performance, there was also an interaction between, group * rise-time. The relationship between this interaction and WM load can be examined further in future experiments in order to determine if individuals with dyslexia have an impairment for short rise-time tones at larger WM load conditions. The descriptive data shows that in the 3-back condition, individuals with dyslexia show a trend for an impairment for short rise-times, but this effect is not significant. A suggestion for future research might be to conduct this experiment with higher N-back values to determine if a group effect emerges at the short rise times. However, given 50% was the change level in this experiment, 4-back might put dyslexic participants at chance level of

performance. Alternatively, the experiment could be repeated for low rise-time tones only, increasing to higher N values in order to determine if there is an N * group *rise-time interaction. Or, the interaction could be probed further by using a task that has a lower demand upon executive functioning. This would increase overall performance, and allow the relationship between rise time perception and WM processing to be examined. Finally, now the behavioural effects have been explored, a follow up experiment could be conducted with EEG, in order to examine ERP effects associated with rise-time processing during the N-back task. Furthermore, one could correlate phase-locking to the AE with WM performance, in order to directly test the relationship between perceptual processes and WM performance.

This research has the potential to inform the design of training experiments. Given that impaired phase locking or entrainment to slow rise-times may contribute towards the WM impairment in dyslexia, entrainment to auditory or visual stimuli within the theta range could act as WM training in dyslexia. As mentioned previously, the auditory system synchronises with ongoing oscillations to the modulation rates in the stimulus, realigning the phase of neural activity so that peaks in excitability co-occur with peaks in amplitude modulation (Zion Golumbic et al., 2012). A theoretical investigation could be conducted to examine entrainment effects in dyslexia.

Specifically, entrainment could be conducted through visual or auditory domains, or through brain stimulation (transcrandial direct current stimulation (tDCS) or Transcranial magnetic current stimulation (TMS)). In the visual domain it has been shown that delivering TMS shortly after the end of a visual stimulus can cause a TMS induced echo of the visual percept (Liao, Wu, Halelamien, Shimojo, 2013). Furthermore, neurons in the visual cortex synchronize their firing rates to the frequency of a flickering light (Herrmann 2001; Williams et al., 2004). In the auditory domain, presenting the auditory stimuli in phase with the TMS

entrainment may lead to enhanced auditory perception of the stimuli. Entrainment could be conducted simultaneously in auditory or visual domains, exploiting audio visual integration.

Visual entrainment may be able to aid, or scaffold, auditory entrainment in developmental dyslexia. Recent research has demonstrated that visual entrainment should also play a crucial role in the mechanisms that underpin the development of speech representations (e.g., Power, Mead, Barnes, & Goswami, 2012). The preferred phase of auditory entrainment was altered in the presence of congruent visual entrainment. There is therefore a visual influence over auditory processing, with visual rhythmic stimulus streams modulating auditory oscillations to the optimal phase for auditory processing and audio-visual integration (Schroeder et al., 2008). For example, one could conduct an auditory N-back task where stimuli presented before the target in the theta frequency, could aid target detection when the target occurs in phase with the stream. However, if the target occurs out of phase, perception may decrease. These effects could be explored in dyslexia, particularly to assess entrained versus non-entrained conditions. Furthermore, an additional experiment could be conducted where the stimuli preceding the target are presented at random frequencies, which should not entrain the theta range, or in the theta frequency. One could also explore the long term learning effects associated with entrainment at the theta frequency. Entrainment at the theta range could lead to increased phase locking over time, thus, when stimuli occur in phase, phase locking should be enhanced in dyslexia.

To conclude, the current chapter has provided some initial evidence that individuals with dyslexia may have impaired WM for long rise-times, over tones with short rise-times. This suggests that the WM difficulty in dyslexia might be confounded by lower level auditory processing difficulties. Such experiments would also lead to a theoretical advancement, given that they would provide more knowledge about the contribution of the phase of the stimuli to

successful WM encoding in dyslexia. This might have implications for future WM training studies examining neural entrainment in dyslexia. Finally, if more support can be given to demonstrate that individuals with dyslexia have impaired WM for long rise-times, then this may have implications for behavioural learning interventions, in that individuals with dyslexia might maintain information in WM more successfully when speech processing is optimal (short, sharp beats).

Chapter 7: Discussion and General Conclusions.

In the final chapter of this thesis, the aims of the thesis are reviewed. Following this, an overview of the experimental work is presented, along with a discussion of the key findings, and how they link in with previous research in this area. Finally limitations and theoretical implications are addressed. This chapter ends with a new hypothesis regarding phonological WM processing in dyslexia, and some proposals for future experiments which build upon the experimental work in this thesis.

Summary of original contribution, aims and rationale.

The general aims of this thesis can be readdressed with a summary of chapters 1 and 2. Particularly Chapter 2 raised two important questions regarding WM processing and dyslexia. The first questioned the extent to which the WM deficit in dyslexic adults was due to a specific phonological loop deficit, or a general impairment with central executive processing. The second question examined in this thesis regarded the extent to which the phonological loop deficit in dyslexia can be influenced by lower level difficulties with language. Indeed, individuals with developmental dyslexia have a difficulty with the temporal sampling of language (see Goswami, 2011). Thus, for the first time, the temporal sampling theory of developmental dyslexia (Goswami, 2011) was applied to a WM context, in order to understand the extent to which individuals with developmental dyslexia are impaired or unimpaired for auditory sound modulations that they are able to process efficiently, versus inefficiently.

In terms of the central executive hypothesis, similar questions have been asked in recent years, predominantly examining WM performance across simple and complex span tasks, in children (e.g., Wang & Gathercole, 2013; Menghini, Finzi, Carlesimo & Vicari,

2011). Work in dyslexic adults is sparse, however across two publications Smith-spark et al. (2003) and Smith-Spark and Fisk (2007) have argued for a central executive impairment in dyslexia. However, this work did not dissociate active versus passive WM processes within a single experiment, but instead controlled for simple span task performance when analyzing complex span performance. Furthermore, all previous investigations have been in the behavioural domain. Thus, some of the original contributions of Experiments 1-7 are that they examine central executive (or phonological loop) processing in dyslexic adults. Specifically, Experiments 1, 2, 3, 6, 7 and 8 use the N-back task, while Experiments 4 and 5 use a task paradigm specifically designed to tap into passive storage versus active maintenance. The electrophysiological response during these tasks is measured, and compared between groups. The P300 is predominantly investigated, as a measure of resource allocation (amplitude). The latency of the component indexes speed of the matching process (see Chapter 1, and Watter, Geffen and Geffen, 2001 for a full review), and thus, analysis of the P300 can provide additional insights into the speed at which individuals perform the matching process in a WM paradigm.

Thesis summary: Overview of Chapters, and Experiments.

In Chapter 1, the paradigms and event-related potential (ERP) technique was reviewed. In the ERP literature, Gevins et al. (1996) conducted the first WM ERP studies with the N-back task. Using dense-array EEG (dEEG), these authors noted early, mid and late latency components which differed as a function of WM load. Watter, Geffen and Geffen (2001) employed a visual- spatial N-back paradigm, with four different memory loads (0, 1, 2 and 3), to examine the dual task nature of this task. The results provided strong evidence for the dual task nature of the N-back paradigm. P300 latency was consistent in the 1, 2 and 3 back tasks, indicating that the cognitive requirements for selecting a match or mismatch were

equivalent for the different N-back tasks. This contradicts studies that used Sternberg's paradigm (e.g., Kramer et al., 1991), where P300 latency increased with increased WM load. Watter, Geffen & Geffen, (2001) demonstrate that latency remains consistent in the N-back task because participants have already sufficiently selected the appropriate N-back stimulus in WM, in preparation for comparison. Therefore, if participants are able to maintain N items, in order to compare it to the upcoming stimuli, P300 latency remains constant as N increases. However, P300 amplitude decreases progressively as WM load increased, and resource allocation decreases. This was consistent with previous findings (Gevins et al., 1996; McEvoy et al., 1998). Studies that employ ERP methodology can make use of both experimental design and the temporal properties of ERP features to infer functional significance. Therefore, in Experiments 1-7, the time course (latency) and amplitude of the P300 were assessed in dyslexia, across different modalities of memory. Typically, ERP experiments of visual WM have also examined the N2 (Riby & Orme, 2013), and thus in Experiments 1, 2, 3, 4 and 5 of this thesis, where stimuli were presented in the visual domain, the N2 is also examined. Riby and Orme (2013) interpret increased task demands in WM processing to result in a larger N2 response due to the demands placed upon visual attention.

Experiments 1, 2, 3, 6, 7 and 8 in this thesis, use the N-back task. The N-back task has been used as a robust measure of WM since 1958 (Kirchner, 1958), because cognitive load (the number of items that are stored within WM) can be easily manipulated. In addition, different stimulus features and modalities are easily incorporated into the paradigm (Luu, Caggiano, Geyer, Lewis, Cohn, & Tucker, 2014). Furthermore, the more general central executive components of WM can be kept constant between experiments of different modalities, and assessed at different WM load conditions across modality. This property allowed it to be used to study WM across various sensory modalities, in developmental

dyslexia, where very little is known about the time course of these ERPs during WM functioning.

As outlined in Chapter 2 of this thesis, controversial results been reported regarding the central executive system functioning in dyslexia. Indeed, while some studies have documented reduced efficiency of this system in dyslexics (Smith-Spark, Fisk, Fawcett & Nicolson, 2003; Smith-Spark & Fisk, 2007; Wang & Gathercole, 2013) many have not (for a full review see Snowling, 2001). Smith-Spark and colleagues (Smith-Spark et al., 2003) suggested a possible link between deficits in visual-spatial span and reduced efficiency of the central executive in dyslexia. These effects were probed in Chapter 3. In this chapter, visual presentation was adopted across verbal, and visual-objects (pictures, and Chinese characters stimuli), in order to compare verbal and visual WM processing in developmental dyslexia. In Experiments 1 (verbal) and 2 (pictures) a 2: 1 ratio of non-target: target trials were presented, whereby the probability was 2 times greater on each trial that the stimuli would be a non-target trial. Presentation times were also very short for an N-back task (500ms, followed by a delay period of 500ms), but are consistent with short encoding times often used in visual WM presentation tasks (e.g., Luck and Vogel, 1997). These short presentation times allowed us to tax WM processing, in order to uncover any underlying impairment. Visual impairments are reported in the literature (e.g., Smith-Spark & Fisk, 2007), but the finding is inconsistent, with many researchers arguing for a verbal deficit only (e.g., Vellutino, Fletcher, Snowling, & Scanlon, 2004), and have failed to uncover a visual-spatial WM impairment (e.g., O'Shaughnessey & Swanson, 1998; Schuchardt et al., 2008).

The results of Experiments 1 and 2 suggest that dyslexic individuals were impaired for verbal information only. In Experiment 1, individuals with dyslexia have impaired accuracy across all N-back tasks, with a reduced hit rate. However, accuracy for non-target

trials (correct– rejects) was not reduced. This was reflected in an increased criterion score in the dyslexic group, which suggests that individuals with dyslexia used this probabilistic information to perform the task. Thus their overall accuracy, across hits and correct rejects was adequate (no significant difference between dyslexic and non-dyslexic individuals). In contrast, in Experiment 2 and 3, this criterion difference was not significant. Experiment 2 used pictorial visual-object information, still maintaining a 2: 1 ratio of non-target: target trials. Experiment 3 used Chinese characters, which could not be verbally recoded, and did not contain any semantic information, but there was a 1: 1 ratio of non-target: target stimuli. In both Experiments 2 and 3, individuals with dyslexia did not show this criterion effect.

Furthermore, when auditory verbal stimuli were used, and the trial ratio was made equivalent (as in Experiments 6 and 7), this criterion shift was not observed. It is likely that individuals with dyslexia become more conservative and use this probabilistic information only in contexts where they have an impairment (the verbal stimuli in Experiment 1). Critically, this impairment was also associated with a reduced P300 amplitude response (as in Experiment 1). The phonological loop (Baddeley & Hitch, 1974, Baddeley, 2000) is responsible for the re-coding of visually presented verbal material into a phonological format, and is thus a likely candidate for the impairment observed in Experiment 1.

However, the results of Experiments 2 and 3 of chapter 3 were inconsistent with a general central executive impairment in dyslexia, as visual-object processing even at higher N-back levels was unimpaired. Thus we might question why previous research has demonstrated a central executive impairment, and the current results did not. Only one experiment to my knowledge has demonstrated impaired visual object WM in dyslexic children (Menghini et al., 2011). However, Smith-Spark et al. (2003) and Smith Spark and Fisk (2007) demonstrated an impairment for visual WM when assessing spatial information

only. The impairment was found for complex span tasks, which they attributed to a general central executive impairment. One possibility is that we do not replicate central executive impairments, because Experiments 2 and 3 use visual-object information.

Indeed the visual spatial sketchpad can be differentiated into two components: visual object, and visual spatial. Visual object information includes item information e.g., colours, texture, shape, while visual-spatial, information refers to the spatial location of objects (where). Both are processed by functionally independent subsystems (Smith et al., 1995; Vicari, Bellucci &, Carlesimo, 2006), with the dorsolateral pre frontal cortex (DLPFC) becoming active for spatial WM processing (e.g., Curtis, & D'Esposito, 2003). One possibility is that the DLPFC is responsible for the visual spatial impairment in dyslexia observed by previous researchers (Smith-Spark et al., 2003; Smith Spark & Fisk, 2007; Wang & Gathercole, 2013). This is likely, given that the DLPFC is a neural candidate for manipulation and visual – spatial WM processing. This might explain why the impairment is often found for spatial but not object WM. Thus, the following two experiments (Experiments 4 and 5) were designed to tap into visual spatial WM processing in dyslexia.

As described in Chapter 4, the DLPFC is also associated with the active manipulation of items in WM. Previously in the WM literature, maintenance and manipulation, or central executive related processes have been difficult to dissociate (Glahn et al., 2002). Therefore, to examine the extent to which the central executive is impaired in developmental dyslexia, the visual-spatial sketchpad was probed further, in Experiments 4 and 5. Experiments 4 and 5 examine visual-spatial WM processing in dyslexia with a spatial delay response task (SDRT; Glahn et al 2002).

In Experiment 4, participants were required to encode and maintain, 1, 3, 5, and 7 dots, which were presented at pseudorandomised locations on the screen. After a fixed delay,

participants were required to state if a probe did, or did not occur in the same location as a dot from the target array. In Experiment 5 passive maintenance versus active manipulation were directly compared within a single experimental design. Participants saw a pre-cue which instructed them to maintain, or manipulate three dots presented above a horizontal meridian line. In the manipulate condition, participants were required to flip the dots across the horizontal line, and then respond 'yes' if the cue display matched their mental representation. Across Experiments 4 and 5 it was hypothesized that if individuals with dyslexia have a general central executive impairment, they should show an impairment at high WM loads (5 and 7 targets) in Experiment 4, and in the manipulation condition only in Experiment 5.

The results of Experiment 4 argue against a general visual – spatial WM impairment in dyslexia, even in higher WM load conditions. Participants with dyslexia showed comparable accuracy (hit rate, d' and RTs) as non-dyslexic individuals. Furthermore, ERP analysis did not reveal any between group differences in terms of amplitude or latency. Thus, even for higher WM loads, individuals with dyslexia were unimpaired for visual-spatial WM load maintenance. In Experiment 5, we hypothesised a more robust group difference for the manipulation condition, if individuals with dyslexia have a general central executive impairment. Once again, the accuracy (hit rate and d'), and ERP results, did not support the idea that individuals with dyslexia have impaired central executive functioning. However, a significant trial (target versus non target) * group (non-dyslexic versus dyslexic) * condition (maintenance versus manipulation) interaction was observed in the RT data. This interaction was due to individuals with dyslexia, who for target trials were significantly slower than non-dyslexic individuals, when they were required to manipulate information in WM. However, when examining target trials only, the between group difference was only marginally significant.

Further, in Experiment 4, for the P300 amplitude analysis, the P300 peaked in the right hemisphere for dyslexic individuals only. Overall, there was a comparable P300 amplitude at both the midline and lateral electrodes. For latency, which indexes stimulus evaluation time, though, a midline analysis demonstrated an interaction between load * group, whereby for non-dyslexic individuals there was no effect of WM load upon latency. For dyslexic individuals, there was an effect of WM load upon the P300 latency, whereby the P300 peaked later as WM load increased. In Experiment 5, these P300 latency differences were not observed between groups[19]. Across Experiments 4 and 5, the results suggest that in both conditions of maintenance and manipulation, attentional resource allocation, and processing demands did not differ between groups. However, as shown in Experiment 4, there might be subtle differences in stimulus evaluation time, with individuals with dyslexia showing longer latencies as N increases at encoding. This might reflect early attempts to rehearse visual information online, and earlier engagement of the memory matching process.

However, these subtle ERP effects were predominantly topographic, and a more general central executive, or WM difficulty should result in impaired N2 amplitude, or a P300 amplitude difference in the visual domain, as we saw in the auditory domain (Experiment 1). Given that hit rate also did not differ between groups, the overall conclusions of Experiments 4 and 5 are that individuals with developmental dyslexia do not have impairments in maintaining visual spatial information online, even as WM load is increased. Thus, the current results do not lend great support to Mehini et al (2011), Smith-Spark et al. (2003), Smith-Spark and Fisk (2007) or Wang and Gathercole (2013). If, as argued by the authors, individuals with

[19] For the N2 analysis there were subtle topographic differences in the brains electrophysiological response between groups. There was a significant region by group interaction, whereby the N2 was more negative in anterior regions, and more positive in posterior regions for individuals with dyslexia. However, these effects should not be overly interpreted. Analysis at the anterior, and posterior regions separately did not give rise to any between group differences in N2 amplitude.

dyslexia had a general central executive impairment, then one would expect this impairment to persist regardless of the modality of visual memory (visual-object, or visual-spatial). However, in Experiment 5 there was a trend towards individuals with dyslexia being slower for target trials where they were required to manipulate information. However, their accuracy was comparable, which suggests that there was no difference in the mnemonic representation between groups. Furthermore, one would expect reduced accuracy, and reduced ERP response in the manipulation task if there was a domain general central executive impairment.

The final two ERP experiments were presented in Chapter 5 of this thesis. The aims of these experiments were to explicitly examine ERP responses associated with phonological WM, and to remove some of the limitations present in Experiment 1 Chapter 3. Specifically, in Experiment 1, there was a reduced P300 response in individuals with dyslexia. It is likely that participants phonologically recoded the letters, but not certain. In Experiment 6, participants took part in a verbal N-back task, in the auditory domain, which unlike Experiment 1 is a direct test of phonological WM. Furthermore, stimulus presentation time was lengthened compared to Experiments 1, 2 and 3, and the non-target: target ratio was made equivalent. The findings demonstrated that without probabilistic information, individuals with dyslexia had reduced d' values relative to controls, which demonstrates that they were unable to discriminate signal (target items) from noise (non-target items). In this context, adopting a more conservative response bias did not have an accuracy advantage, because the 1: 1 ratio of non-target: target trials means that responding 'no' is less likely to result in a correct reject. Thus, signal discrimination became harder. Individuals with dyslexia show a significant P300 amplitude reduction in posterior regions, compared to non-dyslexic individuals. In Experiment 7, auditory words were presented, which were manipulated by their AoA. In this context, the effects were slightly weaker, which might be due to the concrete and complex nature of the stimuli, and because the words also contained semantic

information. Indeed, semantic information is able to scaffold WM processing (Riby & Orme, 2013). However, group differences are observed in the behavioural hit rate, and P300 differences were seen at higher WM load conditions. Furthermore, individuals with dyslexia showed latency differences for words learnt later in life. During the AoA task, latency became shorter as N increased, which might suggest that evaluation time reduced as individuals became more practiced at the task. However, this effect was not observed for dyslexic individuals for words learnt later in life. This might be due to impaired access to phonological stimuli, which prevails in the 'late' learned word condition.

The ERP and behavioural results (reduced P300 amplitude, and decreased hit rate) in Experiments 6 and 7 are consistent with Vellutino (1979) who suggested that individuals with dyslexia have a systematic difficulty on tasks which incorporate a verbal component, however, they perform at the same level with non-dyslexics on tasks without a verbal component. Therefore, the findings from WM experiments, that tapped into phonological processes (including Experiment 1, 6, and 7) are in accordance with previous research which has demonstrated that individuals with dyslexia have difficulty on tasks that involve phonological storage (e.g., Ackerman & Dykman, 1993; Mann, Liberman, & Shankweiler, 1980; Roodenrys & Stokes, 2001) and on measures of verbal complex span (e.g., de Jong, 1998; Pickering, 2006b; Swanson, 1999; Swanson & Ashbaker, 2000).

A general central executive impairment, or a more specific lower level phonological impairment?

A large body of research suggests that dyslexia stems from an underlying deficit in the phonological processing system (e.g., Beitchman & Young, 1997; Lyon, 1995). The research presented and discussed thus far argues against a general central executive dysfunction. Previously, it was considered that visual spatial information may be impaired,

particularly when participants were required to manipulate it. However, the evidence for this was weak. While the results of Experiments 4 and 5 are not conclusive for arguing against a visual-spatial WM impairment (subtle RT differences were observed), a general central executive impairment is not concluded. A *general* central executive impairment, as suggested by Smith-Spark et al. (2003), Smith-Spark and Fisk (2007) or Wang and Gathercole (2013), would predict that individuals with dyslexia should be impaired for visual-object information too. Even under conditions of high demand (as in the rapid presentation rate in Experiments 2 and 3), these differences were not observed. Thus, from the current results, it seems unlikely that adults with developmental dyslexia have a domain general difficulty manipulating, and actively holding information online.

Thus, in response to the initial aims and questions of this thesis, the current results suggest against a general central executive difficulty in adults with dyslexia, particularly because there were no accuracy or ERP differences between individuals with or without dyslexia for Experiments 2, 3, 4 and 5, and critically individuals with dyslexia were not impaired at manipulating items in visual WM (Experiment 5). Instead they point towards a more specific phonological loop dysfunction. In Experiment 6, the results suggested that the auditory WM impairment in dyslexia may be confounded by a lower level perceptual difficulty with language processing. In vision WM research, the influence of attentional or perceptual biases upon WM maintenance are explored, and research suggests that lower level bottom up factors that influence encoding, can effect WM maintenance (see Shapiro & Miller, 2011). Here, perceptual difficulties with auditory WM are thought to be partly responsible for the overall WM impairment in dyslexia.

This does not mean that the central executive is not implicated in the WM impairment in dyslexia at all, there might be some more subtle difficulties with manipulation that depend

on the type of information being held online. Indeed, there are different levels of central executive processing which might operate at different levels of the WM model (Baddeley & Hitch, 1974; Baddeley, 2000). For example, the central executive system may interact differently with visual-spatial, as opposed to visual-object information. Furthermore, accessing phonological constructs might differ depending on the nature and accessibility of the phonological stimuli (e.g., AoA effects). Both of these processes are very different to actively manipulating the information over time. This suggestion is in accordance with Ramus and Szenkovits (2008) who suggest that phonological STM processes in dyslexia are limited, specifically in terms of conscious access to phonological material. This is an executive task, but it is domain specific.

Therefore, in Chapter 6 of this thesis, a single behavioural experiment was presented that tests the extent to which the WM impairment in developmental dyslexia is just a secondary effect of a low level auditory processing deficit (see Beneventi, Tonnessen, Ersland, & Hughdahl, 2010 for a review of this debate). Indeed, the Temporal Sampling Theory of Developmental Dyslexia (Goswami, 2011) proposes that individuals with dyslexia have a difficulty phase locking to slower modulations in speech (the theta frequency), and thus have a difficulty processing slower rise-times. Experiment 8 aimed to determine how an impairment in rise time modulation affects working memory performance, and the extent to which these two processes are independent. An N-back task was used, where stimuli consisted of 10 sinosoidal tones, either with short or long rise-times.

The results of Experiment 8 demonstrated that tones with shorter rise-times have an advantage in phonological WM, as reflected in higher accuracy scores, and a higher d'. However, when conducting this analysis for non-dyslexic and dyslexic individuals separately, the results suggest that this effect was driven by the dyslexic group, who showed a reduced

hit rate for long rise-time tones. For non-dyslexic individuals, the effect of rise-time was not significant, but the effect of rise-time was significant for dyslexic individuals. This analysis was conducted based upon a strong a-priori hypothesis we had about rise-time processing in developmental dyslexia, and upon a marginally significant group * rise-time interaction. The results suggest that an impairment with slow amplitude modulations does contribute towards the phonological impairment in dyslexia. Although here, it is difficult to determine if this is a perceptual difficulty, or a maintenance difficulty – the latter suggesting a primary role of WM. However, as discussed in Chapter 6, even perceptual difficulties are tied in very closely to encoding difficulties, and thus cannot be dissociated from WM processing. Here, in a WM task, it is demonstrated that the WM difficulty is specifically for tones with slower modulations. The results were therefore largely consistent with our predictions, and support work from Goswami (2011).

These results raise the question as to why auditory entrainment at certain modulations may lead to a disadvantage for dyslexic individuals in WM. As suggested by Goswami (2011), individuals with dyslexia show impaired phase locking, in the theta range, to slower amplitude modulations. Recent experiments suggest a powerful role of neural coherence between neural areas for the integration and binding of information (e.g., Steinmetz et al., 2000; Nakatani & van Leeuwen, 2006). Thus, this impaired phase locking process to the amplitude envelope can be considered at the local level. However, along with the study of local oscillatory processes, connections between areas are essential for adequate processing. Cross- frequency coupling and phase coding may serve as an important neural mechanism underlying WM processes (see Fell & Axmacher, 2011). If individuals with developmental dyslexia have impaired phase locking in the theta range, specifically to longer rise-times (see Goswami, 2011) then, neuronal spiking and LFP phase (entrainment or realignment) may be

reduced. This is because the phase of the underlying neuronal oscillations that generate the LFP will have reduced temporal control over neuronal excitability, so will be unable to gate spiking activity in such a way that it occurs at the most relevant times (e.g., Lakatos et al., 2005; Schroeder & Lakatos, 2009).

Theoretical implications: a new hypothesis.

To some extent, the conclusions made in this thesis fit and have new implications for the phonological access hypothesis, proposed by Ramus and Szenkovits (2008). Ramus and Szenkovits (2008) argue against degraded phonological representations in dyslexia, and instead argue for impaired phonological access. However, it might be argued that impaired phonological representations, which would emerge from a difficulty with phase locking to certain auditory sound modulations, may result in impaired phonological access. As described in Chapter 2, the phonological access hypothesis describes the process by which lexical and sub-lexical phonological representations are accessed for external computations. This process could become impaired via the faulty timing mechanism mentioned above. Verbal STM requires access to phonological representations for the purpose of copying them into buffers, then access to phonological buffers for retrieval.

If the phonological loop requires conscious access to input representations, timing is essential. It is here that impaired phase locking could impair the gating of spiking activity in such a way that the system becomes temporally inefficient. This proposal is consistent with previous research which argues for auditory STM temporal order errors in dyslexia (Trecy, Steve & Martine, 2013). This will also have consequences for the conscious access to input representations which is required to recycle the phonological representation to an output representation. The conscious access to phonological representations may place special demands on executive mechanisms, which control access to phonological representations.

While the conclusions of this thesis have been that overall there is no general central executive impairment, lower level difficulties with auditory processing specifically in terms of timing and entrainment could have consequences for the WM system. This could still be in terms of encoding, or re-access and recycling of information within the phonological loop. Therefore, impaired phase locking in the theta range might have more widely distributed consequences.

As discussed previously, it is unlikely that adults with dyslexia have a global and general central executive impairment. However, the impairment may arise through central executive processes interacting with a particular modality (as seen in Experiment 6, where group differences only emerged at higher WM load conditions), or when specific complex stimuli are used (long rise times). The current results suggest it is important to fractionate the WM model (Baddeley & Hitch, 1974) when considering WM impairments. The model provides an excellent framework for isolating specific impairments in disorders. However, the model also specifies links between the central executive and subsystems. Thus, it is important to move beyond classifying the WM impairment in developmental dyslexia into a specific component of the model, but to consider interactions (the links) between the different components of the model. For example, the interaction between the phonological loop, and the central executive. One can also assess the influence of sensory processing upon these interactions.

Thesis Limitations

Though showing a consistent pattern of results, the current work has limitations, which will now be considered. The first considered here is the picture stimuli used in Experiment 2, which were semantic in nature and also have the potential of being verbally recoded. However, because the group effect was not found, and short presentation times were used, it

is unlikely that participants were verbally recoding. However, since semantic information may have still facilitated WM performance, this limitation was addressed in Experiment 3, which used Chinese characters that could not contain any semantic information, and could not be verbally recoded. In the first empirical chapter, the presentation times and ISI for all experiments were very short (i.e. 500ms for encoding, and 500ms for the delay period), especially for a task that involves active manipulation of stimuli. These presentation times were chosen to tax WM and to reveal any underlying visual WM impairments. However, for some individuals, this made the task quite difficult. Critically, in the 4-back conditions, accuracy dropped to chance for dyslexic individuals. Low accuracy is not ideal for ERP research which in this case required the analysis of correct trials only, where an ERP is a signal to noise ratio.

The P300 analysis aimed to counteract this by collapsing across ERP conditions (1 and 2 back, and 3 and 4 back) to ensure there was enough signal within the average. An ERP is a measure of signal to noise, so collapsing across conditions ensured that the signal to noise ratio was higher, and thus the statistics reflected signal. However, one limitation which could not be addressed was the influence of encoding time. If encoding time differs between groups, then one group (potentially individuals with dyslexia) could be limited more than the non-dyslexic group. Furthermore, a control WM condition was not implemented, so we could not identify if participants with dyslexia were just impaired at processing the letters overall. Both these limitations were addressed in Chapter 5, where the stimulus presentation time was increased to 1500ms, and the ISI was increased to 1600 Ms. With the addition of a baseline 0-back condition, and with slower presentation times, the results demonstrated that individuals with dyslexia were not impaired in the 0-back or 1-back conditions. However, the between group impairment emerged as WM load increased.

We also averaged the ERP signal across conditions in Chapter 5, by collapsing the 0 and 1, 2 and 3, and 4 and 5 back conditions, to compare low, medium and high WM load conditions respectively. Collapsing across conditions prevents too many comparisons at a later stage especially once behavioural effects have already been established at the individual N-back levels. Collapsing across conditions is common in the ERP literature, because it results in fewer *p*-values (especially when electrode and or topographic location are factors), fewer spurious interactions, and smaller experiment wise error. Without the collapsing of conditions, the AoA ERP analysis would consist of 12 waveforms (WM load = 6, and AoA = 2) for non-dyslexic participants, and 12 waveforms (WM load = 6, and AoA = 2) for dyslexic participants. Furthermore, it allows us to increase signal within the ERP and ensure that the average reflects signal, as opposed to noise. However, one potential limitation is the averaging of the 0 and 1 back condition, as the two reflect independent processes. In many ways these conditions are alike in terms of their low cognitive load. However, in the 1 back condition there is no lure, while in the 0-back condition, there are lures before a target is reached. Furthermore, in the 1 back condition, WM updating occurs, whereas WM updating is minimal in the 0-back condition. Collapsing 2 and 3 back, and the 4 and 5-back conditions, should not provide any limitations. Therefore, the analysis was repeated without the 0-back and 1-back condition, and the between group statistical effects did not change. Thus, any difference between conditions is likely to be affected by cognitive WM load, as opposed to averaging across conditions.

A third limitation relates to the timings of the SDRT in Chapter 4. These experiments were replications of the fMRI tasks used by Glahn et al (2002), and thus we replicated the methodology with the same timings. Furthermore, it was considered important to give participants enough time to perform the manipulation condition. It was essential to keep this

timing consistent between the load experiment, and the passive and active WM conditions, in order to make direct comparisons. Replicating Glahn et al. (2002)'s specific timings was essential given we were relying on the activation of specific neural areas (the DLPFC and the VLPFC). Given the poor temporal quality of fMRI, it was unclear when these different components of the WM system became active in this specific task. Future research could reduce the encoding presentation time, and compare between group differences. Although this was a limitation, it is still important to note that individuals with dyslexia show a phonological impairment even for tasks which are not temporally demanding (such as digit span, where participants have 1s to encode each stimulus). Furthermore, Luck and Vogel (1997) demonstrated that stimuli encoding time did not lead to improved visual STM performance, indicating that the time dedicated to perceiving and encoding the stimuli was not a limiting factor for visual STM. However, reducing encoding, or maintenance time might have led to between group differences, as it would have limited any involvement that LTM could have played in supporting performance. Thus, this task could be repeated in the future, with a 500ms encoding time, and 1500ms maintenance time.

Furthermore Experiment 8 includes a potential limitation. It was acknowledged that the tones were different lengths, reflecting the short / long rise-times. To address this problem, during task design we added blank time to the end of each recording, so that overall participants would have the same time to encode each stimulus. Adding white noise, as in Evans, Selinger and Pollak (2011), was considered, however, it was unclear how that would affect phase locking to each stimulus, or influence signal detection (via perceptual masking). The effect of increasing the steady state of the stimuli was unknown, and thus it was considered important to replicate Richardson, Thomson, Scott, Goswami (2004)'s stimuli directly. Furthermore, if participants used the length of the stimuli to discriminate them as the N-back stimuli or not, then this would not lead to a specific difference in hit rate or d'

between long versus short rise-times. In addition, an influence of length cannot explain the rise-times effect in the dyslexic group only. Thus, it is likely that the effect of rise-time results in genuine between group effects in Experiment 8.

Finally, across experiments, N was sequentially manipulated across blocks from low to high WM load. It was considered too cognitively demanding to start the experiment at a high N, and we anticipated that a different criterion might be adopted between groups. The possibility that dyslexic individuals became tired during the experiment, which gave rise to the between group results is possible but unlikely, given that between group results did not emerge in the same experimental design when visual information was used.

Future research

There are key aspects of this work which could be developed for future research, both in the visual and phonological domains. In the visual domain, work conducted in this thesis found weak effects. In Experiment 5, there was a RT difference, which manifested as a task * group * trial interaction, which did not reach significance upon simple effects analysis. The visual WM impairment in some contexts (which may not have been examined, or highlighted here), could obtain. For example, as stated previously, Experiments 4 and 5 could be conducted again, but with shorter presentation times to prevent any information transfer between short term, and LTM. Furthermore, Experiment 5 could be conducted without a pre cue (instruction to maintain or manipulate). A retro cue could be presented in order to instruct participants to maintain or manipulate the information within the maintenance period. This would prevent participants from asserting any top down control over the task, and would prevent them manipulating any information within the encoding period. If an effect of manipulation could be seen during the delay period for a retro-cue, but not a pre-cue, this would suggest that individuals with dyslexia were able to use top – down control to override any WM difficulty.

Furthermore, it could be that only sub-groups of dyslexic individuals have visual WM impairments. Thus, in groups of individuals who do show visual processing impairments, or visual object span differences, these impairments can be probed further b using an analysis based upon individual differences, rather than group differences. Visual object and visual-spatial information are just two types of visual information, and both have varying levels of complexity. Individuals with dyslexia might have more difficulty with fine grained discrimination of visual location information. Future research could address the influence of low level visual processing upon visual WM. For example, here we have shown that auditory perceptual process might influence auditory WM, however it might be the case that individuals with magnocellular impairments, who show a lower level visual impairment, might show impairments with visual-WM, particularly when the task involves fine grained spatial decisions.

If visual WM impairments are identified in a sub-group of dyslexic individuals, more specific research questions can be addressed. The current research measures hit rate, and d', which are indicative of and correlated to WM capacity. However, future research might assess WM precision in dyslexia. Research within the visual WM domain has established that one can maintain item information (hit), but with low grade precision. Recent research has demonstrated that instead of slot storage, (e.g., Cowan, 2005; Luck & Vogel, 1997), resources can be distributed, thus effecting the precision with which an item is held in memory (see Alvarez & Cavanagh, 2004; Bays & Husain, 2008; Bays, Catalao & Husain, 2008), such that increasing numbers of objects are stored with increasing variability, or error. Assessing precision within dyslexia (through orientation decision tasks) would allow us to uncover how resources are distributed between items in WM. Precision could also be examined in the auditory domain (with dyslexia), where research assessing precision and

verbal WM is sparse. It will be interesting to see in upcoming years how the slots versus resource models of WM (although not mutually exclusive), can inform theories of WM, and how these may be applied to individuals with developmental disorders. For example, an individual may be able to store two items in WM (equivalent capacity), but with lower resolution. Furthermore, some individuals may be able to compensate for capacity differences by pooling resources to enhance the resolution of certain targets. Between group differences may be drawn out in this example by increasing capacity.

In the phonological domain, the effect of neural entrainment and WM processing can be addressed. For example, one could conduct an auditory N-back task where stimuli are presented at particular frequencies. Particularly, it has been identified that individuals with dyslexia are impaired in the theta range (Goswami, 2011). The effect of presenting target stimuli in phase or out of phase with the preceding stream could be examined, predicting that individuals with dyslexia might be less sensitive to presenting stimuli rhythmically. However, one could also explore the long term learning effects associated with entrainment at the theta frequency. Entrainment at the theta range could lead to increased phase locking over time, thus, when stimuli occur in phase, phase locking should be enhanced in dyslexia. Furthermore, the use of visual entrainment can be used as an additional scaffold in order to assist verbal WM processing in dyslexia.

Finally, in this chapter, a hypothesis regarding temporal sampling of information is provided, and how impaired phase locking might have consequences across the entire WM system. It is important to note that this is a hypothesis formed from the current results, as opposed to a definite conclusion. Future research is needed to address and test these predictions, as an alternative hypothesis might be that impaired phase locking to an auditory stimulus, just results in weaker item representation. An initial step might be to compose an

oscillatory computational neural network model to test the proposed hypothesis (See Chapter 6 for a full description). Predictions from the model can then be directly tested with EEG experimentation. Although previous research has observed oscillatory activity between neural regions, very little is known about their functional role, or about how oscillations directly transfer information. Therefore, a neural network connectionist model would have implications not only for WM processing in dyslexia, but would inform how oscillations are able to form WM representations. The model could assess the interaction between low level sensory processing stages and the formation of WM representations. EEG testing could also be used to examine the effect of auditory phase locking and entrainment, and successful coherence between distal neuronal regions. Connectivity analysis would allow for the visualization of information flow between various sources of electrophysiological activity.

Thesis Conclusion

The central aim of this thesis was to examine the extent to which adults with developmental dyslexia have a WM impairment that could be attributed to the phonological loop, or a more domain general central executive impairment. Furthermore, the nature of auditory processing was assessed. To address this, a series of 8 experiments were conducted, across sensory modalities, using behavioural and ERP measures to assess WM proficiency in individuals with and without developmental dyslexia. The experimental findings showed support for a phonological loop impairment, where individuals with dyslexia showed reduced P300 amplitude for auditory stimuli (Chapter3, and 5) which was accompanied by a behavioural impairment (reduced d' and hit rate). They provide strong support against a domain general central executive impairment, because an impairment is not identified for visual-object information (as in Chapter 3). Although there were subtle timing differences in Chapter 4, Experiment 5; overall, individuals with dyslexia were not impaired in their accuracy, or ERP

analysis in contexts where they are required to manipulate information online (Chapter 4). Furthermore, the final experiment provides initial evidence that lower level auditory processing difficulties might contribute towards the WM impairment in developmental dyslexia (Chapter 6). It is concluded that individuals with dyslexia do not have a global or general central executive impairment, however, they show robust phonological WM impairments. One possibility, is that the phonological WM impairment may be influenced by a lower level auditory impairment for tones with long rise-times, which may suggest that the phonological WM impairment in dyslexia may be partly influenced by perceptual difficulties with auditory information. Thus, any differences in WM performance between groups, may become more apparent when stimuli that are perceptually difficult for dyslexic individuals are used in a WM task.

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List of appendices

Appendix A: Ethical Approval for Experiments run during year 1, 2, and 3.

Ethical approval Year 1

PPROVAL BY PSYCHOLOGY RESEARCH ETHICS COMMITTEE

The following research project has been approved by The Psychology Research Ethics Committee

This project requires a valid CRB check in addition to this approval. It is your responsibility to provide it to the departmental office before you begin collecting data.

Date: 2011/01/18
Code: 20111705

Applicant details:
Name: Jumana Ahmad
Status: PhD Student
Email address: ja337@kent.ac.uk

Title of the research:
Negation processing in Dyslexia.

When carrying out this research you are reminded to
* follow the Departmental Guidelines for Conducting Research with Human Participants
* comply with the Data Protection Act 1998
* refer any amendments to the protocol to the Panel

Please keep this form in a safe place. You may be asked to present it at a later stage of your study for monitoring purposes. Final year project students and MSc students will need to submit a copy of this form with their project.

You can log in at http://www.kent.ac.uk/psychology/technical/ethics/index.php to copy or print pregenerated handouts for this study.

Ethical approval Year 2

APPROVAL BY PSYCHOLOGY RESEARCH ETHICS COMMITTEE

Your study has been approved. You can now proceed to do your study without resubmitting documents to the ethics committee. However, before proceeding with the research, please ensure you deal with all the issues outlined below. You MUST deal with these issues prior to data collection, otherwise this Ethics approval is not
valid.

This project requires a valid CRB check in addition to this approval. It is your responsibility to provide it to the departmental office before you begin collecting data.

Date: 2011/09/13
Code: 20111935

Applicant details:
Name: Jumana Ahmad
Status: PhD Student
Email address: ja337@kent.ac.uk

Title of the research:
Auditory working memory processing in Dyslexia: An N-back task.

When carrying out this research you are reminded to
* follow the School Guidelines for Conducting Research with Human Participants
* comply with the Data Protection Act 1998
* refer any amendments to the protocol to the Panel

Please keep this form in a safe place. You may be asked to present it at a later stage of your study for monitoring purposes. Final year project students and MSc students will need to submit a copy of this form with their project.

You can log in at http://www.kent.ac.uk/psychology/technical/ethics/index.php to copy or print pregenerated handouts for this study.

Ethical approval Year 3

APPROVAL BY PSYCHOLOGY RESEARCH ETHICS COMMITTEE
The following research project has been approved by
The Psychology Research Ethics Committee

Date: 12:14 31-10-2012
Code: 20122445

Applicant details:
Name: Jumana Ahmad
Status: PhD Student
Email address: ja337@kent.ac.uk
Title of the research:
Auditory and Visual working memory ability in Dyslexia. An intervention with Galvanic Vestibular Stimulation.
When carrying out this research you are reminded to
* follow the Departmental Guidelines for Conducting Research with Human Participants
* comply with the Data Protection Act 1998
* refer any amendments to the protocol to the Panel
Please keep this form in a safe place. You may be asked to present it at a later stage of your study for monitoring purposes. Final year project students and MSc students will need to submit a copy of this form with their project.
You can log in at http://www.kent.ac.uk/psychology/technical/ethics/index.php to copy or print pregenerated handouts for this study.

Appendix B: Example information sheets, consent forms, and debriefing sheets.

Example Information Sheet

Auditory working memory processing in Dyslexia: An N-back task.

Who is Organising This Study?

This research is organised by the Psychology Department of the University of Kent. The researcher is Jumana Ahmad, a PhD Student. The research supervisor is Heather Ferguson.

What Are the Aims of the Study?

This research is organised by the Psychology Department of the University of Kent. The researcher is Jumana Ahmad, a PhD Student. The research supervisor is Heather Ferguson. This study aims to examine working memory processing in Dyslexia. Specifically we are looking at the effects of working memory for information that you hear. Working memory can be defined as our memory for recent information e.g., a phone number you have just heard. It differs from long term memory, which is our memory for information that you may have heard a long time ago. This study is divided into two parts. You can take part in both today, or arrange the second part at another time. Part 1: In the first part of the study you will take part in an N-back task. During the N-back task you will have to make a yes or no decision in order to indicate if you have heard a stimulus before in a specific position. While you are doing this task, we will take EEG recordings. This lets us know when the brain is responding to a task. We analyse EEG results as a group, and take grand averages for each condition. These recordings allow us to determine how hard certain conditions in the task were. Detailed instructions will be given on the computer screen before the experiment starts. Part 2: In the second part of the study, we gather background information on IQ and a short dyslexia assessment. For the IQ task, you will take part in short 5 minute tasks, such as 'block design' The purpose of the dyslexia screening, is not to diagnose dyslexia in individuals, but is purely for research purposes. This involves measuring reading and spelling ability or taking part in basic proof reading tasks. Please note: If you have any questions, please ask the researcher at any point during the experiment. The dyslexia screening procedure (part 2) in this study is by no means used to diagnose dyslexia. If you are worried that you might have dyslexia, please contact the Dyslexia and disability service at the University of Kent. The researcher will provide you with details about the Dyslexia and Disability service here at the University of Kent on your Debrief sheet. This service will be able to advise and help you.

Who Can Take Part?

British (English as a first language). We are looking to recruit participants with and without dyslexia.

Who Can Not Take Part?

Not Native English Speakers.

What Happens to the Information I Provide?

Participation in this study guarantees confidentiality of the information you provide in line with the UK Data Protection Act 1998. Only researchers involved in the study and, if required, the body funding this research will be authorised to access the data. Your name and any other identifying information will be stored separately from your data in a securely locked filing cabinet. Questionnaires will be stored in a securely locked room for as long as is required by the Data Protection Act. The data collected for this study will be used for a student project. Once the data is analysed a report of the findings may be submitted for publication. Only broad trends will be reported and it will not be possible to identify any individuals. A summary of the results will be available from the researcher on request.

Contact for Further Information

If you require any further information or have any queries about this study please contact the researcher:

Jumana Ahmad
email: ja337@kent.ac.uk

Or the research supervisor:

Dr Heather Ferguson
Tel: 01227 827120
e-mail: H.Ferguson@kent.ac.uk
Address: Psychology Department, Keynes College, University of Kent, CT2 7NP

If you wish to withdraw your data from this study, please contact the Psychology Department Office on:

Tel: 01227 823699

If you have any serious concerns about the ethical conduct of this study, please inform the Chair of the Psychology Research Ethics Panel (via the Psychology Department Office) in writing, providing a detailed account of your concern.

KENT
UNIVERSITY OF KENT

Example Consent Form

Consent Form - copy 1 (for participant)

Title of project: Auditory working memory processing in Dyslexia: An N-back task.

Name of Researcher: Jumana Ahmad

1. I Confirm that I have read and understand the information sheet for the above study and have the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree to take part in the above study ☐

Name of the Participant: _____

Signature: _____ Date: _____

Example Debriefing Sheet

Auditory working memory processing in Dyslexia: An N-back task.

Thank you very much for your participation in this research. We would like to provide some further information about the purpose of the study and what we expect to find.

The aim of the current experiment was to examine working memory processing in individuals with and without dyslexia. Today you completed an N-back task, whereby you had to remember certain letters or words and decide whether or not you had heard them in the 0, 1, 2, 3, 4 or 5 back condition. In the second part of the study we also carried out a dyslexia screening and IQ measurement. These scores are not used to diagnose you with dyslexia. However, if you have any concerns that you might be dyslexic, please contact the Dyslexia and Disability service at the University of Kent. Support at the Canterbury campus The DDSS is based in Keynes College and the office is open from 09.00 – 17.00, Monday to Friday. For further information, email accessibility@kent.ac.uk or call +44 (0) 1227 823158. Support at the Medway campus Full details of disability and dyslexia support for Medway

students will be available from the Student Services department located in the Gillingham Building on the Medway campus. For further information contact Graham Gorvett by email medwaystudentservices@kent.ac.uk or telephone +44 (0)1634 888804 Website: http://www.kent.ac.uk/ddss/ The service will be able to address any concerns you have. If you have any questions about the research conducted today, please contact the researcher, Jumana Ahmad at ja337@kent.ac.uk or supervisor at H.Ferguson@kent.ac.uk. Many thanks for your help with this research.

If you have any queries about this research or would like to ask any further questions, please contact the researcher or research supervisor using the contact details below.

If you would like to withdraw your data at any point, please contact the Psychology departmental office on **01227 823961**. If you have been given a participant code you need to cite this. You do not have to give a reason for your withdrawal.

Once again, we would like to thank you for your valuable contribution to this research. Your participation is greatly appreciated.

Yours sincerely,

Jumana Ahmad

Researcher contact details:

Jumana Ahmad
ja337@kent.ac.uk

Supervisor contact details:

Dr Heather Ferguson
Tel: 01227 827120
e-mail: H.Ferguson@kent.ac.uk
Address: Psychology Department, Keynes College, University of Kent, CT2 7NP

If you have any serious concerns about the ethical conduct of this study, please inform the Chair of the Psychology Research Ethics Panel (via the Psychology Department Office) in writing, providing a detailed account of your concern.

Appendix C: AoA Stimuli and Raw Scores for Counterbalanced Measures

WORD	GROUP	AOA	FAM	IMG	KFFRQ	NLET	NSYL	HAL Freq	NPhon	NSyll
ADDER	Early	356	361	583	-	5	2	5.861	3	2
DIP	Early	331	466	456	6	3	1	8.527	3	1
ELF	Early	272	355	543	-	3	1	8.887	3	1
FAIRY	Early	242	471	536	4	5	2	7.76	4	2
GAIN	Early	389	543	307	74	4	1	10.082	3	1
HAIL	Early	354	440	477	10	4	1	8.079	3	1
HOWL	Early	269	447	536	4	4	1	8.079	3	1
HUSH	Early	246	415	467	4	4	1	6.248	3	1
MAKER	Early	358	487	379	12	5	2	8.997	4	2
OX	Early	329	364	548	5	2	1	7.082	3	1
PEEP	Early	289	418	437	2	4	1	6.433	3	1
POUCH	Early	367	373	513	2	5	1	6.723	3	1
RHYME	Early	253	480	475	3	5	1	7.396	3	1
TRIM	Early	367	456	378	20	4	1	8.332	4	1
TUCK	Early	342	452	416	2	4	1	6.912	3	1
WAND	Early	272	381	513	1	4	1	8.598	4	1
WHACK	Early	311	350	486	1	5	1	7.485	4	1
WHIRL	Early	364	423	499	3	5	1	5.958	4	1
WORTH	Early	369	542	275	94	5	1	11.307	3	1
YELP	Early	378	333	499	2	4	1	5.357	4	1
BOOTH	Late	508	444	486	7	5	1	8.404	3	1
CLOVE	Late	500	395	446	1	5	1	6.236	4	1
CULT	Late	586	437	386	11	4	1	9.292	4	1
FONT	Late	547	338	497	-	4	1	9.884	4	1
GIG	Late	564	425	437	1	3	1	8.834	3	1
HUE	Late	544	404	399	1	3	1	7.208	3	1
KINK	Late	523	356	480	-	4	1	6.56	4	1
LUNGE	Late	508	394	459	4	5	1	6.157	4	1
OMEN	Late	558	394	413	2	4	2	6.903	4	2
QUAIL	Late	536	376	505	-	5	1	5.858	4	1
REALM	Late	561	376	324	19	5	1	8.888	4	1
RYE	Late	500	388	459	4	3	1	7.137	2	1
SNUB	Late	522	386	348	-	4	1	5.198	4	1
SPASM	Late	558	422	486	3	5	2	6.261	5	2
SPREE	Late	531	410	425	4	5	1	6.279	4	1
STRUT	Late	511	368	437	3	5	1	6.594	5	1
UNION	Late	503	595	526	182	5	2	10.328	6	2

VENOM	Late	531	375	456	2	5	2	8.425	5	2
VIGIL	Late	550	370	426	1	5	2	6.457	5	2

Appendix D: Grand average ERPs at the midline electrodes for Experiment 4.

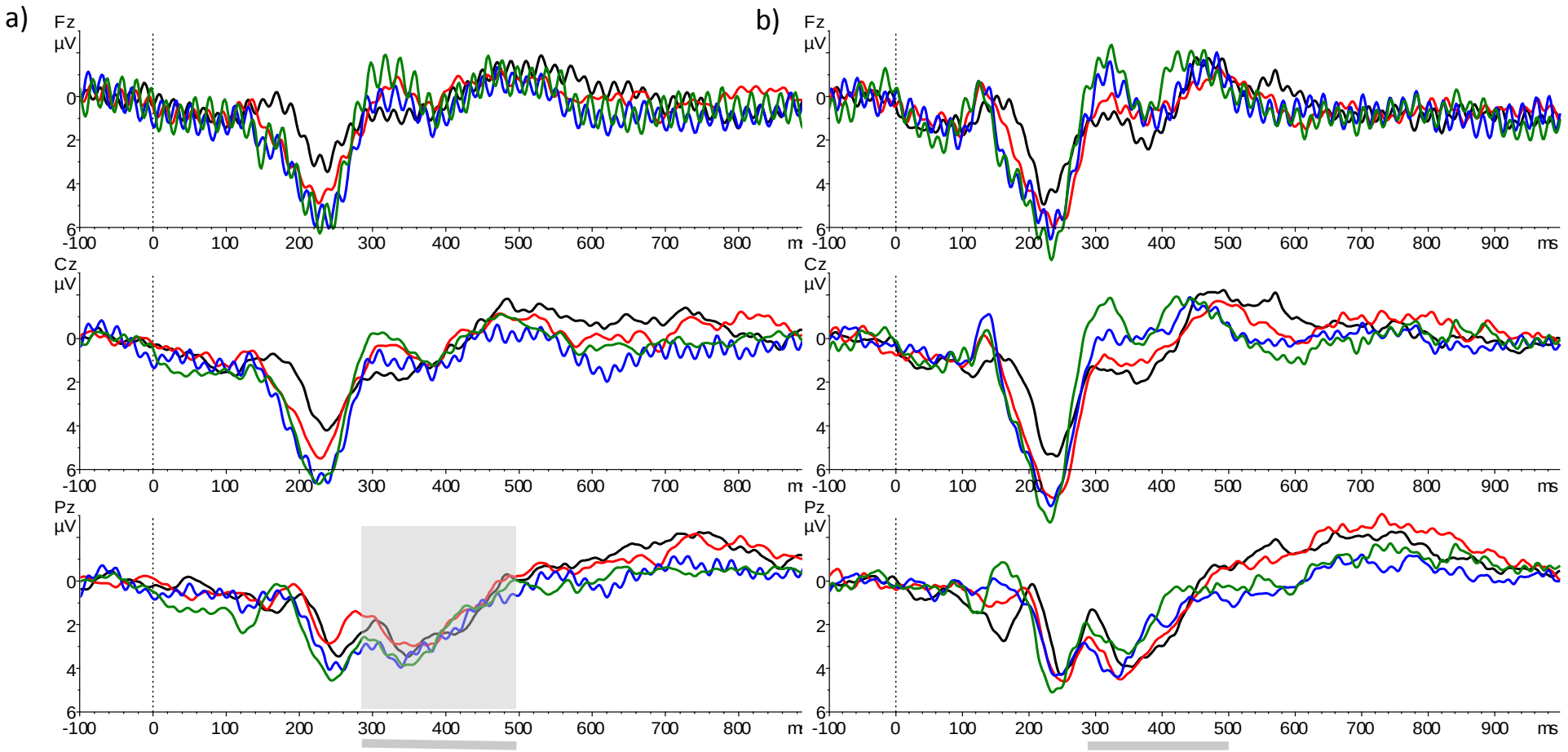

Figure D1: a) ERPs for non-dyslexic individuals at encoding, for WM load conditions of 1 (black), 3 (red), 5 (blue) and 7 (green). b) Grand average ERP waves for dyslexic individuals, at WM load conditions of 1 (black), 3 (red), 5 (blue) and 7 (green).

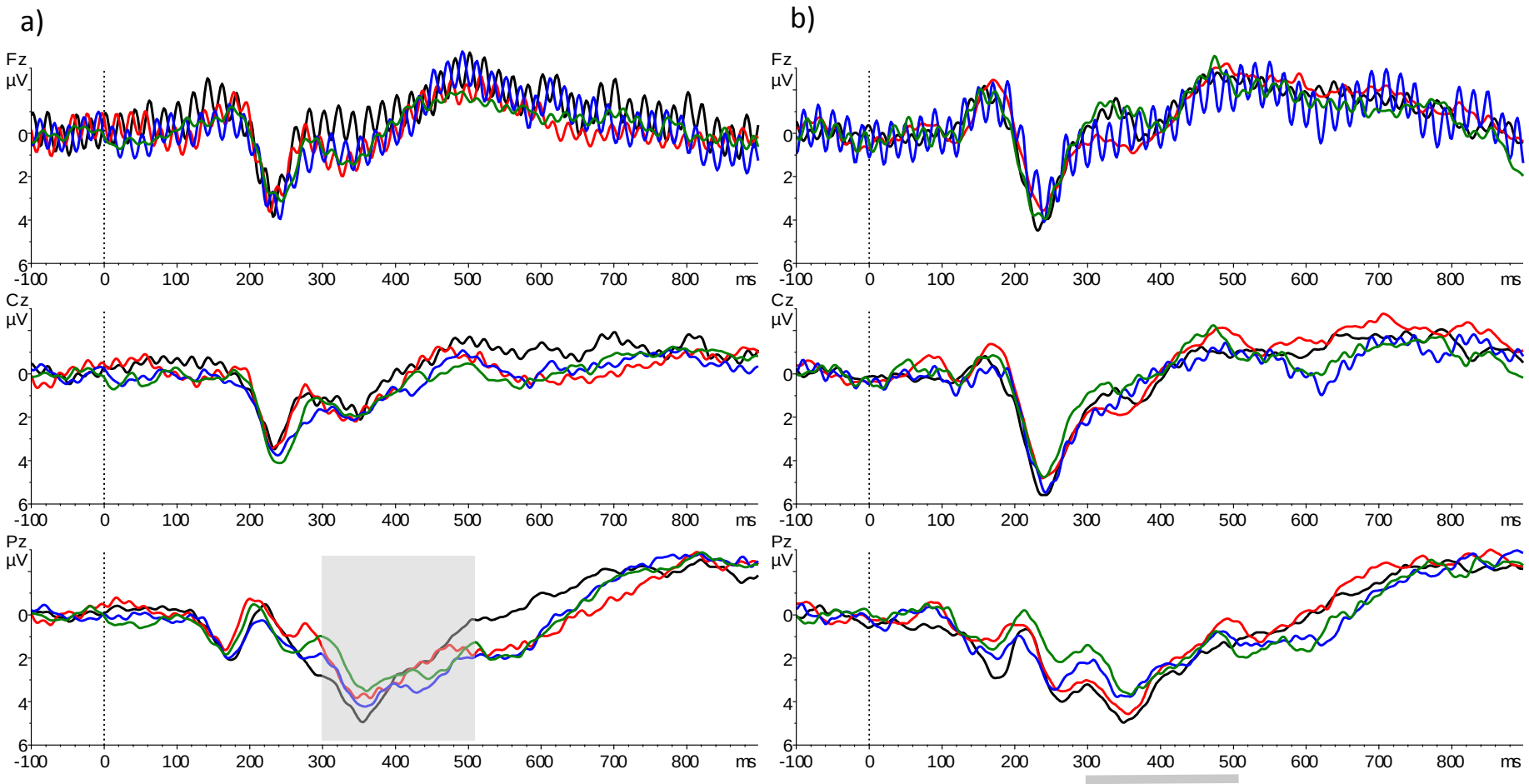

Figure D2: a) ERPs for non-dyslexic individuals at retrieval, for WM load conditions of 1 (black), 3 (red), 5 (blue) and 7 (green). b) Grand average ERP waves for dyslexic individuals, at WM load conditions of 1 (black), 3 (red), 5 (blue) and 7 (green).

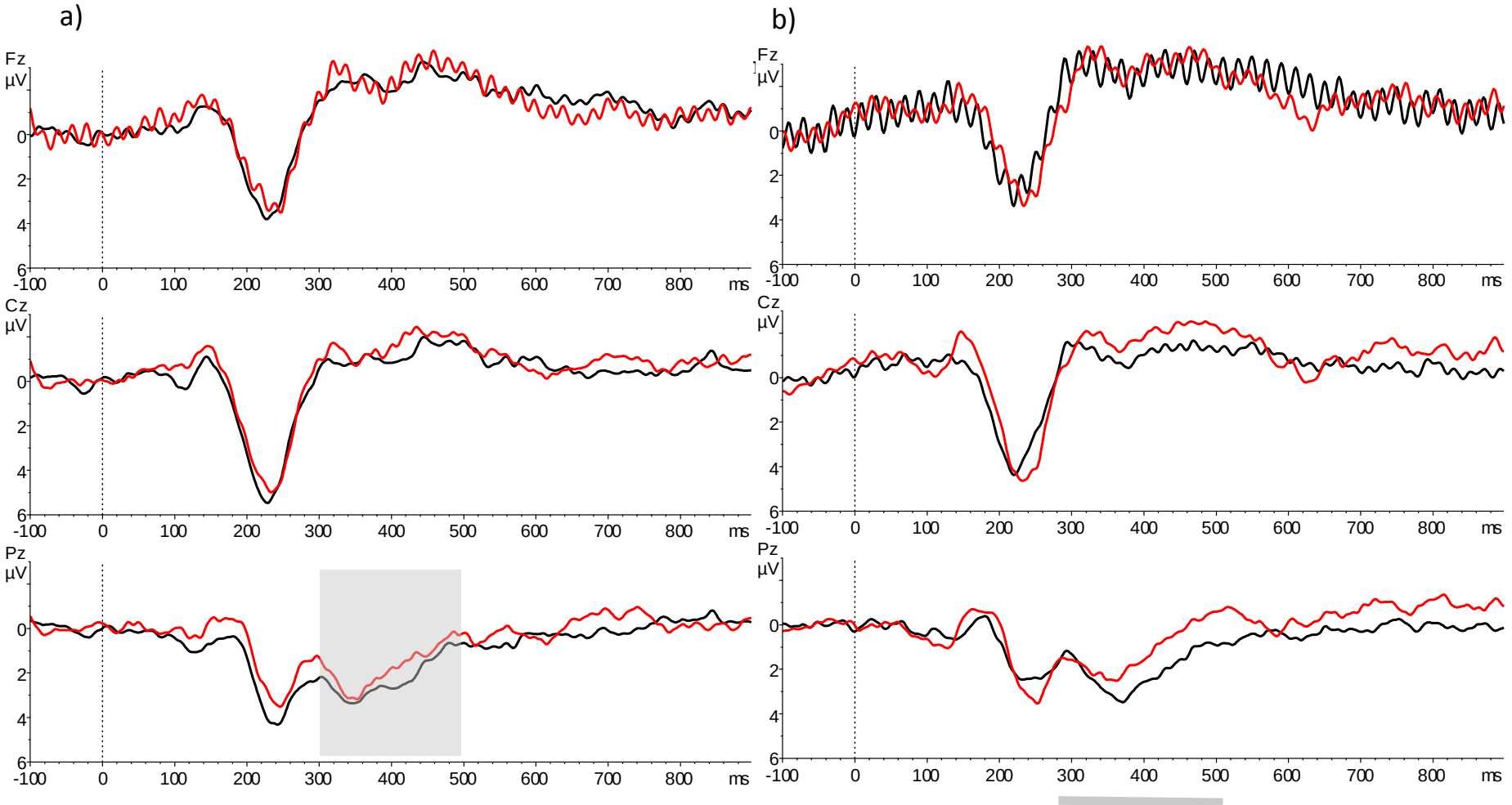

Figure E1: a) Grand average ERPs at encoding, for **non-dyslexic (black)** and dyslexic (red) individuals in the maintenance condition. b) Grand average ERP at encoding waves for **non-dyslexic (black)** and dyslexic (red) in the maintenance plus manipulation condition.

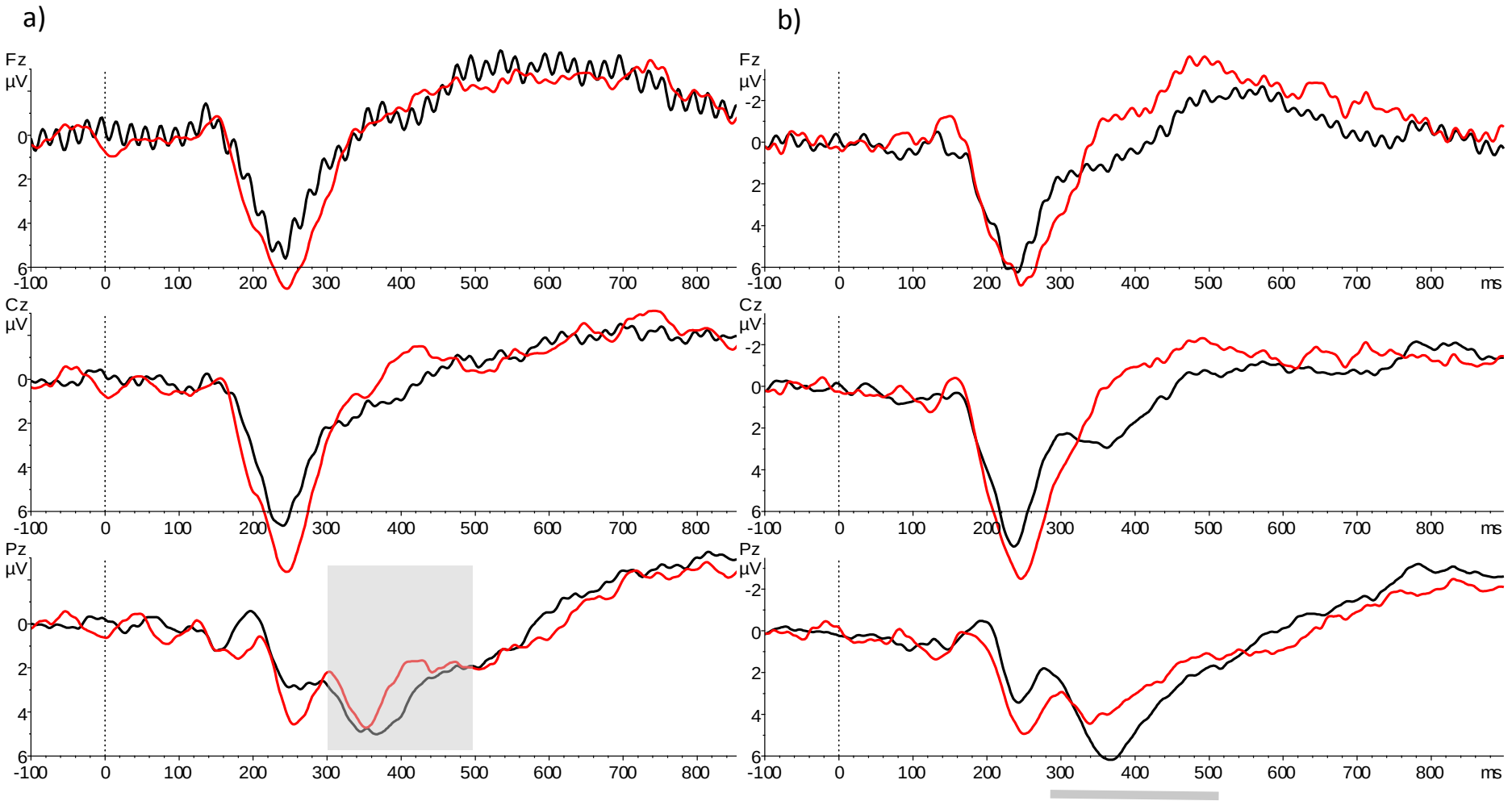

Figure E2: a) ERPs at retrieval, for **non-dyslexic (black)** and dyslexic (red) individuals in the maintenance condition. b) Grand average ERP waves for **non-dyslexic (black)** and dyslexic (red) in the maintenance plus manipulation condition.

Appendix F: Grand average ERPs at the midline electrodes for Experiment 6**.**

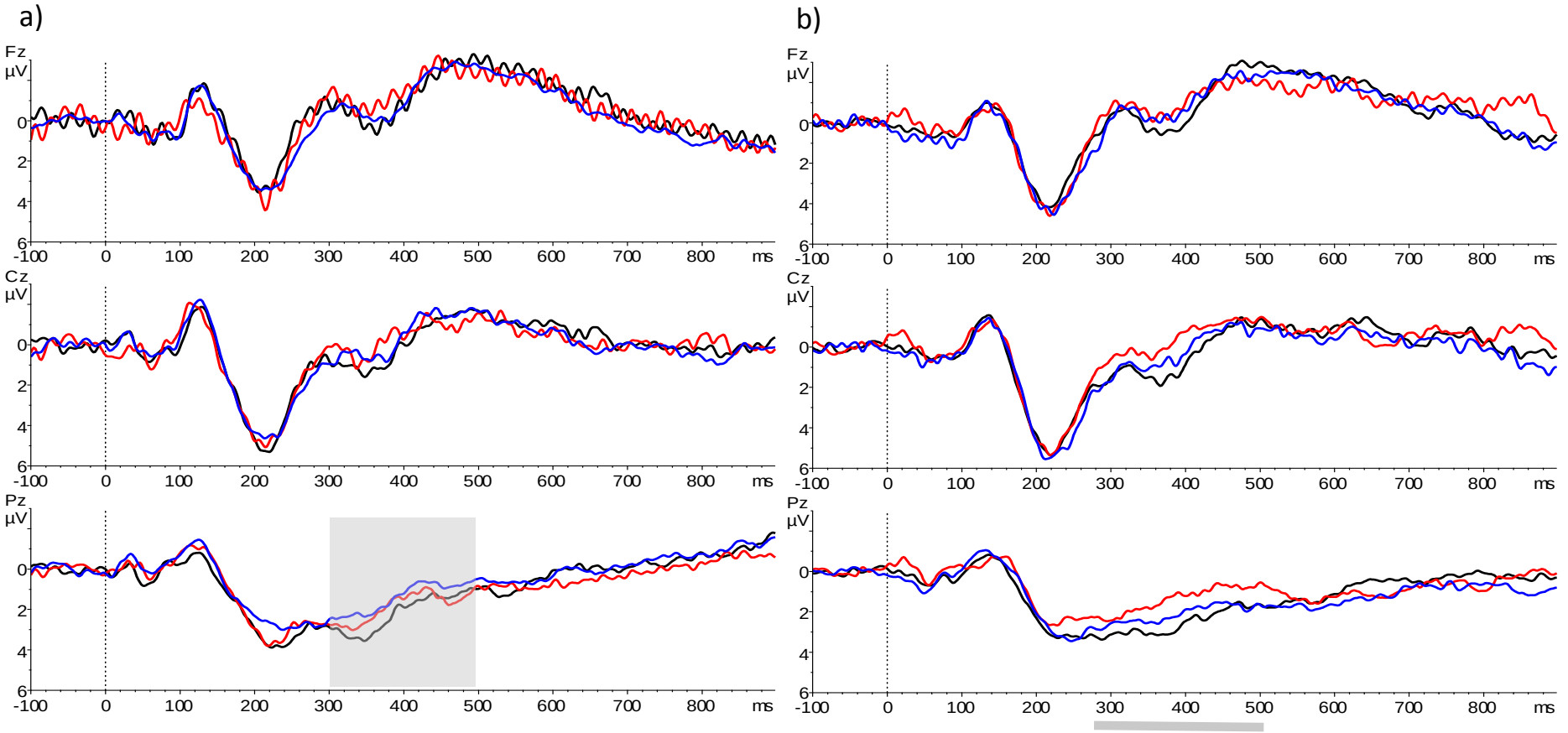

Figure F1: ERPs at the midline, time-locked to the onset of each letter, for a) non-dyslexic, and b) dyslexic groups. Showing conditions of **low (black)**, medium (blue), and high (red) WM load conditions.

Appendix G: Grand average ERPs at the midline electrodes for Experiment 7

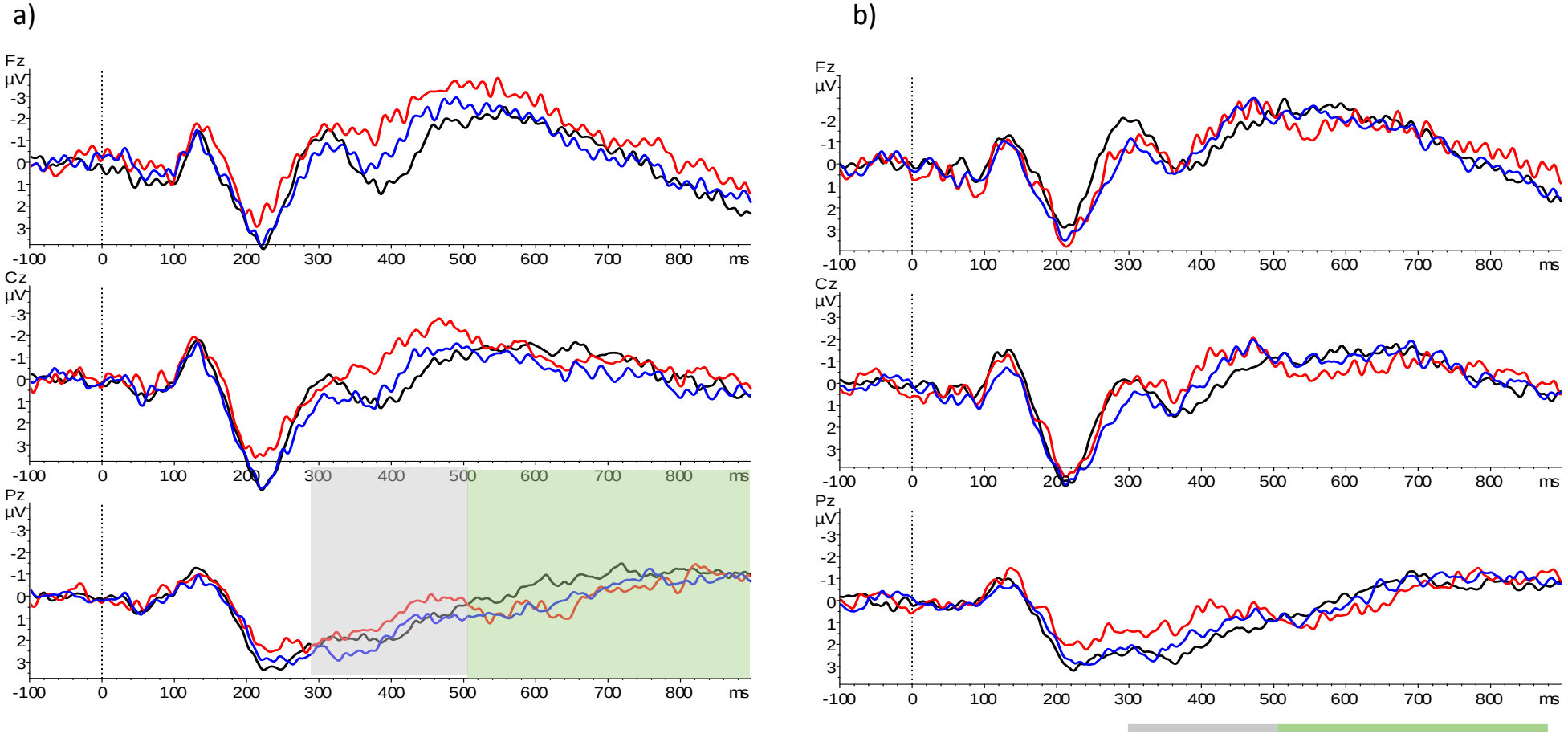

Figure G1: ERP plots for Experiment 7. The grand average ERPs time locked to the onset of each words. ERPs were plotted for a) early words, non-dyslexic participants and b) early words, dyslexic conditions. ERPs are plotted for l**ow WM load (black), medium WM load (blue), high WM load (red).**

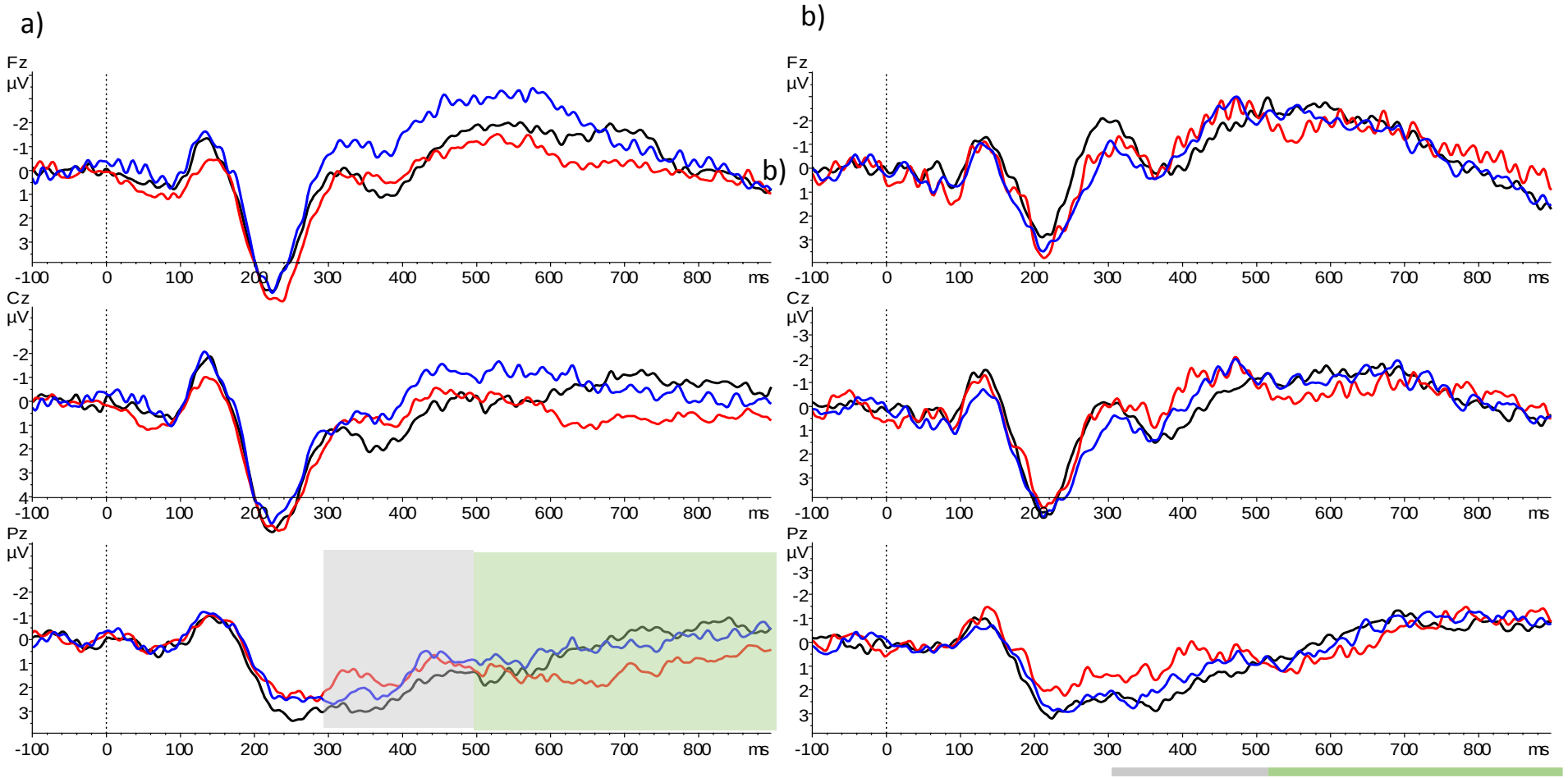

Figure G2 ERPs for Experiment 7. Time-locked to the onset of late learned words for a) non-dyslexic and b) late learned words dyslexic conditions. ERPs are plotted for l**ow WM load (black), medium WM load (blue), high WM load (red**