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Systematic Review on Neural Basis of Dyslexia

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Abstract

Dyslexia is a learning disability, affecting the literacy skills like reading, writing, spelling or mathematical, occurring in children (Developmental Dyslexia) or in adults (Acquired Dyslexia). In the Indian context, at the school level, it is estimated to range from 3 to 21%, based on the area of study and the methodology used. Research has shown that the causes for Dyslexia are multifactorial, ranging from neuro-developmental, genetic, linguistic to neurobiological in nature. Neurobiology of dyslexia is born from the construct that phonological deficits seen in dyslexics have multiple components (like poor phonological awareness, impaired lexical retrieval and poor verbal short-term memory), and each of these involve different neural networks. The present study is based on a systematic review and analyses of 17 selected research articles. This review revealed that, for good readers, there were patterns of coordination among different regions of the brain, especially the left parieto-temporal region, occipito-temporal and inferior frontal language regions. Within the lobes there were regions with more activation during some tasks than others. Whereas, for children with Dyslexia, different regions of the brain showed, inactivation, under activation or reduced activation in left parieto-temporal and occipito-temporal regions along with increased activation in frontal and right hemisphere language related regions during activities of reading, writing and spelling. Studies also demonstrated changes in the brain activation patterns after intensive evidence-based reading interventions in the dyslexics. Based on neurobiological factors, dyslexics have been explained as a deficit in automatization, a cerebellar deficit and deficits in magnocellular pathways.

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Keywords: Dyslexia, Neurobiology, Systematic Review, Neuroimaging

1. Introduction

Learning to read and write are multidimensional processes, rooted in Language and Cognition. Dyslexia is a form of Learning Disability, neuro-developmental and neurobiological in nature, which affects literacy and/or academic skills (Reading, Writing and Spelling and Mathematics) in children, adolescents and adults, despite having adequate intelligence and instruction. It has drawn attention of researchers from diverse fields such as Cognitive Psychology, Neurosciences, Linguistics, Speech-Hearing Sciences and Education.

Earlier, studies on Dyslexia have indicated that a deficiency in phonological processing was thought to underpin the reading difficulties of individuals with dyslexia. A variety of explanations have been proposed in this regard including deficits in phonological awareness, lack of awareness of the sound structure of words in their language, difficulty in breaking down a spoken word into its individual sounds, difficulty in spelling etc. These deficits were thought to co-occur with visual processing deficits, which were particularly salient for languages having visually complex stimuli (representation of spoken form in the written form) such as Chinese or Telugu. Cross-linguistic research has shown that Individuals with dyslexia performed worse than typically developing readers on phonological tasks, specifically when discriminating between novel visual patterns and in visuo-spatial working memory, which requires greater attention control.

According to International Dyslexia Association (IDA), Dyslexia is defined as 'difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction' (Lyon, Shaywitz & Shaywitz, 2003). Note that not only does this definition indicate exclusionary criteria specifying that dyslexia is not a word level disorder but also that dyslexia occurs because of a specific cognitive deficit, evidence of adequate classroom instruction and absence of other disabilities that would explain the reading problem.

More recently, with advent of neuroimaging techniques, studies using CT scan, MRI, fMRI or combination with other non-invasive techniques, a multidisciplinary approach to Dyslexia has shed light on the involvement of the brain mechanisms and multifactorial causes. Thus, the aim of the current study was to note the neural mechanisms underlying dyslexia.

2. Methodology

The present study is based on systematic review and analyses of research studies in the areas of Phonological Awareness, Working Memory, and Neurobiological aspects of Dyslexia, which was carried out during Covid-pandemic. However, this paper focuses on the neurobiological findings, for comprehensive understanding of Dyslexia. The criteria for selection of the studies for review of this aspect were - a) any one or combination of

neuroimaging techniques used, b) the sample is based on normal / dyslexic population (no restrictions on the sample size), and c) was an open access full article. Using the above-mentioned criteria, conducting extensive search at each level, 17 articles were finally selected and systematically reviewed. All studies included non-invasive procedures. A chronological approach to present the studies was used as this gives us a chance to note the changes in terms of procedures and equipment used over a period of time. Different languages were included to get a broader perspective.

3. Summary of Systematic Review

Of the 17 studies taken for systematic review, 15 were individual studies, whereas 2 were review studies. The languages used in the individual studies were English (9), German (2), Chinese (1), Chinese-English Bilingual (1), Hindi-English Bilingual (1) and Farsi (1). The neuro-imaging techniques used were Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET) and evoked response potentials (ERP) in combination with behavioral methods. The findings discussed here are on the involvement of the different areas of the brain and their connections in relation to the linguistic aspects (specifically the reading, writing and spelling) in dyslexia.

3.1. Cerebellar Deficits and Dyslexia

Reading and Writing, spelling, rapid automatized naming (picture naming speed), phonemic segmentation, automatization, memory, nonsense word repetition is markedly impaired in Dyslexic Children, which is assumed due to cerebellar deficit. Dyslexics also have less increase in activation of the right cerebellar hemisphere and Vermis, as reported in a PET study (Nicolson & Fawcett et. al., 2001) Problems in the rate of reading single words and text reflect the role of cerebellum in precise timing (Eckert et. al., 2003) Decreased activation of left cerebellar regions was seen in poor spellers and readers (Gabauer, et. al., 2012). At the same time in the context of non-dyslexics it was observed that, bilateral cerebellar activities were observed in phrase reading in Hindi—English bilinguals (Kumar et. al. 2010). It was also noted that deficits in balance and muscle tone were greater than other impairments in children with Dyslexia.

3.2. Planum –Temporale and Dyslexia

Dyslexic Children have a larger right planum temporal in the brain. Besides, auditory association cortex in Wernicke's area in Temporal lobe, larger planum temporal is also found in Occipital and Temporo-Parietal regions. Processing of Language in Dyslexic children is possibly affected, as the over activation of the right temporal area and under activation of the left temporal area is due to interference of a larger right planum temporal (Bloom & Garcia et. al. 2013; Morgan&Hynd,1998). Extreme left ward asymmetry of the Planum temporal (PT) and Planum Parietal in Dyslexics were reported by Eckert, et. al. (2003), whereas Hynd et. al. (1990) reported leftward asymmetry of PlanumTemporale in 10% of Dyslexic Children (70% in normal and ADHD children). Typical leftward asymmetry of the planum temporal disrupts lateralization, which results in Linguistic difficulties in dyslexia. Symmetrical Plana were also

reported to be larger than asymmetrical plana after postmortem of Dyslexic brains (Eckert, et. al., 2003; Morgan & Hynd, 1998).

Symmetrical plana temporal results in poor verbal comprehension and phonological decoding skills, reduced confrontation naming abilities in Dyslexics. Major proportion of Dyslexics has rightward asymmetrical or symmetrical plana. However, there is disagreement, as to whether or not the symmetrical plana are due to a larger right planum or smaller right planum (Morgan & Hynd, 1998).

3.3. Occipito-Temporal Cortex (OTC) and Dyslexia

Occipito-Temporal Cortex (OTC) includes fusiform gyrus and the inferior temporal gyrus. This region is very close to the parts of the brain that process visual information. Researchers believe that this region is used to process familiar visual information- including letters and words. A portion of the fusi form gyrus, called Visual Word Form area has shown activation, when readers process other types of visual information (e.g., images of objects) (Kearns, et. al., 2019).

Decreased activation in left OTC results in impaired object names, letters, words and non words, numbers and abstract symbol strings, written words and sentence reading, which makes dyslexics poor Spellers and readers (Gebauer, et. al., 2012). Damage to fusiform gyrus in the left OTC results in dyslexia and dyscalculia. A reduced activation within the left lingual / calcarine cortex was found when impaired readers orthographically process single letter pairs (You,et al., 2011). There is decreased neural adaptation in OTC in dyslexic readers, while processing written words. Altered activation is also seen in pre-reading studies suggesting early developmental brain alternations prior to reading acquisition. OTC plays a role in reading acquisition and reading proficiency. There was an under-activation in the left Ventral OTC in child and adult dyslexic readers, which is unaffected by Orthographic depth, since decreased neural responses were identified in deep (English) as well as in shallow (e.g., Dutch, Italian) orthographies. Developmental Dyslexia is frequently associated with a decreased activation in OTC for letters and words (Kronbichlar, & Kronbichlar, 2018).

3.4. Brain Connectivity and Dyslexia

Absence or Permanent disruption of connectivity between Visual Word Form area in OTC and left inferior frontal gyrus as well as left inferior parietal lobule results in impairment of orthographical and phonological processing of words during reading. This led to insufficient and slow reading performance. Research has shown that Visual Word Form Area is involved in spelling deficits, while accessing the orthography of auditorily presented words.

Dyslexics show increased connectivity to the left middle and superior occipito - temporal gyri and the left insula (Van der Mark, 2011). An evidence-based intervention at an early age improves reading fluency and facilitates the development of Occipito-temporal neural region that underlies skilled reading (Shaywitz & Shaywitz, et. al., 2004).

Decreased activation was also seen in inferior fronto-parietal cortex for sentence reading and in inferior parietal region for semantic processing. However, increased activation was seen for incongruous (compared to congruous) sentence endings in inferior frontal and superior temporal areas and bilateral parietal cortices and the precuneus. It suggests that reduced inferior parietal activation during sentence reading is due to a semantic impairment. Functional and structural MRI studies point to a link between Phonological processing and left temporo-parietal cortex, including the arcuate fasciculus and corona radiata.

Impairments in Fronto-Cerebellar network may play a role in delayed reading development in dyslexia (Ebrahimi, et. al., 2019; Eckert, et. al., 2003). Functional imaging studies show activation in the left anterior frontal and right cerebellar hemisphere during fluency tasks, passive listening to clicks, linguistic working memory tasks and rapid production of consonant-vowel stimuli. Study by Richards & Berninger (2008) has documented fMRI changes in the brain of the dyslexic children before and after treatment and found that there is increased brain connectivity after treatment. Predominantly, they brought about significant and durable changes in brain reorganization so that the brain activation patterns resemble those of typical readers.

3.5. Bilingual Activation and Dyslexia

Studies on Chinese -- English (You, et al., 2011) and Hindi --English bilingual (Kumar et al., 2009) dyslexics report that former had reduced left Temporo-parietal (e.g., angular gyrus) activation in phonological processing in reading English, whereas latter had activation of anterior temporal pole, right caudate nucleus, right superior temporal gyrus (STG), left insula and left putamen due to deficits in slow reading and writing in English. Chinese impaired English readers also showed reduced activation with left lingual/ calcarine cortex. There seems to be a universal underlying mechanism in all dyslexics for orthographic processing of reading due to neural disruption in left OTC (You et al., 2011; Kumar et al., 2009).

3.6. Magno-Cellular Pathways and Dyslexia

The visual magnocellular pathway originates in the magnocellular cells in the retina and projects via the magnocellular layers of the lateral geniculate nucleus (LGN) to the primary visual cortex (VC) situated in the back of occipital lobe. This pathway plays a crucial role for focusing visual attention and letter decoding. It is responsible for timing visual events when reading. A deficit in this pathway is likely to be an important cause of reading difficulties. It is involved in normal eye movement control, visuo-Spatial attention, visual search, letter encoding and peripheral vision, which are involved in the development of orthographic skills. Magnocellular system allows the rapid perception of movement form and changes in brightness, but is relatively insensitive to stimulus location and color. Dysfunction of Magnocellular effects have been demonstrated in transparent / opaque as well as alphabetic and logographic scripts.

Magnocellular system is poorly developed in many dyslexics. Dyslexics often complain that small letters appear to blur and moving around, when they read. These are the result of abnormalities of the magnocellular component of the visual system, which is specialized in processing fast temporal information. - Dyslexics may be unable to process fast incoming information adequately in phonological or visual or motor systems. This may lead to phonological, Visual or motor deficits (Stein, & Walsh, 1997). Visual Magnocellular training improves detection of coherent motion, control of saccadic eye movements in dyslexic children. These improvements result in increased reading accuracy and reduced visual reading errors-like omissions and additions (Ebrahimi, et. al., 2019).

Dyslexics have great variety of visual, kinesthetic, sequencing memory and motor deficits due to differences in the particular magnocellular Systems (Stein, 2008). Poor reading performance of Dyslexics is due to abnormally reduced sensitivity in the magnocellular system. In particular, the visual sensory abnormalities in Dyslexics are the result of magno cellular shrinkage and organization in Lateral geniculate nucleus, whereas the parvo-cells are intact. Postmortem Histology reflects that magnocellular cell in the LGN are significantly smaller and more disorganized in Dyslexic brains than controls. Dyslexics have diminished visually evoked potentials to rapid, low contrast stimuli, but normal responses to slow or high contrast stimuli (Livingstone, et. al., 1991; Greatrex & Dresdo, 1995). Children with high motion detection thresholds would be more likely to make orthographically nonsense 'letter' errors than children with low motion thresholds. Impairment of the visual magnocellular pathway revealed by poor performance on motion detection task could degrade the encoding of the position of letter features, when children read.

Phonological factors play an important role in explaining children's "letter" errors, but these factors are independent of magnocellular function. Phonological or orthographic deficit could produce the same errors in letter/letter cluster to sound/sound cluster mapping (grapheme-phoneme correspondence), which could lead to children making "letter" errors. Hence, reading can be described by a multi-channel model comprising visual and phonological processing and short-term memory components (Cornelissen, et. al., 1998). Research has also noted that Lateral geniculate nucleus in dyslexic individuals contains thinner magnocellular layers in the left hemisphere (Girardo-Chica, et. al., 2015).

4. Conclusion

Neurobiology of dyslexia is born from the construct that Phonological deficits seen in them have multiple components - poor phonological awareness, impaired lexical retrieval and poor verbal short-term memory, each of which involve different neural networks. It was observed that there was inactivation, under activation or reduced activation of brain during reading, writing, and spelling in Dyslexics. It also explains dyslexia based on Automatization deficit, Cerebellar deficit, and Magnocellular pathway deficit. The implications of these finding for parents of children who are acquiring reading skills are many folds, for example they can play phoneme games, can ask the child to segment words and repeat nonsense words etc. These

simple tasks can help the child to not only acquire reading skills faster but also help to form better neural networks for long lasting memory.

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