Neural evidence of allophonic perception in children at risk for dyslexia

M.W. Noordenbos [a,*], E. Segers [a], W. Serniclaes [b,c], H. Mitterer [d], L. Verhoeven [a]

[a] Behavioural Science Institute, Radboud University Nijmegen, The Netherlands
[b] CNRS, Laboratoire de Psychologie de la Perception, Université Paris Descartes, France
[c] UNESCOG, Brussels' Free University, Belgium
[d] Max Planck Institute for Psycholinguistics, The Netherlands

ABSTRACT

Learning to read is a complex process that develops normally in the majority of children and requires the mapping of graphemes to their corresponding phonemes. Problems with the mapping process nevertheless occur in about 5% of the population and are typically attributed to poor phonological representations, which are – in turn – attributed to underlying speech processing difficulties. We examined auditory discrimination of speech sounds in 6-year-old beginning readers with a familial risk of dyslexia ($n=31$) and no such risk ($n=30$) using the mismatch negativity (MMN). MMNs were recorded for stimuli belonging to either the same phoneme category (acoustic variants of /bə/) or different phoneme categories (/bə/ vs. /də/). Stimuli from different phoneme categories elicited MMNs in both the control and at-risk children, but the MMN amplitude was clearly lower in the at-risk children. In contrast, the stimuli from the same phoneme category elicited an MMN in only the children at risk for dyslexia. These results show children at risk for dyslexia to be sensitive to acoustic properties that are irrelevant in their language. Our findings thus suggest a possible cause of dyslexia in that they show 6-year-old beginning readers with at least one parent diagnosed with dyslexia to have a neural sensitivity to speech contrasts that are irrelevant in the ambient language. This sensitivity clearly hampers the development of stable phonological representations and thus leads to significant reading impairment later in life.

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1. Introduction

Developmental dyslexia is a specific and persistent failure to acquire efficient reading and spelling skills despite average or above average intelligence, adequate and effective classroom instruction, and good socio-cultural opportunities (Démonet, Taylor, & Chaix, 2004). The disorder typically persists into adulthood and is characterized by slow and error-prone reading, poor non-word reading, and weak spelling. Although there is still no consensus on the causes of developmental dyslexia, it is agreed that problems with phonological awareness (i.e., the ability to identify and manipulate speech elements such as phonemes and syllables) constitute the core deficit (Ramus, 2003; Snowling & Hulme, 2010; for a review see Vellutino, Fletcher, Snowling, & Scanlon, 2004). Impaired phonological processing prohibits the establishment of stable phonological representations, and thus affects the mapping of graphemes onto their corresponding phonemes (Anthony & Francis, 2005; Elbro, Borstrom, &

Petersen, 1998; Goswami, 2002). And, indeed, deficits in the perception of contrastive speech sounds have been found to positively relate to phonological awareness, reading ability, and speech-in-noise perception (Hornickel, Skoe, Nicol, Zecker, & Kraus, 2009; McBride-Chang, 1995).

Speech perception involves the mapping of a spectrally complex and rapidly changing acoustic signal onto discrete phonological units. A basic property of speech perception is that listeners perceive speech sounds categorically. That is, most listeners attend to acoustical cues that signal phonologically relevant speech contrasts and have learned to ignore cues that signal irrelevant distinctions (Liberman, Harris, Hoffman, & Griffith, 1957). Deficits in the detection of acoustic speech cues may thus play a role in difficulties with the development of stable phonological representations (McBride-Chang, 1996; Studdert-Kennedy, 2002). Numerous behavioral studies have shown that individuals at-risk or with dyslexia present poorer categorization for consonants in both identification tasks (e.g., Boets et al., 2011; Breier et al., 2001; Chiappe, Chiappe, & Siegel, 2001; Gerrits & De Bree, 2009; Joanisse, Manis, Keating, & Seidenberg, 2000; Maassen, Groenen, Crul, Assman-Hulsmans, & Gabreëls, 2001; Manis et al., 1997) and discrimination tasks (e.g., Bogliotti, Serniclaes, Messaoud-Galusi, & Sprenger-Charolles, 2008; Breier, Fletcher, Denton, & Gray, 2004; Maassen et al., 2001;

* Correspondence to: Behavioural Science Institute, Radboud University Nijmegen, P.O. Box 9104, 6500 HE Nijmegen, The Netherlands.
Tel.: +31 24 3612070; fax: +31 24 3616211.
  E-mail address: m.noordenbos@bsi.ru.nl (M.W. Noordenbos).

Mody, Studdert-Kennedy, & Brady, 1997; Serniclaes, Van Heghe, Mousty, Carre, & Sprenger-Charolles, 2004). For example, Maassen et al. (2001) has shown children with dyslexia to have less sharply defined phoneme boundaries along both voicing and place-of-articulation continua than control children.

The reduced between-category discrimination in individuals with dyslexia suggests that their phonological representations are not properly developed. It has been further suggested that their phonological representations are over-specified, as reflected by better behavioral discrimination of well-specified allophonic contrasts within the same phoneme category than controls (Bogliotti et al., 2008; Serniclaes et al., 2004). Recent neuroimaging data suggest that when individuals with dyslexia do not show heightened sensitivity to allophonic contrasts behaviorally, it might still be present in the form of neural activation (Dufor, Serniclaes, Sprenger-Charolles, & Démonet, 2009). Note, however, that not all studies have found better within-category discrimination in individuals with dyslexia (e.g., Breier et al., 2004; Van Beinum, Schwippert, Been, Van Leeuwen, & Kuijpers, 2005). This could be due to the different features of the speech continua being used, as the speech perception deficits in dyslexia are quite subtle. For example, Breier et al. (2004) investigated within-category discrimination in general and not specifically allophonic perception in English speaking children; they used a continuum with only positive voice-onset-times (VOT) with the phonemic boundary placed around $+30$ ms VOT, but allophonic boundaries for VOT continua are located around -30 and $+30$ ms VOT as evidenced by studies in infants (Aslin, Pisoni, Hennessy, & Perey, 1981; Hoonhorst et al., 2009). These ± 30 ms VOT boundaries are phonemic in a three voicing category language, such as Thai (Lisker & Abramson, 1970). Furthermore, the continuum used by Breier et al. contained no well-specified allophonic boundaries, contrary to the full VOT continuum used in the study of Bogliotti et al. (2008). This means that discrimination of within-category differences in studies without well-specified allophonic contrasts (e.g., Breier et al., 2004; Van Beinum et al., 2005) might arise from the discrimination of simply any kind of acoustic contrast rather than those that straddle an allophonic boundary per se.

Studies showing better discrimination of stimuli crossing allophonic boundaries suggest that individuals with dyslexia perceive speech using allophonic rather than phonemic units and are thus sensitive to phonetic variation that is actually irrelevant for the ambient language. The perception of speech using an allophonic mode is the same ability that all newborns have—an ability that allows them to acquire the language that they hear (Kuhl et al., 2006; Werker & Tees, 2002). This ability is reorganized during the first year of life in accordance with the relevance of the allophonic contrasts within the language being acquired (Hoonhorst et al., 2009; Kuhl, 2004). Stable grapheme–phoneme correspondences are then easily established by most children when they start to read but not by children with dyslexia.

A heightened allophonic sensitivity in individuals diagnosed with dyslexia does not – in and of itself – demonstrate a possible causal relationship between speech perception difficulties and dyslexia (Bogliotti et al., 2008; Dufor et al., 2009; Serniclaes et al., 2004). In the present study, we therefore examined auditory discrimination of speech sounds belonging to either the same phoneme category (acoustic variants of /bə/) or different phoneme categories (/bə/ vs. /də/) in 6-year-old beginning readers with a familial risk of dyslexia by means of event-related potentials (ERP).

Event-related potentials have the advantage of being considerably less affected by attentional, motivational, and task-related artifacts than behavioral tasks. The Mismatch Negativity (MMN) is a negative deflection of the event-related potential and is elicited by any noticeable change in the preceding auditory stimulus sequence—irrespective of attention or the behavioral task (for reviews see Näätänen, Paavilainen, Rinne, & Alho, 2007; Näätänen & Winkler, 1999). The MMN usually reaches its maximum amplitude on the fronto-central scalp about 100–250 ms after deviance onset, but its amplitude is enlarged and its peak latency is shortened as the degree of stimulus change increases (Pakarinen, Takegata, Rinne, Huotilainen, & Näätänen, 2007; Sams, Paavilainen, Alho, & Näätänen, 1985; Tiitinen, May, Reinikainen, & Näätänen, 1994). Several studies have also shown better pre-attentive discrimination of phonetic contrasts to be reflected by larger MMN amplitudes (Cheour et al., 1998; Dehaene-Lambertz & Baillet, 1998; Näätänen et al., 1997; Winkler et al., 1999). For example, Näätänen et al. (1997) showed the MMN amplitude to be larger in healthy adults when the *infrequent* deviant stimulus reflects a relevant contrast in the participant's native language (Finnish) as opposed to an irrelevant foreign contrast (Estonian). In still other cross-linguistic research using the MMN, Cheour et al. (1998) showed memory traces for language-specific speech sounds to develop between 6 and 12 months of age. The finding of larger MMN amplitudes to familiar speech sounds compared to unfamiliar sounds suggests the activation of language-specific memory traces and is therefore increasingly being used in research on developmental language disorders.

In dyslexia research, several studies have shown both children and adults with dyslexia to have diminished MMN amplitudes for changes of consonants (Lachmann, Berti, Kujala, & Schröger, 2005; Schulte-Körne, Deimel, Bartling, & Remschmidt, 1998, 2001; Sharma et al., 2006) and tone frequencies (Baldeweg, Richardson, Watkins, Foale, & Gruzelier, 1999; Kujala, Lovio, Lepistö, Laasonen, & Näätänen, 2006). Normal MMNs have been found for duration changes in only adults with dyslexia (Baldeweg et al., 1999; Kujala et al., 2006) but not in children with dyslexia (Corbera, Escera, & Artigas, 2006). Studies of children and infants with a familial risk for dyslexia have also shown diminished MMN responses for changes in phonemes (Maurer, Bucher, Brem, & Brandeis, 2003; Van Leeuwen et al., 2008), vowels (Lovio, Näätänen, & Kujala, 2010), and duration (Leppänen et al., 2002). However, the pre-attentive auditory processing of allophonic variants was not investigated in these studies while such allophonic processing may be an important marker for dyslexia.

In the present study, we therefore investigated the auditory discrimination of phonemic and allophonic contrasts in 6-year-old beginning readers at risk for dyslexia using the MMN. These children were tested after about six months of formal reading instruction, the first moment that reading problems can be detected despite formal reading instruction, because differences in reading performance at this time can be a possible indication of later dyslexia. We recorded MMNs to speech sounds belonging to either the same or different phoneme categories. If children at risk for dyslexia are sensitive to acoustic properties that are irrelevant for their language, this can be hypothesized to cause more phonological variants (i.e., allophones) to be used to process the ambient language than necessary, lead to grapheme–phoneme mismatches, and thereby impair later reading. Furthermore, the only neural evidence for an allophonic mode of speech perception so far comes from a PET study with adults diagnosed with dyslexia (Dufor et al., 2009). These authors demonstrated that the discrimination of within-category pairs was related to reduced activation in the left inferior premotor cortex in non-dyslexic adults while discrimination of the same pairs was related to enhanced activation in the same region in dyslexic adults. It has yet to be demonstrated, however, that children at risk for dyslexia are similarly sensitive to such allophonic variants

of phonemes. Neural evidence of an allophonic mode of speech perception in children at risk for dyslexia is thus needed and, when found, suggests a possible explanation of later reading problems.

2. Methods

2.1. Participants

Sixty-one first-grade children that were followed up in the context of a longitudinal study that started in kindergarten participated in this study. At the time of inclusion, all of the children were in the year before formal reading instruction is initiated and children at risk for dyslexia were selected based on the presence of at least one parent diagnosed with dyslexia. At the time of testing, these children received about six months of formal reading instruction and 31 (15 boys and 16 girls) were at risk for dyslexia. As an extra control for the reading problems of the parents of the at-risk children, the reading skills of these parents were tested using a standardized word reading task (Brus & Voeten, 1999) and a standardized non-word reading task (Van den Bos, Lutje Spelberg, Scheepstra, & De Vries, 1994). All the parents of the at-risk children scored below the 10th percentile on both reading tasks. Thirty children (15 boys and 15 girls) with parents who reported no familial history of reading problems served as a control group. All children were native Dutch speakers. Written consent was obtained from the parents of all the children after the nature of the study was explained. Nonverbal intelligence was measured in kindergarten using the Raven Coloured Progressive Matrices (Raven, 1965) and did not differ for the control ($M=22.93$, $SD=4.74$) versus at-risk children ($M=21.94$, $SD=4.59$; $t(59)=.84$, $p=.41$, $d=.21$). Skills relevant for the development of reading were assessed using the following behavioral tests.

2.1.1. Verbal short term memory

The *Woorden en zinnen nazeggen* [Repeating Words and Sentences] subtest from the standardized *ESM-toets* ([Test for children with Specific Language Impairment], Verhoeven, 2004) was used to assess verbal short term memory (vSTM). The task consists of two parts. In the first part, the child is instructed to repeat an increasing number of CVC words that can range from two words at the beginning to a maximum of seven words at the end. Each correctly repeated group of words scores as one point. The words are presented by the instructor at normal articulation speed. In the second part, the child is instructed to repeat sentences of an increasing length, ranging from seven words at the beginning to 17 words at the end. Once again, the sentences are presented by the instructor. Each correctly repeated sentence counts as two points. If only one error is made during the repetition of the sentence, one point is assigned. Both tasks are terminated when the child makes four successive errors. The maximum score for this subtest was 36 (12 for words and 24 for sentences). The Cronbach's alpha reliability coefficient for the subtest, as listed in the manual, is .88 (Verhoeven, 2004).

2.1.2. Phonological awareness

Phonological awareness (PA) was assessed using the *Screeninginstrument Beginnende Geletterdheid* ([Screening Instrument for Emerging Literacy], Vloedgraven, Keuning, & Verhoeven, 2009; Vloedgraven & Verhoeven, 2007). Two phonological tasks were used (phoneme segmentation and phoneme deletion). Each phonological task starts with the presentation of three response alternatives both visually and auditorily, followed by the auditory presentation of the target word. For *phoneme segmentation*, the individual phonemes in the target word are pronounced. The child must then select the picture that corresponds to the target word. For *phoneme deletion*, the target word is pronounced along with a phoneme to be deleted. The child must then select the picture of the word that remains after deletion of the pronounced phoneme. Both of the phonological tasks consist of high-frequency monosyllabic words selected from the Dutch word frequency list (Schaerlaekens & Kohnstamm, 1999). Each task is composed of two practice items and 15 test items. Feedback is only given on the practice trials. The number of correct items for both tasks were summed to produce a raw PA score, which fulfilled the assumption of normality.

2.1.3. Rapid automatic naming

Naming speed was assessed using a Rapid Automatic Naming task for letters (RAN; Van den Bos & Lutje Spelberg, 2007). The card with letters consists of 50 randomly listed lowercase letters in five columns ('d', 'o', 'a', 's' and 'p'). The child must name the letters as fast and accurately as possible. The time to complete the 50 items is measured and converted into standard scores.

2.1.4. Standardized word and non-word reading tasks

The word reading level of the children was assessed using a standardized Dutch word reading test, the *Drie-Minuten-Toets* (DMT, [Three-Minutes-Test], Verhoeven, 1995). The DMT consists of three cards: two containing 150 words each and one

containing 120 words. The words presented on the cards differ in complexity. The children are instructed to read as many words out loud as possible in 1 min for each of the cards. The test score is the total number of correctly produced words. For the present study, only the first card (DMT1: CVs, VCs, or CVCs) and the second card (DMT2: monosyllabic words containing consonant clusters) were used. The third card contains words with multiple syllables and was thus considered too difficult for the children who were just starting to receive reading instruction. The Cronbach's alpha reliability coefficients for cards 1 and 2 in first grade, as listed in the manual, are .88 and .96, respectively. The scores for the two cards were summed to produce a word-reading score.

Non-word reading was assessed using a standardized Dutch non-word reading test (Klepel; Van den Bos et al., 1994). The test consists of 116 non-words of increasing difficulty. The children are instructed to read the non-words out loud as quickly as possible without making errors. The score on this task is the number of non-words read correctly in 2 min. The Cronbach's alpha reliability coefficient of the non-word task, as listed in the manual, is .93.

The characteristics of the participants and their test scores are displayed in Table 1. As multiple tests were performed, the obtained p-values were corrected using the Holm–Bonferroni step-down approach (Holm, 1979). While the groups were comparable with regard to age and nonverbal intelligence, the at-risk children performed significantly lower on the reading tasks and all of the skills relevant for the development of reading.

2.2. Stimuli and procedure

Stimuli were the sinewave analogs of speech sounds that consisted of the consonants /b/ and /d/ followed by the neutral vowel /ə/. The stimuli were generated using parallel formant synthesis (Carré, 2004). The difference in the place-of-articulation was created by modifying the onset of the initial frequencies for F2 and F3. The end frequencies for the F2 and F3 transitions were fixed at 1500 and 2500 Hz, respectively. The initial frequency of the first formant (F1) was 300 Hz; the end frequency was 500 Hz. The voice onset time was −80 ms; the duration of all frequency transitions was 40 ms; and the duration of the stable vocalic segment was 80 ms. Each stimulus thus had a total duration of 200 ms.

Two stimulus conditions – a between-category condition and a within-category condition – were administered with the order of presentation counter-balanced across participants. The standard stimulus in the two conditions was the syllable /bə/ with rising transitions of the F2 and F3 (rising F2: from 1094 to 1500 Hz; rising F3: from 2024 to 2500 Hz) and a probability of 0.88. In the between-category condition, two stimuli from *different* phoneme categories were presented. The deviant stimulus was thus the syllable /də/ with falling transitions of F2 and F3 (falling F2: from 1853 to 1500 Hz; falling F3: from 3429 to 2500 Hz). In the within-category condition, two acoustically different stimuli that nevertheless belong to the *same* phoneme category were presented. The deviant stimulus was thus an acoustically different exemplar of the syllable /bə/ with a rising F2 transition (from 1033 to 1500 Hz) and a falling F3 transition (from 2692 to 2500 Hz). The contrast between the two /bə/ syllables is allophonic because it crosses a natural psychoacoustic boundary (i.e., a change in the rising/falling direction of the F3 transition), which is nevertheless relevant for the operative phoneme distinctions in other languages (Serniclaes & Geng, 2009). The probability of the deviant stimulus in each condition was 0.12. Each condition contained two blocks of 400 stimuli (48 deviants and 352 standards) with a stimulus-onset asynchrony of 600 ms. Each block had a duration of approximately 4 min and was separated by a short break. The participants had a longer break in between conditions. The stimuli were presented in a pseudo-random order with the restriction that the first 16 stimuli in each block were always a standard and at least three standard stimuli occurred between two deviant stimuli. For each participant, a unique stimulus presentation list was created.

In a mini-van equipped with a custom-built EEG lab and parked near the children's school, the children individually watched a self-selected silent movie while auditory stimuli were binaurally presented through a set of Sennheiser HD 555 headphones at a comfortable hearing level of approximately 65 dB. The

Table 1
Descriptive Statistics for the Sample.

	Control (n=30)		At-Risk (n=31)			
	M	SD	M	SD	p	d
Age (years)	6.93	.33	6.94	.30	.46	.03
vSTM	16.90	4.69	13.77	5.24	.03	.63
RAN	45.30	8.03	40.84	8.76	< .01	.53
Phonological awareness	10.27	3.40	8.77	2.54	.04	.50
Standardized word reading	67.97	32.56	37.71	14.07	< .001	1.21
Standardized non-word reading	10.47	2.19	7.90	1.76	< .001	1.29

Note. vSTM=verbal Short-Term Memory; RAN=Rapid Automatic Naming.

children were instructed to ignore the auditory stimuli while EEG recordings were made. The procedure was approved by the Central Committee on Research Involving Human Subjects, The Netherlands.

2.3. EEG recording

The EEG (amplified by BrainAmp DC amplifier, band pass 0.1–200 Hz, sampling rate 500 Hz) was recorded with Ag/AgCl-electrodes from 26 scalp sites according to the International 10–20 system using the ActiCap system (Brain Products GmbH, Gilching, Germany). In addition, electrodes were placed on the left and right mastoids while the ground was placed on AFz. The electrodes were referenced to the left mastoid. The horizontal and vertical electro-oculograms (EOG) were monitored by electrodes placed on the left and right external canthi of the eyes and above and below the left eye. For all of the electrodes, impedances were kept below 20 kΩ, which is the default setting for active electrodes. The EEG and EOG were recorded and digitized using Brain Vision Recorder software (1.03, Brain Products GmbH, Gilching, Germany).

2.4. Data analysis

The continuous EEG was corrected offline for ocular artifacts (Gratton, Coles, & Donchin, 1983) and analyzed using the Matlab-based FieldTrip toolbox (Oostenveld, Fries, Maris, & Schoffelen, 2011). The EEG was re-referenced to the mean of the two mastoid electrodes, and the ERPs were calculated by averaging the epochs of 600 ms including a 100 ms pre-stimulus interval separately for the standards and deviants in each condition. Epochs containing voltage changes exceeding ± 100 μV at any electrode were omitted for averaging. The epochs were digitally filtered using a 1–30 Hz band pass filter and baseline-corrected with respect to the 100 ms pre-stimulus interval. Only the standard immediately preceding the deviant was included in the analysis to thereby obtain a similar signal-to-noise ratio for both the standard and deviant. Data from one participant was discarded from further analysis due to technical problems during the measurement.

The mean number (s.d.) of accepted epochs for the control and at-risk children was 93 (3) and 94 (2) for the deviants and 93 (3) and 94 (2) for the standards in the between condition; 94 (2) and 94 (2) for the deviants and 94 (2) and 94 (2) for the standards in the within condition. The number of accepted epochs in each condition did not differ significantly for the control versus at-risk participants (*F*s < 1).

For each participant, the average ERPs were computed across all remaining trials per condition. The MMN was obtained by subtracting the ERPs elicited by the standard stimuli from those elicited by the deviant stimuli separately for each condition. As there is ample evidence that the amplitude of the MMN is maximal over fronto-central scalp locations and particularly at Fz, F3, and F4 (Duncan et al., 2009; Kujala, Tervaniemi, & Schröger, 2007; Näätänen et al., 2007), these recording sites were included in the analyses. The MMN was identified at electrode Fz where the MMN response was clearly detectable in both groups and also with an inverted polarity below the Sylvian fissure, at the mastoid electrodes. For each participant, the MMN peak latencies were determined within a 65 ms time window defined by the grand-mean latency of the most negative peak occurring between 150 and 300 ms post stimulus-onset at the F3, Fz, and F4 electrodes for each group and each stimulus condition. The individual mean MMN amplitudes were averaged across a 50 ms time window surrounding the individual MMN peak latency. One-tailed *t*-tests, corrected for multiple comparisons using the Holm–Bonferroni step-down approach (Holm, 1979), were conducted to determine whether the MMN mean amplitudes at F3, Fz, and F4 significantly differed from zero at the group level.

The mean MMN amplitudes and peak latencies were analyzed in separate three-way repeated measures analyses of variance (ANOVAs) with Electrode (F3, Fz, F4) as the within-subjects factor and Group (control, at-risk) and Condition (between, within) as between-subjects factors. Significant group differences were analyzed further in two-way repeated measures ANOVAs with Electrode (F3, Fz, F4) as the within-subjects factor and Group (control, at-risk) as the between-subjects factor. Greenhouse–Geisser corrections for violations of the sphericity assumption were applied when appropriate; the uncorrected degrees of freedom and *p*-values after the correction are reported.

3. Results

We first determined if the deviant stimulus in each condition elicited significant MMNs. In the between-category condition, the phonemic deviant elicited MMNs that differed significantly from zero in both the control and at-risk children (Table 2, Fig. 1). In the within-category condition, the allophonic deviant elicited only a significant MMN in the children at risk.

Three-way repeated measures ANOVAs (Group × Condition × Electrode) were next conducted and showed a significant main

Table 2
Mean MMN Amplitude (μV) and Peak Latencies (ms) at F3, Fz, and F4.

Condition	Amplitude				Latency			
	Control		At-Risk		Control		At-Risk	
	M	SD	M	SD	M	SD	M	SD
Between								
F3	−4.0**	3.4	−1.8*	3.8	232	20	259	20
Fz	−4.1**	3.1	−1.5*	4.1	236	18	260	21
F4	−3.8**	3.6	−1.6*	3.7	229	19	263	23
Within								
F3	−0.2	2.9	−2.5**	2.9	219	25	240	22
Fz	−0.5	3.5	−2.0**	3.2	216	26	249	23
F4	−0.4	3.4	−2.0**	3.1	221	21	243	24

Note. The amplitudes significantly differing from zero are marked with asterisks (one-tailed *t*-tests).

* *p* < .05.
** *p* < .001.

effect of Condition ($F(1,116)=7.05$, $p=.01$, $\eta_p^2=.06$) and a Group x Condition interaction ($F(1,116)=13.14$, $p<.001$, $\eta_p^2=.10$) for MMN amplitude and significant main effects of both Condition ($F(1,116)=20.50$, $p<.001$, $\eta_p^2=.15$) and Group ($F(1,116)=63.46$, $p<.001$, $\eta_p^2=.35$) and a Group × Condition × Electrode interaction ($F(2,232)=3.71$, $p=.03$, $\eta_p^2=.03$) for peak latency.

The control versus at-risk children were next compared in two-way repeated measures ANOVAs (Group × Electrode) for MMN amplitude and peak latency per condition. In the between-category condition, a clearly lower MMN amplitude for the phonemic deviant was found in the at-risk children (mean − 1.63 μV; Table 2) when compared to the control children (mean − 3.97 μV; $F(1,58)=7.03$, $p=.01$, $\eta_p^2=.11$). Furthermore, the MMN peaked significantly earlier in the control children (mean latency: 232 ms; Table 2) than in the at-risk children (mean latency: 260 ms; $F(1,58)=38.90$, $p<.001$, $\eta_p^2=.40$); the at-risk children thus needed significantly more time to process the speech stimuli. No differences in the MMN amplitudes and peak latencies at the frontal electrodes F3, Fz, and F4 were further observed for the groups in the between-category condition (both *F*s < 1; Fig. 2a).

In the within-category condition, the difference in MMN amplitudes and peak latencies for the allophonic deviant was significant for the at-risk versus control children ($F(1,58)=6.15$, $p=.02$, $\eta_p^2=.10$; $F(1,58)=25.88$, $p<.0001$, $\eta_p^2=.31$, respectively). However, the MMN amplitude in the within-category condition only differed significantly from zero for those children at-risk (see Table 2). No differences in the MMN amplitudes ($F(2,60)=1.22$, $p=.30$, $\eta_p^2=.04$; Fig. 2b) or peak latencies ($F(2,60)=2.14$, $p=.13$, $\eta_p^2=.07$) at the frontal electrodes F3, Fz, and F4 were further observed for the at-risk children on the allophonic deviant in the within-category condition.

Given that only the at-risk children showed a significant MMN in the within-category condition, the phonemic and allophonic contrasts were next compared for only these children. The MMN amplitudes were comparable ($F < 1$) while the latencies differed significantly ($F(1,60)=12.68$, $p=.001$, $\eta_p^2=.18$) with the MMN peaking significantly earlier for the allophonic contrast than for the phonemic contrast (Table 2).

Correlations between MMN amplitudes and peak latencies averaged over the frontal channels for each condition and the reading and phonological measures for the whole sample are displayed in Table 3. Significant correlations were observed between peak latencies and standardized word and non-word reading tasks, but not for MMN amplitude. Within-group correlations for both MMN amplitude and peak latencies were not significant.
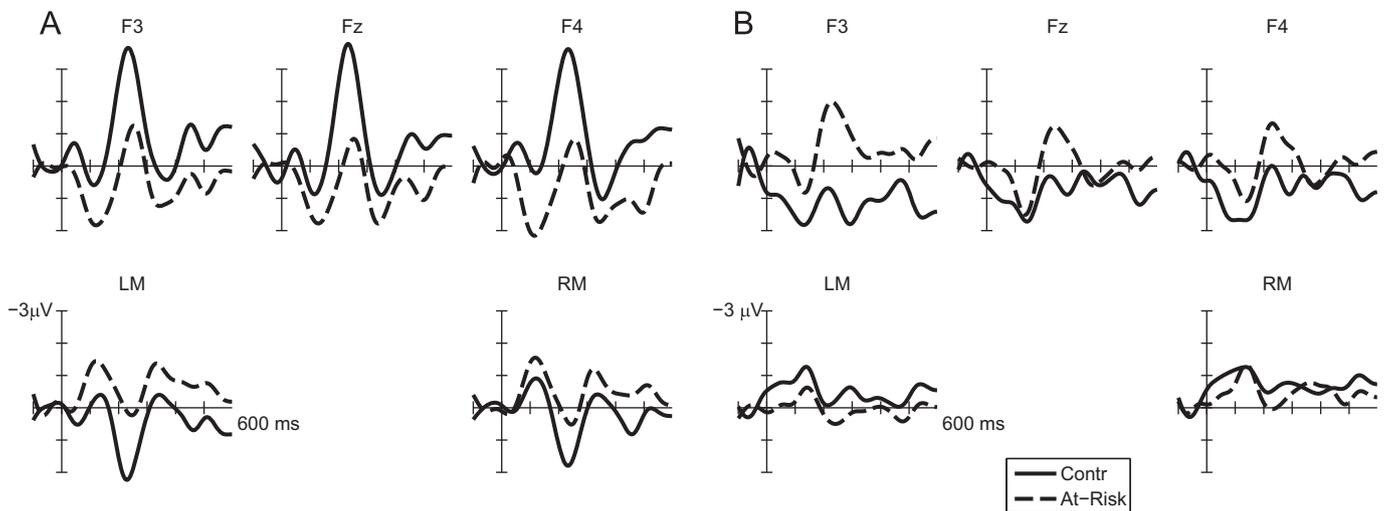
Fig. 1. Grand-average difference waves at frontal and mastoid electrodes. MMN responses at the mastoids were re-referenced to the average of all electrodes in order to illustrate the polarity reversal at the mastoid electrodes. **A**. In the between-category condition, the phonemic deviant stimulus elicited significant MMN responses in both the control and at-risk children. **B**. In the within-category condition, a significant MMN was only present in the children at risk for dyslexia.
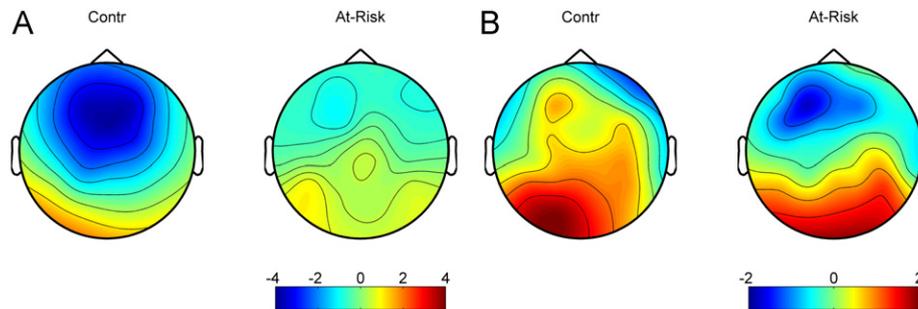

Fig. 2. MMN topography at grand-average peak latency. **A**. Higher MMN activation at fronto-central scalp locations for phonemic deviant stimulus in control children compared to children at risk for dyslexia in the between-category condition. **B**. MMN activation present only in children at risk for dyslexia in the within-category condition.

Table 3
Pearson's correlations between MMN amplitude, latency and reading and phonological measures for all subjects.

	Amplitude		Peak latency	
	Between-category	Within-category	Between-category	Within-category
vSTM	−.10	−.01	−.20	−.24[b]
RAN	−.14	−.03	−.24[b]	−.24[b]
Phonological awareness	−.12	.09	−.24[b]	−.25[a]
Standardized word reading	−.18	.00	−.35**	−.29*
Standardized non-word reading	−.17	.01	−.38**	−.31*

Note. vSTM=verbal Short-Term Memory; RAN=Rapid Automatic Naming.

[a] $p < .06$.
[b] $p < .07$.
* $p < .05$.
** $p < .01$.

4. Discussion

In the present study, we investigated the pre-attentive discrimination of speech sounds in children at risk for dyslexia and children without such risk by comparing MMN responses to well-defined phonemic and allophonic contrasts. Stimuli from different phoneme categories elicited MMNs in both the control and at-risk children while the stimuli from the allophonic contrast elicited an MMN in only the children at risk for dyslexia. These results provide neural evidence for the pre-attentive discrimination of speech sounds within the same phoneme category by children at risk for dyslexia.

Children at risk for dyslexia showed a significantly lower MMN amplitude for the phonemic deviant, which is in agreement with previous MMN results for not only children and adults with dyslexia when exposed to changes in consonants (Schulte-Körne et al., 1998, 2001; Sharma et al., 2006) but also children and infants with a familial risk for dyslexia when exposed to changes in both phonemes (Maurer et al., 2003; Van Leeuwen et al., 2008) and vowels (Lovio et al., 2010). Furthermore, the MMN for the phonemic deviant peaked earlier in the control children than in the at-risk children, which shows the at-risk children to need significantly more time to process the speech stimuli than the control children (Sprenger-Charolles, Colé, Lacert, & Serniclaes, 2000). These findings are in agreement with a large body of behavioral studies showing that individuals with dyslexia are less categorical in how they perceive phonemic contrasts (e.g., Chiappe et al., 2001; Joanisse et al., 2000; Maassen et al., 2001) and suggesting that the phonological representations needed for effective grapheme–phoneme mapping are not properly acquired by children at risk for dyslexia. It can be concluded that the phonological representations of the control children in the present study were more accurate, as reflected by their significantly

better performance on the skills relevant for the development of reading, leading to more rapid selection of the relevant cues in the auditory signal. Accurate phonological representations allow fast, automatic discrimination of speech sounds resulting in significantly higher MMN amplitude and earlier latency (Pakarinen et al., 2007; Sams et al., 1985). In contrast, inaccurate phonological representations lead to small or even absent MMNs (e.g., Dehaene-Lambertz, 1997; Näätänen et al., 1997).

According to the allophonic explanation of dyslexia, children with dyslexia maintain a sensitivity to phonemic distinctions that are irrelevant in their native language and a lack of a one-to-one relationship between allophones and graphemes is the origin of such children's failure to master the alphabetic principle. These deficiencies may cause later reading problems as processing a written language using more phonological categories (i.e., allophones) than is necessary in the ambient language can generate grapheme–phoneme mismatches and thereby impair the reading process.

Behavioral studies have shown that children with dyslexia have a greater sensitivity to the fine-grained acoustic properties of the acoustic signal than children without dyslexia (Bogliotti et al., 2008; Goswami, Fosker, Huss, Mead, & Szűcs, 2011). In addition, children with dyslexia have been shown to have better within-category discrimination for well-specified allophonic contrasts compared to controls (Bogliotti et al., 2008; Serniclaes et al., 2004). Nevertheless, some studies have not found better within-category discrimination in individuals with dyslexia (e.g., Breier et al., 2004; Van Beinum et al., 2005), which is probably due to the use of continua that did not contain well-specified allophonic boundaries. In the present study, allophonic perception in children at-risk for dyslexia was investigated using carefully designed stimuli (i.e., a continuum with well-defined allophonic boundaries). And we showed a significant MMN for the allophonic contrast to be elicited in only the children at-risk for dyslexia. This finding is thus in agreement with the notion that individuals with dyslexia use an allophonic as opposed to phonemic mode of speech perception (Serniclaes et al., 2004).

Our results and the reflection of language-specific memory traces by the MMN, in particular, are supported by the results of other research showing the MMN amplitude to be larger in healthy adults when the infrequent deviant stimulus reflects a relevant contrast in the participant's native language as opposed to an irrelevant foreign contrast (Dehaene-Lambertz, 1997; Näätänen et al., 1997). Furthermore, we found that the MMN for the allophonic contrast to peak significantly earlier than the MMN for the phonemic contrast in at-risk children when compared to control children. As previous research has shown that an increased degree of stimulus change is associated with a higher MMN amplitude and shorter latency (Pakarinen et al., 2007; Sams et al., 1985), our findings suggest that the allophonic contrast was more easily discriminated by the children at risk for dyslexia than the phonemic contrast that requires accurate representation of the native language for discrimination. Furthermore, peak latencies in both the between and within-category condition were negatively correlated with word and non-word reading performance. That is, children with higher scores on the standardized word or non-word reading tasks showed shorter MMN latencies, indicating that they needed less time to process the speech stimuli.

Several studies have shown that MMN amplitude and behavioral parameters such as reaction time and discriminability are highly correlated (for a review see Kujala et al., 2007). This shows that a difference between sounds is usually not behaviorally detected in the absence of an MMN (Winkler et al., 1999). Behavioral performance and MMN are nevertheless not identical processes. The MMN mainly reflects pre-attentive central

auditory processing whereas behavioral performance also includes more higher-order processes, such as those involved in attention and decision making. Given that the MMN is thought to reflect an automatic change detection process in the brain, it is considerably less affected by attentional, motivational, and task-related artifacts than behavioral tasks. This makes the MMN a suitable tool for the clinical evaluation of auditory discrimination in especially young children and special populations (see e.g., Davids et al., 2011).

The finding of pre-attentive discrimination of a within-category contrast by children at risk for dyslexia shows that our results are robust as only about half of the children at risk for dyslexia actually develop dyslexia (Grigorenko, 2001). Studies relating the brain responses of children to their later language performance (Espy, Molfese, Molfese, & Modglin, 2004; Guttorm et al., 2005; Leppänen et al., 2010; Molfese, 2000), moreover, suggest that problems with auditory processing indeed interfere with the development of stable phonological representations and thus with a prerequisite for becoming literate. Still other studies have reported deviant cortical discrimination of speech sounds by not only infants and children at risk for dyslexia (Maurer et al., 2003; Schulte-Körne et al., 1998) but also adults with dyslexia (Schulte-Körne et al., 2001). However, the processing of allophonic variants was not investigated in these studies. Prior to the present study, which has shown children at risk for dyslexia to pre-attentively discriminate allophonic variants within the same phoneme category, the only neural evidence for an allophonic mode of speech perception was for adults diagnosed with dyslexia (Dufor et al., 2009). Our findings point to a possible cause of dyslexia and supplement the neural evidence for deviant brain responses to speech sounds already at birth (Guttorm et al., 2005) and also later in adulthood (Dufor et al., 2009) for individuals who are either at risk for dyslexia or later diagnosed with dyslexia.

Note that the present study has some limitations. To begin with, we investigated auditory perception in beginning first-grade readers at risk and not at risk for dyslexia. To further disentangle the allophonic mode of speech perception in children at risk for dyslexia and shed greater light on the auditory development of young children, however, longitudinal studies that start even before the initiation of formal reading instruction should be undertaken, and follow the children until dyslexia has been evidenced. Furthermore, the present study provides neural evidence of allophonic perception in children at risk for dyslexia for stimuli differing in the place-of-articulation but allophonic boundaries are present on both place-of-articulation and VOT continua. Future studies should extend the present evidence of a heightened sensitivity to allophonic contrasts in at-risk children to include VOT continua with well-specified allophonic boundaries. Finally, in future research, behavioral measures should be included to extend the results of neural measures, but care must be taken when comparing these measures as the pre-attentive processing and behavioral measures do not reflect identical processes. It can nevertheless be hypothesized that children with no risk of dyslexia will not detect an allophonic contrast behaviorally (cf. Bogliotti et al., 2008; Serniclaes et al., 2004).

Our research provides direct neurophysiological evidence for an allophonic mode of speech perception in individuals at risk for dyslexia. The results show children at risk for dyslexia to be sensitive to contrasts that are irrelevant in the ambient language and thus a sensitivity that can hamper the development of stable phonological representations. Additionally, the reduced sensitivity to relevant speech contrasts by these children may be a consequence of an allophonic perception. Therefore, an allophonic mode of speech perception possibly leads to significant reading impairment later in life.

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