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Speech perception deficit in dyslexic adults as measured by mismatch negativity (MMN)

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Abstract

Deficits in phonological processing are known to play a major role in the aetiology of dyslexia, and speech perception is a prerequisite condition for phonological processing. Significant group differences between dyslexics and controls have been found in the categorical perception of synthetic speech stimuli. In a previous work, we have demonstrated that these group differences are already present at an early pre-attentive stage of signal processing in dyslexic children: the late component of the MMN elicited by passive speech perception was attenuated in comparison to a control group. In this study, 12 dyslexic adults and 13 controls were assessed using a passive oddball paradigm. Mismatch negativity (MMN) was determined for both tone and speech stimuli. The tone stimuli yielded two MMN components, but no group differences. Three components were found for the speech stimuli. Multivariate testing for group differences yielded a significant result, and univariate *P* values revealed significant differences between dyslexics and controls in two of the three time windows. This suggests that speech perception as measured on an early, pre-attentive level plays a major role in dyslexia not only in children (as shown in our previous study) but also in adults.

Keywords: Dyslexia; Mismatch negativity; Speech perception; Auditory processing; Adults; Passive oddball paradigm

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1. Introduction

Dyslexia is a specific disability in reading and spelling despite adequate educational resources, a normal IQ, no obvious sensory deficits, and adequate sociocultural opportunity (Dilling et al., 1991). Dyslexia occurs in all languages, and spelling disability in particular often persists into adulthood. Prevalence estimates range from 4 to 9% (Shaywitz et al., 1990).

Deficits in phonological processing are known to play a major role in the aetiology of dyslexia (Elbro, 1996), and speech perception was found to be a prerequisite condition for phonological processing (Watson and Miller, 1993; McBride-Chang, 1995; Schulte-Körne et al., 1999b). In several studies, significant group differences have been found between dyslexic children and normals regarding the categorical perception of synthetic /ba/–/da/–/ga/ syllables (Godfrey et al., 1981; Manis et al., 1997; Werker and Tees, 1987). These studies used stimulus identification and discrimination tasks, which required the subjects to focus on the relevant stimulus dimension. These cognitive processes could have been influenced by attention, motivation, and memory-span performance, all of which have been demonstrated to be abnormal in dyslexia (Jorm, 1983; Schulte-Körne et al., 1991). Thus it remains unclear whether the deficits in speech perception demonstrated represent an underlying deficit in dyslexia, reflect a secondary effect, or are caused along with dyslexia by the same underlying, as yet unknown, deficit. Therefore, the question arises as to whether the speech perception deficit described in dyslexics occurs on the level of sensory perception which is characterised by pre-attentive and automatic processing.

A neurophysiological paradigm well-suited to examine pre-attentive and automatic central auditory processing is the mismatch negativity (MMN). This is a negative component of the event-related brain potential (ERP), elicited when a detectable change occurs in repetitive homogeneous auditory stimuli (Näätänen, 1992). The most commonly described MMN occurs at 100–300 ms post-stimulus onset although other studies have found later MMNs between 300 and 600 ms (Kraus et al., 1996). The MMN is elicited by any change in frequency, intensity or duration of tone stimuli, as well as by changes in complex stimuli such as phonetic stimuli (Näätänen, 1992). It is assumed to arise as a result of a mechanism that compares each current auditory input with a trace of recent auditory input stored in the auditory memory. The MMN usually reaches its amplitude maximum over the fronto-central scalp (Näätänen, 1992).

In a study with dyslexic children we have already demonstrated that the late component of the MMN elicited by passive speech perception was attenuated in comparison to a control group (Schulte-Körne et al., 1998). This attenuation was detected only with speech but not with tone stimuli, supporting the hypothesis that dyslexics have a specific speech processing deficit at a sensory level. Further evidence for a specific speech processing deficit in dyslexic children and adults came from the studies of Watson and Miller (1993) and Schulte-Körne et al. (1999b). They found no influence of the ability to discriminate tone stimuli or detect a gap between bursts on reading and spelling ability.

It is well described that dyslexic children often continue to have difficulties into adulthood, especially in spelling (Dilling et al., 1991), however, the role of speech perception in dyslexia in adults has not yet been extensively examined. Dyslexics have been shown to have difficulties in speech identification and discrimination tasks (Liberman et al., 1985; Steffens et al., 1992; Cornelissen et al., 1996), but tasks in all of these three studies required subjects to focus on the stimuli, thus the results may have been influenced by factors such as motivation and attention. In the current study we used a passive oddball paradigm which requires the subjects to focus on a sensory modality other than that of the test stimuli. We have examined a sample of spelling disabled adults to clarify whether the previously described deficits in speech perception in children can also be found in adults. To elicit an MMN we used synthetic speech as well as tone stimuli. The latter served as a control condition to examine whether the auditory processing deficit is specific for speech

Table 1
Sample characteristics of dyslexics and controls

	n	M:F	Age	IQ	Spelling percentage rank
Controls	13	4:9	29.0 ± 5.9	119.7 ± 14.6	78.2 ± 21.3
Dyslexics	12	8:4	30.5 ± 8.6	110.3 ± 14.6	11.1 ± 9.7

stimuli. It was hypothesised that as with children, adult dyslexics would have an attenuated MMN in the speech but not in the tone condition.

2. Methods

Twelve dyslexic adults were recruited through an adult education centre and an announcement in the Dyslexia Association Journal. The history revealed that all had had difficulties as children in reading and spelling, and a reading test (Schulte-Körne, 2000) revealed a significant difference in reading speed between them and the controls. Spelling was measured by an age-appropriate German spelling test (Jäger and Jundt, 1981) and spelling disability was diagnosed if there was a discrepancy of at least 1 S.D. between actual spelling ability and that predicted on the basis of IQ (linear regression model, Schulte-Körne et al., 1996). The 13 controls were recruited through advertisements in the local press, notices in the Psychology Department of the Philipps University of Marburg, and personal contacts. Exclusion criteria were: an uncorrected visual deficit, a hearing deficit, relevant psychiatric or emotional disorder, a history of fits, other neurological disorders, or a bilingual family. All subjects reported themselves to be strongly right-handed, had a good school attendance record and an IQ (measured by German adaptation of the Culture Fair Intelligence Test, CFT20; Weiß, 1987) greater than 85. For sample characteristics see Table 1.

Tone stimuli were produced by 2200 Hz (standard) and 2640 Hz (deviant) sine waves of 90 ms duration with 3 ms rise and 3 ms fall time. Synthesised speech stimuli were created by a Klatt synthesiser (Klatt, 1980), the standard stimulus was *da* and the deviant was *ga*. Each speech stimulus was of 110 ms duration. Stimuli were presented in two blocks: speech and tone. In each block, 1800 standard stimuli and 200 deviant stimuli were presented in a pseudorandom order with at least five standards between any two deviants and a constant onset to onset interval of 590 ms between stimuli. The stimuli were presented binaurally by insert earphones. The sound level was set to 70 dB SPL. Calibrations were performed with a Bruel and Kjaer 2235 sound level meter and an artificial ear (Bruel and Kjaer, model 4152) with a 6-cm^3 coupler to approximate the volume of the external ear. Subjects were seated in a comfortable chair in a quiet room. They were specifically instructed not to attend to the presented stimuli, and to aid them in this, a self-selected film was shown.

Electrodes were placed at 19 scalp sites based on the International 10–20 system: Fp1, Fp2, F7, F8, F3, F4, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, and O2 with reference to the linked ears. The ground electrode was positioned at Fpz. Horizontal and vertical eye movements and blinks were detected with two additional electrodes placed below the subjects' right and left eyes and the Fp1 and Fp2 electrodes. Electrode impedances were kept below 10 kΩ. The EEG was amplified by Schwarzer amplifiers, with time constant set to 0.6 s and using a low pass filter with half amplitude at 85 Hz. The EEG was recorded continuously and A/D converted with 12-bit resolution at a sampling rate of 172 Hz.

Artefact detection was facilitated by the use of a computer program developed at our Institute. EEG epochs in which either the EEG exceeded ± 70 μV or the electro-ocular (EOG) activity exceeded ± 30 μV relative to the baseline were automatically excluded from averaging, leaving a minimum of 68 deviant and 580 standard trials for averaging. This procedure was checked visually to verify its accuracy. ERPs were calculated

by averaging epochs of 750 ms (including a 50-ms prestimulus interval) separately for both standard and deviant stimuli. The first two standard EEG epochs following each deviant epoch were omitted from averaging because they were assumed to represent a mismatch process related to the preceding deviant. Data from T3 and T4 had to be excluded from further analyses because of an accumulation of muscle artefacts. Difference waveforms (MMN curves) were calculated by subtracting the averaged standard from the averaged deviant ERP. Peak amplitudes and latencies were calculated for each subject searching the maximum peak in the respective intervals (Figs. 1 and 2) gained from visual grand average inspection. Since the assumed maximum of activity was over Fz (Näätänen, 1992), data from this electrode were used for statistical calculations.

3. Results

3.1. Analysis of tone stimuli

Fig. 1 shows 2 MMN peaks (Fz), an early one (70–230 ms) and a late one (370–590 ms). Table 2 shows the peak amplitudes and the latencies of the two MMN components. According to visual inspection of the data, there is no evidence for latency differences between the groups.

A MANOVA was performed to examine the effect of group (dyslexics vs. controls) on the peak amplitudes. No significant differences were found. In order to compare the spatial distribution of the MMN activity between the two groups (dyslexia vs. controls), brain maps were calculated to show the mean amplitudes for the two time windows (MMN 1 and 2, Fig. 2).

There is no suggestion of a different lateralisation between the groups. There seem to be group differences at some of the electrodes regarding the average activation, but over the expected frontal leads group differences were not significant (Table 2).

3.2. Analysis of speech stimuli

Compared with the tone stimuli, the speech stimuli did not produce a comparable, clearly differentiated MMN. A number of different MMN components were produced, all of comparably low amplitude. In order to check which parts of the curve contain meaningful negativity rather than mere background noise, all values of the control

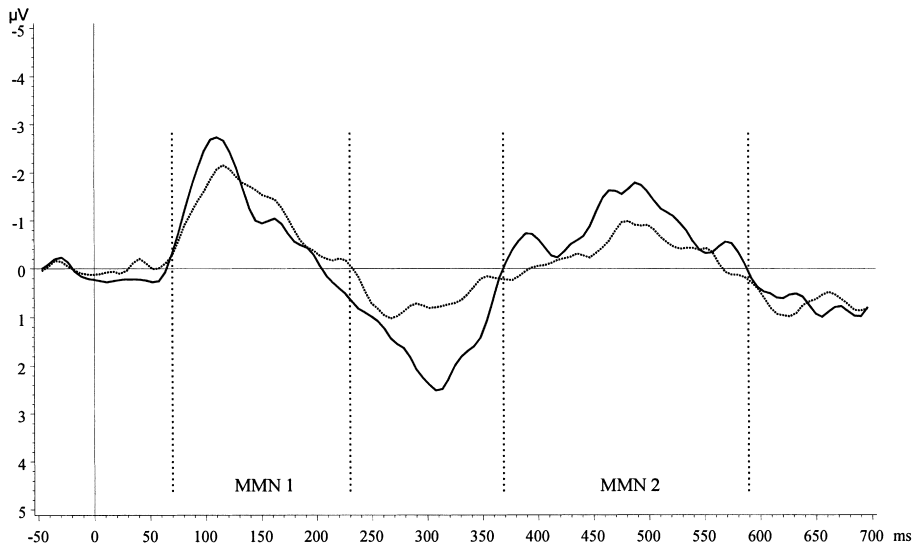

Fig. 1. Grand average of the mismatch negativity (MMN) for tone stimuli in dyslexic subjects (broken line) and controls (solid line) at Fz (fronto-central lead). The responses to the standard stimulus have been subtracted from those to the deviant stimulus.

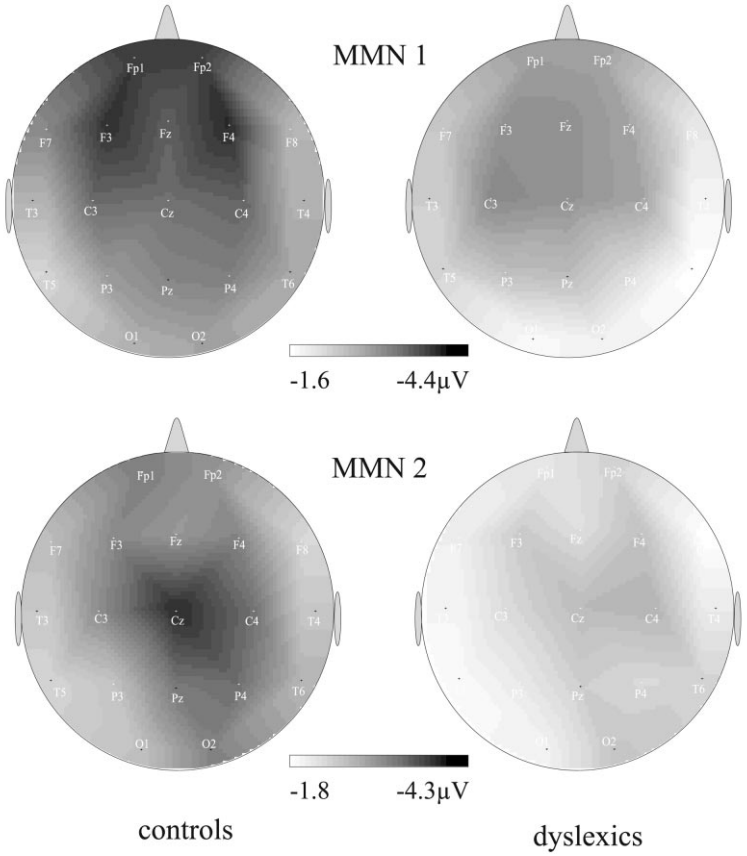

Fig. 2. Scalp distribution of the tone MMN in the two time windows. The grey steps indicate the amount of activation. The corresponding range (in μV) was set individually for each pair of scalp distributions.

group were tested against zero using *t*-tests. Values[1] in three areas proved to be significantly different from zero, and therefore the peak amplitudes from these three time windows were used for the purpose of further analyses (Fig. 3). In the group of dyslexics, however, only in window 1 an MMN could be found.

The time windows between MMN1 and MMN2, and between 100 and 220 ms also contained peak values comparable to those in the MMN windows 1–3. These parts of the graph were, however, not significant because the underlying variances were too large. According to visual inspection of the data, there is no evidence for latency differences between the groups. A further illustration of the data is provided by Figs. 4 and 5, which show not only the MMN graphs, but also the underlying curves of the standard and deviant stimuli.

Table 2
Means and standard deviations of the MMN peak amplitudes and latencies for tone stimuli

	Controls	Dyslexics
MMN1, amplitude	-3.88 ± 1.78 μV	-3.29 ± 1.19 μV
MMN2, amplitude	-3.22 ± 1.90 μV	-2.31 ± 1.90 μV
MMN1, latency	124.15 ± 28.51 ms	135.83 ± 46.55 ms
MMN2, latency	457.70 ± 54.22 ms	493.92 ± 52.16 ms

[1] Each value (data point) of the curve is the average of all subjects' amplitude values of that particular time and in the respective group. Visual inspection of the curve of the control group yielded four possible analysis windows with distinguishable peaks. *t*-tests in 3 of the 4 possible windows resulted in a considerable number of significant values.

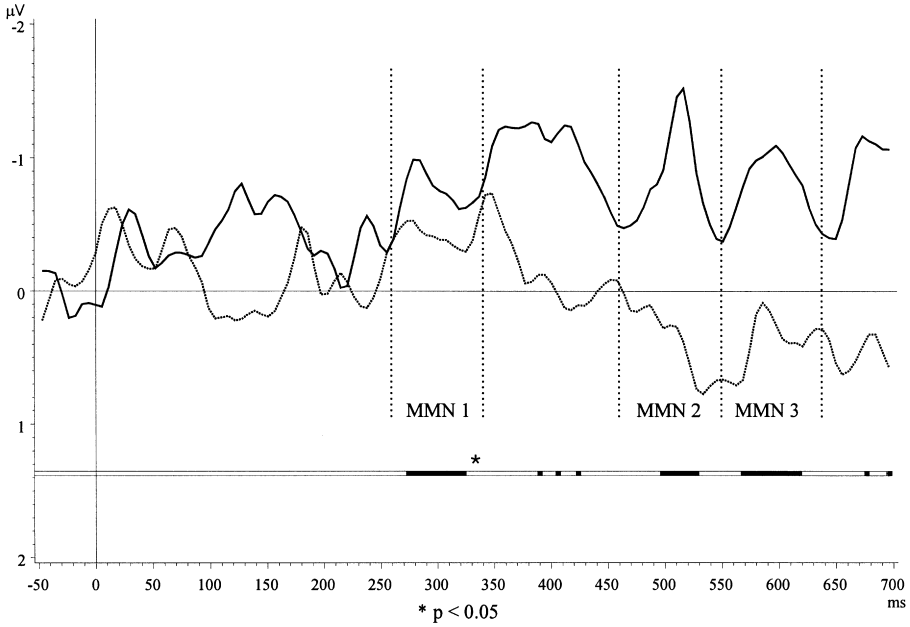

Fig. 3. Grand average of the MMN for speech stimuli in dyslexic subjects (broken line) and controls (solid line) at Fz. The responses to the standard stimulus have been subtracted from those to the deviant stimulus. The black bars indicate ranges of the curve with significant MMN values in the control group.

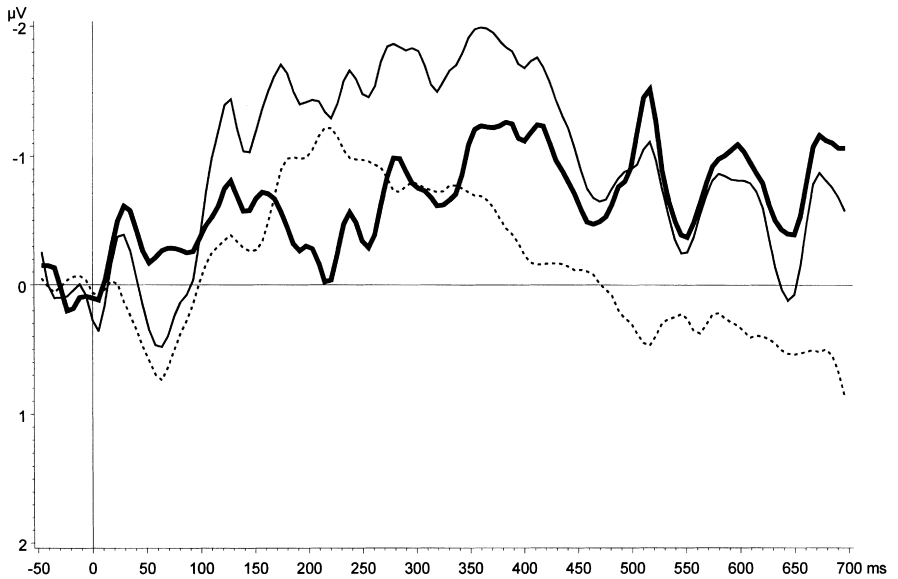

Fig. 4. Grand average of the standard stimuli (dotted line), the deviant stimuli (solid line), and the mismatch negativity (MMN, bold line) for speech stimuli in control subjects at Fz (fronto-central lead).

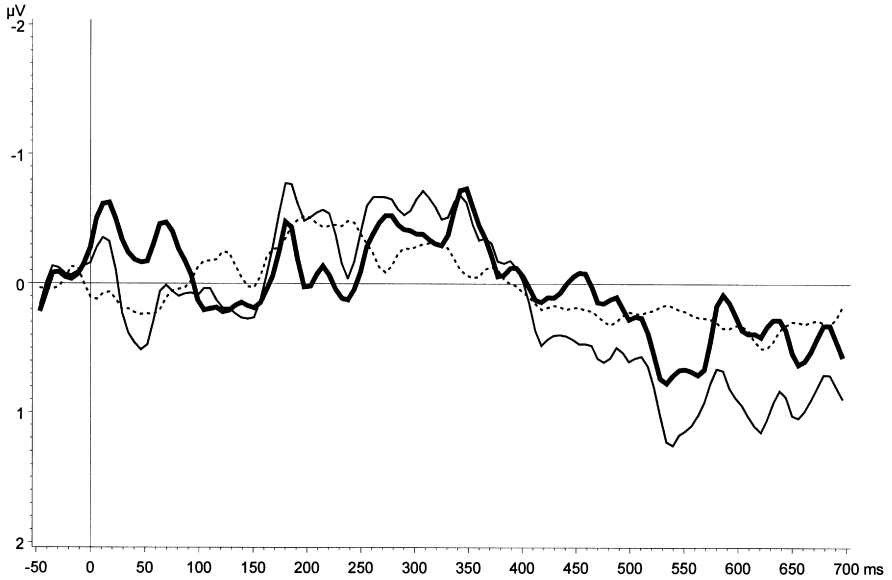

Fig. 5. Grand average of the standard stimuli (dotted line), the deviant stimuli (solid line) and the mismatch negativity (MMN, bold line) for speech stimuli in dyslexic subjects at Fz (fronto-central lead).

A MANOVA was also undertaken for the peaks of the three speech MMNs (main effect group, three dependent variables). The multivariate F value was significant ($P = 0.039$). Because of this borderline result, the univariate P values were also examined. These were $P = 0.24$ (peak 1), $P = 0.03$ (peak 2) and $P = 0.005$ (peak 3). Whilst caution must be exercised in interpreting these a-posteriori-analyses, it could be argued that the group differences are only present in the latter two time windows (Table 3).

In order to compare the spatial distribution of the MMN activity between the two groups (dyslexia vs. controls), brain maps were calculated to show the mean amplitudes for the three time windows (MMN 1, 2, and 3; Fig. 6).

Table 3
Means and standard deviations of the MMN peak amplitudes and latencies for speech stimuli

	Controls	Dyslexics
MMN1, amplitude	-2.51 ± 2.19 μV	-1.63 ± 1.27 μV
MMN2, amplitude	-2.53 ± 2.21 μV	-1.00 ± 0.70 μV
MMN3, amplitude	-2.53 ± 1.59 μV	-0.87 ± 0.99 μV
MMN1, latency	291.23 ± 26.89 ms	301.92 ± 27.39 ms
MMN2, latency	511.31 ± 21.25 ms	498.67 ± 30.84 ms
MMN3, latency	597.54 ± 24.01 ms	590.25 ± 28.53 ms

Fig. 6 demonstrates higher MMN activation in the control group over all three time windows. The maximal activation occurs over the right frontal region (F4). The lateralisation increases as the latency of the components increases. There is no suggestion of a different lateralisation between the groups.

4. Discussion

We have tried to find evidence supporting the hypothesis of a specific speech perception deficit in dyslexia by looking for a reduced MMN amplitude following the presentation of speech stimuli.

The group comparison yielded a significantly reduced MMN on speech stimuli in the dyslexic group. This difference appears to be speech specific[2] since it was not detected with the tone

[2] However, it is unclear which attribute of the speech stimuli, e.g. the rapid transition of formants, leads to the group differences.

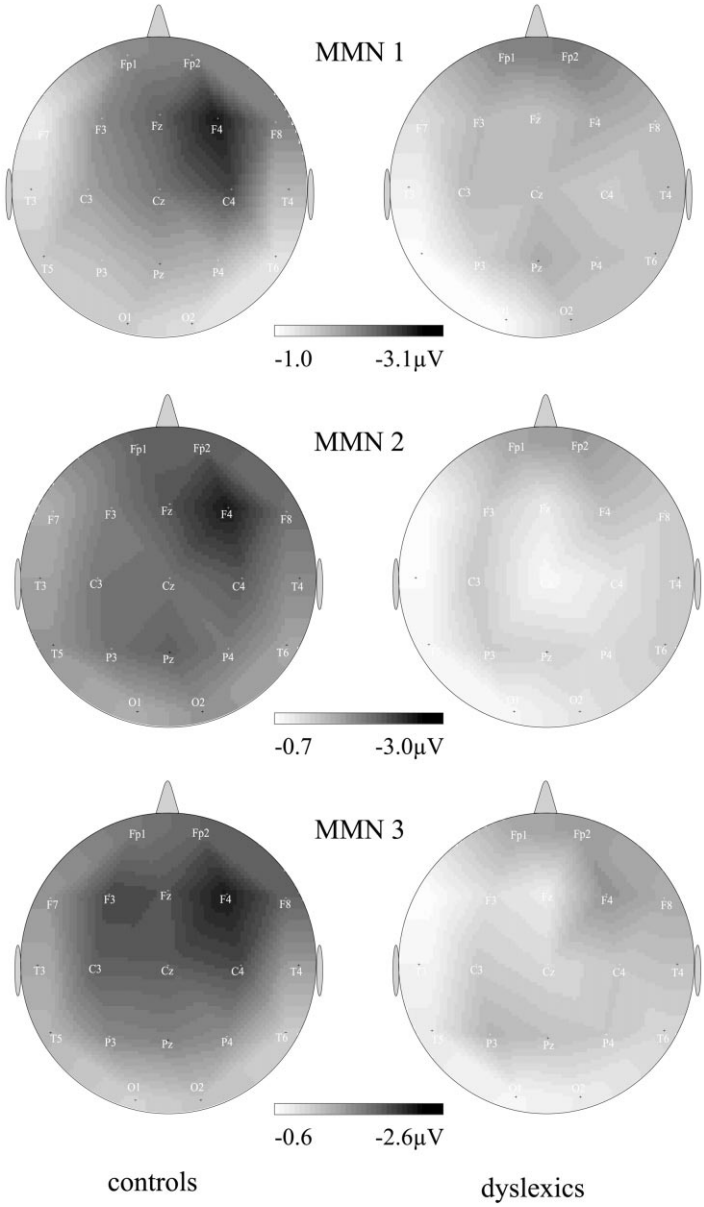

Fig. 6. Scalp distribution of the speech MMN in the three time windows. The grey steps indicate the amount of activation. The corresponding range (in μV) was set individually for each pair of scalp distributions.

stimuli. It can be concluded that dyslexic adults have a specific weakness in the passive perception of speech, as already shown in dyslexic children (Schulte-Körne et al., 1998). Another study supporting this result was conducted by Uwer et al. (2000). The authors found a reduced MMN in dyslexic children with speech, but not with tone stimuli.

However, Baldeweg et al. (1999) found a reduced area of the frequency MMN in dyslexics with tone stimuli. Although a total of three studies now favors a speech specific deficit in dyslex-

ics, with the deviating result of Baldeweg et al. (1999) this issue remains controversial.

An interesting finding was that the MMN appears to have more than one component, both for tone as well as speech stimuli. Only in the later components of the speech MMN were significant group differences detected. This finding concurs with the results of our previous work where dyslexic children and adults were found to have a significantly attenuated MMN elicited by speech and complex tone patterns between 300 and 600 ms (Schulte-Körne et al., 1998, 1999a). Several other groups have also found MMN activity later than 300 ms (Sams et al., 1990; Sandridge and Boothroyd, 1996; Sharma et al., 1993). In some other studies the interstimulus interval was so short that it would not have been possible to detect the presence of any later MMN component (Alain et al., 1998); whilst in others, the traces were discontinued after 300 ms (Nyman et al., 1990; Näätänen et al., 1997), such that the analysis of the later components was no longer possible. The works of Tremblay et al. (1997) and Winkler et al. (1999) have clearly documented the presence of MMN components in the time window from 300 to 500 ms in response to presentation of consonant vowel clusters. We feel it is important to further investigate the significance of these components, and in particular to clarify whether this represents an additional mismatch process that is tied to a speech specific deficit in dyslexics.

Fz was used to calculate the group differences; the brain maps show that there is lateralisation to the right hemisphere. Since this is true for both groups, it can be assumed that testing over F4 would result in the same group differences than over Fz. Testing over F4 was not carried out because a-posteriori testing is not recommendable for statistical reasons (the resulting p values would no longer be appropriate). The finding that the late component of the speech MMN is lateralised to the right frontal hemisphere might correspond to results of Kasai et al. (1999) who found that there are several generators of MMN in the temporal and frontal lobes of both hemispheres. Giard et al. (1990) found an MMN component over the right frontal area which might be related to an automatic attention switch process. In comparison to the more central speech MMN found in our study with dyslexic children, the late component of the speech MMN in adults has been lateralised to the right.

The processing deficit of the stop consonant speech stimuli which are characterised by brief and rapid spectral changes might correspond to the finding of Tallal (1980) that dyslexics are impaired when processing brief, rapidly changing auditory stimuli. We have recently found evidence for this hypothesis (Schulte-Körne et al., 1999a). Dyslexic adults were found to have a significant pre-attentive deficit in processing of rapid temporal patterns suggesting that it may be the temporal information embedded in speech sounds, rather than phonetic information per se, that resulted in the attenuated MMN found in dyslexics in previous studies.

The most likely interpretation of our data is that the attenuated MMN in dyslexics is speech specific. The works of Mody et al. (1997) and Adlard and Hazan (1998) also support the theory that dyslexics have a specific speech perception deficit. In these studies, however, active speech perception was investigated. The results of this study and the study with dyslexic children (Schulte-Körne et al., 1998) provide strong evidence that the speech discrimination difficulties of dyslexics occur already before conscious perception. The finding that speech perception is developed in early years of childhood (Kuhl et al., 1992) suggests that deficits in pre-attentive speech processing are a basic dysfunction in dyslexics which continues to have an important impact in dyslexia into adulthood.

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References

Adlard, A., Hazan, V., 1998. Speech perception in children with specific reading difficulties (dyslexia). Q. J. Exp. Psychol. A. 51, 153–177.

Alain, A., Woods, D.L., Knight, R.T., 1998. A distributed cortical network for auditory sensory memory in humans. Brain Res. 812, 23–37.

Baldeweg, T., Richardson, A., Watkins, S., Foale, C., Gruzelier, J., 1999. Impaired auditory frequency discrimination in dyslexia detected with mismatch evoked potentials. Ann. Neurol. 45, 495–503.

Cornelissen, P.L., Hansen, P.C., Bradley, L., Stein, J.F., 1996. Analysis for perceptual confusions between nine sets of consonant–vowel sounds in normal and dyslexic adults. Cognition 59, 275–306.

Dilling, H., Mombour, W., Schmidt, M.H., 1991. International Classification of Mental Diseases, ICD-10, German edition Huber, Bern.

Elbro, C., 1996. Early linguistic abilities and reading development: a review and a hypothesis. Read. Writ. 8, 453–485.

Giard, M.H., Perrin, F., Pernier, J., Bouchet, P., 1990. Brain generators implicated in the processing of auditory stimulus deviance: a topographic event-related potential study. Psychophysiology 27, 627–640.

Godfrey, J.J., Syrdal-Laskey, A.K., Millay, K.K., Knox, C.M., 1981. Performance of dyslexic children on speech perception tests. J. Exp. Child Psychol. 32, 401–424.

Jäger, R.S., Jundt, E., 1981. Mannheimer Rechtschreib-Test (M-R-T), Hogrefe, Göttingen.

Jorm, A.F., 1983. Specific reading retardation and working memory: a review. Br. J. Psychol. 74, 311–342.

Kasai, K., Nakagome, K., Itoh, K. et al., 1999. Multiple generators in the auditory automatic discrimination process in humans. Neuroreport 10, 2267–2271.

Klatt, D.H., 1980. Speech perception. A model of acoustic-phonemic analysis and lexical access. J, Phon. 8, 279–312.

Kraus, N., McGee, T., Carell, T., Zecker, S., Nicol, T., Koch, D., 1996. Auditory neuro physiologic response and discrimination deficits in children with learning problems. Science 273, 971–973.

Kuhl, P.K., Williams, K.A., Lacerda, F., 1992. Linguistic experience alters phonetic perception in infants by 6 months of age. Science 255, 606–608.

Liberman, P., Meskill, R.H., Chatillon, M., Schupack, H., 1985. Phonetic speech perception deficits in dyslexia. J. Speech Hear. Res. 28, 480–487.

Manis, F.R., McBride-Chang, C, Seidenberg, M.S. et al., 1997. Are speech perception deficits associated with developmental dyslexia? J. Exp. Child Psychol. 66, 211–235.

McBride-Chang, C., 1995. Phonological processing, speech perception, and reading disability: an integrative review. Educ. Psychol. 30, 109–121.

Mody, M., Studdert-Kennedy, M., Brady, S., 1997. Speech perception deficits in poor readers: auditory processing or phonological coding? J Exp. Child Psychol. 64, 199–231.

Näätänen, R., 1992. Attention and Brain Function. Lawrence Erlbaurn, Hillsdale.

Näätänen, R., Lehtokoski, A., Lennes, M. et al., 1997. Language-specific phoneme representations revealed by electric and magnetic brain responses. Nature 385, 432–434.

Nyman, G., Alho, K., Laurinen, P. et al., 1990. Mismatch negativity (MMN) for sequences of auditory and visual stimuli: evidence for a mechanism specific to the auditory modality. Electroencephalogr. Clin. Neurophysiol 77, 436–444.

Sams, M., Aulanko, R., Aaltonen, O., Näätänen, R., 1990. Event-related potentials to infrequent changes in synthesized phonetic stimuli. J. Cogn. Neuro. 2, 344–357.

Sandridge, S.A., Boothroyd, A., 1996. Using naturally produced speech to elicit the mismatch negativity. J Am. Acad. Audiol. 7, 105–112.

Schulte-Körne, G. (2000) Lese-Rechtschreibschwäche und Sprachwahrnehmung, Waxmann, Münster (in print).

Schulte-Körne, G., Deimel, W., Bartling, J., Remschmidt, H., 1998. Auditory processing and dyslexia: evidence for a specific speech processing deficit. Neuroreport 9, 337–340.

Schulte-Körne, G., Deimel, W., Bartling, L., Remschmidt, H., 1999a. Pre-attentive processing of auditory patterns in dyslexic human subjects. Neurosci Lett. 276, 41–44.

Schulte-Körne, G., Deimel, W., Bartling, J., Remschmidt, H., 1999b. The role of phonological awareness, speech perception, and auditory temporal processing for dyslexia. Eur. Child Adolesc. Psychiat Suppl. 3, 28–34.

Schulte-Körne, G., Deimel, W., Müller, K., Gutenbrunner, C., Remschmidt, H., 1996. Familial aggregation of spelling disability. J. Child Psychol. Psychiat. 37, 817–822.

Schulte-Körne, G., Remschmidt, H., Warnke, A., 1991. Selective visual attention and continuous attention in dyslexic children. An experimental study. Z. Kinder Jugendpsychiatr 19, 99–106.

Sharma, A., Kraus, N., McGee, T., Carrell, T., Nicol, T., 1993. Acoustic versus phonetic representation of speech as reflected by the mismatch negativity event-related potential. Electroencephalogr. Clin. Neurophysiol. 88, 64–71.

Shaywitz, S.E., Shaywitz, B.A., Fletcher, J.M., Escobar, M.D., 1990. Prevalence of reading disability in boys and girls. J. Am. Med. Assoc. 264, 998–1002.

Steffens, M.L., Eilers, R.E., Gross-Glenn, K., Jallad, B., 1992. Speech perception in adult subjects with familial dyslexia. J. Speech Hear. Res. 35, 192–200.

Tallal, P., 1980. Auditory temporal perception, phonics, and reading disabilities in children. Brain Lang. 9, 182–198.

Tremblay, K., Kraus, N., Carrell, T.D., McGee, T., 1997. Central auditory system plasticity: generalization to novel stimuli following listening training. J. Acoust. Soc. Am. 102, 3762–3773.

Uwer, R., Albrecht, W., v. Suchodoletz, W. (2000) Die Mismatch Negativity auf Sprach- und Tonstimuli bei Kindern mit Lese-Rechtschreibschwäche. Paper presented at the Meeting of the German Association of Child and Adolescent Psychiatry, Jena.

Watson, B.U., Miller, T.K., 1993. Auditory perception, phonological processing, and reading ability/disability. J Speech Hear. Res. 36, 850–863.

Weiß, R.H., 1987. Grundintelligenztest Skala 2 CFT 20, 3rd edition Westermann, Braunschweig.

Werker, J.F., Tees, R.C., 1987. Speech perception in severely disabled and average reading children. Canadian J. Psychol. 41, 48–61.

Winkler, I., Lehtokoski, A., Alku, P. et al., 1999. Pre-attentive detection of vowel contrasts utilises both phonemic and auditory memory representations. Brain Res. 7, 357–369.