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Neural encoding of the speech envelope by children with developmental dyslexia

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ABSTRACT

Developmental dyslexia is consistently associated with difficulties in processing phonology (linguistic sound structure) across languages. One view is that dyslexia is characterised by a cognitive impairment in the "phonological representation" of word forms, which arises long before the child presents with a reading problem. Here we investigate a possible neural basis for developmental phonological impairments. We assess the neural quality of speech encoding in children with dyslexia by measuring the accuracy of low-frequency speech envelope encoding using EEG. We tested children with dyslexia and chronological age-matched (CA) and reading-level matched (RL) younger children. Participants listened to semantically-unpredictable sentences in a word report task. The sentences were noise-vocoded to increase reliance on envelope cues. Envelope reconstruction for envelopes between 0 and 10 Hz showed that the children with dyslexia had significantly poorer speech encoding in the 0–2 Hz band compared to both CA and RL controls. These data suggest that impaired neural encoding of low frequency speech envelopes, related to speech prosody, may underpin the phonological deficit that causes dyslexia across languages.

1. Introduction

Children with developmental dyslexia have difficulty in processing the phonological aspects of speech, across languages (Ziegler & Goswami, 2005, for review). For example, they are poor at making decisions about whether words rhyme with each other ("cat" "hat"), at counting syllables in words ("caterpillar" has 4 syllables), at detecting syllable stress ("difficulty" has first syllable stress) and at deleting individual speech sounds (phonemes: "star" without the "s" sound leaves "tar") (e.g., Bradley & Bryant, 1978, English; Wimmer, 1993, 1996, German; Share & Levin, 1999, Hebrew; Kim & Davis, 2004, Korean). These phonological difficulties are found not only when children with dyslexia are compared to chronological age-matched children without reading difficulties (the CA match design), but also when children with dyslexia are compared to younger children matched for reading level (the RL match, designed to equate the effects of reading experience on the brain; Goswami, 2003). Furthermore, training phonology improves reading acquisition for all children (e.g., Bradley & Bryant, 1983; Lundberg, Frost, & Petersen, 1988; Schneider, Kuespert, Roth, Vise, & Marx, 1997), and also improves visual processing in dyslexia (Olulade et al., 2013). Accordingly, the phonological difficulties experienced by children with dyslexia are considered a causal factor in this developmental disorder (Goswami, 2015). Consequently, current remediation relies on intensive phonological training at the phoneme level accompanied by training in letter-sound correspondences (e.g., Brem et al., 2010; Schneider, Roth, & Ennemoser, 2000).

Accurate encoding of the phonological structure of words requires efficient auditory processing. Recent studies with adults and children with developmental dyslexia have consistently reported atypical neural activity related to auditory processing (Abrams, Nicol, Zecker, & Kraus, 2009; Lehongre, Ramus, Villiermet, Schwartz, & Giraud, 2011; Poelmans et al., 2012; Hornickel & Kraus, 2013; Power, Mead, Barnes, & Goswami, 2013; Lizarazu et al., 2015). However, none of these recent auditory studies has used a reading level (RL) match control group, an important research design for helping to distinguish cause from effect in studies of developmental disorders (Goswami, 2003). When children with dyslexia show impairments compared to *both* age-matched peers and to younger children matched for reading achievement, this suggests a causal role, as impairments occur despite matching for both developmental level and reading level. Intervention studies can then be used to investigate the causal status of identified factors. Accordingly, inclusion of an RL-matched

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control group may help to determine whether the observed differences in neural activity in recent auditory studies are a cause of dyslexia or a consequence of the atypical (severely reduced) reading experience that accompanies having dyslexia.

Some neural studies using developmental research designs are now beginning to emerge in the literature. These include longitudinal studies (Krafnick, Flowers, Luetje, Napoliello, & Eden, 2014), studies incorporating an RL-matched group to control for reading experience (Clark et al., 2014; Olulade et al., 2013), studies of pre-readers (Saygin et al., 2013) and studies including unaffected at-risk groups in an attempt to find endophenotypic traits (Khan et al., 2011; Leppänen et al., 2012; Neuhoff et al., 2012). For example, Saygin et al. (2013) studied pre-reading children with a range of phonological abilities. They found significant links between pre-reading phonological skills and the integrity of white matter organisation in the left arcuate fasciculus (Saygin et al., 2013). Krafnick et al. (2014) used an RL- matched control group and fMRI to show that previously-reported differences in grey matter volume between dyslexics and controls arise largely from the disordered reading experience that ensues from being dyslexic, rather than being causal to the disorder (Krafnick et al., 2014). In a longitudinal neuroanatomical study beginning with pre-reading children at-risk for dyslexia, abnormalities in the left-lateralised reading network were only observed *after the children had learned how to read* (Clark et al., 2014). In this small-scale study, the neuroanatomical *precursors* to dyslexia were restricted to the primary sensory cortices (Clark et al., 2014; Goswami, 2014). Meanwhile, an RL-match study exploring the role of visual sensory processing in dyslexia showed that abnormal visual motion processing was a result of impaired reading experience rather than a cause of dyslexia (Olulade et al., 2013).

These recent studies show the power of developmental research designs in distinguishing the causes and consequences of developmental dyslexia. In the electrophysiological (EEG) literature, however, developmental research designs are largely absent. For example, Schulte-Korne and Bruder (2010) reviewed over 30 EEG studies of sensory processing in children and adults with developmental dyslexia, yet none of the studies reviewed included an RL-matched control group to control for the effects of reading experience on the brain. Hämäläinen, Salminen, and Leppänen (2013) reviewed over 50 studies of non-speech auditory processing in developmental dyslexia, including 17 EEG studies. Again, none of the EEG studies reviewed included an RL-matched control group. Some EEG studies have employed an unaffected at-risk group in an attempt to control for reading experience. For example, Neuhoff et al. (2012) compared dyslexic children to unaffected age-matched siblings as well as to unaffected not-at-risk CA controls. The unaffected siblings had a genetic risk for dyslexia but had normal reading and spelling abilities. Neuhoff et al. reported that the late MMN to tone burst stimuli was diminished in both the dyslexic and the unaffected at-risk siblings compared to the CA controls. To our knowledge, the encoding of *connected speech* in developmental dyslexia has not yet been investigated electrophysiologically using an RL-matched group of younger children. Here, we investigate the encoding of sentences in children with developmental dyslexia using EEG and both CA- and RL-matched control groups. An RL control group is crucial in order to disambiguate the effects of reading experience on neural aspects of spoken language processing.

We explored the neural processing of slow temporal information in connected speech as a test of Temporal Sampling theory (Goswami, 2011). Temporal sampling theory predicts impaired neural encoding of speech envelope information in developmental dyslexia. We designed a novel test of temporal sampling theory using recent technical advances that enable speech resynthesis using EEG data (e.g., Mesgarani, David, Fritz, & Shamma, 2009). The resynthesis technique enables the speech stimulus to be reconstructed from the responses of the neuronal populations that encode it. A reverse reconstruction approach is used to find the best approximation of the input stimulus, and this best approximation is then compared to the original stimulus, for example via a linear mapping between features. The accuracy of the reconstruction is described as a correlation. Speech resynthesis techniques thus enable stimulus envelope reconstruction at the level of individual sentences and items (Mesgarani et al., 2009; O'Sullivan et al., 2014).

Accordingly, by reconstructing individual speech stimulus envelopes from their resultant EEG patterns, a direct measurement of the neural encoding of speech by children becomes possible. This was our approach in the current study. We administered a word report task using noise vocoded speech that had been developed for children (Johnson, Pennington, Lowenstein, & Nittrouer, 2011), while simultaneously recording EEG. Noise vocoding degrades the temporal fine structure (TFS) of speech (see Fig. 1) while leaving the low frequency envelope intact. When the TFS of speech is degraded, listeners are forced to rely largely on the preserved envelope information in order to perceive the words and the sentences accurately. Although accurate listening is also supported by semantic information, here we deliberately used sentences that were semantically unpredictable (while being syntactically appropriate, e.g., "Arcs blew their cough"). Therefore, childrens' ability to report the words and sentences accurately should enable assessment of the quality of their neural encoding of low frequency envelopes in speech. On temporal sampling theory, the quality of neural encoding for these low frequency envelopes should be impaired for children with dyslexia.

Utilising a developmental research design, we compared the neural encoding of low frequency speech envelopes by children with dyslexia with neural encoding by both CA-matched and RL-matched typically-developing control children. If children with dyslexia show significantly poorer speech encoding compared to younger children who can read the same number of words (the RL match design), the dyslexic deficit is less likely to arise from reduced reading experience (Goswami, 2003, 2015). We assessed envelope reconstruction accuracy in 5 frequency bands (0–2, 2–4, 4–6, 6–8, 8–10 Hz). Following prior work, reconstruction accuracy was estimated by the Pearson correlation between the actual stimulus envelope of each sentence and the EEG reconstruction (Mesgarani et al., 2009; O'Sullivan et al., 2014). In speech resynthesis studies to date, reconstruction effects for neurotypical adults listening to connected speech (e.g., in a cocktail party paradigm) have yielded significant median Pearson correlations in the range of 0.05 (e.g., O'Sullivan et al., 2014).

All our sentences consisted of four monosyllabic words, hence had a relatively predictable temporal pattern. Recent research on temporal prediction has highlighted the relevance of delta-beta phase-amplitude cross-frequency coupling (Arnal, Doelling, & Poeppel, 2014). To explore the potential contribution of these temporal prediction networks to our sentence encoding task, we compared the topographies of delta-beta phase-amplitude coupling between our groups. Beta band activity has also been characterised recently as playing a privileged role in speech processing (see Poeppel, 2014). Accordingly, we also explored topographical differences in beta power between the children.

2. Materials and methods

2.1. Participants

Forty-six children participated in the study, who were all taking part in a longitudinal behavioural study of auditory processing (Goswami et al., 2013). Participants comprised all children in the