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Early dynamics of white matter deficits in children developing dyslexia

Jolijn Vanderauwera[a,b,*], Jan Wouters[b], Maaike Vandermosten[a,b,1], Pol Ghesquière[a,1]

[a] Parenting and Special Education Research Unit, Faculty of Psychology and Educational Sciences, KU Leuven, Belgium
[b] Research Group ExpORL, Department of Neurosciences, KU Leuven, Belgium

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ABSTRACT

Neural anomalies have been demonstrated in dyslexia. Recent studies in pre-readers at risk for dyslexia and in pre-readers developing poor reading suggest that these anomalies might be a cause of their reading impairment. Our study goes one step further by exploring the neurodevelopmental trajectory of white matter anomalies in pre-readers with and without a familial risk for dyslexia (n = 61) of whom a strictly selected sample develops dyslexia later on (n = 15). We collected longitudinal diffusion MRI and behavioural data until grade 3. The results provide evidence that children with dyslexia exhibit pre-reading white matter anomalies in left and right long segment of the arcuate fasciculus (AF), with predictive power of the left segment above traditional cognitive measures and familial risk. Whereas white matter differences in the left AF seem most strongly related to the development of dyslexia, differences in the left IFOF and in the right AF seem driven by both familial risk and later reading ability. Moreover, differences in the left AF appeared to be dynamic. This study supports and expands recent insights into the neural basis of dyslexia, pointing towards pre-reading anomalies related to dyslexia, as well as underpinning the dynamic character of white matter.

1. Introduction

Reading is a relatively recent cultural invention (around 5000 years ago) (Dehaene, 2009). In evolutionary terms this is a relatively short time span for our brain to develop a genetically imprinted reading network. Hence, when a child learns to read, pre-existing brain networks are reorganized within only a few years of time (Dehaene, 2009). Although the vast majority of children becomes literate rather easy within their early lives, 3–7% of the children struggles with learning to read and/or write. They are diagnosed with developmental dyslexia (Peterson and Pennington, 2015; Snowling, 2000), a learning disability characterized by severe and persistent reading and/or spelling impairments not accounted for by intellectual and sensory deficits (Peterson and Pennington, 2015; Vellutino and Fletcher, 1964). The diagnosis of dyslexia is typically given after several years of reading/writing instruction, when the impairments show up to be severe and persistent. This implies that targeted interventions do not start during the first stages of literacy acquisition, when they are most effective (Ozernov-Palchik and Gaab, 2016; Torgesen, 2002). Yet, early intervention is important because children experiencing a lifelong reading failure, are likely to display lower educational attainment and more psychiatric and health problems (Undheim, 2003). A thorough understanding of neurodevelopmental reading processes can aid our understanding of the aetiology of developmental dyslexia. Moreover, a thorough understanding might enhance early detection of dyslexia, that can consequentially lead to more effective remediation. The neural reading network consists of three distinct left hemispherical regions in advanced readers, i.e. inferior frontal, temporo-parietal and occipito-temporal cortex (for reviews, see Martin et al., 2015; Norton et al., 2015; Paulesu et al., 2014; Sandak et al., 2004). Functional neural deficits associated with dyslexia have been consistently shown in the left posterior regions of the reading network, mostly in adults (Richlan et al., 2009; Turkeltaub et al., 2003) but also in children (Richlan et al., 2011) and pre-readers at risk for dyslexia (Vandermosten et al., 2016), with mixed evidence for deficits in frontal or right hemispheric regions (Richlan et al., 2009). Given the interactive and dynamic character of the brain, previous research suggested that the neural deficit in dyslexia might not originate within cortical regions, but rather in the white matter connections between them (Boets et al., 2013; Saygin et al., 2013; Wang et al., 2016). The three main regions of the reading network are dorsally connected through the arcuate fasciculus (AF), while a ventral connection is sustained by the inferior fronto-occipital fasciculus (IFOF) (Vandermosten et al., 2012a). In adults and school-aged children with dyslexia, white matter anomalies have been shown in the left dorsal pathway, more specifically in the direct segment of the AF connecting the frontal region to the temporo-parietal region

* Corresponding author at: L. Vanderkelenstraat 32, PO Box 3765, 3000 Leuven, Belgium.
E-mail address: Jolijn.Vanderauwera@kuleuven.be (J. Vanderauwera).
[1] Both should be considered as 'joint last author' of this paper.

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(Rauschecker et al., 2009; Vandermosten et al., 2012a, 2012b). Recent studies have demonstrated that these anomalies are already present prior to reading onset in those children at familial risk (Langer et al., 2015; Wang et al., 2016; but see Vandermosten et al., 2015) or at cognitive risk for dyslexia (Saygin et al., 2013). These studies point towards a causal role of dorsal white matter anomalies in developmental dyslexia, rather than these anomalies being a consequence of reading failure. However, evidence is scarce on the developmental trajectory of white matter anomalies paralleling the very first stages of reading acquisition, specifically in those children who demonstrate severe and persistent reading deficits, i.e. children who develop dyslexia. First attempts have recently been made to address this gap of knowledge. Wang et al. (2016) demonstrated slower development of white matter fractional anisotropy (FA) in a temporo-parietal node of the left direct AF segment in children who developed poor relative to good reading skills. Another recent study by Kraft et al. (2016) showed higher T1 intensities, an indirect measurement of myelin, in the anterior segment of the AF in pre-readers who developed poor reading skills. However, they did not observe differences in white matter FA, the most common used measure for quantitative description of white matter pathways. In addition to the involvement of the dorsal AF in reading, the ventral IFOF has been suggested to be involved in orthographic aspects of reading in adults (Vandermosten et al., 2012a). Yet, the specific role of this pathway is unclear in early reading stages, which rely more on phonological processes. The investigation of ventral white matter connections prior to reading onset and through the first stages of reading acquisition is therefore important, as this might entail information on the neurodevelopmental reorganization paralleling reading acquisition, and plausibly reading failure. One study that investigated the IFOF in the pre-reading brain, by means of T1 intensities, demonstrated no effect of family risk (Kraft et al., 2016).

The last decade, studies have indicated that a familial risk for dyslexia is related to neural deviances (e.g. Raschle et al., 2011; Raschle et al., 2012). It is, however, not clear whether these neural differences are associated with both the disorder and the familial risk (Leppänen et al., 2010), or with the familial risk regardless of reading/writing outcome (Hakvoort et al., 2015; Vanderauwera et al., 2016). The present study fills this gap by investigating structural white matter reorganization through the very first stages of reading and writing acquisition both in children who develop dyslexia and in children who are merely at familial risk for dyslexia. The first aim of the present study is to investigate whether atypical pre-reading neural connectivity is specific to those children developing *severe and persistent* reading and/or spelling difficulties, i.e. developmental dyslexia. This study thereby aims at enlarging recent insights in poor readers (Kraft et al., 2016; Wang et al., 2016). The dynamic pattern of the potentially observed deficits through the initial stages of reading acquisition will also be tracked, by means of a longitudinal study design (n = 61). We hypothesize that if neural differences will be found prior to reading onset in those children developing dyslexia, these differences will be located in the left long AF segment, as differences in this pathway have most consistently been demonstrated in older subjects (e.g Vandermosten et al., 2012a). Second, we aim to investigate whether we can observe, in addition to dyslexia-related white matter anomalies, white matter differences that are merely related to the familial risk for dyslexia, which would define dyslexia as a continuum, also at the neural level. These differences can be expected in the ventral IFOF (Kraft et al., 2016) or in the dorsal AF (Langer et al., 2015; Wang et al., 2016). Finally, the predictive value of cognitive, familial risk and neural factors for developmental dyslexia will be investigated. It is our special interest to investigate whether potentially observed pre-reading white matter differences between children developing typical reading skills and children developing dyslexia can attribute to the prediction, on top of familial risk and cognitive predictors including phonological skills, that we hypothesize will provide the strongest prediction.

2. Methods

2.1. Participants

MRI scans were administered in 75 children before the start of literacy acquisition, i.e. during the summer holidays prior to first grade when the children were aged 5–6 years old. Since acquiring MRI scans in this population is challenging, a submarine protocol was developed that sufficiently prepared the children on the MRI assessment (Theys et al., 2014). After two years of reading and writing instruction, i.e. during summer holidays prior to third grade, MRI scans were conducted again in a subsample of 65 children using a knight and damsel protocol. Similar as in the submarine protocol (Theys et al., 2014), the knight and damsel protocol prepared the children for the MRI scanner in a playful manner. In a first step, the child watched a movie at home with his/her parents, in which an introduction and explanation was given of the scanning session. Second, before entering the MRI examination room, a set of different games was played together with the child in a small castle, explaining every aspect of MRI scanning and training adequate within scanner behaviour. Because of inadequate data acquisition in four participants, longitudinal diffusion images are available of 61 children. Thirty-four of these children had a familial risk for dyslexia (FRD^+), defined by having at least one first-degree relative with dyslexia, while 27 children had no familial risk (FRD^-). In the initial sample (Vanvooren et al., 2014), children with and without a familial risk were pairwise matched based on sex, age, parent's socio-economic status (SES) assessed with the Family Affluence Scale (Boudreau and Poulin, 2009; Boyce et al., 2006), non-verbal intelligence assessed with the Coloured Progressive Matrices (Raven et al., 1984), and school environment (i.e. same class). In the sample included in this study, no group differences are present in these matching variables (see Table 1). Participants were selected in kindergarten based on five inclusion criteria (Vanvooren et al., 2014): (1) a non-verbal IQ above 80, (2) normal hearing (i.e. a Fletcher index of less than 20 dB HL), (3) monolingual native Dutch speaking, (4) no history of brain damage, vision deficits, or articulatory problems, and (5) no high risk for developing ADHD. Non-verbal intelligence has again been tested at the start of second grade by the WISC-III-NL subtest Block Design (Wechsler, 2005) (Table 1). One child was diagnosed with attention deficit disorder (ADD), however, results did not change by removing this participant. At the early reading stage, all participants had two years of reading instruction, except for one child who had one, and another child who had three years of reading instruction. One child changed after one year of reading instruction from a Dutch school to a French school. Similarly, removing these participants from the analyses did not change the results.

Two sets of criteria were applied to classify children as dyslexic readers (DR) or as typical readers (TR). For these classifications word reading, pseudo-word reading and spelling scores conducted at the start of the second and third grade were used. The first set of criteria selected children with reading problems that are severe and persistent, i.e. a score below percentile 10 on the same reading test at the two time points. Based on this set of criteria, 11 children were classified as dyslexic. The second set of criteria selected children with severe and persistent spelling problems, i.e. below percentile 10 at the two time points. As dyslexia is mostly defined as a reading impairment, children that were selected based on severe and persistent spelling problems were additionally required to have reading scores below percentile 16 at both measurement times, to assure that these children also had poor reading skills. Based on this set of criteria, four additional children were classified as dyslexic. Hence, of the 61 children included in this study, 15 children developed dyslexia: 7% (n = 2) of the children without a familial risk and 38% (n = 13) of the children with a familial risk. These results are in line with the expected prevalence described in these two populations (Gilger et al., 1991; Snowling, 2000).

In kindergarten, the children of this study did not receive reading