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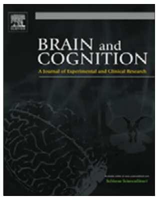

Electrophysiological correlates of impaired reading in dyslexic pre-adolescent children

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ABSTRACT

In this study, event related potentials (ERPs) were used to investigate the extent to which dyslexics (aged 9–13 years) differ from normally reading controls in early ERPs, which reflect prelexical orthographic processing, and in late ERPs, which reflect implicit phonological processing. The participants performed an implicit reading task, which was manipulated in terms of letter-specific processing, orthographic familiarity, and phonological structure. Comparing consonant- and symbol sequences, the results showed significant differences in the P1 and N1 waveforms in the control but not in the dyslexic group. The reduced P1 and N1 effects in pre-adolescent children with dyslexia suggest a lack of visual specialization for letter-processing. The P1 and N1 components were not sensitive to the familiar vs. less familiar orthographic sequence contrast. The amplitude of the later N320 component was larger for phonologically legal (pseudowords) compared to illegal (consonant sequences) items in both controls and dyslexics. However, the topographic differences showed that the controls were more left-lateralized than the dyslexics. We suggest that the development of the mechanisms that support literacy skills in dyslexics is both delayed and follows a non-normal developmental path. This contributes to the hemispheric differences observed and might reflect a compensatory mechanism in dyslexics.

1. Introduction

Reading is a complex skill that entails a multi-stage process, including visual feature processing, orthographic and phonological analysis, semantic retrieval, and articulatory processes. Due to advances in brain imaging technologies in recent years, it is now possible to investigate the cortical dynamics of the component processes involved in reading and to provide a neurobiological signature for reading failure, as in developmental dyslexia (Dehaene, 2010). A number of brain imaging studies have provided converging evidence that three important neural systems support reading: one anterior system around the inferior frontal gyrus involved in word analysis (decoding), and two posterior systems, one in the parietotemporal region also involved in word analysis, and the other more inferior in the occipitotemporal region which is responsible for fluent and automatic reading (for an overview, see Price & Mechelli, 2005; Schlaggar & McCandliss, 2007; Shaywitz & Shaywitz, 2008; Shaywitz et al., 2002). A recent review and meta-analysis of neuroimaging studies of dyslexia found evidence for an atypical neural organization of the reading process in dyslexics relative to non-impaired readers. This is mostly expressed as an underactivation of the left temporoparietal language regions (posterior aspect of the superior temporal gyrus/sulcus, supramarginal gyrus) and the left occipitotemporal cortex, while the inferior frontal system, related to covert articulatory processes, shows an overactivation (as premotor/motor cortex; Maisog, Einbinder, Flowers, Turkeltaub, & Eden, 2008; Richlan, Kronbichler, & Wimmer, 2009).

At present, the dominant explanatory framework for dyslexia is that this disorder is caused by a language-specific deficit within the phonological processing system. The *phonological deficit explanation* of dyslexia contends that dyslexic readers are unable to process the phonological structure underlying word reading, which includes difficulty in manipulating the basic components of language (i.e., graphemes and phonemes). Specifically, poorly specified phonological word representations give rise to a phonemic awareness deficit which hinders the extraction of grapheme-phoneme associations on which sublexical reading is dependent (Ramus et al., 2003; Snowling, 2000; Vellutino & Scanlon, 1987).

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A recent reformulation proposes that the deficit may have less to do with the quality of phonological representations and more with access/accessibility to these representations (Ramus & Szenkovits, 2008).

In support of the phonological deficit hypothesis, dyslexics perform below average on a variety of experimental tasks that require phonological skills. These include verbal short-term memory (e.g., digit span), phonological awareness (e.g., phoneme deletion and rhyme judgments) or phonological decoding (e.g., pseudoword reading) tasks (Ramus et al., 2003; Shaywitz, 2003; Tijms, 2004; Wagner, Torgesen, & Rashotte, 1994). Moreover, electrophysiological and brain imaging studies have described different brain activation patterns in dyslexic and non-dyslexic readers during performance of such tasks (Georgiewa et al., 1999, 2002; Rumsey et al., 1997; Shaywitz & Shaywitz, 2005; Shaywitz et al., 1998; Simos, Breier, Fletcher, Bergman, & Papanicolaou, 2000). For example, the event related potentials (ERPs) literature on phonological effects tends to report a delayed onset of responses that reflect phonological processing in dyslexic compared to normal readers in the N400 range (e.g., Ackerman, Dykman, & Oglesby, 1994; Rüsseler, Becker, Johannes, & Münte, 2007). A relatively early negative potential peaking at about 320 ms (N320) has been also implicated in phonologic transcription (e.g., Bentin, Mouchetant-Rostaing, Giard, Echallier, & Pernier, 1999). Recently, Jednoróg and colleagues demonstrated that in a phonological priming task, the dyslexics differentiate from controls in both the phonologically incongruent and congruent conditions, that is, dyslexics exhibited reduced N400 in the former (less negative-going) and enhanced N400 in the later (more negative-going). This pattern was taken to indicate that in dyslexia the integration of phonologically related information and the ability to detect deviant stimuli are impaired (Jednoróg, Marchewka, Tacikowski, & Grabowska, 2010). Further, FMRI studies have indicated that there is a relative underactivation in dyslexics in the left posterior parts of the reading network, including the temporal and temporoparietal regions; these are believed to be central for phonological decoding and integration processes (Georgiewa et al., 2002; Pugh et al., 2000a; Sandak, Mencl, Frost, & Pugh, 2004; Shaywitz et al., 2001). Yet, these deviant brain responses in dyslexic individuals reflect mainly their difficulties with explicit phonological tasks (e.g., many studies have typically focused on indirect meta-phonological awareness tasks like rhyme judgments) and the way in which phonological information is processed in these tasks is not necessarily the same as under normal perceptual conditions. On-line phonological processing capacities in dyslexics lack further investigation.

Moreover, recent research has suggested that visual-orthographic processing may also characterize dyslexic reading. This hypothesis is supported, for example, by behavioral indices showing dyslexic group performance to be poor in tasks that emphasize lexical orthographic processing (e.g., orthographic judgment tasks, Marinelli, Angelelli, Notarnicola, & Luzzatti, 2009; orthographic lexical decision tasks, Bergmann & Wimmer, 2008). Neuroimaging studies have offered further insight, suggesting evidence for a reduced level of automaticity in visual word processing in dyslexia, which is subserved by the left occipitotemporal region (e.g., Cao, Bitan, Chou, Burman, & Booth, 2006; Maurer et al., 2007, 2011; van der Mark et al., 2009; Wimmer et al., 2010).

The left occipitotemporal region has attracted the attention of reading researchers in the recent years. Development of brain processes within this region that are specifically tuned to recurring properties of a writing system allows, within less than 250 ms, the rapid extraction of linguistic information from sequences of letters (separable from higher-order linguistic properties) and the integration of letters into a perceptual word-form (Brem et al., 2006; Parviainen, Helenius, Poskiparta, Niemi, & Salmelin, 2006; Tarkiainen, Helenius, Hansen, Cornelissen, & Salmelin, 1999). The left occipitotemporal region corresponds closely to the visual word form area (VWFA) of Cohen and colleagues (Cohen et al., 2002; McCandliss, Cohen, & Dehaene, 2003), whose primary function during reading is to "support a form of perceptual expertise for visual word recognition that enables rapid perception of visual words in one's own language" (Schlaggar & McCandliss, 2007, p. 480). More specifically, recent evidence indicates that at least two levels of VWFA specialization exist: a fast, coarse specialization for print (letter sequences vs. visual control stimuli) and a specialization for processing letter sequences also at the whole-word level, as reflected in VWFA sensitivity to the orthographic familiarity of letter sequences (e.g., Bruno, Zumberge, Manis, Lu, & Goldman, 2008; van der Mark et al., 2009). A number of FMRI studies have suggested that disruption of this region in dyslexic when compared to normal-reading subjects is evident at both coarse, low-level letter-sequences processing, and, also, dyslexics failed to exhibit a second level of VWFA specialization at the higher whole-word level, that is, increased activity for unfamiliar than familiar word-forms (the so-called *orthographic familiarity effect*; van der Mark et al., 2009; Wimmer et al., 2010; see also, Cao et al., 2006).

Time-sensitive event related potentials have identified the N1 component that peaks within 150–200 ms as the strongest index of specialized letter-sequences processing (e.g., Bentin et al., 1999; Maurer, Brem, Bucher, & Brandeis, 2005; Maurer et al., 2007). In addition, some studies have found similar effects in the preceding P1 (~50–150 ms; Maurer et al., 2005, 2011). Other orthographic characteristics have been shown to modulate the N1, including word frequency. Higher frequency words generally evoke lower amplitude neural responses (Assadollahi & Pulvermüller, 2003; Hauk & Pulvermüller, 2004; Sereno, Rayner, & Posner, 1998). It has been suggested that this effect reflects facilitated access to lexical information (e.g., Hauk & Pulvermüller, 2004), though the variable word frequency can also affect later stages of word processing (e.g., Hauk, Davis, & Pulvermüller, 2008).

While a range of studies demonstrate enhanced and left-lateralized N1 to sequences of letters (vs. control stimuli) in skilled readers, some evidence suggests an absence of this N1 effect in the dyslexic population. For example, Maurer et al. (2007) used ERPs to investigate for the first time how tuning for print develops in young dyslexic children and matched, normally developing, readers. The authors demonstrated that over the course of kindergarten to the second grade, there is an increased activation in normal readers in response to visual words as compared to similar control symbols that occurs around 150–270 ms (N1 component). In contrast, this was absent in the children who developed dyslexia. The authors concluded that the earliest cortical activation that is specifically related to prelexical orthographic analysis is abnormal in dyslexia. Maurer and colleagues' results contrasted with those from a previous study that failed to report a reduction in the N1 response in dyslexics (Simos et al., 2000). However, in a magnetoencephalographic (MEG) study with dyslexic adults, Helenius and colleagues confirmed a delayed activation in left occipito-temporal areas in dyslexics at the point in time during which letter sequence specific signals first emerge during reading (~150 ms; Helenius, Tarkiainen, Cornelissen, Hansen, & Salmelin, 1999). These findings were restricted to the most severe cases, which raises the possibility that at some point during development, some dyslexics are able to attain an appropriate brain sensitivity to text. This is a relevant topic on the basis of the developmental trajectory of print specialization in non-impaired readers (i.e., tuning for print plays an important functional role in the early phase of reading acquisition and follows an inverted non-linear U-shaped developmental time course, probably reflecting a full specialization involving more selective brain processes; Brem et al., 2009; Maurer et al., 2006). An open question is whether impaired print specialization is a