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Event-related potentials to tones show differences between children with multiple risk factors for dyslexia and control children before the onset of formal reading instruction

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ABSTRACT

Multiple risk factors can affect the development of specific reading problems or dyslexia. In addition to the most prevalent and studied risk factor, phonological processing, auditory discrimination problems have also been found in children and adults with reading difficulties. The present study examined 37 children between the ages of 5 and 6, 11 of which had multiple risk factors for developing reading problems. The children participated in a passive oddball EEG experiment with sinusoidal sounds with changes in sound frequency, duration, or intensity. The responses to the standard stimuli showed a negative voltage shift in children at risk for reading problems compared to control children at 107–215 ms in frontocentral areas corresponding to P1 offset and N250 onset. Source analyses showed that the difference originated from the left and right auditory cortices. Additionally, the children at risk for reading problems had a larger late discriminative negativity (LDN) response in amplitude for sound frequency change than the control children. The amplitudes at the P1–N250 time window showed correlations to letter knowledge and phonological identification whereas the amplitudes at the LDN time window correlated with verbal short-term memory and rapid naming. These results support the view that problems in basic auditory processing abilities precede the onset of reading instruction and can act as one of the risk factors for dyslexia.

1. Introduction

Specific reading difficulty or dyslexia is one of the most common learning difficulties, affecting approximately 5–10% of the population (Vellutino et al., 2004). Besides problems in learning to read and write, individuals with dyslexia often have problems in phonological processing, verbal short-term memory, and rapid naming (Lyon et al., 2003; Vellutino et al., 2004). These three deficits have been found consistently across studies and phonological deficits are thought to be directly causal to the development of reading problems (Bradley and Bryant, 1983; Wagner and Torgesen, 1987). However, other deficits have also been observed with varying consistency in, for example, more general language skills (Lyytinen et al., 2005), fine motor learning (Nicolson et al., 2001), binocular control (Stein et al., 2000) and auditory processing (Goswami, 2011; Tallal and Gaab, 2006).

The role of these other deficits remains unclear and the role of auditory deficits in the development of reading problems has been discussed extensively in the literature (e.g., Goswami, 2011; Hämäläinen et al., 2013b; Ramus, 2004; White et al., 2006). The connection between auditory and reading problems has been suggested to, be directly causal, further aggravating the already existing problems, one of many accumulating risk factors or co-occurring with no direct association (e.g., Bishop et al., 1999; Pennington, 2006; Ramus, 2004; Tallal, 1980). Most previous studies have examined the auditory processing abilities of either school-age children or adults with dyslexia (e.g., Corbera et al., 2006; Khan et al., 2011; Kujala et al., 2000; Lachmann et al., 2005; Hämäläinen et al., 2007, 2008) and thus the developmental relationship between auditory and reading or pre-reading skills is not clear. Only a handful of studies have examined the auditory processing abilities of children at risk for dyslexia before formal reading instruction has begun (e.g., Boets et al., 2006; Hämäläinen et al., 2013a; Leppänen et al., 2010; van Leeuwen et al., 2006; Lovio et al., 2010; Maurer et al., 2003).

Examining the auditory processing abilities of children before reading instruction has started is important in disentangling the effect of reading acquisition, pre-reading skills and general brain maturation. During childhood, relatively fast maturation occurs in the brain and in the auditory cortices due to, for example, changes in synaptic connections, number of neurons, and myelination (Moore and Guan, 2001; Petanjek et al., 2011). One method to examine the time course of brain maturation is electroencephalography (EEG), which measures

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the fluctuations of electrical fields generated by the brain on the scalp surface. The majority of developmental EEG studies have been carried out with school-age children (e.g. Corbera et al., 2006; Hämäläinen et al., 2007; Lachmann et al., 2005). However, maturational changes in EEG occur rapidly already before school age (Albrecht et al., 2000; Choudhury and Benasich, 2011; Ponton et al., 2000). The present study examined electrophysiological indices of auditory discrimination in children at preschool with and without risk factors for dyslexia.

Most sounds that generate activity in the cochlea nerve produce a series of responses in EEG, often called obligatory event-related potentials (ERPs), even without active attention to the sounds. In adults, this series of responses consists of P1, N1, P2, and N2 responses. They occur at approximately 50 ms, 100 ms, 150 ms, and 200 ms after the stimulus onset. The obligatory responses in children, on the other hand, show a drastically different morphology. With inter-stimulus intervals (ISIs) shorter than one second, the waveform of children younger than 10 years is characterized by P1 and N250 responses, emerging approximately 100 ms and 250 ms after stimulus onset (Sharma et al., 1997; Sussman et al., 2008). After 9 years of age, the N1–P2 complex starts to emerge and gradually grows in amplitude to adult values while the P1–N250 complex reduces in amplitude (Albrecht et al., 2000; Ceponiene et al., 2002; Ponton et al., 2000, 2002; Tonnquist-Uhlen et al., 1996; Wunderlich et al., 2006). The P1–N250 complex shows a fronto-central voltage distribution and has sources close to the auditory cortex (Parviainen et al., 2011; Ponton et al., 2002). The exact functional significance of the obligatory responses is not clear, but they most likely reflect sound detection and complexity, formation of memory representations, categorization, and feature extraction processes (Karhu et al., 1997; Näätänen and Picton, 1987; Ceponiene et al., 2001).

Several studies have examined the obligatory responses in children with dyslexia. Some studies carried out at school age and using fast stimulation rates (below 1 s) have not found P1 or N250 amplitude or latency differences between individuals with dyslexia and controls (Lachmann et al., 2005; Sharma et al., 2006). However, group differences have been found in experiments where long ISIs (above 1 s) have been used and thus the N1–P2 responses are elicited. In an experiment where tones were presented with 2–2.5 second intervals in an active listening task, the P2 response was found to be enhanced in poor reading children compared to controls (Bernal et al., 2000). On the other hand, in a passive listening experiment, the P2 amplitude was reduced in children with dyslexia compared to controls when 1–5 second sound intervals were used (Hämäläinen et al., 2007). In the same experiment, the N1 response was found to be larger in amplitude in individuals with dyslexia compared to controls in a condition where sound pairs were presented and the second sound in the pair had a long rise time (Hämäläinen et al., 2007). However, in one study the same children as in Hämäläinen et al. (2007) were examined using source localization methods and smaller left hemispheric responses were found in children with dyslexia than children in the control group in an experiment where the ISI was short (610 ms) (Khan et al., 2011). The source activity was smaller at latencies centered around 128 ms and 180 ms (corresponding to the P1 and emerging P2), but only in a condition where tone pairs with short gaps (10 ms) were used (Khan et al., 2011). Thus the previous literature suggests differences between typically reading school-age children and children with dyslexia to emerge in obligatory ERP responses at 100–200 ms, particularly, but not exclusively, when long ISIs are used.

Only two studies have been carried out at kindergarten age and examined the obligatory ERP responses in children at risk for dyslexia. The N250 response has been found to be larger in 6.5-year-old children who developed dyslexia at school age compared to controls and children who had a familial risk for dyslexia but developed reading skills at the normal level for both speech sounds and complex non-speech sounds (Hämäläinen et al., 2013a). Although it is not clear what processes the N250 response reflects, a larger N250 could be a marker of a less mature auditory cortex or a marker of more effortful memory trace formation (e.g., Karhu et al., 1997; Ponton et al., 2000). Additionally, the P1 response has been found to be smaller in 6.5-year-old children at risk for dyslexia compared to control children for speech sounds (Lovio et al., 2010). This finding was interpreted as a difficulty in establishing sound representations in the at-risk children (Lovio et al., 2010).

The above reviewed studies examined the obligatory ERPs. However, auditory discrimination ability can be studied more specifically using a response called the mismatch negativity (MMN; Näätänen et al., 1978, 2010). The MMN is generated when repetitive background stimulation is given and a sound is played that differs in some acoustic or more abstract feature from the background. This deviant sound elicits an ERP with negative voltage at frontocentral electrodes and positive voltage in areas below the Sylvian fissure, indicative of sources near the auditory cortex. There are also reports of a frontal source for the MMN response (e.g., Opitz et al., 2002; Rinne et al., 2000; Deouell, 2007). The MMN peaks between 150 and 250 ms after the onset of the deviant sound feature. Many types of deviant features elicit the MMN response, including changes in sound frequency, duration, and intensity, but also in sound onsets, sound omissions, and violations in abstract sound feature rules (Näätänen et al., 2010). Several studies have also shown that the amplitude of the MMN response correlates with behavioral deviant sound detection accuracy (e.g., Novitski et al., 2004; Pakarinen et al., 2007) and that the MMN amplitude increases with improvement in behavioral discrimination ability after training (e.g., Atienza et al., 2002; Kraus et al., 1995; Näätänen et al., 1993).

In children between the ages of 6 and 13 years, MMN to frequency and duration changes has been reported to have similar or slightly longer peak latency (130–250 ms) compared to adults and to have a similar frontocentral negative topography (e.g., Ahmmed et al., 2008; Ceponiene et al., 1998; Corbera et al., 2006; Gomes et al., 1999; Huttunen et al., 2007; Hämäläinen et al., 2008; Korpilahti and Lang, 1994; Lachmann et al., 2005; Shafer et al., 2000; Sharma et al., 2006; Uwer and von Suchodoletz, 2000). At this age, MMN often seems to be accompanied by a second, longer latency frontocentral negative deflection starting after around 350 ms (Ceponiene et al., 2004; Hämäläinen et al., 2008; Schulte-Körne et al., 1998). In different studies, this late discriminative negativity (LDN) has also been termed late negativity or late mismatch negativity. However, its function is still unknown, with suggestions of involvement in further cognitive processing of the sound change, long-term memory storage, or activation of attention mechanisms (Ceponiene et al., 2004; Cheour et al., 2001; Shestakova et al., 2003; Zachau et al., 2005).

In preschool or kindergarten children there are fewer and more varied reports on the latency and topography of MMN compared to school-age children. Maurer et al. (2003) reported a positive frontocentral component at the latency of 150 ms in response to pitch and duration changes in both tones and speech stimuli and interpreted it as an MMN or mismatch response. They also observed a later negativity at circa 450 ms. However, Shafer et al. (2000) found a negative amplitude response to frequency change at a latency of 226–238 ms in children aged between 4 and 6 years. Holopainen et al. (1997) examined MMN to frequency change in children aged 5.2 years and found frontocentral negative voltages peaking at 283 ms. Similarly, Korpilahti et al. (2001) reported an adult-like frontocentral negative component with a latency between 150 and 350 ms to a duration change in tones in children aged 5.7 years. Lovio et al. (2010) examined speech stimuli in children aged 6.4 years and also found a negative response with 150 ms latency elicited by changes in vowel frequency, duration, and intensity, as well as in vowel identity and consonant identity. Thus there are a few studies characterizing the MMN waveform at preschool age with most of them showing an adult-like frontocentral negativity with slightly longer peak latency than in adults.

Several studies have examined the MMN response in school-age children with dyslexia. Most of these studies have found a reduced MMN amplitude in response to small frequency changes and duration

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