

# Dyslexia – A Correlational Study Between Brain and Hypothalamus That How They Are Affected in Dyslexic Child

Manisha Agrahari<sup>1,\*</sup>, Raj Kamal Sharma<sup>2</sup>

## Abstract

*The 'dyslexia dispute' is tenacious. A crucial component of the media argument is the belief that dyslexia does not exist, which has been popularized by several outspoken commentators. For them, dyslexia are the creation of excessively anxious parents, backed up by a clique of private educational psychologists eager to deliver a diagnostic – for a charge – even when no Conditional exists. Critiques of dyslexia can also be found in academic settings, particularly in psychology. The main point in these is that the name 'dyslexia' is unhelpful because it is more of an emotive word used to attract funds than a clearly established scientific disease. The debate surrounding dyslexia continues, with emerging research revealing neurobiological changes influenced by educational therapies. Dyslexia is a lifelong neurodevelopmental disorder that typically arises early and is heritable. Its prevalence is challenging to determine due to the varied reading abilities and profiles of poor readers, which include both dyslexic individuals and those who struggle in specific academic areas. While dyslexia cannot be cured, targeted educational strategies can help improve reading skills. Diagnosis can occur at any age, with differing tests for children and adults. Future research should explore the relationship between dyslexia, the hypothalamus, and socioemotional factors, including its impact on hormonal balance and mental health.*

**Keywords:** Dyslexia, dyslexia dispute, hormonal balance, mental health, neurobiological changes, socioemotional factors

## INTRODUCTION

The incapacity to learn language person receives. Although the mind is often average and highly “smart,” it has strengths outside of the linguistic domain. Dyslexia is characterised by difficulties with accurate and fluent word reading and poor spelling and decoding abilities that do not progress as expected with the provision of well-intentioned and targeted intervention. These discrepancies may have been partially caused by the emphasis on various writing systems used in various research, according to a recent meta-analysis. The hypothalamus is a tiny structure deep inside the brain, making

### \*Author for Correspondence

Manisha Agrahari  
E-mail: manisha\_agrahari@rediffmail.com

<sup>1</sup>Assistant Professor, Department of Nursing, St. Stephene's College of Nursing Supaul, Pipra, Bihar, India

<sup>2</sup>Associate Professor, Department of Morphology, Karaganda Medical University, Karaganda, Kazakhstan

Received Date: October 03, 2024

Accepted Date: October 16, 2024

Published Date: November 08, 2024

**Citation:** Manisha Agrahari, Raj Kamal Sharma. Dyslexia – A Correlational Study Between Brain and Hypothalamus That How They Are Affected in Dyslexic Child. Research & Reviews: A Journal of Neuroscience. 2024; 14(3): 28–36p.

it prone to signal drop-out in functional magnetic resonance imaging (fMRI) and challenging to examine at voxel sizes routinely utilized in fMRI (~30 voxels at 3 mm<sup>3</sup>). Furthermore, the hypothalamus is not homogeneous, but rather comprises a complex substructure of more than 10 nuclei [1]. For instance, in temporoparietal, occipitotemporal and cerebellar cortices in alphabetic languages, DD demonstrated reduced gray matter volume (GMV) in comparison to TD controls but in morpho-syllabic languages, the GMV of the left inferior frontal gyrus was more affected. Specifically, modifications to the temporo-parietal junction may provide a possible

---

indicator of pre-readers' susceptibility to dyslexia. Stanovich was among the first to criticize the discrepancy definition of dyslexia (1991). Stanovich opposed this strategy based on two major points. To begin with, reading improves vocabulary and language abilities. An expression like “Ns source neurons and Nt target neurons are connected randomly with connection probability p” can be used to characterize the connectivity structure of a neural network model. An algorithm that links each potential pair of source and destination neurons with probability after considering each pair precisely once might be one way to understand this statement. Alternative readings of the same sentence could permit numerous connections between the same pair of neurons, apply a non-uniform connection probability to distinct pairs of neurons, or make additional assumptions about the distribution of incoming and outgoing connections for each neuron. Decisions significantly influence network dynamics beyond just structure. To illustrate this, we simulate two balanced recurrent networks. Abnormalities in white matter related to dyslexia extend beyond the reading network and thalamocortical projections, affecting the limbic and motor systems, particularly in the cerebellum and corona radiata. The arcuate fasciculus plays a crucial role in dyslexia, linking key language areas, such as Wernicke's and Broca's regions, the visual word form area, and the frontal lobe. Dyslexia affects 5–17% of children and is marked by slow, effortful reading stemming from phonological processing difficulties. Traditional assessments include tests for working memory and reading abilities, and digital tools have made various screening tests widely accessible. Studies show reduced brain activity in critical language regions for children with dyslexia, though reading patterns can resemble those of children with normal vision [2, 3].

### **INTRINSIC FACTORS UNDERLYING HYPOTHALAMIC REGULATION AND NEUROCHEMICAL MODULATION**

Numerous internal and external cues that either independently or in concert with one another influence the orchestrations of hypothalamic functioning, as evidenced by decades of study. Consequently, to get mechanistic insights into the functioning of hypothalamic neurons, a deeper exploration of the intricate dynamics underlying the interplay between extrinsic and intrinsic inputs is necessary. Neurotransmitters, hormones, circadian cycles, metabolic status, genetic susceptibility, feedback mechanisms, neuropeptides, and neural circuits are examples of intrinsic factors. GABA, serotonin, and dopamine are examples of neurotransmitters that have been well studied and shown to be important regulators of hypothalamic processes. For instance, Saper and associates discovered that the arousal system is inhibited, and sleep is promoted when GABA is released from inhibitory neurons in the ventrolateral preoptic nucleus. An essential element influencing hypothalamus function is hormone regulation. As a major center for hormone regulation, the hypothalamus secretes, releases, and inhibits substances that regulate the pituitary gland's production of hormones. The intricate workings of complex hormonal networks regulate important physiological functions like as development, metabolism, stress response, and reproduction. Adrenocorticotrophic hormone (ACTH) and cortisol levels that are excessively elevated have been linked to significant abnormalities in the regulation of sleep, specifically regarding the hypothalamic–pituitary–adrenal (HPA) axis. These alterations have been linked to the emergence of persistent insomnia and elevated central nervous system arousal. Six Furthermore, the corticotropin-releasing factor (CRF) neurotransmission activity in the lateral hypothalamus is highly significant in controlling the tachycardic response, or accelerated heart rate. Neuropeptides assume a pivotal role as intrinsic determinants influencing the intricate activities of the hypothalamus. The hypothalamus exerts its influence on the modulation of alcohol consumption via the actions of neuropeptides, which are specialised neurochemicals instrumental in shaping brain function [4, 5].

### **EXTERNAL FACTORS MODULATE HYPOTHALAMIC FUNCTIONS**

HCG and leptin are two examples of the hormones that the hypothalamus uses to control appetite and fullness. Positive effects on blood sugar normalization and weight loss are demonstrated by leptin receptor expression on POMC neurons in obese and diabetic mice. It is also known that changes in ambient temperature can affect hypothalamic activity, which in turn can cause reactions like sweating and shivering to control body temperature [6].

### **Reading Brain Systems**

Reading involves coordinated activity of both lower- and higher- order processing brain regions, making it difficult to classify as a single group acting independently. Neuroimaging studies identify unique brain regions for reading-related subskills, which influence dyslexics' fluent reading development. Research on unskilled and late scholars has made it possible for us to see firsthand how learning to read alters fundamental brain functions. A type of "cultural recycling" of brain regions that have evolved for many fundamental activities and may be specialized to carry out a skill that has been acquired culturally, like reading, has been proposed as the reason for this shift. Additionally, the developmental trajectory of certain brain activations associated with reading ability has been shown by longitudinal study. Furthermore, lesion studies looking at patients with brain injury leading to poor reading have validated the causal function of several of these brain systems in literacy [7].

### **Brain Correlates of Attentional and Visuo-Attentional Deficits in Dyslexia**

Compared to the "linguistic" subtype, the brain imaging results for this type of dyslexia are very different. They involve the parietal areas bilaterally, in areas that are known to be activated in different attentional tasks like a flanked-letter categorization task, which assesses visual attention mechanisms involved in multi-letter processing. The existence of a visuo-attentional mechanism in dyslexia has garnered occasional support, despite strong opposition from proponents of an exclusively phonological origin for the disorder. Additionally, several independent demonstrations have suggested that targeted attentional competency training may reverse brain abnormalities associated with dyslexia, possibly by strengthening white matter connectivity between posterior visual and frontal executive networks [8].

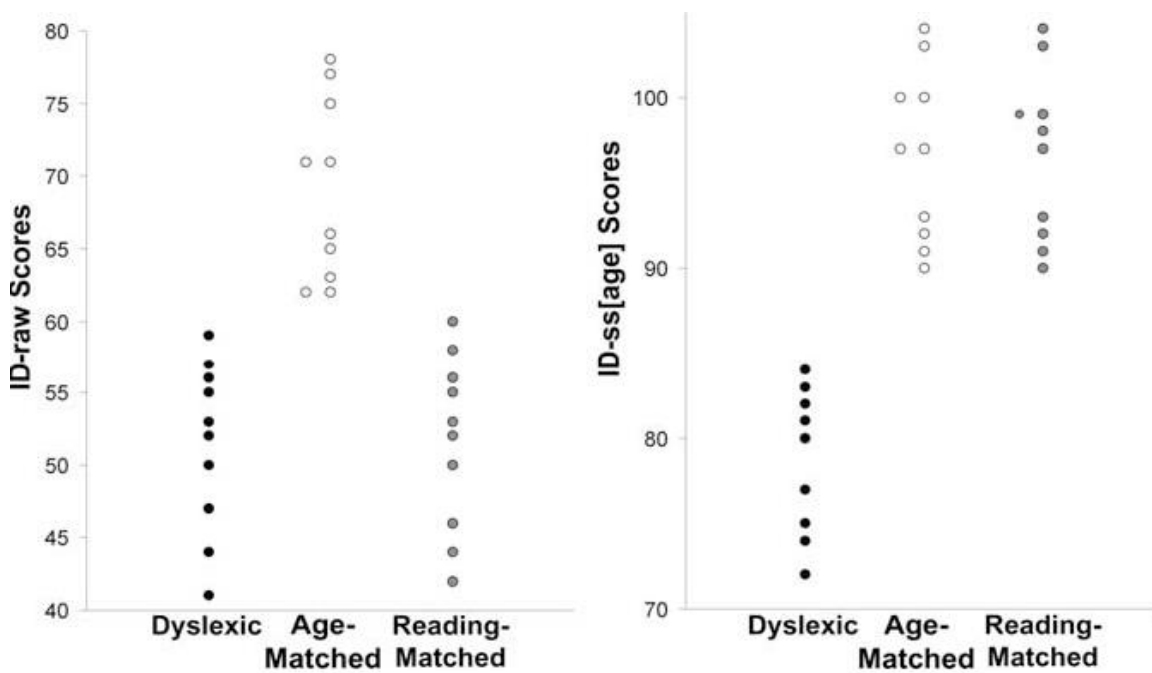
### **Children Who Read Normally and Those Who Are Dyslexic Compared**

We investigated the  $\beta$  estimates independently for the two circumstances for each of the six areas to see if activation differences were predominantly influenced by the rhyming or rest conditions. In the right parietotemporal region (R Par-temp;  $F(2,27) = 3.92$ ;  $p = 0.02$ ), there was a main effect of group only for  $\beta$  estimates of the resting baseline condition. This was predominantly due to the substantially greater  $\beta$  values in the age-matched ( $t(18) = 2.4$ ;  $p = 0.01$ ) and reading-matched ( $t(18) = 2.5$ ;  $p < 0.02$ ) groups when compared to the dyslexic group. In five of the six areas, the rhyming condition  $\beta$  projections revealed significant main effects (or a trend) of group [left inferior parietal lobule 1 (L Par-temp 1):  $F(2,27) = 3.00$ ,  $p = 0.03$ ; L Par-temp 2:  $F(2,27) = 3.44$ ,  $p = 0.02$ ; right superior frontal gyrus (R Frontal). Comparing the activation of each dyslexic participant with their individually chosen age- or reading-matched control revealed these group disparities in each of the six ROIs. For instance, compared to their age- or reading-matched peers, nine out of ten dyslexic children had reduced activity in the left parietotemporal area (L Par-temp 2). Nine to ten of the ten dyslexic children displayed less activation than their corresponding age-matched child across all six ROIs, and seven to nine of the ten dyslexic children, despite having matched reading proficiency (ID-raw score), displayed less activation than their corresponding reading-matched child. Binomial testing revealed that all six locations had a significant impact (i.e., substantially bigger proportion than 0.5) for the age-matched and dyslexic peers [9, 10]. The relationship between the resting baseline and rhyming condition activation was an unexpected discovery. In all six locations, the dyslexic group consistently had higher activation for the resting baseline than for the rhyming task ( $t(9) = 4.09 \sim 8.69$ ;  $p = 0.003 \sim < 0.001$ ). Just the age-matched group exhibited higher activation for the rhyming task; however, this only became significant in the left (L Par-temp 2,  $t(9) = 3.31$ ;  $p = 0.009$ ) and right parietotemporal regions ( $t(9) = 2.52$ ;  $p = 0.003$ ). The reading-matched group demonstrated similar activation in the rhyme task and resting baseline. Performing the same analysis without global scale produced findings that were comparable (Figure 1 and Table 1) [11].

### **Neural Enigma**

The medical phrase "neural angina" is not widely used. But it can just be a misunderstanding or mismatch of terminology. "Neural" describes nerves, but "angina" usually refers to chest discomfort brought on by diminished circulation to the heart. Please add more context so that I can correctly assist

you if you meant to refer to a condition connected to nerve pain or neuralgia, or if this is another phrase you’ve encountered. Numerous significant advances in neuroscience and psychiatry have been made possible by the ENIGMA Consortium. The ENIGMA Consortium developed consensus protocols for data quality control and outlier handling, standardized processes for extracting brain metrics (such as cortical thickness, cortical surface area, and subcortical volume) from raw neuroimaging data, and invented new meta-analytic techniques for the analysis of aggregated statistical data (Figures 2 & 3) [12].



**Figure 1.** Comparison of ID-Raw Scores and ID-ss(Age) Scores between Dyslexic, Age-Matched, and Reading-Matched groups.

**Table 1.** Brain regions with significant activation differences between age-matched controls and dyslexic participants.

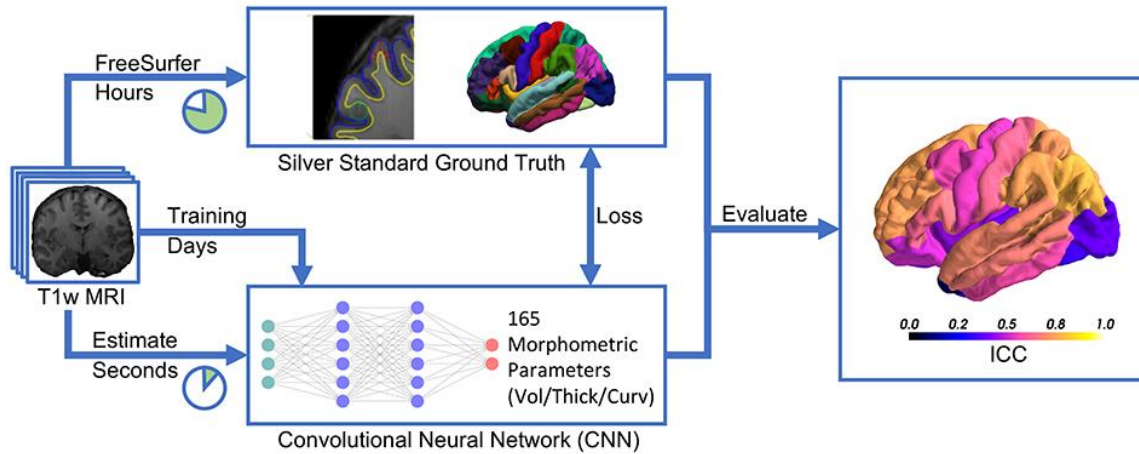
Region	Brodmann Area	Talairach Coordinates (x, y, z)	Z	p	Volume (ml)
Age-matched > dyslexic					
Frontal lobe					
L Middle frontal gyrus	8	-38, 19, 34	3.38	< 0.001	0.08
R Superior frontal gyrus	9	20, 41, 38	3.63	< 0.001	0.22
Parietal lobe					
L Inferior parietal lobule 1	40	-50, -49, 39	4.23	< 0.001	0.60
L Inferior parietal lobule 2	40	-36, -49, 37	4.11	< 0.001	0.41
R Inferior parietal lobule	39	40, -62, 44	5.48	< 0.001	2.68
Temporal lobe					
R Middle temporal gyrus	37	59, -47, -6	3.92	< 0.001	0.42
Dyslexic > age-matched	n/a	n/a	n/a	n/a	n/a

Note: ET, 10; p = 0.001, uncorrected.

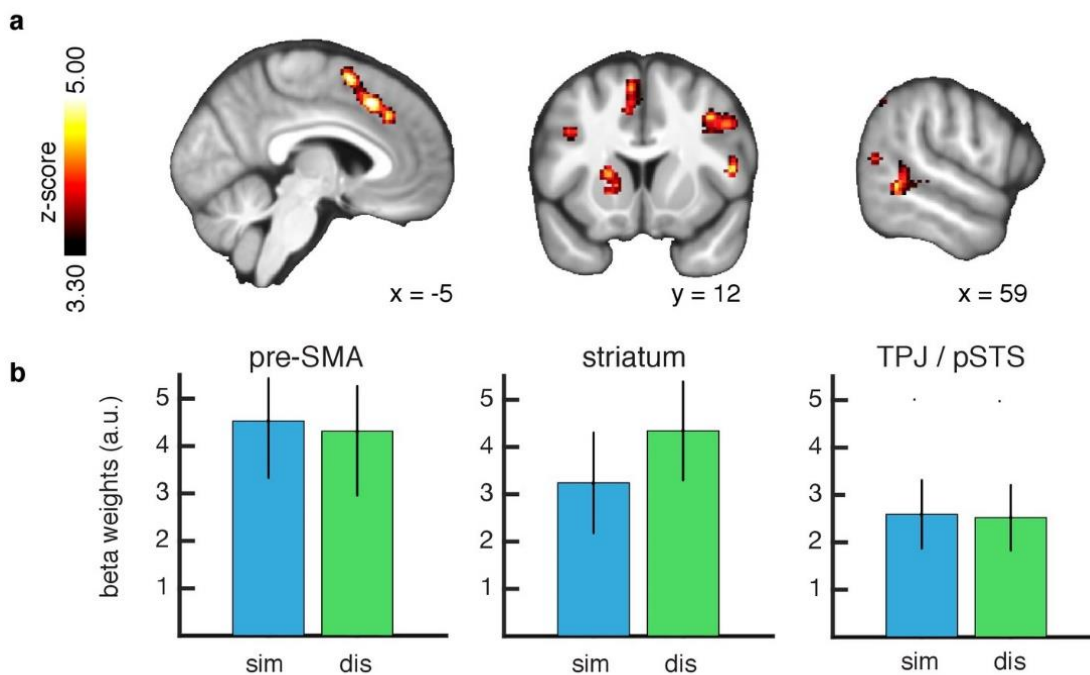
### The Neurological Underpinnings of Dyslexia: Exploring the Developmental Trajectories of the Dyslexic Brain

Dyslexia, a complex neurodevelopmental disorder, has long captivated scientific interest, as researchers work to unravel the intricate neurological underpinnings of this reading disability.

Numerous neuroimaging studies have illuminated the atypical brain activity and connectivity patterns observed in individuals with dyslexia, offering insights into the dynamic neural systems that shape the reading process (Figures 4 and 5) [13].



**Figure 2.** Workflow for estimating morphometric parameters from T1-weighted MRI using a CNN.

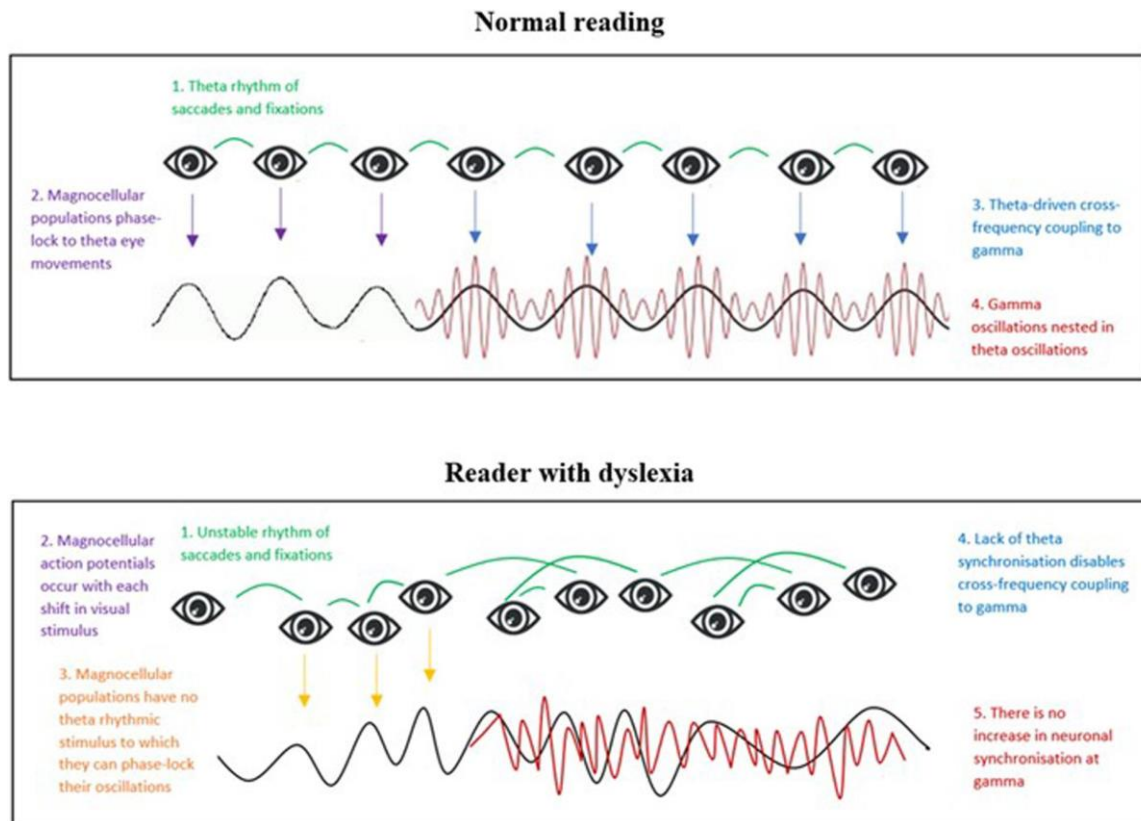


**Figure 3.** Brain activation and beta weights for similar and dissimilar conditions in pre-SMA, striatum, and TPJ/pSTS.

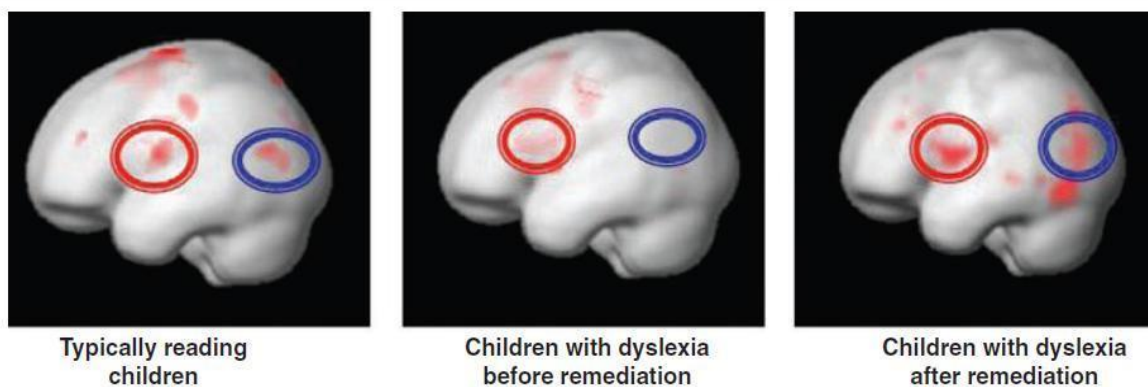
### Implications for Research

An unrepresentative sample for research and potentially unrepresentative findings in areas other than reading arise when dyslexia is classified by discrepancy from IQ, which effectively raises the average IQ of study participants and overlooks poor readers with low IQ. The requirement of at least average intellect has been preserved in current research practice, but discrepancy criteria have been removed. The primary goal of this is to allay concerns that certain findings – which go beyond reading proficiency – may be attributable to poor cognitive capacity. Another goal is to make study samples more homogeneous. Research on reading accuracy and the language and cognitive abilities that underpin precise decoding will inevitably center on dyslexia when it is believed to have a particular impact on

understanding. Because English is an outlier orthography in which learning to read properly is challenging, compared to other European orthographies, this is mostly an artifact of English being spoken in the nations where research was better financed.



**Figure 4.** Neural rhythm differences in normal readers and dyslexic readers, focusing on theta and gamma oscillation synchronization.



**Figure 5.** Brain activation differences in dyslexia and its treatment (from (36)). Functional magnetic resonance imaging activations shown on the left hemisphere for phonological processing in typically developing readers (left), age- matched dyslexic readers (middle), and the difference before and after remediation in the same dyslexic readers (right). Red circles identify the frontal region, and blue circles identify the temporo-parietal region of the brain. Both regions are hypoactivated in dyslexia and become more activated after remediation.

Thankfully, advances in cross-linguistic research and the availability of data on numerous languages and orthographies have made it evident that fluency development is equally crucial and needs

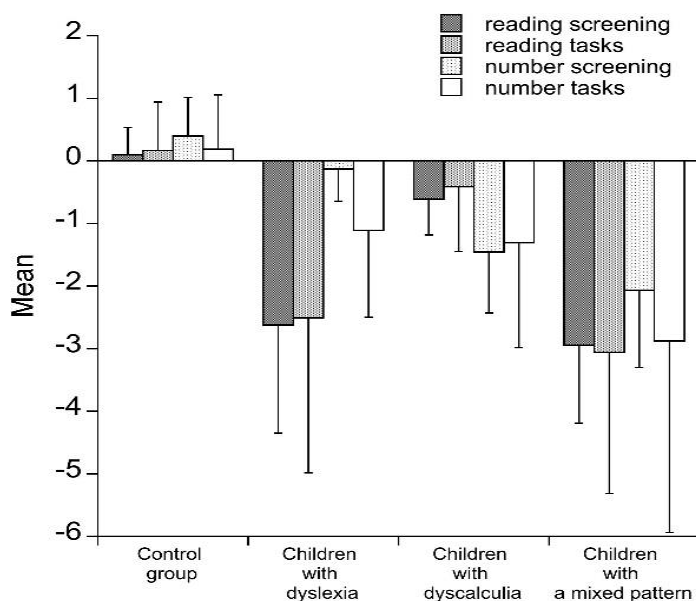
independent study in addition to accuracy. Comparably, if we assume that a certain component, or combination of variables, causes dyslexia, we will naturally focus our efforts on figuring out what that factor is and how it affects people. For decades, this has been the focus of multiple fruitful groups, and there is no end to the endless stream of hypotheses on the various origins of dyslexia [14–18]. Accordingly, if dyslexia are considered a condition, then something is wrong, and it is our responsibility as researchers to identify, define, and attempt to treat it. On the other hand, if reading difficulty is viewed as a normal aspect of cognitive variability, rather than an aberrant occurrence, then our task becomes studying and comprehending this variability, its dimensions, causes, and correlations.

Lastly, if dyslexia are considered a disorder that is innate in certain children, then it is reasonable to wonder just what dyslexia is. It is a natural, unique thing. But if we see a brain's inadequacy for learning to read not as the result of a single factor but rather as the outcome of a complex developmental pathway where environment and genetics interact to produce a complex phenotype, then the focus shifts to comprehending these developmental pathways, their differences, and how sensitive they are to various manipulations. This research program is very different in that it studies the graded effects of a multitude of risk and protective factors over multiple levels (genetic, neural, cognitive, behavioral), and their associations and interactions across levels of reading skill, ages, and environments. Instead of adopting simplistic dichotomies between “dyslexic” and “non-dyslexic” groups and their – often uninformative and potentially misleading [19].

## Outcomes

Allocation of networks in the resting state among reading domains.

A comparison of the NeuroSynth “reading” activations to the 7-network parcellation from Yeo and colleagues demonstrates that reading is extensively spread across resting-state networks (Figure 1). The visual and somatomotor-auditory RSNs accounted for around 25% of the NeuroSynth activations (17.5 and 8.2%, respectively) in the forward-inference map, whereas attention networks accounted for 37%. Additionally well-represented were the fronto-parietal (19.3%) and default mode (17.8%) networks. The only RSN that did not significantly overlap with the reading network was the limbic network. The visual, dorsal attention, ventral attention, and fronto-parietal networks constituted a bigger fraction of the activation compared to the baseline distribution of the Yeo parcellation; the limbic, somatomotor, and default modes exhibited lesser proportions Figure 6 [20, 21].



**Figure 6.** Mean performance scores on reading and number tasks across control, dyslexia, dyscalculia, and mixed difficulty groups.

## CONCLUSIONS

We have discussed how dyslexia develop. We chose the least committal form of theory – in terms of increased brain noise – to avoid making hasty theoretical decisions. We investigated how it may affect the procedures involved in development. We found that DNC would have a significant role in the formation of integrative brain networks as well as individual talents. DNC explains the delayed development of specialized skills and the persistence of older, less effective neural circuits, which are crucial for internalized speech and executive function. This framework aligns well with existing dyslexia theories and clarifies the comorbidities associated with the condition. It shifts the perspective on dyslexia from merely a reading impairment to a broader definition that encompasses overall poor performance. Dyslexia is characterized by ongoing difficulties in developing age-appropriate reading skills, without primary issues in text comprehension, which should be regarded as a separate concern.

## REFERENCES

1. Wilmot A, Hasking P, Leitão S, Hill E, Boyes M. Understanding Mental Health in Developmental Dyslexia: A Scoping Review. *Int J Environ Res Public Health*. 2023;20(2):1653. <https://doi.org/10.3390/ijerph20021653>
2. Team U. Understanding dyslexia in your child. Understood. 2023 Nov 27; Available from: <https://www.understood.org/en/articles/dyslexia-in-children>
3. Anuradha D. A study on the challenges faced by dyslexic children. *Sci Educ Innov Context Mod Probl*. 2022;5(3):133. Available from: [https://imcra-az.org/uploads/public\\_files/2022-05/39](https://imcra-az.org/uploads/public_files/2022-05/39)
4. Snowling MJ, Hulme C, Nation K. Defining and understanding dyslexia: past, present and future. *Oxford Rev Educ*. 2020;46(4):501–13. <https://doi.org/10.1080/03054985.2020.1765756>
5. *Understanding the Brain: The Birth of a Learning Science*. OECD eBooks; 2007. <https://doi.org/10.1787/9789264029132-en>
6. Hoeft F, Hernandez A, McMillon G, Taylor-Hill H, Martindale JL, Meyler A, et al. Neural basis of dyslexia: A comparison between dyslexic and nondyslexic children equated for reading ability. *J Neurosci*. 2006;26(42):10700–8. <https://doi.org/10.1523/jneurosci.4931-05.2006>
7. Richlan F, Kronbichler M, Wimmer H. Meta-analyzing brain dysfunctions in dyslexic children and adults. *Neuroimage*. 2011;56:17352011; 56:1735–42. <https://doi.org/10.1016/j.neuroimage.2011.02.040>
8. Richlan F, Kronbichler M, Wimmer H. Functional abnormalities in the dyslexic brain: a quantitative meta-analysis of neuroimaging studies. *Hum Brain Mapp*. 2009;30:32992009;30:3299–308. <https://doi.org/10.1002/hbm.20752>
9. Goswami U. Sensory theories of developmental dyslexia: three challenges for research. *Nat Rev Neurosci*. 2015;16:43–54. <https://doi.org/10.1038/nrn3836>
10. Phan TV, et al. Structural brain dynamics across reading development: a longitudinal MRI study from kindergarten to grade 5. *Hum Brain Mapp*. 2021;42:4497–509. <https://doi.org/10.1002/hbm.25560>
11. Brem S, et al. Visual word form processing deficits driven by severity of reading impairments in children with developmental dyslexia. *Sci Rep*. 2020;10:18728. <https://doi.org/10.1038/s41598-020-75111-8>
12. Paz-Alonso PM, et al. Neural correlates of phonological, orthographic and semantic reading processing in dyslexia. *Neuroimage Clin*. 2018;20:433–47. <https://doi.org/10.1016/j.nicl.2018.08.018>
13. Finn ES, et al. Disruption of functional networks in dyslexia: a whole-brain, data-driven analysis of connectivity. *Biol Psychiatry*. 2014;76:397–404. <https://doi.org/10.1016/j.biopsych.2013.08.031>
14. Turkeltaub PE, Gareau L, Flowers DL, et al. Development of neural mechanisms for reading. *Nat Neurosci*. 2003;6:767–73. <https://doi.org/10.1038/nn1065>
15. Eckert M. Neuroanatomical markers for dyslexia: a review of dyslexia structural imaging studies. *Neuroscientist*. 2004;10:362–71. <https://doi.org/10.1177/1073858404263596>
16. Skeide MA, Bazin PL, Trampel R, et al. Hypermyelination of the left auditory cortex in developmental dyslexia. *Neurology*. 2018;90. <https://doi.org/10.1212/WNL.000000000000493>

17. Wallace MT, Stevenson RA. The construct of the multisensory temporal binding window and its dysregulation in developmental disabilities. *Neuropsychologia*. 2014;64:105–23. <https://doi.org/10.1016/j.neuropsychologia.2014.08.005>
18. Ozernov-Palchik O, Gaab N. Tackling the ‘dyslexia paradox’: reading Reading brain and behavior for early markers of developmental dyslexia. *Wiley Interdiscip Rev Cogn Sci*. 2016;7:156–76.
19. González GF, Karipidis II, Tijms J. Dyslexia as a neurodevelopmental disorder and what makes it different from a chess disorder. *Brain Sci*. 2018;8(10):189. <https://doi.org/10.3390/brainsci8100189>
20. Wahlberg-Ramsay M, Nordström M, Salkic J, Brautaset R. Evaluation of aspects of binocular vision in children with dyslexia. *Strabismus*. 2012;20:139–44. <https://doi.org/10.3109/09273972.2012.735335>
21. Christian LW, Nandakumar K, Hrynychak PK, Irving EL. Visual and binocular status in elementary school children with a reading problem. *J Optom*. 2018;11:160–6. <https://doi.org/10.1016/j.optom.2017.09.003>