A Review on Dyslexia

S.Ramyasilpa  
Department of pharmacology,  
Nargund college of pharmacy, Bengaluru, Karnataka, India  
shilpasankarapu@gmail.com

ABSTRACT

Dyslexia is a specific reading disorder. It is common childhood disorder affecting around 5% of school aged children. The main deficits of dyslexia include inability to process sensory input (i.e., acoustic information) that comes into the nervous system rapidly, and an impaired reading ability. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge. The dyslexia is associated with alteration of several biological levels like genetic, biochemical, physiological, cognitive levels.

Keywords: Dyslexia, human language writing systems, logographic nature

INTRODUCTION

Dyslexia is a specific reading disorder. [1] It is common childhood disorder affecting around 5% of school aged children. [2,3,4] It is neurological disorder that is characterized by difficulty in reading or learning, gross neurological deficits, uncorrected visual or auditory problems, emotional disturbances or inadequate schooling. [2,3] It is characterized by dysfunction of the normal left hemisphere language network and also implicates abnormal white matter development. [5] It is associated with human language writing systems, alphabetical or syllabic characters and logographic nature. [6] This disease is present throughout their life span and interferes with academic achievement or activities of daily living that require reading skills. [4] The main deficits of dyslexia include inability to process sensory input (i.e., acoustic information) that comes into the nervous system rapidly, and an impaired reading ability. [2] Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge. [4]

Prevalence:

Approximately 5–17% of the population is considered to have dyslexia disease. [7] The prevalence differs with the use of different diagnostic criteria. The sex ratio may be influenced by severity, IQ and assessed cognitive profiles. Boys are usually more susceptible to girls. In children, 20% of patients associated with dyslexia have ADHD disorder. But in adults, dyslexia is coordinated with depressive disorders and disorders of social behavior. A child with an affected parent has a risk of 40–60% of developing dyslexia. This risk is increased when other family members are also affected. Dyslexia is both familial and heritable, which provides opportunities for early identification of affected siblings and often for delayed but helpful identification of affected adults. Thus, up to 50% of children of dyslexic parents, 50% of siblings of dyslexic children, and 50% of parents of dyslexic children may have

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the disorder. Replicated linkage studies implicate loci on chromosomes. [8]

Etiology:
It occurs due to dysfunction of CNS that further leads to core deficit in reading. The cause of dyslexia is multi-factorial. It is associated with alteration of multiple genes and environmental risk factors. [9] These genes results in disturbance in cortical neuron migration and diminished activity in left hemispheric brain regions. [8] There are 6 candidate genes that are identified for pathophysiology of dyslexia. 

DYX1C1 in the DYX1 locus on chromosome 15q21; DCDC2 and KIAA0319 in the DYX2 locus on chromosome 6p21; C2orf3 and MRPL19 in the DYX3 locus on chromosome 2p16–p15; and ROBO1 in the DYX5 locus on chromosome 3p12–q12. [10] DCDC2 and KIAA0319 are more involved in pathophysiology.

Pathophysiology:
The dyslexia is associated with alteration of several biological levels like genetic, biochemical, physiological, cognitive levels. [2] One of the cause of dyslexia is malfunctioning of cerebellum. As cerebellum functions results in regulation of motor control, verbal working memory. So, malfunctioning of cerebellum leads to dyslexia. This results in alteration of magnocellular pathway. [11]

1. Neuroanatomical features:
The left planum temporale on the superior temporal lobe of the brain is believed to be responsible for language processing in the majority of people. In dyslexic individuals, ectopia and heterotopias within the left perisylvian cortical area were detected, and the usual asymmetry between the left and right brains were absent. The relationship between planum temporale morphology, language and psycholinguistic function is significant in understanding the etiology of dyslexia. [6]

2. Genetic features:
Several genes have been identified in pathophysiology of dyslexia.

DYX1:-
DYX1C1 gene in DYX locus is present on 15q21. It is usually functioning with reading ability, spelling. If DYX1C1 gene is mutated, it results in altering function of reading ability. The mutation of DYX1C1 gene occurs due to translocation that interrupts its sequence. This gene is present in tissues and lung, hepatic, testicular and brain. Mutations of this gene results in malfunctioning of hippocampus that cause conformational change in normal anatomical structure and affects quarter of pyramidal neurons that further leads to appearance of localized heterotopias. [6]

DYX2:-
Mutation of this loci leads to alteration of chromosome 6p2287-89 area and results in dyslexia. Alteration of this gene leads to change in phonological and orthological nature. So, 2 genes DCDC2 and KIAA0319 gene in DYX2 loci alterations are found to be seen during dyslexia. DCDC2 is mainly expressed in endorhinal cortex, inferior temporal cortex, medial temporal cortex, hypothalamus, amygdale, hippocampus. [6]

DYX3:-
Alterations of c2orf3 and MRPL19 genes on DYX3 loci leads to dyslexia. This loci is present on chromosome 2p16-p15 area 109. [6]

DYX4:-
Gene on this loci that is altered is not yet found. This loci is expressed in chromosome 6q11.2-q12 area resulting in functions like spelling ability and phonological encoding.

DYX5:-
ROBO1 gene is present on DYX5 loci relating to chromosomal area 3p21-q13. This gene is
usually associated by axon growth regulation, probably in those that cross from one brain hemisphere to other.\cite{6,12}

So, the protein encodes ROBO1 gene intervenes with axon or fibres growth regulation projected outside the brain cortex together with those that form part of thalamocortical projections. Alterations of ROBO1 gene leads to dyslexia disorder.\cite{13}

DYX6:-
This loci functions are associated with single word reading ability and phonological awareness.\cite{14}

DYX7:-
This loci includes several genes that are involved in pathophysiology of dyslexia. They are SCT gene, STIM1 gene, MTR1 gene and HRAs gene.\cite{15}

DYX8:-
This loci is associated with functioning of reading efficacy, quick naming of objects and colours. It is present on chromosomal area 1p34-p36.\cite{16,17}

DYX9:-
This loci is located in xq27. This gene is related to fragile x syndrome, hereditary mental retardation that includes many speech disturbances.\cite{17}

Treatment:-
Testing and screening for dyslexia are available and it is necessary to find out at early stage. Without proper diagnosis and instruction, dyslexia can lead to frustration, school failure, and low self-esteem.
An assessment for dyslexia includes reading or writing while the tester looks for signs of dyslexia, such as adding, dropping, or changing words; pulling words from other lines; or reversing or transposing words and letters.
While not diagnostic in itself, body language may provide a clue: A person with dyslexia may frequently clear his or her throat, tap a pencil, or fidget during the testing out of anxiety about performing on the test.
Dyslexia cannot be prevented or cured, but it can be managed with special instruction and support. Early intervention to address reading problems is important. Parents must understand that children with dyslexia can learn, but probably need to learn in different ways than children without the condition. Teaching should be individualized and may involve modeling letters and words in clay or other three-dimensional techniques to help the child learn letters and words.
If you notice any of the signs of dyslexia, your child’s doctor can help determine whether there are physical problems, such as vision problems, that are causing or contributing to your child’s condition, and he or she can refer you to specialists who can diagnose and treat learning differences. These may include an educational specialist, an educational psychologist, or a speech therapist.\cite{17}

CONCLUSION
Dyslexia is a specific reading disorder. It is common childhood disorder affecting around 5% of school aged children. It occurs due to dysfunction of CNS that further leads to core deficit in reading. The cause of dyslexia is multifactorial. It is associated with alteration of multiple genes and environmental risk factors. The dyslexia is associated with alteration of several biological levels like genetic, biochemical, physiological, cognitive levels. One of the cause of dyslexia is malfunctioning of cerebellum. As cerebellum functions results in regulation of motor control, verbal working memory. So, malfunctioning of cerebellum leads to dyslexia. This results in alteration of magnocellular pathway. Dyslexia cannot be prevented or cured, but it can be managed with special instruction and support. Early
intervention to address reading problems is important. Such information will definitely provide us with much needed new targets for treatment. So, early diagnosis and cessation of disease is associated with better outcome.

REFERENCES

4. Lauren M. McGrath1, Shelley D. Smith2 and Bruce F. Pennington1 Breakthroughs in the search for dyslexia candidate genes. Trends in Molecular Medicine2006;12(7): 333-41.
6. A. Benitez-Burraco Neurobiology and neurogenetics of dyslexia Neurología 2010;25(9):563-581
11. A. Benitez-Burraco Neurobiology and neurogenetics of dyslexia Neurología 2010;25(9):563-581