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Neurobiology of Dyslexia

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Abstract

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Dyslexia is one of the most common learning disabilities, yet its brain basis and core causes are not yet fully understood. Neuroimaging methods, including structural and functional magnetic resonance imaging, diffusion tensor imaging, and electrophysiology, have significantly contributed to knowledge about the neurobiology of dyslexia. Recent studies have discovered brain differences prior to formal instruction that likely encourage or discourage learning to read effectively, distinguished between brain differences that likely reflect the etiology of dyslexia versus brain differences that are the consequences of variation in reading experience, and identified distinct neural networks associated with specific psychological factors that are associated with dyslexia.

1. Introduction

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Developmental dyslexia, an unexplained difficulty in word reading accuracy and/or fluency, affects 5–12% of children [1,2]. Dyslexia is associated with many undesirable outcomes, including reduced educational attainment and academic self-esteem [3]. Furthermore, children with dyslexia tend to read far less outside of school than their peers [4], resulting in a widening gap in reading skills. Over the past 15 years, neuroimaging has made visible and quantifiable the brain differences that are associated with dyslexia; here, we review progress in the past few years in understanding the biological basis of dyslexia at a neural systems level.

Reading is a complex and slowly learned skill requiring the integration of multiple visual, linguistic, cognitive, and attentional processes. Neuroimaging methods including functional magnetic resonance imaging (fMRI), electroencephalography (EEG, and event-related potentials or ERPs), and magnetoencephalography (MEG), have revealed the brain regions most consistently involved in single word reading. In typically reading adults, these regions are lateralized to the language-dominant left hemisphere, and include inferior frontal, superior and middle temporal, and temporo-parietal regions [5]. In addition, experienced readers recruit an area of the left fusiform gyrus, termed the *visual word form area* (VWFA), which becomes preferentially engaged for orthographic (print) processing with reading experience [6–8]. This reading network (Figure 1) develops over years as children gain both specific reading skills and other abilities relevant to reading (e.g., 9). White-matter pathways that connect the components of the reading network can be quantified in size and strength by diffusion tensor imaging (DTI). Major tracts involved in reading include the left arcuate/superior longitudinal fasciculus, which connects frontal and temporal language regions, the inferior longitudinal fasciculus, which connects occipital and temporal lobes, and the corona radiata, which connects cortex to subcortical structures [10].

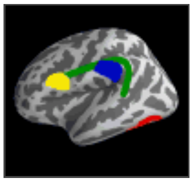

Figure 1
Schematic of the aspects of the reading brain in the left hemisphere. The inferior frontal gyrus (yellow) and the inferior parietal area (blue) are connected by the arcuate fasciculus (green). The fusiform gyrus, which includes the visual word form area, **...**

2. Psychological Bases of Dyslexia

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Because reading involves multiple linguistic, visual, and attentional processes, it is likely that variable patterns of weakness may contribute to reading difficulty across children. Although it is unlikely that there is a single causal mechanism of dyslexia, some frequent likely causes have been identified (Table 1). The best understood cause for
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Key Constructs in Reading and Potential Deficits in Dyslexia

Construct	Definition	Example Tasks
Phonological Awareness (PA)	Knowledge of, and ability to manipulate, the sound structure of words	- Say *game* without the /g/ - What word do these sounds make? /s/ - /i/ - /t/ - Name a word that rhymes with *star*
Rapid Automatized Naming (RAN)	Speed with which a series of familiar stimuli can be named aloud, reflecting efficient visual-verbal connections	Name, as quickly as possible, a 10×5 array of 5 randomly repeated objects, colors, letters, or numbers
Reading	Ability to read single words and connected	- Read aloud a list of common

3. Functional an

Meta-analyses of primary research findings have identified broad patterns of functional and structural differences between typical and dyslexic readers. The most common functional brain differences, in children and adults, are reduced activations (hypoactivations) in left temporal, parietal, and fusiform (VWFA) regions [19–22]. In most cases, these hypoactivations arise from comparisons between two tasks or conditions, and thus reflect a lack of differential sensitivity to reading demands rather than a broader dysfunction of those brain regions. Increased activations in dyslexia are sometimes, but not consistently, observed in left inferior frontal and right-hemisphere regions. Variability across these findings may reflect differences in reading tasks, ages of participants, diversity among dyslexic groups, and other factors. Additionally, structural gray matter differences in dyslexia tend to co-localize with regions that show functional differences [23], but are also observed in the cerebellum, particularly in lobule VI [24, 25]. DTI studies often find reduced organization or volume in the left superior longitudinal fasciculus, including the arcuate fasciculus, and corona radiata fibers [26].

Because most neuroimaging studies of dyslexia have been conducted with children or adults who have had years of reading difficulty, it has been impossible to determine whether the brain differences are associated with the underlying neurobiological etiology of dyslexia, or are instead the consequence of years of altered and often vastly reduced reading experience (including compensatory alterations in reading networks). One approach to dissociating the cause and consequence of dyslexia in the brain has been to compare dyslexic children not only to age-matched typically reading children, but also to “ability-matched” children who are years younger than the dyslexic children but read at the same level. Ability-matched children are conceptualized as having approximately the same amount of reading experience as older dyslexic children. In one such study, dyslexic children exhibited reduced left parietal and occipito-temporal activations relative to both age- and ability-matched children, suggesting that these hypoactivations were related to the cause of dyslexia (in contrast, left prefrontal activations tracked ability level) [27].

A similar design challenged another idea about dyslexia, the magnocellular hypothesis of dyslexia. Previously, postmortem evidence from individuals with dyslexia revealed smaller magnocellular neurons in the lateral geniculate body [28], part of the visual pathway that is associated with motion perception. Accordingly, reduced activation for moving gratings in area MT, the cortical region most associated with motion perception, was found in adults with dyslexia [29]. When, however, children with dyslexia were examined, their MT activations were equivalent to ability-matched younger children, suggesting that the MT hypoactivation in dyslexia reflected reading experience [30]. This conclusion was further supported by evidence that remediation of the reading difficulty also enhanced MT activations in children with dyslexia [30]. These findings suggest that reduced MT activation for visual motion in dyslexia is a consequence, not a cause, of dyslexia. Similarly, many structural brain differences in dyslexia among age-matched groups were eliminated when a group with dyslexia was compared to ability-matched children [31].

Another strategy for identifying brain differences that underlie dyslexia has been the study of pre-reading children, typically in kindergarten, for whom brain differences cannot be the consequence of altered reading experience. Although pre-reading children cannot have a formal diagnosis of dyslexia, children can be identified as at-risk for dyslexia because of either a family history of dyslexia, which increases their risk of dyslexia by four times or more [32], or low performance on tests of pre-reading skills that tend to predict future reading difficulty (e.g., PA or RAN). Often, these children are followed longitudinally to determine which at-risk children actually progress to dyslexia.

Several neuroimaging studies have found brain differences preceding formal reading instruction in pre-reading children that resemble those observed in older children and adults. ERP studies of the mismatch negativity (MMN), an automatic response to an oddball auditory stimulus that is reduced in adults with dyslexia, have observed differences between infants with versus without a family history of dyslexia [33], and infants who do or do not develop dyslexia [34, 35]. Thus, the MMN may be a promising early endophenotype of dyslexia [36].

In MRI, pre-reading kindergartners with familial risk for dyslexia exhibited reduced bilateral occipitotemporal and left temporo-parietal activations for PA [37] and also bilaterally reduced grey matter volumes in similar posterior cortical regions [38]. Decreased grey-matter volumes in prefrontal and parieto-temporal regions were also found in 5- and 6-year-olds with maternal histories of reading difficulty [39]. In a heterogeneous sample of kindergartners, pre-reading children exhibited a positive correlation between measures of PA and both the size and microstructural white-matter organization of the left arcuate fasciculus [40]. Although it is not yet known which of these children will develop dyslexia, these studies support the idea that the most commonly observed functional and structural brain differences characterizing dyslexia are present before significant reading experience and therefore are more likely causes rather than consequences of dyslexia.

5. Advances in Understanding the Brain Basis of Aspects of Dyslexia

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Brain Basis of Phonological Awareness (PA) Deficits

Impaired PA in dyslexia could reflect either a deficit in *representing* phonetic sounds and/or a deficit in *access* to and *manipulation* of those sounds (e.g., for mapping phonemes to print). Previously, a review of behavioral studies of dyslexia concluded that phonetic representations are intact, but access to those representations may be impaired [41]. Recently, a neuroimaging study with adults found that phonetic representations, as measured by multivoxel pattern analysis of activations in bilateral auditory cortices, were intact in dyslexia, but that functional and structural (DTI) connectivity between auditory cortices and left inferior frontal gyrus was reduced [42]. These findings favor the interpretation of dyslexia as being characterized by weakness in access to otherwise intact phonetic representations. Consistent with this conclusion is the finding that children with dyslexia exhibited reduced prefrontal activation when engaging in an auditory PA task, but no difference in temporal-lobe activation, as compared with both age- and ability-matched children [43].

Brain Basis of Rapid Automatized Naming (RAN) Deficits

RAN has been partially dissociated from PA as a skill essential for learning to read [12, 13], but now there is evidence for a neurobiological distinction between the two skills. A large structural MRI study of typical adult readers of Chinese found that phonological decoding ability was related to gray matter volume in left perisylvian cortex, whereas naming speed was related to volume in a more distributed network across all four lobes [44]. Further, functional activation to a PA task differed among groups of children with PA and RAN deficits, as predicted by the double deficit hypothesis. Activation in left inferior parietal lobule showed a gradient associated with PA ability, whereas activation in right cerebellar lobule VI showed a gradient with RAN ability [45].

Brain Basis of Reading Fluency Deficits

For older children with dyslexia who must read longer texts, slow reading is a major problem. Both the psychological and brain bases of reduced fluency for connected text, such as sentences and paragraphs, have been poorly understood relative to the many studies focusing on single-word reading. Two studies, however, examined reading fluency directly in dyslexia during fMRI by presenting sentences word-by-word at varying rates and testing comprehension, but the two studies reported disparate results [46, 47]. Both studies reported that more rapid reading resulted in greater activation of left fusiform cortex in the VWFA region. One study reported that children with dyslexia exhibited reduced activation related to fluency exclusively in left fusiform gyrus despite no significant differences in comprehension accuracy [46]. The other study reported that adults with dyslexia exhibited disproportionately worse comprehension accuracy and lesser activation in left prefrontal and superior temporal regions as a function of reading speed, but found no group difference in the VWFA region [47]. Although the populations and outcomes of the two studies differed, they have initiated the analysis of the brain basis of impaired reading fluency in dyslexia.

Brain Basis of Basic Perceptual Processes

Neuroimaging findings have reported neural correlates of atypical basic perceptual processes in dyslexia. Successful parsing of the speech signal depends on the ability of left auditory cortex to selectively amplify phonemic information in the 30 Hz (low gamma) range [48]. MEG revealed reduced entrainment, or synchronization of neural firing, to the 30 Hz frequency range in dyslexia, as well as reduced left-hemisphere specialization for such oscillations [49, 50]. These differences may impede the efficient transfer of acoustic information into more abstract phonemic representations. Individuals with dyslexia also exhibited reduced neural entrainment in response to linguistic stimuli [51, 52], differences in EEG signals that reflect integration of auditory and visual stimuli [53], and greater variability of auditory brainstem responses to speech sounds [54].

An advantage of understanding dyslexia in terms of basic perceptual processes is that the neural mechanisms of those processes can be studied in animals. Animal research has linked dyslexia-associated genes such as KIAA0319 with atypical neural migration [55] and impaired speech sound discrimination [55, 56], suggesting that the mechanism by which cortical abnormalities result in behavioral deficits is through the disruption of synchronous firing in response to oral language [57]. In humans, variation in KIAA0319 and two other dyslexia susceptibility genes has been associated with variation in left-hemisphere white matter and reading skill [58]. Such research may integrate findings from the genetic, cellular, cognitive, and behavioral levels in understanding the core deficits in dyslexia.

6. Conclusion

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Progress in understanding the cognitive neuroscience of dyslexia may be approaching translation from basic research to intervention for children who will struggle to read. Remediation is known to be most effective in beginning readers, so early and accurate identification may promote effective intervention for children before they experience prolonged reading failure. Neuroimaging has identified biomarkers that enhance or outperform current behavioral measures in predicting long-term reading outcomes [59–63]. With further progress in understanding specific components of dyslexia (e.g., PA, RAN, fluency) it may also become possible to develop personalized interventions that target the specific patterns of weaknesses that undermine learning to read in individual children.

Highlights

- Neuroimaging is identifying brain differences related to causes of dyslexia.
- Brain bases of specific aspects of dyslexia have been better identified.
- Genetics may bridge study of neural mechanisms to dyslexia in humans.

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Footnotes

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