Iran J Psychiatry 2023; 18: 4: 503-509

Short Communication

An Overview on Electrophysiological and Neuroimaging Findings in Dyslexia

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Abstract

Objective: Dyslexia is a prevalent neurodevelopmental condition that is characterized by inaccurate and slow word recognition. This article reviews neural correlates of dyslexia from both electrophysiological and neuroimaging studies.
Method: In this brief review, we provide electrophysiological and neuroimaging evidence from electroencephalogram (EEG) and magnetic resonance imaging (MRI) studies in dyslexia to understand functional and structural brain changes in this condition.
Results: In both electrophysiological and neuroimaging studies, the most frequently reported functional impairments in dyslexia include aberrant activation of the left hemisphere occipito-temporal cortex (OTC), temporo-parietal cortex (TPC), inferior frontal gyrus (IFG), and cerebellar areas. EEG studies have mostly highlighted the important role of lower frequency bands in dyslexia, especially theta waves. Furthermore, neuroimaging studies have suggested that dyslexia is related to functional and structural impairments in the left hemisphere regions associated with reading and language, including reduced grey matter volume in the left TPC, decreased white matter connectivity between reading networks, and hypo-activation of the left OTC and TPC. In addition, neural evidence from pre-reading children and infants at risk for dyslexia show that there are abnormalities in the dyslexic brain before learning to read begins.
Conclusion: Advances in comprehending the neural correlates of dyslexia could bring closer translation from basic to clinical neuroscience and effective rehabilitation for individuals who struggle to read. However, neuroscience still has great potential for clinical translation that requires further research.

Key words: *Brain Waves; Dyslexia; Electroencephalogram (EEG); Neuroimaging; Neuropathology*

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Article Information:
Received Date: 2023/05/03, Revised Date: 2023/05/31, Accepted Date: 2023/06/08

Over the past two decades, a large body of research has been devoted to the neurobiology and neural correlates of developmental dyslexia. Dyslexia is an inability to read in spite of adequate education, normal intelligence, and the absence of obvious neurological or sensory impairment (1). It is a very common reading problem that affects 3-7% of people worldwide (2). In addition, about 5-17% of children are affected by developmental dyslexia (3). Dyslexic boys often receive more clinical attention than dyslexic girls, as they apparently suffer from a larger rate of externalizing disorders such as attention deficit hyperactivity disorder (4). Affected children have been shown to be at risk for various persistent psychosocial and educational problems, such as stress, anxiety, and deficits in cognitive processing (5). Weakness in phonological awareness, rapid automatic naming, and reading fluency are considered psychological correlates of dyslexia (6, 7). Although its neurobiological bases are not fully understood, dyslexia has been shown to reflect abnormal patterns of activations in some brain areas, including left occipito-temporal cortex (OTC), temporo-parietal cortex (TPC), and frontal area (8). Various neuroimaging (e.g., functional magnetic resonance imaging and diffusion tensor imaging) and electrophysiological (e.g., electroencephalography and magnetoencephalography) techniques have been utilized to explore the neural underpinnings of reading problems in individuals with dyslexia (9). Despite the progress made in understanding the neural substrates of this neurodevelopmental disorder in recent years, there are still contradictions in this field, and strong evidence is needed on the neural mechanisms associated with dyslexia symptoms. Therefore, in this short review, we attempted to provide a comprehensive model for the neural correlates of dyslexia.

Electrophysiological Evidence

Electroencephalography (EEG) is a highly popular electrophysiological modality that reflects the direct electrical activity of cortical neurons (10). The spectral power of well-known EEG frequency bands is a simple and rich source of information obtained from the quantitative frequency analysis of EEG signals (11, 12). It has been shown that these standard frequency bands reveal important concepts and meanings of various cognitive, perceptual, and sensorimotor functions (13). Therefore, in the last three decades, many studies have investigated various EEG indices in dyslexia. In general, individuals with dyslexia show aberrant increases in delta (0.5-4 Hz) and theta (4-8 Hz) activities in different areas of the cerebral cortex, including frontal, central, temporal, and parietal lobes, compared to typical subjects (14-16). In addition, the aberrant reduction of alpha activity (8-12 Hz) in the frontal, temporal, and parietal lobes has been consistently reported in dyslexic subjects (17, 18) (Figure 1). These abnormalities appear to be located mostly in the TPC, which is believed to be the decoding center of the reading network in the brain to link the sounds and letters within words (19). Furthermore, it is important to consider aberrant activity in EEG frequency bands, where delta and theta frequencies are associated with prosodic and syllabic perceptions, respectively (20, 21). A major neurobiological hypothesis assumes an important role for lower frequency bands, especially theta waves. Disruption of theta activity is believed to alter the encoding of syllables with lower temporal modulation traces and even multisensory processing, with implications for attention and visual-auditory integration (22). Moreover, some studies have linked the reduction of alpha activity to phonological processing problems in dyslexia. It should be noted that alpha waves are the dominant oscillations in the human brain that play an active role in information processing (23).

In addition to the study of the power of frequency bands, the characteristic of EEG connectivity in different frequency bands has also been investigated in electrophysiological studies. These studies suggest that the global organization of functional networks may be compromised in dyslexia (24). Indeed, reading is a complicated skill controlled through a distributed network of language and visual-related areas, and disruption of connectivity in this network is associated with dyslexia (25). EEG studies have indicated reduced connectivity between occipital to inferior-temporal areas, as well as between left to right temporal regions, in dyslexic children (26, 27). Additionally, some studies have shown reduced long-range connectivity between distant cortical sites within and beyond the reading network in dyslexia, particularly in the theta band (28). Deficient functional connectivity in the superior temporal gyri, middle occipital gyrus, OTC, and angular gyri in the theta frequency band has often been reported in dyslexic children compared to typically reading children (29-31). Moreover, a global network deficiency in the beta band has been reported in dyslexic children (19). On the other hand, as a compensatory neural mechanism, increased connectivity from the left central to right inferior-temporal and occipital regions was found in severely dysfluent children during a task involving visual word and false font processing (27).

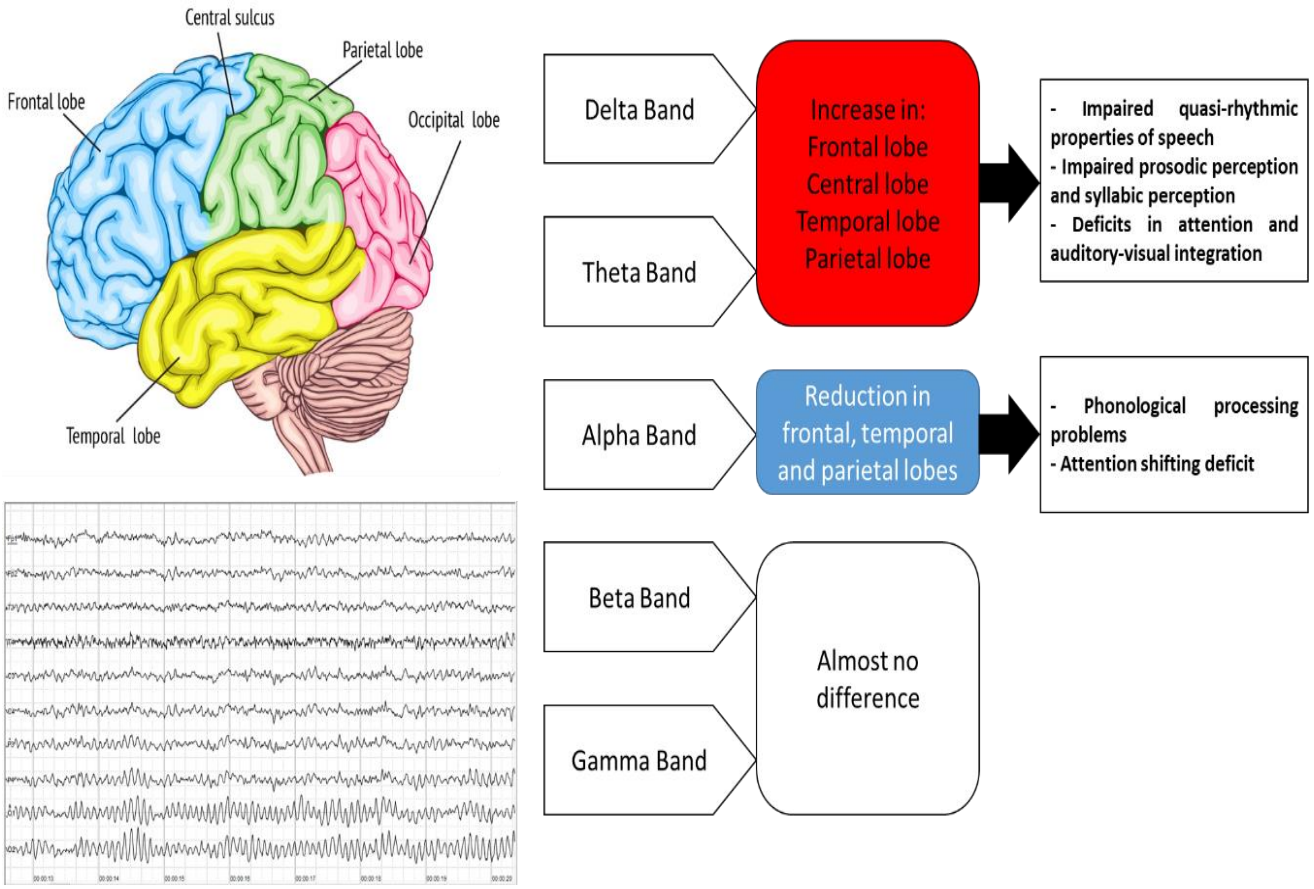

Figure 1. Current Evidence from Electrophysiological Studies for the Neural Correlates of Dyslexia

Neuroimaging Evidence

Dyslexia has been proven to be related to functional and structural impairments in the reading and language regions of the left hemisphere (32). Structural neuroimaging studies have consistently demonstrated reduced grey matter volume in the left TPC (33, 34). Moreover, decreased white matter connectivity between reading networks is another frequently reported finding that has been shown to be linked to orthographic and phonological deficits in dyslexia (35, 36). These structural abnormalities have been suggested to be caused by failures of neuronal migration throughout the language and reading networks (37). In addition, the most frequently reported functional impairments in dyslexia include aberrant activation of the left hemisphere OTC, TPC, and inferior frontal gyrus (IFG) areas (Figure 2). Hypo-activation and functional disorganization of the left OTC in response to pseudo-words and words is a consistent and persistent abnormality among dyslexic children and adults, indicating that dyslexia might be related to an early failure in recruiting this brain area to perform reading tasks during development (38). Aberrant OTC activation has also been shown to be linked to rapid automatic naming and deficits in letter and word recognition in dyslexia (6). Furthermore, the hypo-activation of the left TPC during phonological processing tasks among dyslexic children and adults is a consistent finding in dyslexia (23). Activation in TPC is normally increased as a function of greater efforts in mapping printed letters to phonology. However, dyslexic people have no raised activation in this area during such increased efforts (9). As opposed to the hypo-activation noted in the left OTC and TPC, research frequently reports the hyper-activation of the IFG in dyslexia. Typical readers have an age-dependent decrease in IFG activation, whereas dyslexics show increased activation with age, which may be a neural compensatory mechanism (39). However, this is not a consistent finding in dyslexia, and some studies have reported conflicting results; some studies have found no difference in IFG activation between younger individuals with and without dyslexia, while others have reported the hypo-activation of this brain region in dyslexics (40). Furthermore, cerebellar abnormalities are often found in neuroimaging research of dyslexia. The cerebellum is structurally and functionally connected to the frontal cortex and TPC, which are critical for verbal working memory, phonological processing, and semantics (41). The cerebellum is believed to play a key role in timing functions and procedural learning that are essential for fluent reading and various language functions. Hypo-activation of language-engaged cerebellar areas is a consistent finding in dyslexia (42).

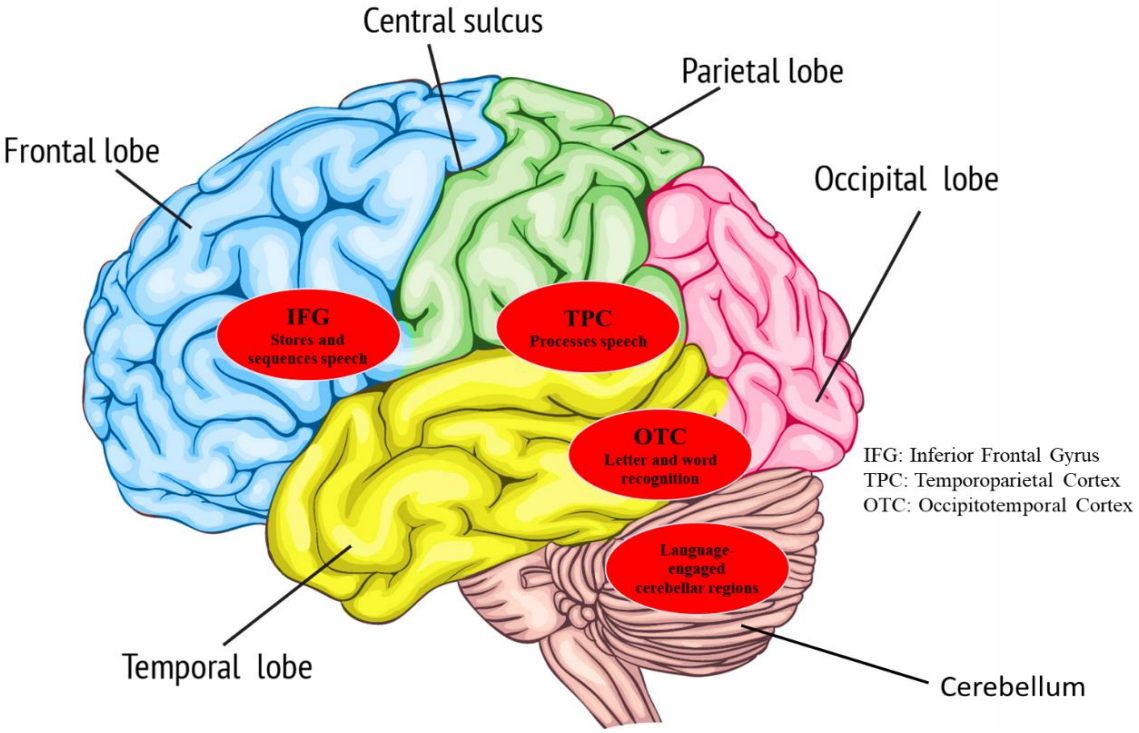

Figure 2. Current Evidence from Neuroimaging Studies for the Neural Correlates of Dyslexia

At-Risk Infants

There is still no consensus among researchers as to whether these neurological abnormalities are the result or cause of reading deficits in dyslexia. However, neural evidence from pre-reading children and infants at risk for dyslexia indicate that there are abnormalities in the dyslexic brain before learning to read begins. For instance, infants at risk of dyslexia show abnormalities in the left arcuate fasciculus structure, which is related to dyslexia in both adults and children (35). Further, six-month-old infants at a familial risk of dyslexia show abnormalities in early neural responses within the left hemisphere to letter sounds (43). At-risk pre-reading children show both functional and structural deficits in the left hemisphere reading regions, including the OTC and TPC, in line with abnormalities reported in children and adults with dyslexia (44). Therefore, neural evidence may play an important role in the early diagnosis of dyslexia in the future.

Discussion

Dyslexia is a multifactorial disorder related to reading problems in spite of enough instruction. Neuroimaging and electrophysiological studies suggest that this disorder is linked to abnormalities in brain networks involved in normal reading development (45). Neural signatures that can be indicative of future reading difficulties are present from early childhood (46). In fact, we can identify reading difficulties before a child struggles to learn to read and falls behind. Therefore, timely appropriate interventions can alleviate the symptoms of the disorder and promote the normalization of the neural abnormalities (both structurally and functionally) through remediation-related brain plasticity (47, 48). Previous studies have shown increased activation in the TPC and IFG regions along with improvements in reading functions such as fluency and phonological awareness as a result of remediation after a treatment period (49, 50). Moreover, reading interventions have led to increased white matter connectivity and grey matter volume in dyslexic individuals (51, 52). Interestingly, such neuroplasticity is not limited to children and has also been observed in adults (51).

The literature demonstrates that computational and translational neuroscience can effectively help us gain more insights into complex disorders such as dyslexia and manage affected individuals (53). While dyslexia is currently addressed through different speech therapy techniques, understanding the neural correlates of dyslexia can provide new therapeutic targets to achieve better outcomes. In other words, considering the neural abnormalities in people with dyslexia, therapists can benefit from different brain stimulation techniques such as transcranial magnetic stimulation as adjuvant treatments to facilitate brain plasticity towards normal status.

Limitations

Here, we attempted to provide a brief comprehensive review of the neural correlates of dyslexia. However, not conducting a systematic review to retrieve all relevant studies is the main limitation of our study.

Future Directions

First of all, we need well-designed longitudinal studies with sufficient sample sizes to examine developmental changes in the neural correlates of dyslexia. The evidence from such studies can contribute to better planning for the effective management of this disorder. Second, neural signatures can be explored to predict differential treatment response in dyslexic individuals. In other words, by using neural data, it is possible to predict with relatively good accuracy whether patients will respond to a specific intervention or not. Such research can contribute to optimizing dyslexia intervention methods and personalizing treatment approaches. In addition, due to the highly heritable nature of dyslexia (9), it is suggested to conduct brain genetic research to investigate the relationship between brain functional and structural findings and dyslexia candidate genes.

Conclusion

Advancements in comprehending the neural correlates of dyslexia could bring closer translation from basic to clinical neuroscience and effective rehabilitation for individuals who struggle to read. Both electrophysiological and neuroimaging studies with their unique properties (high temporal and spatial resolution, respectively) could provide valuable neural evidence for dyslexia. This evidence is present from infancy and early childhood, allowing for the early identification of children at risk for dyslexia, when remediation is known to be most effective. However, neuroscience still has great potential for clinical translation, which requires further research.

Acknowledgement

The authors thank the institutions where they work for encouraging the development of research.

Conflict of Interest

None.

References

1. Hedenius M, Persson J. Neural correlates of sequence learning in children with developmental dyslexia. Hum Brain Mapp. 2022;43(11):3559-76.
2. Wagner RK, Zirps FA, Edwards AA, Wood SG, Joyner RE, Becker BJ, et al. The Prevalence of Dyslexia: A New Approach to Its Estimation. J Learn Disabil. 2020;53(5):354-65.
3. Surushkina SY, Yakovenko E, Chutko L, Didur M. Dyslexia as a multifactorial disorder. Neurosci Behav. 2021;51:303-8.
4. McGrath LM, Stoodley CJ. Are there shared neural correlates between dyslexia and ADHD? A meta-analysis of voxel-based morphometry studies. J Neurodev Disord. 2019;11(1):31.
5. Boyes ME, Leitão S, Claessen M, Badcock NA, Nayton M. Correlates of externalising and internalising problems in children with dyslexia: An analysis of data from clinical casefiles. Aust Psychol. 2020;55(1):62-72.
6. Norton ES, Wolf M. Rapid automatized naming (RAN) and reading fluency: implications for understanding and treatment of reading disabilities. Annu Rev Psychol. 2012;63:427-52.
7. Melby-Lervåg M, Lyster SA, Hulme C. Phonological skills and their role in learning to read: a meta-analytic review. Psychol Bull. 2012;138(2):322-52.
8. Norton ES, Beach SD, Gabrieli JD. Neurobiology of dyslexia. Curr Opin Neurobiol. 2015;30:73-8.
9. D'Mello AM, Gabrieli JDE. Cognitive Neuroscience of Dyslexia. Lang Speech Hear Serv Sch. 2018;49(4):798-809.
10. Mohammadi MR, Khaleghi A, Nasrabadi AM, Rafieivand S, Begol M, Zarafshan H. EEG classification of ADHD and normal children using non-linear features and neural network. Biomed Eng Lett. 2016;6:66-73.
11. Khaleghi A, Sheikhani A, Mohammadi MR, Moti Nasrabadi A. Evaluation of Cerebral Cortex Function in Clients with Bipolar Mood Disorder I (BMD I) Compared With BMD II Using QEEG Analysis. Iran J Psychiatry. 2015;10(2):93-9.
12. Moeini M, Khaleghi A, Amiri N, Niknam Z. Quantitative electroencephalogram (QEEG) Spectrum Analysis of Patients with Schizoaffective Disorder Compared to Normal Subjects. Iran J Psychiatry. 2014;9(4):216-21.
13. Moeini M, Khaleghi A, Mohammadi MR. Characteristics of Alpha Band Frequency in Adolescents with Bipolar II Disorder: A Resting-State QEEG Study. Iran J Psychiatry. 2015;10(1):8-12.
14. Bosch-Bayard J, Peluso V, Galan L, Valdes Sosa P, Chiarenza GA. Clinical and Electrophysiological Differences between Subjects with Dysphonetic Dyslexia and Non-Specific Reading Delay. Brain Sci. 2018;8(9).
15. Mahmoodin Z, Mansor W, Khuan L, Mohamad N, Amirin S. Feature extraction of electroencephalogram signal generated from writing in dyslexic children using daubechies wavelet transform. J Teknol. 2016;78(6-8):119-25.
16. Mahmoodin Z, Lee KY, Mansor W, Zainuddin AZA. Support vector machine with theta-beta band power features generated from writing of dyslexic children. Int J Integr Eng. 2019;11(3).
17. Babiloni C, Stella G, Buffo P, Vecchio F, Onorati P, Muratori C, et al. Cortical sources of resting state EEG rhythms are abnormal in dyslexic children. Clin Neurophysiol. 2012;123(12):2384-91.
18. Papagiannopoulou EA, Lagopoulos J. Resting State EEG Hemispheric Power Asymmetry in Children with Dyslexia. Front Pediatr. 2016;4:11.
19. Xue H, Wang Z, Tan Y, Yang H, Fu W, Xue L, et al. Resting-state EEG reveals global network deficiency in dyslexic children. Neuropsychologia. 2020;138:107343.

20. Ghitza O, Greenberg S. On the possible role of brain rhythms in speech perception: intelligibility of time-compressed speech with periodic and aperiodic insertions of silence. Phonetica. 2009;66(1-2):113-26.
21. Poeppel D, Idsardi WJ, van Wassenhove V. Speech perception at the interface of neurobiology and linguistics. Philos Trans R Soc Lond B Biol Sci. 2008;363(1493):1071-86.
22. Goswami U. A temporal sampling framework for developmental dyslexia. Trends Cogn Sci. 2011;15(1):3-10.
23. Cainelli E, Vedovelli L, Carretti B, Bisiacchi P. EEG correlates of developmental dyslexia: a systematic review. Ann Dyslexia. 2023;73(2):184-213.
24. Seshadri NPG, Geethanjali B, Singh BK. EEG based functional brain networks analysis in dyslexic children during arithmetic task. Cogn Neurodyn. 2022;16(5):1013-28.
25. Yu X, Ferradal S, Dunstan J, Carruthers C, Sanfilippo J, Zuk J, et al. Patterns of Neural Functional Connectivity in Infants at Familial Risk of Developmental Dyslexia. JAMA Netw Open. 2022;5(10):e2236102.
26. Martinez-Murcia FJ, Ortiz A, Gorriz JM, Ramirez J, Lopez-Abarejo PJ, Lopez-Zamora M, et al. EEG Connectivity Analysis Using Denoising Autoencoders for the Detection of Dyslexia. Int J Neural Syst. 2020;30(7):2050037.
27. Žarić G, Correia JM, Fraga González G, Tijms J, van der Molen MW, Blomert L, et al. Altered patterns of directed connectivity within the reading network of dyslexic children and their relation to reading dysfluency. Dev Cogn Neurosci. 2017;23:1-13.
28. Fraga González G, Van der Molen MJW, Žarić G, Bonte M, Tijms J, Blomert L, et al. Graph analysis of EEG resting state functional networks in dyslexic readers. Clin Neurophysiol. 2016;127(9):3165-75.
29. Taskov T, Dushanova J. Functional connectivity in developmental dyslexia during speed discrimination. Symmetry. 2021;13(5):749.
30. Dushanova JA, Tsokov SA. Small-world EEG network analysis of functional connectivity in developmental dyslexia after visual training intervention. J Integr Neurosci. 2020;19(4):601-18.
31. Dushanova JA, Tsokov SA. Altered electroencephalographic networks in developmental dyslexia after remedial training: a prospective case-control study. Neural Regen Res. 2021;16(4):734-43.
32. Peterson RL, Pennington BF. Developmental dyslexia. Lancet. 2012;379(9830):1997-2007.
33. Kronbichler M, Wimmer H, Staffen W, Hutzler F, Mair A, Ladurner G. Developmental dyslexia: gray matter abnormalities in the occipitotemporal cortex. Hum Brain Mapp. 2008;29(5):613-25.
34. Hoeft F, Ueno T, Reiss AL, Meyler A, Whitfield-Gabrieli S, Glover GH, et al. Prediction of children's reading skills using behavioral, functional, and structural neuroimaging measures. Behav Neurosci. 2007;121(3):602-13.
35. Langer N, Peysakhovich B, Zuk J, Drottar M, Sliva DD, Smith S, et al. White Matter Alterations in Infants at Risk for Developmental Dyslexia. Cereb Cortex. 2017;27(2):1027-36.
36. Wang Y, Mauer MV, Raney T, Peysakhovich B, Becker BLC, Sliva DD, et al. Development of Tract-Specific White Matter Pathways During Early Reading Development in At-Risk Children and Typical Controls. Cereb Cortex. 2017;27(4):2469-85.
37. Myers CA, Vandermosten M, Farris EA, Hancock R, Gimenez P, Black JM, et al. White matter morphometric changes uniquely predict children's reading acquisition. Psychol Sci. 2014;25(10):1870-83.
38. Richlan F, Kronbichler M, Wimmer H. Meta-analyzing brain dysfunctions in dyslexic children and adults. Neuroimage. 2011;56(3):1735-42.
39. Price CJ. A review and synthesis of the first 20 years of PET and fMRI studies of heard speech, spoken language and reading. Neuroimage. 2012;62(2):816-47.
40. Richlan F, Kronbichler M, Wimmer H. Functional abnormalities in the dyslexic brain: a quantitative meta-analysis of neuroimaging studies. Hum Brain Mapp. 2009;30(10):3299-308.
41. Stoodley CJ. The cerebellum and cognition: evidence from functional imaging studies. Cerebellum. 2012;11(2):352-65.
42. Stoodley CJ. The Cerebellum and Neurodevelopmental Disorders. Cerebellum. 2016;15(1):34-7.
43. Leppänen PH, Richardson U, Pihko E, Eklund KM, Guttorm TK, Aro M, et al. Brain responses to changes in speech sound durations differ between infants with and without familial risk for dyslexia. Dev Neuropsychol. 2002;22(1):407-22.
44. Raschle NM, Chang M, Gaab N. Structural brain alterations associated with dyslexia predate reading onset. Neuroimage. 2011;57(3):742-9.
45. Carrasco A, Carrasco KD. The use of neuronal response signals as early biomarkers of dyslexia. Adv Neurodev. 2022;6(4):389-96.
46. Kere J. The molecular genetics and neurobiology of developmental dyslexia as model of a complex phenotype. Biochem Biophys Res Commun. 2014;452(2):236-43.
47. Van Hirtum T, Ghesquière P, Wouters J. A Bridge over Troubled Listening: Improving Speech-in-Noise Perception by Children with Dyslexia. J Assoc Res Otolaryngol. 2021;22(4):465-80.
48. Meyler A, Keller TA, Cherkassky VL, Gabrieli JD, Just MA. Modifying the brain activation of poor readers during sentence comprehension with extended remedial instruction: a longitudinal study of neuroplasticity. Neuropsychologia. 2008;46(10):2580-92.
49. Reetzke R, Xie Z, Chandrasekaran B. Neurobiology of literacy and reading disorders. The Frequency-Following Response: A Window into Human Communication. 2017:251-66.
50. Hoeft F, McCandliss BD, Black JM, Gantman A, Zakerani N, Hulme C, et al. Neural systems

predicting long-term outcome in dyslexia. Proc Natl Acad Sci U S A. 2011;108(1):361-6.
51. Krafnick AJ, Flowers DL, Napoliello EM, Eden GF. Gray matter volume changes following reading intervention in dyslexic children. Neuroimage. 2011;57(3):733-41.
52. Keller TA, Just MA. Altering cortical connectivity: remediation-induced changes in the white matter of poor readers. Neuron. 2009;64(5):624-31.
53. Khaleghi A, Mohammadi MR, Shahi K, Nasrabadi AM. Computational Neuroscience Approach to Psychiatry: A Review on Theory-driven Approaches. Clin Psychopharmacol Neurosci. 2022;20(1):26-36.