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Cerebral lateralization of language in children at risk for dyslexia: A review of neuroscientific evidence

Anastasia-Konstantina Papadopoulou[a,b], Filippos Vlachos[c], Marietta Papadatou-Pastou[a,b]

a School of Education, National and Kapodistrian University of Athens, Greece

b Biomedical Research Foundation, Academy of Athens, Greece

c Department of Special Education, University of Thessaly, Greece

Abstract

Dyslexia is a neurodevelopmental disorder of word-level processing. It is typically diagnosed at school age, when children are expected to learn to read and spell accurately and fluently. One of the neurobiological traits that characterize children with dyslexia is the atypical cerebral lateralization of language. Typical cerebral lateralization of language -meaning increased activation of the left hemisphere compared to the right one during language tasks- is shown for the majority of neurotypical individuals. However, hemispheric activation in children with dyslexia during language processing is more bilateral or right-lateralized. There is evidence that cerebral lateralization of language is partially or completely established before the onset of literacy training, in infancy or kindergarten years. Therefore, atypical lateralization can also be observed in that age and provide an indication of the risk for dyslexia prior to diagnosis. In the present review, our main focus is to present neuroscientific evidence that has associated different components of the cerebral lateralization of language -namely, functional, structural, functional connectivity, and neurogenetic- with the risk for dyslexia. In addition, we stress out the gap of knowledge regarding the cerebral lateralization of written language in children and we present our future goals to address this gap.

Keywords

dyslexia, cerebral lateralization, risk, language

Corresponding author: Marietta Papadatou-Pastou, School of Education, National and Kapodistrian University of Athens, Greece, Email: marietta.papadatou-pastou@seh.oxon.org , mpapadatou@primedu.uoa.gr (npapadop@bioacademy.gr fvlachos@uth.gr, mpapadatou@primedu.uoa.gr)

"There is no symmetry in nature; one eye is never exactly the same as the other" ~ Edouard Manet (1832-1883; famous French painter).

Introduction

Asymmetry or laterality is evident in every aspect of nature including the human brain. Cerebral laterality provides an evolutionary advantage through better performance and more efficient response (1, 2). There are multiple cognitive and motor functions that display a pattern of cerebral lateralization in the majority of individuals (3-6). One of the most well-studied manifestations of cerebral laterality is the cerebral lateralization of language (7, 8). This lateralization has been found to be weaker in children with dyslexia, but also in children *at risk* for dyslexia, compared to neurotypical children (e.g., 4). Dyslexia, a Greek word indicating an impairment at a word level, is characterized by difficulty in recognizing, storing, retrieving, and representing speech sounds leading to deficient reading accuracy and fluency as well as difficulties in spelling in spite of normal intelligence (4, 9-13) and adequate schooling (12). In the context of this review, we will discuss neuroscientific data that show that dyslexia is associated with atypical lateralization of language-related regions. We will also stress out the paucity of studies of cerebral lateralization of written language in children with dyslexia and how we aim to address this gap in the literature.

Atypical lateralization in dyslexia

Language is a fundamental component of human evolution and society. A plethora of neuroimaging and neurophysiology studies have established that language is typically left-lateralized for the majority of individuals, meaning that the language-related regions of the left hemisphere are more activated compared to the respective regions of the right hemisphere during language tasks (5,14-16). Differences between brain hemispheres in gene expression (3,6,17), volume (18,19), structure (3,6), and connectivity (5,8,18,20-22) might underlie this functional asymmetry. Although it is generally agreed that a clear language lateralization pattern is formed by the age of six or seven (23), there are contradictory findings regarding language lateralization in infancy and in early childhood (10,11,24-38); most findings suggest that brain activation is bilateral during language comprehension for the first three years of age and it progressively becomes more left-lateralized (e.g., 27,32,33). However, there are also studies showing that left lateralization of language is apparent in neonates (30) and that there is no change in the degree of lateralization between ages one and five (29). Atypical language lateralization, meaning bilateral or right-shifted activation, is associated with left- and mixed-handedness in neurotypical individuals (5,15,39,40) and with neurodevelopmental disorders, such as autism spectrum disorder (41) and schizophrenia (42), but also language disorders, such as dyslexia (43-45).

In dyslexia, brain regions that show atypical lateralization during language tasks include the left inferior gyrus and the left temporo-parietal cortex, together with other posterior perisylvian areas of the left hemisphere (44,46-53). These regions are associated with phonological awareness, meaning the ability to comprehend, analyze, and manipulate the sound components of speech (54). The accompanying phonological deficit is considered the most prominent characteristic of dyslexia (9,44,55-57). In fact, appropriate educational interventions focusing on the amelioration of phonological awareness have been shown to lead to the reorganization of the brain to approximate the lateralization profile of typically developing children (58,59). Children are usually entailed in these programs after having developed the symptomatology and having been diagnosed with dyslexia, although earlier intervention has better performance outcomes (58).

Dyslexia also affects other trajectories of information processing (48,60,61). Such aspects, which are also impaired but do not seem to have a causal role in dyslexia, are audiovisual stimuli integration (62-66), working memory (67), and writing (44,57,68), as well as writing problems - the latter being the most difficult to overcome (13,55,57,69). Brain regions associated with visual and auditory processing show impaired functional connectivity and activation patterns (22,66), while aberrant morphology of parietal regions is linked to working memory problems (70). Although there is also evidence of structural and functional abnormalities in regions associated with writing (49,71,72), the neurological underpinnings for cerebral lateralization of written language in children with dyslexia have never been assessed.

When it comes to the factors that might prelude the development and manifestation of dyslexia symptoms, we should take into consideration that dyslexia is a disorder of neurobiological origin (73), while also being subject to environmental conditions. It has a heterogeneous genetic basis and it is highly heritable, but not through Mendelian inheritance (47,55,58,67,74,75). Family history of dyslexia (58) and the general literacy environment (76), but also prenatal exposure to testosterone (77) and the sex of the individual (47) are factors that influence acquiring and expressing the dyslexia phenotype. Dyslexia often coincides with other learning disorders, such as dysgraphia and attention deficit hyperactivity disorder (73,78). Children with dyslexia with or without these comorbidities fall within the normal range of cognitive performance before the occurrence of early symptoms (9,48,58,79-81).

The full symptomatology of the disorder emerges not earlier than school age, when children with dyslexia encounter more difficulties in reading, spelling, and writing compared to their classmates (9,58,73). Having said that, it has been shown that neurodevelopmental disorders can be predicted earlier than the occurrence of the symptoms, using not only behavioral, but also neurobiological indicators (18,58,82). In the next paragraphs, we will review how the risk for dyslexia in pre-readers is associated with one such neurobiological indicator: cerebral lateralization for language. We will specifically review evidence on atypical functional and structural lateralization, but also evidence regarding functional connectivity and neurogenetics.

Neuroimaging studies in children at risk for dyslexia

Starting with functional laterality, in individuals with dyslexia, regions related to phonological processing are often atypically lateralized, as mentioned above. Dębska et al. (2016) found that the same regions that are shown to be atypically lateralized in individuals diagnosed with dyslexia, viz the temporo-parietal region, the occipito-temporal cortex and the inferior frontal gyrus, were less left-lateralized during phonological processing in Polish kindergarten children at familial risk for dyslexia compared to typically developing children of their age group, despite the absence of behavioral indicators (83). In fact, the activation of the left ventral occipito-temporal cortex in phonology-related tasks has been positively correlated with the ability of kindergarten children at varying risk for dyslexia to respond to short educational training (84). Moreover, children at risk for dyslexia, who do not respond to training, display underactivation in the left hemisphere (85). Davis et al. (2011) also showed that the efficacy of an intervention for children at risk for developing dyslexia is associated with the degree of activation of the left superior temporal gyrus (85). Other neuroimaging studies provide evidence that the left superior temporal gyrus participates less in the processing of phonological information in children at risk for dyslexia compared to controls, while the homologous region of the right hemisphere is more engaged (86,87). Decreased leftward lateralization in children at risk for dyslexia has also been related with a rightward shift of activation during phonological processing tasks (87-89). In contrast to these findings, a recent study by Nora et al. (2021) suggested that the underactivation of the left hemisphere and the overactivation of the right in early primary school children at risk for dyslexia are not associated with an impairment in phonological processing as assessed by pseudoword repetition, but rather with a difficulty in performing better over repetition due to an implicit memory deficit (56). This finding is in line with the observation of persistent working memory problems in individuals with dyslexia (9, 67).

Atypical structural laterality often underlies atypical functional laterality, such as in the cases described above. The decreased leftward lateralization observed in temporo-parietal and occipito-temporal regions of young children at risk for dyslexia are indeed accompanied by analogous differences in gray matter areas compared to typically developing children (16,90). For example, the planum temporale is a region that is associated with phonemic processing, one of the initial stages of phonological awareness. In the neurotypical population, the left planum temporale is larger than the right planum temporale (16,66,91). However, the left planum temporale of individuals with dyslexia is smaller than the right homologous region (16). This effect in planum temporale size of twins has been attributed to intrauterine events (16,92). Vanderauwera et al. (2018) showed that even young pre-readers at risk for dyslexia show this atypicality and, hence, a smaller left planum temporale can be considered an early indicator of dyslexia (16).

Differences in the activation of brain regions could be further attributed to changes in functional connectivity. Disconnections between language-related regions of the left hemisphere and failure of regions of the right hemisphere to decouple have been reported in many cases of dyslexia (9,18,93-95). These impairments also seem to precede the occurrence of dyslexia symptoms. Preschool children without risk for dyslexia show left lateralization of white matter tracts interconnecting language-related regions, while in children at familial risk for dyslexia these tracts show a rightward lateralization (16,96,97). The differences in white matter connectivity between infants at familial risk for dyslexia and controls that can be observed even earlier than the second year of life are in fact specific for language-related regions of the left hemisphere and they can help explain the functional disconnection between these regions (98).

Connectivity abnormalities are also evident in the auditory cortex of individuals with dyslexia, along with atypical functional and structural laterality (22,66,99-101). Thus, observations of atypical gyrification and connectivity of the left primary auditory cortex in children with dyslexia, both prior and after literacy training, could indicate the presence of reading problems resulting from preexisting auditory problems (56). In fact, auditory stimuli processing has gained attention as a neurological trait that can lead to early detection of dyslexia risk, potentially because it is more feasible to assess auditory stimuli processing in infants or preschoolers, compared to phonological processing. In this vein, studies using event related potentials (ERP) -meaning small voltage signals generated by different brain regions (102)- have provided electrophysiological evidence for the hemispheric activation in children at risk for dyslexia in response to auditory stimuli. Most studies have shown that ERPs derived from children as young as even 6 months old in response to speech stimuli show the expected atypical asymmetry -specifically decreased left hemispheric involvement and increased right hemispheric involvement- leading to a more bilateral or right-shifted lateralization pattern (82,103-109). However, according to Cartiani et al. (2016) and Thiede et al. (2019), young children of typical development show a rightward lateralization of their responses to speech stimuli, while at-risk children display a more bilateral distribution of response (110,111). Finally, van Zuijen et al. (2013) have found that auditory stimuli were predominantly processed by the right hemisphere in both typically developing and at-risk 2-month-old infants (112). It is apparent that further research is needed on this topic.

Genetic studies on dyslexia

Given the hereditary nature of dyslexia, genetic polymorphisms have been studied as indicators of atypicalities in the cerebral lateralization of language. Such polymorphisms can serve as a very early warning for potential future reading difficulties. An increasing number of genes (up to 13) and chromosomal loci (up to 42) have been identified during the past decades (113,114). At least nine genetic risk loci and ten candidate

genes (e.g., DYX1C1, DCD2, KIAA0319, ROBO1) have been associated with the risk for developing dyslexia, and most of them are involved in brain development (56,98,115). Experimental manipulation of these genes in studies with rodents, accompanied by postmortem studies of individuals with dyslexia, have provided evidence that the majority of these susceptibility genes are implicated in neuronal migration (116,117). Risk alleles for the DCD2 and the KIAA0319 genes have been mainly associated with a reduced volume of white matter tracts lying within the left temporo-parietal area of the brain and, hence, leading to reduced activation of this area during language tasks (50,92,118). In fact, the rs793842 allele of DCD2 is associated not only with a decrease in the volume of the white matter trajectories between the arcuate fasciculus and the posterior superior temporal sulcus, but also with reduced gray matter thickness in the left middle temporal gyrus (50). Overall, the presence of some risk alleles in the genome could play an important role in the development of the atypical asymmetry profile that is associated with dyslexia or with the risk for the disorder.

Conclusions and future directions

To summarize, neuroimaging and genetic studies have allowed us to investigate cerebral lateralization of language prior to reading age and, thus, before the onset of reading difficulties in children at risk for dyslexia. We can conclude that most evidence points to the fact that, on average, children at risk for dyslexia show a different lateralization pattern compared to typically developing children, be it a less left-lateralized, more bilateral, or more right-lateralized pattern. Of note there is also some contradictory evidence, reporting either an absence of a difference between the two groups or a rightward lateralization in controls. However, most studies have sampled a relatively small number of children and even less infants.

An important limitation of the literature on cerebral lateralization of language in children at risk for dyslexia, is that, given the young age of these children, all the previously mentioned findings come from the assessment of oral language comprehension and production. Cerebral lateralization of written language in children has not been investigated to date, even though, worldwide, about 90% of adolescents (119) use written language for communicational and educational purposes. As a consequence, there are no studies regarding the cerebral lateralization of written language in children at risk for dyslexia. Moreover, there are no studies in children that have already developed dyslexia, for whom writing difficulties are a persisting problem. In our future studies, we are planning to assess this dimension of language lateralization in children at risk for dyslexia using functional Transcranial Doppler ultrasonography (fTCD).

FTCD is a non-invasive neurophysiological technique that measures the changes in blood flow velocity in the two middle cerebral arteries, right and left, during a task, as a proxy for the level of activation of each hemisphere (120,121). FTCD provides comparable results to other well-known neuroimaging techniques, such as functional Magnetic Resonance Imaging (fMRI; 121,122), but it has lower spatial resolution and fails to penetrate the scalp in 5-20% of individuals (121,123). The advantages of fTCD over the more popular techniques is that it is inexpensive, portable, and unaffected by bodily movements at a range from facial movements during speech to driving a car (121, 124,125). This is particularly important for the study of writing, which in other techniques can only be studied with the use of additional equipment. Taking these advantages into account, fTCD allows for the relatively easy and inexpensive collection of neurophysiological data in cases that functional scanning is problematic, such as in infants or children (124,126). In fact, fTCD has been applied to infants as young as 1 year of age (29,127). Moreover, this technique is widely used to study cerebral lateralization across different age groups, which makes it eligible for longitudinal studies (e.g., 128-138). Therefore, fTCD can be employed to assess cerebral lateralization of written language in children at risk for dyslexia and typically developing children.

The specific aims of our future studies are: (a) to examine the differences in cerebral lateralization of written language between young primary school students at risk for dyslexia and typically developing age-matched controls, (b) to investigate how more severe than expected difficulties in writing might affect the pattern of cerebral lateralization of written language in at-risk children, and (c) -given the shift in the cerebral lateralization of oral language following phonological educational interventions- to assess whether the pattern of cerebral lateralization of written language in children at risk for dyslexia shifts to approximate the lateralization pattern of typically developing children, following a 3-month-long phonological intervention. This line of research will add to the existing literature by studying cerebral lateralization of written language in children and in particular in children at risk for dyslexia and by evaluating the impact of an intervention in their lateralization profile, both for the first time in the literature.

Overall, the neurological underpinnings of dyslexia are present long before the manifestation of the symptoms that will allow the formal diagnosis of the disorder. Genetic risk and structural or functional atypical lateralization related to oral language could be evident as early as in embryonic life/fetal stage and infancy or kindergarten years, respectively. This evidence -taken together with the observations of atypical cerebral lateralization of oral language in older children and adults with dyslexia- allows us to suggest that atypical cerebral lateralization of oral language is a persisting characteristic of dyslexia. Notwithstanding the importance of written language in communication and in education and the significance of writing difficulties in cases of dyslexia, cerebral lateralization of written language is significantly under-studied. Additionally, no studies have investigated the pattern of cerebral lateralization of written language in children with, at risk, or without dyslexia. Therefore, our future studies will fill this gap in our knowledge, allowing us to better understand the phenomenon of cerebral lateralization of written language.

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