



Speech versus tone processing in compensated dyslexia: Discrimination and lateralization with a dichotic mismatch negativity (MMN) paradigm

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ABSTRACT

This study investigated the possibility of a pervasive auditory-processing deficit in 10 adult dyslexics who had compensated for their reading disability, compared to 10 matched controls. Unlike previous studies [Baldeweg, T., Richardson, A., Watkins, S., Foale, C. & Gruzelier, J. (1999). Impaired auditory frequency discrimination in dyslexia detected with mismatch evoked potentials. *Annals of Neurology*, 45(4): 495–503], the current EEG study used a dichotic presentation of stimuli in order to probe the relationship between ear advantage and left- and right-hemisphere dominance for processing speech and non-speech stimuli respectively. A dichotic presentation is thought to maximise lateralization effects, as well being a more ecologically valid paradigm. The Mismatch Negativity (MMN) was measured for both speech [consonant–vowel pairs: /ta/ vs. /ka/ and ba/ vs. /da/] and non-speech stimuli (pure tones: 1 kHz vs. 1.2 kHz). Smaller MMNs to tone stimuli were obtained for dyslexics versus controls, but no differences were found for speech stimuli. Controls differentiated between speech and tones, with larger MMNs to tone stimuli. Dyslexics showed significantly greater MMNs to one stop consonant discrimination (/ta/ vs /ka/) than the other (/ba/ vs /da/), but did not differentiate speech from tones, and no or minimal lateralization was found for either group or stimulus type, in line with recent studies [Kershner and Micallef, 1992; Bellis, T.J., Nicol, T., & Kraus, N. (2000). Aging affects hemispheric asymmetry in the neural representation of speech sounds. *Journal of Neuroscience*, 20, 791–797]. However, analysis of left and right dipole source activity suggested right hemispheric preference for tones in controls, and the lack of such a preference in dyslexics. Lateralization of the auditory system in general may be less specialized in compensated dyslexia, although no specific differences in speech lateralization were seen. The present study also extends previous findings to show that the frequency range over which dyslexics are shown to display impaired frequency discrimination can be extended to 20% (1 kHz vs. 1.2 kHz) if using a *dichotic* presentation.

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1. Introduction

Developmental dyslexia refers to specific problems learning to read and spell, despite adequate instruction, intelligence, and socio-cultural opportunity (Critchley, 1970). It is suggested that a failure to adequately develop phonological skills may underlie the problems in reading exhibited by individuals with dyslexia (Snowling and Hulme, 2005), and that these problems may persist into adulthood (Bruck, 1992). In some cases poor phonological skills may co-exist with visual and/or auditory deficits (Ramus et al., 2003). With remedial teaching, age-appropriate reading skills can be attained, although spelling skills often do not reach the same level of compensation (Hatcher et al., 2002).

According to one theory, phonological deficits may be a consequence of underlying auditory processing deficits. Tallal and Piercy (1973) initially proposed that an auditory temporal processing deficit may underlie specific language impairment, and this has been extended to dyslexia

(Miller and Tallal, 1995; Temple et al., 2000). For example, Tallal (1980) found dyslexic children to be impaired on a temporal-order judgment task compared to matched controls. However, Marshall et al. (2001) found that temporal impairments were observable in only a small subset of dyslexics.

There is evidence, however, that some forms of auditory processing, particularly of speech stimuli, are more widely impaired in dyslexics. Manis et al. (1997) found that categorical perception of a /ba-/pa/ continuum was less sharply defined in dyslexics than controls. For non-speech stimuli too, McAnally and Stein (1996) showed that compensated dyslexics were impaired at discriminating pitch for a sound with a frequency of 1 kHz, and performance was related to reading ability. It is possible that a sound discrimination deficit, rather than a temporal processing one, may underlie phonological problems in dyslexia, although discrimination may be particularly impaired with a rapid presentation rate (see McArthur and Bishop (2001) for a discussion of this issue).

1.1. The mismatch negativity (MMN) and dyslexia

The neurophysiology of central auditory function in dyslexia can be investigated by studying the mismatch negativity (MMN) component

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of the ERP signal (see Bishop, 2007 for a review). The MMN is measured in an oddball paradigm, in which a cortical response is elicited by an occasional discriminable change along a given dimension (e.g., frequency, duration) of auditory stimuli, in an otherwise repetitive pattern of stimulus presentation (Näätänen et al., 2007; Picton et al., 2000). The MMN can also be used to investigate phoneme-specific changes in detection (Näätänen et al., 1997). If linguistic deficits in dyslexia are caused by a specific phonological deficit rather than an underlying auditory one (e.g. White et al., 2006), then attenuation of the MMN response would be predicted for speech stimuli but not for non-verbal sounds (Bishop, 2007). In contrast, attenuation of the MMN to non-verbal sounds such as tones, particularly at rapid presentation rates, would lend support to the auditory processing hypothesis. To date, MMN studies of dyslexia to date are inconclusive on this point.

A number of previous studies using the MMN to study dyslexia in children and adults have found impaired discrimination (i.e. attenuated MMN) for both speech (Schulte-Körne et al., 1998, 2001; Csépe, 2003) and tone (Kujala et al., 2006; Baldeweg et al., 1999) stimuli, although there have also been null results for both (Paul et al., 2006; Heim et al., 2000; Alonso-Búa et al., 2006). Speech stimuli generally consist of consonant or vowel pairings, e.g. /ba/ vs. /ga/ or /e/ vs. /i/, in which one of the pair acts as the standard and the other as the deviant. Non-speech stimuli are generally pure tones, with deviants differing from the standard along dimensions including frequency and duration. One recent study (Kujala et al., 2006) compared dyslexics and controls on five dimensions of tone discrimination, and found that pitch (i.e. frequency) discrimination was the only dimension on which dyslexics were impaired. Indeed, no study looking specifically at dyslexia has found attenuated MMNs for duration perception using standard pure tone stimuli (e.g. Huttunen et al., 2007; Baldeweg et al., 1999; Kujala et al., 2006), although differences using frequency modulated stimuli (which requires frequency discrimination at rapid durations) have showed the predicted decrement (Stoodley et al., 2006).

MMNs to speech and tone stimuli have also been measured within the same dyslexic participants; while some studies have shown a speech specific deficit (Csépe, 2003; Schulte-Körne et al., 1998, 2001), others have found deficits in both types of stimuli (Lachmann et al., 2005). The contradictory results are further confounded, in that none matched speech and non-speech stimuli in terms of acoustic complexity, frequency range or envelope (Bishop, 2007). For example, using the standard/deviant speech stimuli /da/ and /ga/, which differ in formant frequency but not duration, a significantly smaller late MMN was observed in dyslexics compared to controls (Schulte-Körne et al., 2001), with a right-lateralization of the MMN for both groups. No difference in MMN was observed for tone stimuli between groups. However, the deviant tone stimuli differed from the standard by a relatively large 440 Hz. This may not have provided a sensitive enough comparison between the groups. Previous studies which have found differences for tone stimuli have used much smaller intervals (e.g. Lachmann et al., 2005; Baldeweg et al., 1999), with the latter study finding that MMN attenuation in dyslexics lessened as the frequency difference between standard and deviant increased.

Furthermore, it is so far uncertain how MMN response may relate to performance on behavioural tasks, or to dyslexic profile. Several studies have found a relationship between MMN response and behavioural performance on speech and language tasks (Kraus et al., 1996; Baldeweg et al., 1999; Stoodley et al., 2006). However, diminished MMNs are in no way a physiological 'marker' for dyslexia, with one recent study finding attenuated MMNs in one subtype of dyslexics, but not in another (Lachmann et al., 2005). One related issue is that, rather than reflecting the underlying cause of poor language skills in dyslexia, reduced MMN response may reflect a consequence of poor phonological awareness. For example, in non-dyslexics, Näätänen et al. (1997) found that MMN size depended on whether the standard-deviant contrast carried phonemic information in the participants' native language: Finnish speakers

generated a smaller MMN to a contrast that was meaningful in Estonian compared to Finnish; and the reverse pattern was observed in Estonian participants. However, discrimination of tone stimuli would presumably not be affected in this way.

Studying MMNs to speech and tones in compensated dyslexics is one way to examine this issue. If such participants show a reduced MMN response, particularly to tone stimuli, despite having compensated for their language deficits, then this may reflect a residual auditory processing deficit that has not been addressed by top-down compensatory strategies. One recent study (Stoodley et al., 2006) measured MMNs to frequency modulated tone deviants in compensated dyslexics and controls. Groups were matched for psychophysical performance on a frequency modulated tone detection task, and yet early MMNs (150–300 ms after stimulus onset) in the dyslexic group were still attenuated relative to controls. Additionally, greater discrepancy between literacy scores and general cognitive ability was related to a smaller MMN. It may be that the MMN response represents a more sensitive measure of auditory processing deficits than psychophysical measures. A similar result was obtained by Baldeweg et al. (1999); they found significantly smaller MMNs in dyslexics compared to controls for tone frequency deviants up to differences of 90 Hz. Pitch discrimination deficit as measured by the MMN was correlated with degree of phonological impairment. While causality is hard to establish, this finding does suggest a link between basic auditory processing of pure tones, and phonological abilities.

1.2. Lateralization of the MMN response

There is also some evidence for altered topography of the MMN response in dyslexics compared to controls, particularly regarding lateralization. In non-dyslexic subjects, there is some evidence for larger left hemisphere MMNs to speech stimuli and larger right hemisphere MMNs to tone stimuli (Paavilainen et al., 1991; Alho et al., 1998; Shtyrov et al., 2000); this may be related to left hemisphere dominance for speech. In dyslexic individuals, structural abnormalities such as reduced or reversed asymmetry in language areas such as the planum temporale have been found (Larsen et al., 1990; Galaburda, 1989; although see Heiervang et al., 2000; Foster et al., 2002). Additionally, functional magnetic imaging (fMRI) studies have revealed an underactivation (compared to controls) of left hemisphere posterior networks involved in reading (Shaywitz et al., 2003), and an over-activation of inferior frontal and right posterior regions, possibly indicating regions of compensatory activity (Pugh et al., 2000). Therefore, a reduced left-lateralization of the MMN response to speech stimuli might be predicted, mirroring reduced anatomical asymmetry.

However, the interpretation of lateralization from EEG and other neuropsychological techniques is made difficult by the variation of findings even within the control population. Whilst some studies measuring MMNs for speech stimuli report greater MMNs in the left hemisphere (Näätänen et al., 1997; Rinne et al., 1999; Shtyrov et al., 1998), other studies have failed to show the expected left-hemisphere dominance for speech stimuli (Bellis et al., 2000; Shtyrov et al., 2000). A similar discrepancy is observed in PET and fMRI studies; whilst some studies have shown a clear left-lateralization to speech stimuli [(PET: Scott et al., 2000, 2004) (fMRI: Narain et al., 2003)], others suggest similar involvement of both hemispheres in processing speech stimuli [(PET: Mummery et al., 1999) (fMRI: Scott and Johnsrude, 2003)]. In MMN studies of dyslexia, no group differences were found in two studies which investigated lateralization to speech stimuli (Schulte-Körne et al., 1998, 2001). There is some evidence of altered lateralization to tone stimuli, but this is inconsistent between studies and stimulus types (Kujala et al., 2000, 2003; Csépe, 2003; Huttunen et al., 2007).

One important factor which may underlie these varied laterality effects found in psychophysiological studies of both dyslexic and non-dyslexic subjects is that often auditory stimuli were *not presented*

dichotically. In a dichotic-listening task (Broadbent, 1954), a listener is simultaneously presented with different auditory items to each ear, and, in behavioural paradigms, is asked to report the item heard in either the left or right ear. Tasks using speech sounds such as consonant–vowels (CVs) typically show that right-handed listeners show a right-ear advantage (REA), i.e., they are more accurate in the reporting of stimuli presented to the right ear (Bryden, 1988; Hugdahl et al., 2001). In contrast, some dichotic-listening tasks using non-speech sounds have found a left-ear advantage (LEA) (Bryden, 1986; Kimura, 1964; Piazza, 1980). Ear advantages in dichotic listening are explained by the fact that contralateral projections to the brain from the ear of presentation consist of more fibres and produce more cortical activity than the ipsilateral projections (Philips and Gates, 1982; Rosenzweig, 1951).

Previous studies investigating the laterality of the MMN have used either a monotic presentation (auditory items presented to only one ear) (e.g., Bellis et al., 2000), or a diotic presentation (same auditory item presented to both ears at the same time) (e.g., Alho et al., 1998; Korpilahti et al., 2001; Näätänen et al., 1997; Baldeweg et al., 1999; Pulvermüller et al., 2001; Schulte-Körne et al., 2001; Shtyrov et al., 2000). There is surprisingly little evidence of EEG studies which have actually used a dichotic presentation to investigate laterality. Some early EEG studies did use a dichotic stimulus presentation (Haaland, 1974; Neville, 1974) but limitations in the synchrony of signal generation often led to differences between stimulus onset times across ears. We are aware of only one EEG study which attempted to investigate dichotic processing in dyslexics versus controls, using CV stimuli (Brunswick and Rippon, 1994). Their results showed that whilst controls presented with a left-hemisphere bias for the N100 response in the left hemisphere, dyslexics showed no hemispheric bias for the response. However the participants in this particular study were requested to produce a verbal response whilst listening to the dichotic consonant–vowel stimuli during the EEG recording. In this case the task demands may well have altered the way the speech stimulus information was processed, and influenced the laterality findings.

There are three main advantages to using a dichotic presentation in the current study. First it mimics the task of the auditory system in real life, i.e. to distinguish between competing stimuli. Secondly, since a dichotic presentation has been shown to lead to ipsilateral suppression of auditory pathways (Bryden, 1988), any lateralised responses will appear more pronounced. Third, a dichotic presentation requires the auditory system to carry out a slightly different comparison to a diotic one in producing the MMN: deviant stimuli are compared with a standard presented *at the same time* (but in the opposite ear) as opposed to being compared with a memory trace of a previously presented standard. There have been a limited number of psychological studies carried out using the dichotic task with a dyslexic population. Of the few studies that have been conducted with individuals with dyslexia or language impairments, a reduced REA has been reported (Hynd and Obrzut, 1981; Mercure and Warren, 1978).

1.3. The current study

The aim of this study was to compare auditory discrimination ability as measured by MMN in compensated dyslexic and control individuals. Unlike previous studies, speech and tone stimuli were presented in a dichotic paradigm, as it was hypothesised that lateralization of the MMN to tones and speech would be more pronounced than with a monotic or diotic presentation. We also hypothesised that a competing stimulus presentation might present a greater challenge to the auditory system compared to standard diotic presentation: this might be reflected by smaller MMNs in the dyslexics.

For speech stimuli, stop CV pairings of ba/da and ta/ka were chosen. Stop consonant–vowel pairings are standard stimuli for investigating speech perception, as they are characterised by brief spectral changes in formant frequency, discrimination of which is

particularly challenging to the auditory system. It is of interest to see if a group difference in the early MMN component is found using a dichotic presentation, as none was found by Schulte-Körne et al. (2001) using a diotic presentation with similar participants (adult compensated dyslexics). However, if a difference were found, it would indicate an auditory perceptual deficit for speech at one of the very earliest, pre-attentional, stages of auditory processing. Tone perception was measured using a deviance of 200 Hz (1 versus 1.2 kHz). This represents a deviance of 20%.

Correlations between phonological skills and size of MMN were also undertaken, in an attempt to replicate previous studies that have found a relationship between the MMN and performance on literacy tasks, e.g. Baldeweg et al. (1999). A relationship would suggest a priori that phonological deficits might be underlain by auditory deficits.

2. Materials and methods

2.1. Listeners

Twenty undergraduate or postgraduate students from the Universities of Oxford or Oxford Brookes took part in this study. Ten of the listeners were dyslexic (7 male; age range 18.7–22.9 years; mean age 21.3 years). They had been diagnosed by a qualified clinician between early childhood and late adolescence. However, all had sufficiently compensated for the disorder to enable them to gain a university-level qualification. They were studying a range of degree subjects in both Arts and Sciences. The remaining 10 listeners acted as controls (2 male; age range 20.1–26.7 years; mean age 23.4 years), and had no history of language or reading impairments.

All listeners had English as a first language and were shown to be right handed on the Edinburgh Handedness Inventory (adapted from Oldfield, 1971). All listeners were screened to confirm hearing thresholds were within normal limits, i.e., 20 dB HL or less from 0.25–4.0 kHz, using a hand-held DSP pure tone audiometer (Micro Audiometrics Corp., Murphy, NC, USA) fitted with Telephonics TDH 39p headphones (Telephonics, Farmingdale, NY, USA). Listeners were given a £10 book token for their time. All listeners gave informed consent.

2.2. Cognitive tests

Cognitive tests were administered to confirm individuals in the dyslexic group as dyslexic. All 10 dyslexic listeners completed the cognitive tests during the same test session in which they completed the EEG part of the study. However, controls were given the cognitive tests in a session subsequent to the EEG study. Several listeners could not be contacted; therefore only five (female) controls completed the cognitive tests.

The dyslexic and control groups were compared on reading and spelling accuracy using the 'BLUE' version of the Wide Range Achievement Test – Revision 3 (Wilkinson, 1993; WRAT3). They were compared on digit span using the Wechsler Adult Intelligence Scale – Revised (The Psychological Corporation, 1986). They were also compared on decoding (non-word reading) using two non-word passages taken from Gross-Glenn et al. (1990); after Finucci et al. (1976). The first passage contained 17 non-words embedded in a context of 52 words (e.g. 'once upon a time, a *tawndy rapsig named Gub* found a *tix of pertollic asquees*'), and the second contained 13 non-words embedded in a context of 44 words. Standardised tests were administered according to instructions, and standardised scores for reading, spelling, and digit span accuracy were obtained. For decoding, reading time and error rate were measured for each passage.

As can be seen from independent samples t-tests in Table 1, controls performed significantly better than dyslexics on spelling, and were significantly faster on the non-word passages. Group differences on reading and digit span scores were not significant, although differences were in the predicted direction. This pattern of performance is

Table 1

Background measures comparing dyslexics and controls on tests of spelling, reading, digit span and decoding (non-word passage reading)

	Dyslexic		Control
	Mean (s.d.)	Mean (s.d.)	t(13)
Spelling (standard score)	105 (10.47)	119.4 (6.15)	0.02*
Reading (standard score)	108.4 (8.13)	114.2 (6.42)	0.19
Digit span (standard score)	10.9 (2.47)	12.8 (3.35)	0.23
Non-word passage reading time (s)	31.03 (8.83)	21.67 (3.00)	0.04*
Non-word passage mean error rate	0.23 (.17)	.054 (.04)	0.04*

* $p < .05$ (two-tailed).

consistent with previous profiles of compensated dyslexia (Dilling et al., 1991; Hatcher et al., 2002).

2.3. Stimulus generation

These consisted of speech and tone stimuli. Speech stimuli were CV pairings: 'ba' (standard) with 'da' (deviant), and 'ta' (standard) with 'ka' (deviant). [It was possible that the MMN could be affected by stimulus specific properties of 'ba' versus 'da' and 'ta' versus 'ka', as opposed to merely reflecting that the deviant differed from the standard. Therefore a pilot study reversed the 'ba/da' presentation so that 'da' was the standard and 'ba' the deviant. No difference in MMNs was found in response to 'da/ba' versus 'ba/da,' so it was thought unnecessary to compare their effects in the full study.] Synthesizer parameters for the CV stimuli were generated using the All-Prosodic Speech Synthesis Architecture (IPOX) interface (Dirksen and Coleman, 1994). The sampling rate was set at 11025 Hz and the frame size was 5 ms. The CV parameters were passed to a Klatt speech synthesizer (Klatt, 1980), to synthesize an adult male voice. The standard IPOX stimulus output parameters were used.

Both /ba/ and /da/ stimuli had the same fundamental frequency (f_0); f_0 increased to a steady-state value of 120 Hz over 88 ms. Stimulus /da/ consisted of a formant transition period followed by a 478 ms steady state period. Onset frequencies for the first formant ($F1$) and second formant ($F2$) were 174 Hz and 1597 Hz respectively. These two formants reached the vowel steady-state frequency of 687 Hz and 997 Hz over 66 ms. The third formant ($F3$) onset was at 2429 Hz, reaching a steady-state frequency of 2584 Hz over 30 ms. For /ba/ the onset frequencies for $F1$, $F2$ and $F3$ were 193, 1461 and 2149 Hz respectively. These three formants reached a vowel steady-state frequency of 687, 1006 and 2574 Hz over 40, 50 and 74 ms, respectively.

Both /ta/ and /ka/ stimuli had the same f_0 ; f_0 increased to a value of 120 Hz over 90 ms, remaining at a value of 120 Hz over 108 ms, finally reaching a steady-state value of 54 Hz over 205 ms. Stimulus /ka/ consisted of a formant transition period followed by a 484 ms steady state period. Onset frequencies for $F1$ and $F2$ were 271 Hz and 1974 Hz respectively. These two formants reached the vowel steady-state frequency of 687 Hz and 987 Hz over 64 and 75 ms respectively. $F3$ did not have a transition period, just a steady-state value of 2545 Hz. Stimulus /ta/ consisted of a formant transition period followed by a 481 ms steady-state period. Onset frequencies for $F1$ and $F2$ were 416 Hz and 1606 Hz respectively. These two formants reached a vowel steady-state frequency of 726 Hz and 1016 Hz over 64 ms, respectively. $F3$ did not have a transition period, just a steady-state value of 2584 Hz.

The parameter files were saved as .wav files and stimulus duration was modified in Cool Edit 2000 (Syntrillium Software Corporation, Scottsdale, AZ, USA). The CV stimuli were converted to stereo .wav files in Cool Edit 2000 to construct dichotic standard stimuli of either /ba/ or /ta/. Dichotic deviant stimuli were also constructed so that the standard stimulus /ba/ or /ta/ would be presented to one ear while the deviant stimulus /da/ or /ka/ respectively, was presented to the other

ear. Onset to offset duration was 362 ms. The offset to onset inter-stimulus interval was 638 ms, so that the duration from the onset of one stimulus and the onset of the next stimulus was 1000 ms.

Tone stimuli were sinusoidal pure tones of 1 kHz/1.2 kHz, which were generated in Matlab (The Mathworks Inc) at sampling rate of 44100 Hz. Tones had a steady-state duration of 352 ms and 5-ms raised-cosine on- and off-set ramps (therefore all stimuli had the same total duration of 362 ms). The stimulus onset to offset duration was 362 ms and again an inter-stimulus silent interval of 638 ms was specified so that the total onset-onset length of each trial was 1000 ms. The pure tones and inter-stimulus silent sequences were stored as .wav files. The pure-tone stimuli were converted to stereo .wav files in Cool Edit 2000 to construct diotic standard stimuli (1.0 kHz) and dichotic deviant stimuli (a 1 kHz stimulus presented to one ear and a 1.2 kHz stimulus presented to the opposite ear).

2.4. Conditions

This study had four independent variables. Three were within-subject: type of stimulus presented (speech, tone), head region from which the response was measured (right, mid, left), and ear of deviant sound presentation (right, left). The fourth was the between-subjects variable of group (dyslexic, control). The dependent variable was the mean amplitude of the mismatch negativity in microvolts (μV).

2.5. Apparatus

All stimuli were stored and output via a personal computer (PC) with a 16-bit resolution soundcard (Creative Labs Sound Blaster Live! 5.1) situated outside an IAC electrically-shielded single-walled sound-attenuating booth. The stimuli were presented through Sennheiser HD25 headphones (frequency response of 16–22 kHz; -3 dB/1.0 kHz). The headphone input came directly from the output of the soundcard DAC, and all stimuli were presented at 75 dB SPL.

The EEG activity was recorded using a SynAmps amplifier and the Acquire option of Scan 4.2 software (NeuroScan, El Paso, TX, USA). The AC signals were amplified with a gain of 250 dB (resolution of 0.336 μV /bit) and digitally sampled at an A/D rate of 500 Hz (16-bit resolution). The signals were filtered with a low-pass cut off of 70 Hz and a high-pass cut off of 0.05 Hz. A notched-filter with a centre-frequency of 50 Hz was used to reduce any mains-power generated frequency-interference. An external TTL trigger code was used to trigger event recording; the trigger-delay was maintained at 3 ms throughout testing. The EEG was displayed and stored in a second PC outside the sound booth, with a 24-bit resolution soundcard.

Listeners were fitted with an electrode cap fitted with sintered silver/silver chloride scalp ring electrodes positioned in an electrode 'Easy Cap' (Falk Minow Services, Herrsching-Breitbrunn, Germany) for 32/40 channels. The electrodes used were positioned at the following 10–20 scalp recording positions: FP1, FP2, AFz, Fz, F3, F4, F7, F8, FC3,

Table 2

Mean MMNs (μV) for dyslexics versus controls, divided by stimulus, head region, and ear of deviant presentation

Stimulus type	ba/da	ba/da	ta/ka	ta/ka	Tone	Tone
Ear of presentation	Right	Left	Right	Left	Right	Left
Mean MMNs (μV)						
<i>Controls</i>						
Right head	-0.73	-0.86	-1.04	-1.00	-1.64	-1.80
Mid head	-1.08	-0.68	-0.56	-1.63	-1.68	-1.60
Left Head	-0.82	-0.72	-0.70	-1.09	-1.63	-1.78
<i>Dyslexics</i>						
Right dipole	-0.72	-0.69	-1.39	-0.80	-0.82	-0.96
Mid head	-0.60	-0.77	-1.27	-0.92	-1.07	-0.86
Left head	-0.56	-0.82	-0.86	-0.97	-0.87	-0.86

FC4, FT7, FT8, T7, T8, TP7, TP8, Cz, C3, C4, CP3, CP4, Pz, P3, P4, P8, Oz. The electrode at position AFz served as the ground electrode (Jasper, 1958). Two electrodes were positioned at the following positions: left mastoid (M1 L) and right mastoid (M2R). Two electrodes were placed

above and below the left eye and two were placed 1 cm from the outer canthi of the right and left eye to record vertical and horizontal eye movements. An electrode on the nose was originally used as the reference, but it was later shown to be more reliable to re-reference to

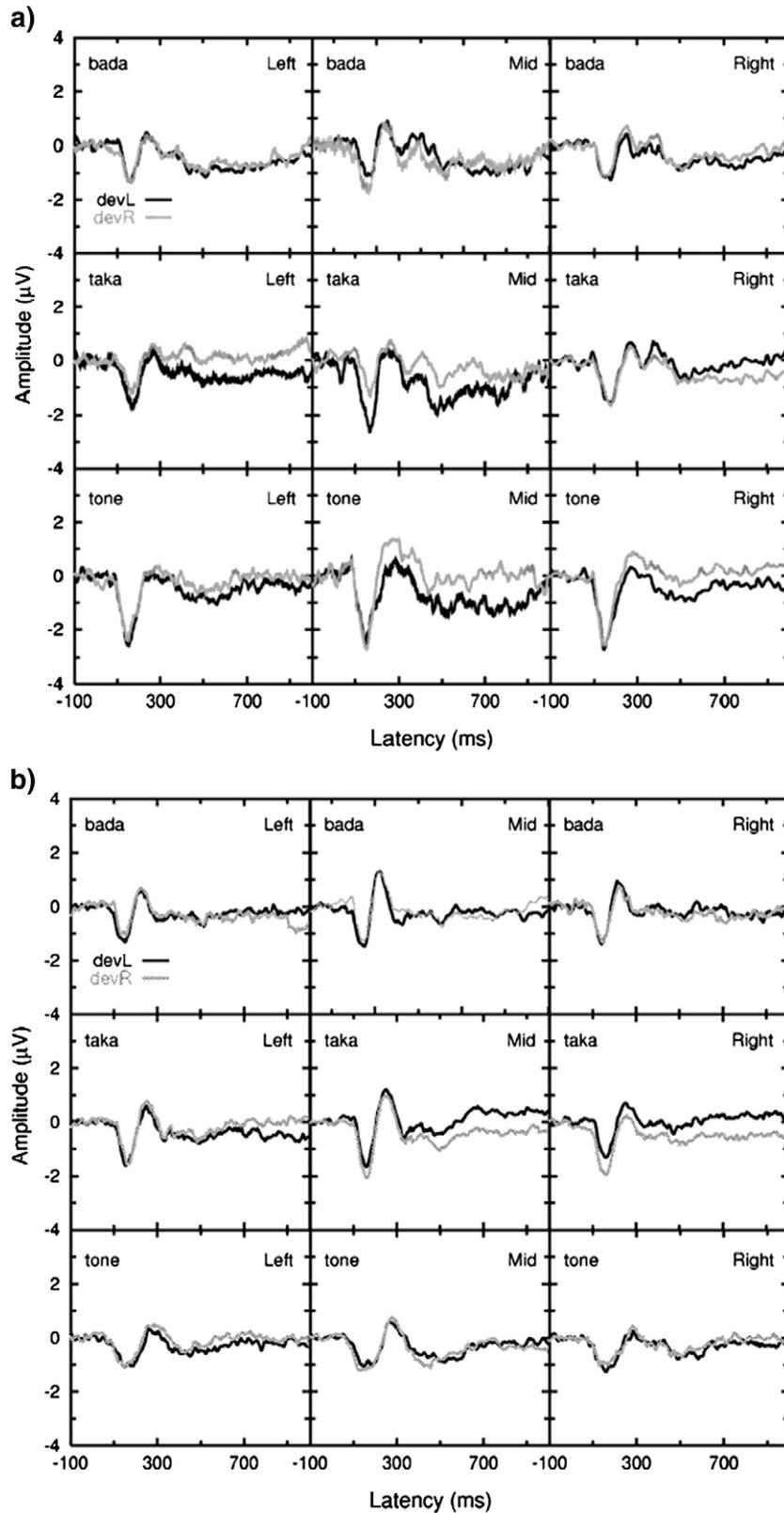


Fig. 1. Mean MMN response to standard versus deviant stimuli at each head region and stimulus type, for a) controls and b) dyslexics. Each row presents the grand average MMN waveforms for the 10 listeners for the stimulus conditions ba/da, ta/ka and tone. Each panel presents the left-ear deviant (dark gray line) and right-ear deviant (light gray line) MMN waveforms for electrode groupings specifying the left-, mid- or right cortical regions (as specified within the text).

all electrodes (minus the eye-electrodes). Inter-electrode impedance was maintained at a minimum with the impedance typically below 5 k Ω . An electrolyte conductive gel (Quick-Gel; NeuroScan, Compumedics, El Paso, TX) was used to reduce skin-electrode conductive impedance. Electrodes were connected to the EEG amplifier (NeuroScan, El Paso, TX, USA) using a 38 channel input box and a 38 channel electrode board adaptor. The data were recorded continuously and stored for later off-line analysis.

2.6. Procedure

The experiment consisted of three blocks (ba/da, ta/ka, and 1 kHz/1.2 kHz). Each was presented once with headphones correctly worn, and once with the headphones reversed as a control measure. Therefore each participant listened to six blocks, presentation of which was randomised. The experiment used a dichotic oddball paradigm. For each block, listeners were presented with the repeating standard from the relevant stimulus pair in both ears simultaneously (ba, ta, or 1 kHz), but the paired deviant stimulus (da, ka, and 1.2 kHz respectively) had a 10% probability of being dichotically presented in either the left or right ear instead of the standard. Each block consisted of 500 stimuli: 400 standard stimuli and 100 deviants, 50 presented in the left ear, and 50 in the right ear. Presentation of dichotic stimuli to either the left or right ear was randomised.

The listeners were seated in a single-walled sound-attenuating booth (IAC, electrically-shielded), 1 m from a television screen (0.2 m \times 0.27 m). They watched a film with the sound muted (and without subtitles) whilst the stimuli were presented through the headphones. Listeners were requested to not actively attend to the auditory stimuli and to keep overt movements to a minimum. Typically listeners would have a break after the first three blocks, and each block lasted nine minutes.

2.7. EEG analysis

The EEG recordings were processed off-line using Scan 4.3 software (NeuroScan Labs, El Paso, TX). After visual inspection of files and removal of bad electrodes (electrode Oz had to be removed from the analysis from all participants (control and dyslexics), in addition P3 was eliminated from the analysis for three participants), the EEG was re-referenced to all electrodes (minus the eye-electrodes). This produced a better signal-to-noise ratio than the nose reference. EEG data from each electrode was divided into epochs lasting 1000 ms (-200 to +800 ms). The VEOU and VEOL electrode activity was used to apply ocular artefact correction, the ocular artefact was applied with a positive-going EEG at 10% with 20 minimum sweeps with a duration of 400 ms. Epochs were rejected if peak amplitude exceeded ± 150 μ V in the 800 ms period after stimulus presentation. Mean epoch rejection rate was 5.93% for dyslexics and 4.87% for controls. An independent samples *t*-test showed that there was no significant difference between the rejection rate for the dyslexic versus control groups: $t(58) = .86$, $p = .40$. The epoched data were low-pass filtered with a low-pass cut off of 30 Hz and a low-pass attenuation of 6 dB.

In order to define the latency intervals for calculation of the greatest response negativity (MMN), the Global Field Power (GFP; an estimate of the signal-to-noise ratio) was calculated from the grand average of all listeners. The region of highest response corresponded to 100–200 ms post stimulus onset.

Average waveforms per electrode were generated based on stimulus type (ba/da, ta/ka, or 1/1.2 kHz), and on whether the stimulus presented was a standard stimulus (i.e. ba, ta, or 1 kHz), or a deviant (i.e. da, ka, or 1.2 kHz). Responses to deviants presented in the right versus the left ear were averaged separately, as ear of presentation was one of the independent variables. This meant there were nine sets of averages per subject (ba/da, ta/ka, and 1/1.2 kHz, for each of standard, left deviant, and right deviant stimuli). Responses collected from stimuli presented with the headphones reversed were at this stage

kept separate from stimuli presented with the headphones the right way round. Therefore each participant had 18 sets of average waveforms for each of the 28 electrodes (504 waveforms in total). These waveforms were filtered at 70 Hz to remove irregular artefacts in the data. Data obtained with headphones the right way round were then averaged with those in which the headphones were reversed. Each participant therefore had 9 rather than 18 sets of data per electrode. Grand averages were then computed at 2 ms bins. These grouped dyslexic ($n = 10$) and control ($n = 10$) listeners separately.

The MMN for the responses measured from each electrode site was then calculated for dyslexics and controls separately. The MMN was defined as the difference waveform for standard versus deviant stimuli at 100–200 ms after stimulus onset. Mean MMN to left-ear presented deviants was calculated by subtracting the mean amplitude for the standard stimuli from the mean amplitude for the left deviants. The same mean amplitude for the standard stimuli was subtracted from the right deviants in order to calculate the mean MMN to right-ear presented deviants.

Electrodes were then chosen for MMN comparison analysis. Equivalent electrodes on the left and right sides of the head were chosen: F3 and C3 on the left side (left cortical region), and F4 and C4 on the right side (right cortical region). More laterally positioned electrodes were originally included in the analysis, but these were found to be unreliable as the N1-P2 waveform had begun to invert. A 'mid head' group of electrodes, Fz and Cz, were also included as a third point of comparison (mid cortical region). MMNs for these pairs of electrodes were averaged, creating three groups: 'right head', 'mid head' and 'left head.'

2.8. Source analysis

Prior to dipole analysis, Principle Component Analysis (PCA) was carried out on the selected latency intervals for the MMN to provide an estimate of the number of components/dipoles. Based on the PCA result, Independent Component Analysis (ICA) was performed to select viable (highest signal-to-noise ratio) components for Source analysis. For latency intervals corresponding to the MMN, ICA typically revealed 2–3 components. Spatiotemporal dipole modeling was then performed using Source (Neuroscan) as follows. Since digitized positions of the electrodes were not available, the standard Source label-matching algorithm was used which is based on a model of a conventional extended 10–20 positioning of electrodes across a median scalp measure. In order to estimate EEG source activity a fixed symmetrical dipole model was applied and dipole location and orientation were jointly fitted for all time points. For each time point the Source strength parameter was iteratively adjusted until the residual variance (difference between the recorded versus modeled activity) was minimized. This model localized 2 hemispherically-symmetrical sources (minimally 10 mm apart) using a three spherical shells volume conductor model. The outermost shell (skin) was fitted to all used electrodes, and the inner shells were specified at 93% (outer skull compartment) and 85% (inner skull compartment, or brain compartment) of the outermost radius.

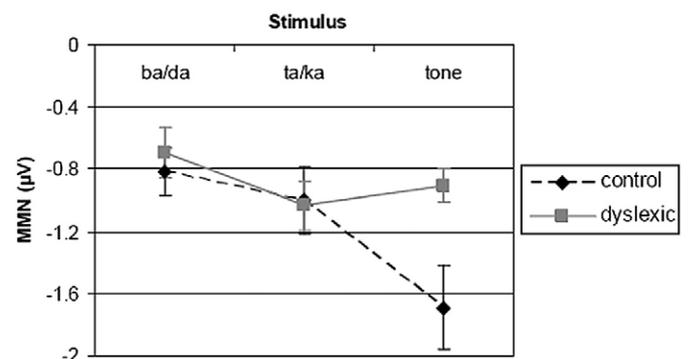


Fig. 2. Two-way interaction between MMN magnitude for stimulus type [tone (1 kHz/1.2 kHz), ba/da and ta/ka] and participant group (control and dyslexic).

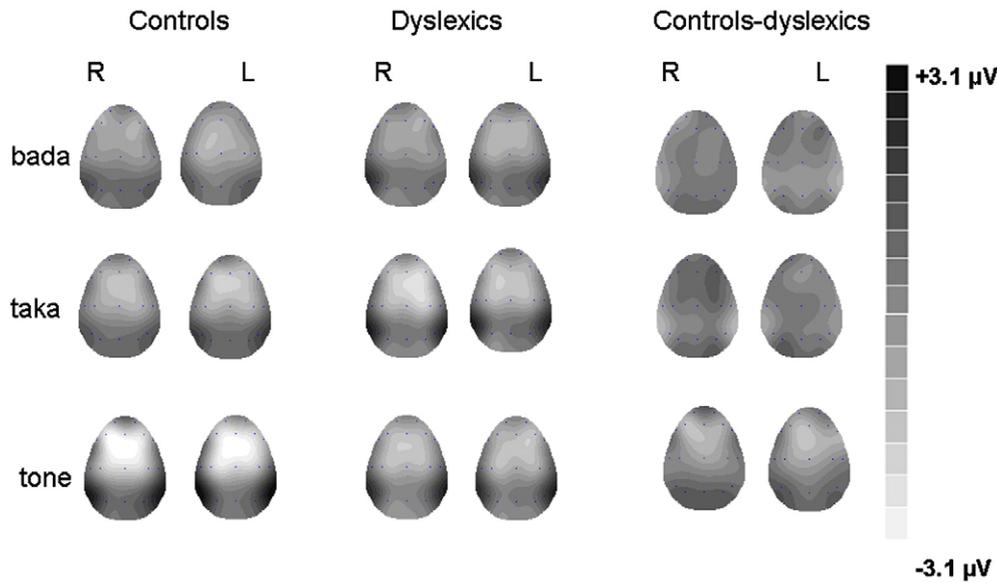


Fig. 3. Topographic maps for the MMN latency interval for controls, dyslexics and controls minus dyslexics (Control-Dyslexics) for the conditions of bada, taka and tone stimuli.

The conductivities were set at 0.33, 0.0042, and 0.33 S/m for the outermost shell, outer skull compartment and inner skull compartments respectively. Dipole solutions were obtained for time windows of 100–200 ms corresponding to the peaks of the MGFP curve. The main criteria used to specify source locations were low standard deviations (typically below 60% although in some cases this value had to be exceeded in order to provide a meaningful fit) and minimal confidence ellipsoids.

3. Results

3.1. MMN

For the early MMN, paired sample t-tests showed that there were no significant differences between the two different speech stimuli used (ba/da and ta/ka) for controls. However, for dyslexics there were significant differences between MMNs to ba/da versus ta/ka when the deviant was presented in the right ear at both mid-head electrodes: $t(9)=3.06, p=.014$, and right head: $t(9)=3.89, p=.004$. Therefore data for the two types of speech stimuli were kept separate for the subsequent analyses. See Table 2 for a table of the mean MMNs used for analysis for controls and dyslexics.

Fig. 1a and b presents the mean MMN response amplitudes at each head region and stimulus type, for controls (Fig. 1a) and dyslexics (Fig. 1b).

A repeated-measures four-way ANOVA was conducted on the MMN data presented in Fig. 1a and b using SPSS version 12.0.1. Within-subject factors were stimulus (ba/da, ta/ka, tones), head (right, mid, and left), and ear (right, left). The between-subjects factor was

Table 3

The grand average dipole source strength (nAm) estimated for the average MMN

Stimulus type	ba/da	ba/da	ta/ka	ta/ka	Tone	Tone
Ear of presentation	Right	Left	Right	Left	Right	Left
Dipole strength (nAM)						
<i>Controls</i>						
Right dipole	16.05	32.50	33.97	35.17	41.22	61.58
Left dipole	51.61	45.75	31.18	39.82	38.53	41.31
<i>Dyslexics</i>						
Right dipole	53.19	55.61	74.18	49.55	35.40	50.54
Left dipole	44.73	51.03	56.70	56.21	36.95	34.78

group (dyslexic, control). As only five controls completed cognitive tests, it was necessary to compare the MMNs of those not tested with those who were, in order to make sure that any differences between dyslexics and controls could be extended to the whole sample. No differences were found between these controls using the repeated measures four-way ANOVA: $F(1,8)=.89, p=.37$, and group did not interact with any other factor. The five controls without cognitive test

Table 4

a) Dipole position [coordinates, x,y,z (mm)] and orientation (unit vector, x,y,z) for all conditions b) average dipole orientation (unit vector, x,y,z) for all conditions

Conditions	Dipole	Dipole position (mm)			Dipole orientation		
		x	y	z	x	y	z
Controls ba/da right	Left	-57.7	30.00	57.10	0.63	-0.68	-0.39
	Right	57.7	30.00	57.10	-0.59	-0.56	-0.58
Controls ba/da left	Left	-63.7	-5.70	107.80	-0.31	-0.73	-0.61
	Right	63.7	-5.70	107.80	0.31	-0.79	-0.53
Controls ta/ka right	Left	-29.9	21.50	74.70	0.73	-0.42	-0.54
	Right	29.9	21.50	74.70	-0.71	-0.57	-0.41
Controls ta/ka left	Left	-44.7	24.20	62.00	0.60	-0.45	-0.66
	Right	44.7	24.20	62.00	-0.62	-0.67	-0.40
Controls tone right	Left	-52.7	-4.00	115.50	-0.45	0.89	0.00
	Right	52.7	-4.00	115.50	0.34	-0.88	0.33
Controls tone left	Left	-43.1	7.40	56.30	0.14	-0.63	-0.77
	Right	43.1	7.40	56.30	-0.12	-0.84	-0.52
Dyslexics ba/da right	Left	-28.4	1.30	42.20	0.66	-0.47	-0.58
	Right	28.4	1.30	42.20	-0.79	-0.47	-0.40
Dyslexics ba/da left	Left	-27.5	-3.90	61.10	0.66	-0.39	-0.65
	Right	27.5	-3.90	61.10	-0.65	-0.68	-0.34
Dyslexics ta/ka right	Left	-27.5	3.90	41.00	0.66	-0.43	-0.62
	Right	27.5	3.90	41.00	-0.53	-0.59	-0.60
Dyslexics ta/ka left	Left	-27.5	-4.40	47.30	0.63	-0.47	-0.62
	Right	27.5	-4.40	47.30	-0.72	-0.57	-0.40
Dyslexics tone right	Left	-28.0	-1.30	38.20	0.31	-0.49	-0.81
	Right	28.0	-1.30	38.20	-0.34	-0.76	-0.55
Dyslexics tone left	Left	-37.3	11.90	38.20	0.37	-0.46	-0.81
	Right	37.3	11.90	38.20	-0.24	-0.71	-0.67
b)							
Average controls (orientation)		x	y	z			
Left dipole		0.22	-0.34	-0.50			
Right dipole		-0.23	-0.72	-0.31			
Average dyslexics (orientation)							
Left dipole		0.55	-0.45	-0.68			
Right dipole		-0.55	-0.63	-0.49			

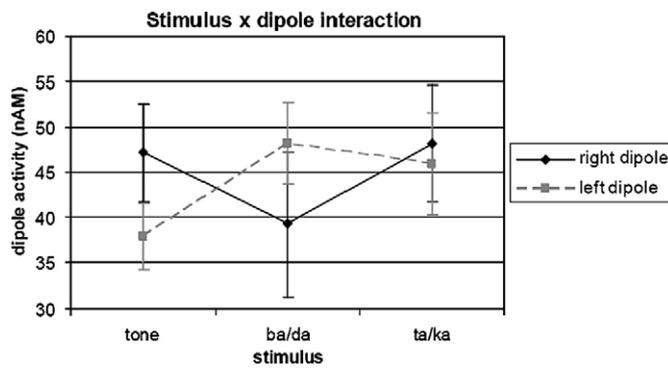


Fig. 4. Two-way interaction between stimulus type [tone (1 kHz/1.2 kHz), ba/da and ta/ka] and dipole activity in the left and right hemisphere.

data were also compared to the dyslexic group and 5 random dyslexics (so as to have equality of sample size). In both analyses, the results obtained closely matched those from the whole sample, and so homogeneity within the control group was assured. Greenhouse Geisser corrections were used where appropriate.

Including all participants, for within-subject factors, there was a main effect of stimulus: $F(2,36)=9.13$, $p=.001$. Analysis of simple effects showed that MMNs for ba/da were significantly smaller than to ta/ka ($p=.024$), as might be expected from the significant difference in the dyslexic group. In turn, MMNs to ta/ka were significantly smaller than to tones ($p=.046$). MMNs to ba/da were significantly smaller than for tones ($p=.001$).

The between-subjects main effect of group was not significant. There was a significant two-way interaction between group and stimulus: $F(2,36)=5.65$, $p=.006$ (see Fig. 2). Analysis of simple effects showed that this was due to a significant group difference for tone stimuli ($p=.014$), with the control group showing greater MMNs, but no group differences were found for either ba/da or ta/ka stimuli. When data from each group were analysed separately, significant differences were found between speech and tone stimuli in the control group: for ba/da versus tone, $p<.001$; for ta/ka versus tone, $p=.002$, but there was no difference between ba/da and ta/ka. This pattern was reversed in the dyslexic group, with the only significant difference being between ba/da and ta/ka ($p=.038$), with MMNs to ta/ka greater than those for ba/da. Group did not interact with any other factor.

Fig. 3 presents topographic voltage maps for the MMN latency interval. For the controls, the topographic maps show a negative focus at the midline fronto-central sites (most prominent for the tonal stimuli), and bilateral positive foci at the posterior-temporal sites (again most prominent for the tonal stimuli). For the dyslexics, the topography of the MMN, as for the controls shows a negative focus at the midline fronto-central sites together with positive bilateral anterior and posterior temporal sites. Dyslexic topographic maps show a markedly smaller negativity to the tonal stimuli compared to the

controls. The topographic maps constructed from a subtraction of the dyslexic maps from the control maps emphasises the differences between dyslexics and controls in the fronto-central sites.

3.2. Dipole source analysis

The grand average dipole source strength estimated for the MMN intervals for right and left dipoles for all experimental conditions is presented in Table 3. The dipole positions [x,y,z coordinates (mm)] and orientations (x,y,z , unit vector) are presented in Table 4a) for all conditions, and Table 4b) for the average across conditions.

A four-way mixed model ANOVA was conducted on the dipole activation data (nAm). Stimulus (tone, ba/da, ta/ka), Ear (right, left), and Dipole (right hemisphere, left hemisphere) were within-subject factors, and Group (dyslexic, control) was a between-subjects factor. Again, dipole activation data from the five controls with cognitive test data was compared with dipole data from those without: no differences were found, suggesting homogeneity within the control group.

There were no main effects of any within-subject factor. There was a two-way interaction between Stimulus and Dipole: $F(2,36)=4.53$, $p=.018$. Although analyses of simple effects did not reveal any significant differences, Fig. 4 suggests a greater response of the right dipole to tone stimuli than to ba/da (though not ta/ka), and a greater response of the left dipole to ba/da (again not ta/ka) than tone. No other two-way interactions involving within-subject factors were seen.

There was a significant three-way interaction between Stimulus, Ear, and Dipole: $F(2,17)=9.24$, $p=.002$. Analysis of simple effects revealed a significant difference between right and left ears for the right dipole, for tone stimuli only: $F(1,18)=5.36$, $p=.033$, with left ear responses being greater than right. This could represent a preferential response of the right hemisphere to tone stimuli presented to the left ear.

There was no main effect of the between-subjects factor Group, and no two-way interactions involving Group. However, there was a three-way interaction between Stimulus, Dipole, and Group (Fig. 5). Analysis of simple effects showed that for controls there was a significant difference in response to tone and ba/da at the right dipole ($p=.029$), and a trend towards significance for a difference in response to tone vs. ta/ka ($p=.057$), both in the direction of a lesser response to the speech stimuli. There were no differences in response to any of the three stimulus types at the left dipole. This would seem to suggest that in controls, the right hemisphere does not respond so strongly to speech as tones, whereas the left hemisphere responds fairly equally (possibly reflecting specialization for speech). Effectively, this result can be interpreted as a two-way interaction between Stimulus and Dipole for controls only.

For dyslexics however, there was a significant difference between responses to tone and ta/ka for both dipoles ($p=.034$ for right dipole and $p=.036$ for left dipole), i.e. no interaction between Stimulus and Dipole. There were no significant simple effects involving ba/da; however it is clear from Fig. 5 that activation to ba/da stimuli is in the

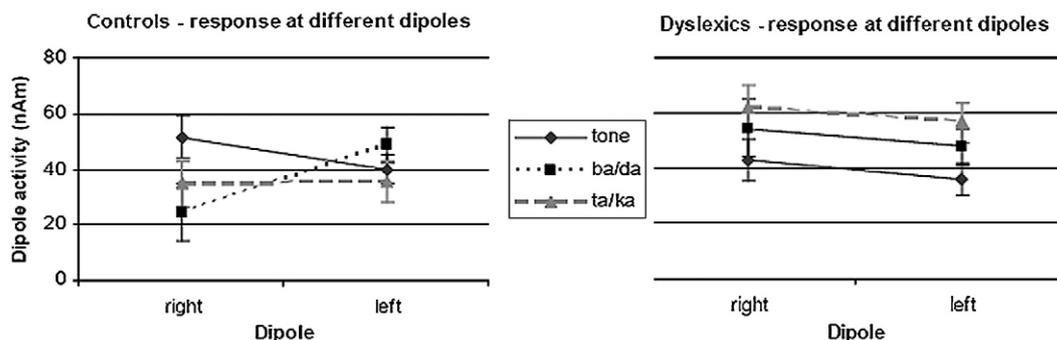


Fig. 5. a) Presents mean dipole activation to each stimulus type [tone (1 kHz/1.2 kHz), ba/da and ta/ka] at dipoles within the right and left dipoles for the control participants. b) Presents mean dipole activation to each stimulus type [tone (1 kHz/1.2 kHz), ba/da and ta/ka] at dipoles within the right and left dipoles for the dyslexic participants.

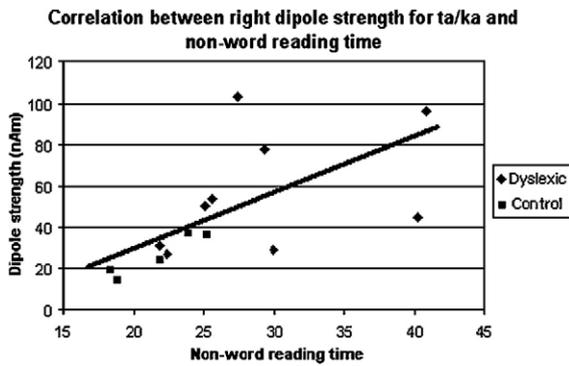


Fig. 6. Correlation between dipole strength for ta/ka and non-word reading time for 10 dyslexic participants and the 5 controls who provided cognitive test data.

same direction to ta/ka activation. The difference between speech and tones is also in the opposite direction to that of controls: for controls, the right dipole was more activated by tones, whereas in the dyslexics both dipoles are more activated by speech (significantly so for ta/ka stimuli). This might reflect a right hemisphere compensatory mechanism in the dyslexic group. The four-way interaction between Stimulus, Ear, Dipole and Group was not significant.

Pearson's correlations were conducted, correlating cognitive test scores with dipole activations for dyslexic individuals ($N=10$), and for the dyslexic group plus the five controls who completed cognitive tests ($N=15$). No significant correlations were found when looking at the 10 dyslexics, possibly due to low power. Including the five controls, two correlations were significant: a positive correlation between right dipole activation to ta/ka stimuli and mean non-word passage reading time: $r=.725$, $p=.002$, and a positive correlation between right dipole activation across stimuli and mean non-word passage reading time: $r=.615$, $p=.015$. The latter correlation was no longer significant when a reading time outlier (Participant 2 in the dyslexic group) was excluded. The correlation between right dipole activation to ta/ka and non-word passage reading time retained significance: $r=.613$, $p=.020$ (Fig. 6). This relationship suggests that the slower the reading time, the larger the right dipole activation to ta/ka stimuli. This may represent more inefficient processing in those with slower reading times. (However, it should be noted that this result is not corrected for multiple comparisons: if the p -value is Bonferroni corrected for 5 comparisons (ta/ka values were correlated with all 5 cognitive test measures), then the relationship would not reach significance at the new p -value of $p=.01$.)

Grand average headmaps depicting the dipole analysis results for all conditions are shown in Fig. 7. Dipole locations are indicated by black circles and dipole orientation by the attached bar. The dipole

sources occupy a location close to the auditory cortex with the dipole in the right temporal lobe in most cases orientated towards the contralateral hemisphere. However, not all dipole sources appear to be located within the temporal lobes. It is of interest to note that for the dyslexic population, in most cases, the dipoles appear to be located within areas more central [average dipole orientation across conditions (see Table 4b) of 0.55, -0.45, -0.68 (left dipole) and -0.55, -0.63, -0.49 (right dipole)] than the temporal dipole location observed within the control population [average dipole orientation across conditions (see Table 4b) of 0.22, -0.34, -0.5 (left dipole) and -0.23, -0.72, -0.31 (right dipole)].

4. Discussion

This study investigated size and lateralization of the MMN component in compensated dyslexics versus controls, for speech versus tone stimuli. There was a main effect of stimulus type, whereby MMNs were larger for tone than speech stimuli for both groups. This is in line with data from previous studies (e.g. Schulte-Körne et al., 2001), and may reflect the simplicity of pure tones compared to CV speech stimuli, making it easier to detect change in the former. An interaction between stimulus type and group was also obtained, with dyslexics and controls showing a similar MMN response to speech stimuli, but controls showing a much larger MMN response to tones than dyslexics. This suggests that dyslexics have an auditory processing deficit specific to tone stimuli. This contradicts several previous studies which have found evidence for a speech specific deficiency in MMN response in dyslexics (Csépe, 2003; Schulte-Körne et al., 1998). When group data were analysed separately, it was found that while the main difference in MMN size for controls fell between tones and speech, the main difference in dyslexics was between ba/da (smaller MMNs) and the other two (ta/ka and tones: larger MMNs). This result was unexpected, especially as dyslexic responses to ta/ka were even slightly greater than to tones. Source analysis also revealed group differences, with reduced lateralization of generators for tone stimuli in dyslexics, and a general right hemisphere preponderance in the dyslexic group for all types of stimuli. This may represent a compensatory mechanism in the current sample.

The finding of a specific deficit for tone stimuli in dyslexics compared to controls can be explained with reference to differences in stimuli and analyses performed between this and previous studies. As mentioned above, a group difference for tones may have been obtained in this study but not Schulte-Körne et al.'s (2001) because the deviant tone differed from the standard by 200 Hz in this study versus 440 Hz in theirs. This latter deviance may not have been sensitive enough to detect differences between groups. Alternatively, differences between studies may be the result of different frequencies used, as Schulte-Körne et al.'s (2001) used a standard of 2.2 kHz whereas the present

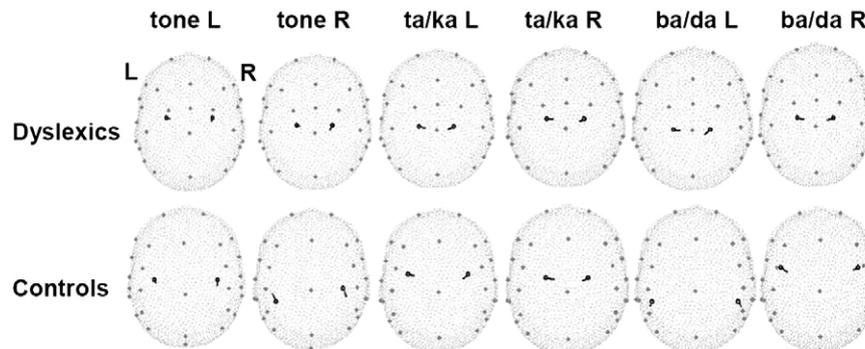


Fig. 7. Grand average headmaps of the dipole source analysis. Dipole location is indicated by the black circle and the dipole orientation is indicated by the attached bar. Small gray discs represent the electrode positions. The top row shows the headmaps for the latency interval corresponding to the MMN for all conditions, whilst the bottom row shows the headmaps for the interval corresponding to the LN for the same conditions. A capital R or L after the word-condition represents the ear of deviant presentation, i.e., either right (R) or left (L) ear.

study used a 1 kHz standard. However, it is still unexpected that dyslexics seemed to show such impairment with a deviance of 200 Hz, when Baldeweg et al. (1999) found that group differences began to subside with deviances of only 90 Hz. This may suggest that tone frequency discrimination can be more impaired than was previously thought, at least in some dyslexics. This finding cannot be attributable to differences in pitch, as Baldeweg et al. (1999) also used a standard stimulus of 1 kHz, with deviances in the same direction (i.e. 90 Hz versus 200 Hz higher in pitch).

It could be that the use of a dichotic method of stimulus presentation can account for these differences. Most studies of frequency difference processing in dyslexics have used a diotic presentation (the same stimulus in both ears), rather than a dichotic (or competing) stimulus presentation. Most diotic studies have reported impaired frequency processing for dyslexics vs. controls when using a frequency deviance of around 10% or less (Baldeweg et al., 1999; Maurer et al., 2003; Lachmann et al., 2005). As the frequency deviance is increased to between 10–20% within a diotic paradigm, there are no longer any observable differences in frequency processing between dyslexics and controls (Schulte-Körne et al., 1998, 2001; Kujala et al., 2006; Meng et al., 2005; Heim et al., 2000). In the present dichotic study a frequency deviance of 20% was used (1 kHz vs. 1.2 kHz), and clear differences in frequency processing were observable between the dyslexic and control groups. This suggests that a dichotic presentation widens the frequency deviance over which dyslexics are thought to be impaired for frequency processing. It may well be the case that when tonal (or speech) stimuli are presented in a competing situation (dichotically) that the inherent extra demands made on the auditory system may make apparent the specific discrimination problems encountered by individuals with dyslexia that differ from the demands made when using a diotic or monotic presentation.

Surprisingly, no group difference was obtained for speech stimuli. This could be due to differences in stimulus duration between this study and that of Schulte-Körne et al.'s (2001). Their CV speech stimuli were of only 110 ms duration, whereas the stimuli used in this study were 362 ms. Perhaps longer stimuli enable a firmer memory trace to be laid down for the standard, with which the deviant could then be compared (as suggested in Näätänen's, 1992 model). This might be particularly important in dyslexia. However, if so, it is hard to explain why duration did not have the same effect on tones. In addition, it could be argued that this would constitute a temporal processing deficit, yet behavioural psychophysical studies such as Marshall et al. (2001) have shown such a deficit in only a subset of dyslexics. Nevertheless, this interpretation does not imply a duration discrimination deficit per se, but only suggests a frequency discrimination deficit at shorter stimulus durations (McArthur and Bishop, 2001; Stoodley et al., 2006).

An alternative explanation is compensation. It could be that the dyslexic group, having compensated for reading deficits, and being educated to a high level, had also compensated for any speech processing deficits. Constant exposure to spoken language, and top down input regarding phonological structure through learning to read, may filter down to affect the perceptual level. In contrast, tone frequency discrimination might not show the same level of compensation, as it is not as well practiced or crucial a task for the type of dyslexic tested in this study. This may also explain the unexpected finding that MMNs to ta/ka were actually slightly more pronounced than to tones in the dyslexic group.

However, this still leaves a question as to why a significant difference between MMNs to ta/ka versus ba/da was found for this group. Both types of stimuli are stop consonants, both thought equally challenging to the auditory system, possibly due to a weaker short-term memory trace being laid down for this type of stimuli (Crowder, 1971). However, a MEG study by Diesch and Luce (1997) showed that the mismatch fields (MMN but recorded magnetically) generated by the contrast between da and ga was located more anterior than the source excited by the contrast between da and ba. While this did not result in any amplitude differences, the study was conducted on

healthy volunteers. It may be that in dyslexics, differences in location of source generation, also observed in this study (thought to reflect featural specificity of the mismatch response) also reflect differences in stimulus discriminability as measured by the mismatch response.

Another explanation for the greater MMN to the ta/ka combination than to the ba/da combination may be phonotactic probability. Phonotactic probability refers to the distribution of phonemes and phoneme sequences in the syllables and words of a language (Trask, 1996). Studies with a dyslexic population presented diotically with non-words with either a high or low phonotactic probability (PP) showed that dyslexics elicited a greater MMN for a low PP non-word than a high PP non-word (Bonte et al., 2007). In contrast, a similar diotic study using a control population found greater MMNs for high PP non-words compared to low PP non-words (Bonte et al., 2005). The present study used a dichotic presentation of CVs, and if the phonotactic probability (Vitevitch and Luce, 2004) of the position-specific occurrence of each phoneme (entered in Klattese) in a CV is calculated then the following values are obtained for the total phoneme-specific probability: ka (0.122), ba (0.0059), ta (0.0039), and da (0.0019). The greatest difference in the total phoneme-specific probability is observed for the ta/ka combination in comparison to the ba/da combination. A greater difference in phoneme-specific probability appears to correspond to the greater MMN amplitude observed in the dyslexic population, compared to the control group. It is not clear why this should be the case. One tentative suggestion is that in a competing presentation some combinations of phonemic-specific probabilities may be overcompensated for, but even so the greater dipole activation to the ta/ka combination within dyslexics is correlated with a slower reading time.

In terms of implications for theories of dyslexia, two possibilities present themselves from these data. Despite showing the hallmarks of impaired phonological representations (poor spelling and decoding), the dyslexic group seemed to show no basic impairment in speech processing. One possibility is that dyslexia is primarily a phonological deficit, not an auditory one, but that perceptual speech impairment could be a commonly co-morbid deficit not detected in the present sample. However, impaired tone perception suggests that some auditory deficits are present. These deficits could be unrelated to phonological difficulties, reflecting comorbidity that may or may not exacerbate the primary phonological impairment. Alternatively, tone perception deficits may reflect an aspect of a causal, underlying auditory processing problem that has not been compensated for. Sample size may have been too small to detect any correlations between phonological skills and MMN, but future studies could use larger samples. Ultimately however, caution is needed when interpreting these data. MMN vulnerability to methodological variation (Tervaniemi and Hugdahl, 2003), may well explain differences between this study and previous MMN investigations on dyslexia, as detailed above. Differences in MMN are in no way a clinical marker for dyslexia.

A further finding of this study regards lateralization. While left hemisphere lateralization for MMN response to speech was predicted for controls but not dyslexics, and right hemisphere lateralization was predicted for tones, in fact no group differences were found. One possible reason why a significant effect of a clear left-hemisphere lateralization was not seen may be that the CVs lacked meaning. Shtyrov et al. (2000) found no lateralization for non-speech complex sounds. As for meaningfulness, Scott et al. (2000) found a pathway specifically for intelligible speech (familiar and meaningful words) in the left temporal lobe. While this pathway is responsible for processing beyond the auditory cortex, where the MMN is mainly thought to be generated, it may influence MMN generation either through top-down modulation, or by making a partial contribution to the MMN signal. Our stimuli may not have activated this pathway, thus decreasing the contribution of the left hemisphere. The general right hemisphere preponderance for all types of stimuli in dyslexics (as suggested by the dipole analysis) might indicate an insufficient distinction between speech and tonal sounds in dyslexics.

A further explanation of our findings is that attention played a role, even though listeners were focussed on a distractor (film). It is thought

that the MMN can be generated pre-attentionally (Näätänen et al., 1978; Näätänen and Alho, 1995), but this doesn't necessarily mean that it can't be affected by attention, either conscious or covert. Schulte-Körne et al. (2001) explain their right hemisphere lateralization findings in terms of an automatic attention switch process mediated by the right frontal area (Giard et al., 1990). While this idea of covert, automatic attention may be controversial so early in processing, our listeners may have been paying some degree of conscious attention to the stimuli and this may have affected lateralization. Using MEG, Hertrich et al. (2002) found that synthetic stimuli elicited a right hemisphere lateralization in the early MMN component when listeners were paying attention, so it is possible that attention modulated MMN response to some extent. This could be investigated by including a condition with an active distractor task.

Given the unexpected nature of our findings, further work should elucidate their causes by varying meaningfulness, attentional load, and whether stimulus presentation is diotic or dichotic. It would also be useful to use a larger sample, so that meaningful correlations between cognitive test results and MMNs could be obtained. EEG measurements could also be related to MEG and fMRI data, so that the temporal accuracy obtained in EEG studies can be related to better spatial localisation of these components. Recent MEG studies on dyslexia have found source localisation differences between groups in the N100 m (Heim et al., 2003a) and P100 m (Heim et al., 2003b) components, and it would be interesting to see whether this would extend to the early MMNm, and whether this might explain the variability in stop-consonant perception in the dyslexic group as measured by MMN in this study. Another interesting comparison would be between adult dyslexics who had compensated versus those who had not. This has already been investigated at the word-reading level using fMRI (Shaywitz et al., 2003).

In conclusion, the current study employs a novel dichotic listening paradigm for investigating the MMN in compensated dyslexics and controls. Dichotic paradigms present a greater challenge to the auditory system than diotic presentation, and this may explain why a diminished MMN to tone stimuli was found in the dyslexic group compared with controls, even when the deviant differed from the standard by 20%. No group differences in MMNs for tone stimuli were found, nor did the lateralization of the MMN differ between groups. While some previous studies have found such group differences, methodological differences between this and other studies may account for our results. Source analysis once again differentiated the groups for tone stimuli, with reduced right hemisphere lateralization in the dyslexic group. This suggests that the auditory system may show less specialisation for different categories of stimuli in dyslexia, and may reflect a compensatory mechanism in the current sample.

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