We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900
Open access books available

185,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Chapter

Learning Disorder (Dyslexia): An Overview Description of the Entity through Available Researches

Sambhu Prasad and Rajesh Sagar

Abstract

Dyslexia is a specific learning disability can be explained with number of biological and neuropsychological theories. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. The available research in this field show that there is impairment in processing the sensory input that enters the nervous system. It also indicate that there are problem in phonological decoding. There are various educational interventions and programs to address dyslexia which includes regular teaching in small group, a learning support assistant like a specialist teacher, policy interventions etc. The basic strategies of intervention focus on phonemic skill such as the ability to identify and process word sounds.

Keywords: dyslexia, fMRI, neurobiology, management

1. Introduction

Specific learning disorder (SLD) is manifested by specific and significant impairments in learning of scholastic skills in children and adolescent. Over the years they have been given different labels like dyslexia, perceptual handicap, neurological impairment, minimal brain dysfunction, congenital word blindness, development aphasia, congenital aphasia, educational handicap. Until very recent times they were often not diagnosed due to lack and paucity of assessment tools. Learning disability is not a single disorder, but is composed of disabilities in any of the 7 specific areas:- like Receptive language (listening), Expressive language (speaking), Basic reading skills, Reading comprehension, Written expression, Mathematical calculation, Mathematical reasoning.

There is heterogeneity in term of etiological factors and manifested as difficulty in reading (dyslexia), writing (dysgraphia) or perform efficient mathematical task (dyscalculia) despite intact sense, intelligence, motivation with adequate socio-cultural opportunity [1, 2]. The term SLD does not include who have learning disability primary the result of any organic lesion in brain, subnormal intelligence, any psychiatric disorders or socio-cultural disadvantages [3]. This disorder is seen worldwide and occurs in students irrespective of their mother tongue and medium of instruction in the school which may be English or any other vernacular language.

Dyslexia was first identified in the latter half of the nineteenth century and subsequently several subtypes have described [4]. In 1993, Castle and Coltheart point out the basic subtypes namely Phonological, Surface and Mixed varieties. The phonological subtype have deficiency in development of graphic phonemes reading ability whereas in surface subtype show difficulty with developmental lexical procedure [5]. The neurobiological aspect of dyslexia has been described as earlier as in 1891 by Dejerine suggesting angular gyrus, supramarginal gyrus in inferior parietal lobe, posterior aspect of supra temporal gyrus and ventral aspect of occipito-temporal were critical for reading [6, 7].

2. Prevalence of dyslexia

Dyslexia is perhaps the most common neurobehavioral disorder affecting children, with prevalence rates ranging from 5 to 17.5 percent [2]. An epidemiological study in British school children in the age range of 8–10 year found the prevalence of 'specific reading difficulties 'to be 3.9%, with the overall prevalence of SLD around 7.5% [8]. According to one Asian study the prevalence of dyslexia and probable dyslexia were found to be 6.3% and 12.6% respectively. The male to female ratio of dyslexia was 3.4:1 [9]. In India, there was about 250 million school going children, 12.5 million (1.25 Crore) children suffering from SLD [10]. Barring arithmetic disorder which may be more common in girls, all other learning disorder seems to be 3–4 times more common in boys. In another study conducted by Sadhu et.al, (2003) reported presence of neurological soft signs in SLD children from Indian context [11]. Agrawal et al. (1991) used Bender Gestalt test, Piaget's test and Indian modification of WISC for the detection of SLD in rural primary school children [12].

3. Diagnostic criteria

Characteristic diagnostic features include difficulty recalling, evoking, and sequencing printed letters and words; processing sophisticated grammatical constructions; and making inferences. There are certain criteria in International Classification of Diseases (ICD-10) [13] and Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [14] for diagnosis of specific reading disorder (**Table 1**).

S. No.	According to ICD 10	DSM 5
1	Specific reading disorder (F81.0)	Reading Disorder (315.00)
2	Specific spelling disorder (F81.1)	Mathematics Disorder (315.1)
3	Specific disorder of arithmetic skills (F81.2)	Disorder of Written Expression (315.2)
4	Mixed disorder of scholastic skills (F81.3)	Learning Disorder Not Otherwise Specified (315.9)
5	Other developmental disorders of scholastic skills (F81.8)	
6	Developmental disorder of scholastic skills, unspecified (F81.9)	

Table 1.
Classification of specific learning disorder.

4. ICD-10, diagnostic criteria for specific reading disorder

A. Either of the following must be present

1. 'A score on reading accuracy and or comprehension that is at least 2 standard errors of prediction below the level expected on the basis of the child's chronological age and general intelligence with both reading skills and I.Q. assessed on an individually administered test standardized for the child's culture and educational system'.

2. 'A history of serious reading difficulties or test scores that met criterion (A) [1] at an earlier age plus a score that is at least 2 standard errors of prediction below the level expected on the basis of the child's chronological age and I.Q'.

B. 'The disturbance described in criterion (A) significantly interferes with academic achievement or with activities of daily living that require reading skills'.

C. 'The disorder is not the direct result of a defect in visual or hearing acuity or of a neurological disorder'.

D. 'School experiences are within the average expectable range'.

E. 'Most commonly used exclusion clause – IQ below 70 on an individually administered standardized test'.

5. DSM-5 criteria for Reading disorder (F81.0)

With word reading accuracy, reading rate or fluency, reading comprehension include:

A. 'Reading achievement, as measured by individually administered standardized tests of reading accuracy or comprehension, is substantially below that expected given the person's chronological age, measured intelligence, and age-appropriate education'.

B. 'The disturbance in Criterion A significantly interferes with academic achievement or activities of daily living that require reading skills'.

C. 'If a sensory deficit is present, the reading difficulties are in excess of those usually associated with it'.

Coding note: If a general medical (e.g. neurological) condition or sensory deficit is present, code the condition on Axis III.

6. Characteristics of reading disorder

History of language delay, or of not attending to the sounds of words (trouble playing rhyming games with words, or confusing words that sound alike), along with a family history, are important red flags for dyslexia [2]. Specific symptoms of reading disorder include difficulties in single word decoding, slow oral reading and poor comprehension of written text. The developmental dyslexia as per

Bakker classification: Linguistic (L) type, perceptual (P) type, and M type [15]. It is also classified by Doehring (1977) into Subtype I (Poor in oral reading of letters, nonsense syllables and words relative to their silent reading skills); Subtype II (Read slowly and made many mistakes) and Subtype III (Better able to read single letters than to read words or syllables during either silent or oral reading) [16]. As per Petrauska and Rourke (1979), developmental dyslexics were classified into those:a) who had language difficulties with intact visual motor skills; b) who had sequencing, visual memory and finger identification difficulties; c) who had language and concept formation difficulties and poor visual motor skills [17]. Rourke also postulated a model which is organized into primary, secondary and tertiary assets and defects [18]. The primary assets has following feature such as repetitive motoric skills, auditory perception, mastery of rote or repetitive material. Its defects include tactile and visual perception complex psychomotor skill and ability to process novel situation. In secondary assets there were selective and sustained attention for simple, repetitive verbal materials and its defects include tactile and visual stimuli and exploratory behavior. The tertiary assets involve rote verbal memory with defects in tactile and visual memory, concept formulation, problem solving, hypothesis- testing skills and understanding the semantic and pragmatic aspects of language [19]. These deficits appear to increase with age. It was also postulated that nonverbal learning disorder is related to dysfunction of white-matter tracts that serve to connect associational areas, with particular involvement of the right hemisphere [18].

7. Etiological factors of dyslexia

A. **Heritability**-It has both familial and heritable cause and found that the rate among siblings of affected persons was around 40 percent and among parents it ranges from 27 to 49.The chromosomal analysis show linkage and implicate loci on chromosomes 2,3,6,15 and 18 [20].

B. Cognitive Influences: Theories of Developmental Dyslexia

1. Cerebellar theory: The studies have shown that the cerebellum of dyslexic have mild dysfunction with number of cognitive difficulties ensue [21].

2. Magnocellular theory: The magnocellular dysfunction is not restricted to the visual pathways but is generalized to all modalities [22].

3. Phonological deficit theory: The dyslexic have a specific impairment in the representation, storage and/or retrieval of speed of sounds [23].

4. Rapid auditory processing theory: This theory says that the primary deficit lies in the perception of short or rapidly varying sounds [24].

5. Visual theory: There is a visual impairment giving rise to difficulties with the processing of letters and words on a page of text [25].

Among investigators in the field there is now a strong consensus supporting the phonological theory.

C. The neural basis of dyslexia

Galaburda et al., 2000 found that there was a microscopic malformations in the perisylvian regions (cortical ectopias and dysplasias) and the geniculate nuclei (size reduction of magnocellular neurons) suggesting abnormal neuronal migration and maturation, prompting research on the neural basis of dyslexia [26]. In skilled adult readers the functional neuroanatomy of reading is widely distributed but dominated by a left-sided network [27]. They also found that the ventral pathway in the posterior fusiform gyrus re[present an automatically assessed visual word-form area [28]; however the dorsal pathway (include angular and supra-marginal gyri) represent phonology based assembly process [29], implicated in the output of phonological and articulatory aspects. Most studies show reduced activity in the left, rather than bilateral perisylvian regions. There was a disconnection within the left perisylvian network, which has a role in phonological processes [30].

D. Neuropsychological Deficits in dyslexia

Visual processing deficit: Both visual auditory and tactile information processing deficit was documented by Laasonen and Tomma, 2000 [31]. There were impairments in executive functioning and deficit in central information processing. It was also shown that there were maturation lag of left hemisphere, also called disconnection syndrome. They also have difficulty with inter-hemispheric transfer and defect in left parietal lobe, temporal lobe, angular gyrus and cerebellum.

E. Neurological Soft Signs in dyslexia

It include abnormalities in: Graphethesia, Stereognosis, Motor task, Face hand face noise test, two point discrimination, maintenance of posture & tapping, more rotation error in BGT. It was also found that arithmetic disabled children had right sided soft signs indicating left hemispheric dysfunction.

8. Neuro-imaging of dyslexia

Given that reading disorder is essentially a language deficit, the left brain has been hypothesized to be the anatomical site of the dysfunction. There are different neuro-imaging studies done including positron emission tomography (PET scanning), event related potentials (ERPs) with auditory and visual stimuli, magnetic evoked potentials by megnetoencephalography (MEG) and magnetic resonance spectrography (MRS) alongwith functional magnetic resonance imaging (fMRI) [32]. The research studies using functional magnetic resonance imaging (fMRI) studies have suggested asymmetrical activation of left brain in children with both language and learning disorders. Functional MRI (fMRI) detects the hemodynamic response related to neural activity in the brain, based on the principal of BOLD (blood oxygen level detection). It has better spatial resolution and BOLD activity from all regions of the brain can be obtained. It can noninvasively record brain signals without risks of radiation inherent in other scanning methods, such as CT and PET scans. Hence, this study was planned with fMRI neuro-imaging technique for studying the areas of activity during different processing tasks in dyslexia.

9. FMRI and dyslexia

Neuroimaging method such as functional magnetic resonance imaging (fMRI) provide evidence of hypoactivation of the left posterior language system

in dyslexia, across different languages. This hypoactivity has been localized to left posterior parietal cortex [33], inferior occipitotemporal cortex, and superior temporal gyrus [34].

Temple et al. 2000, found that the fMRI data revealed largest activation was in the left prefrontal region, between the middle and superior frontal gyri in Brodmann area 46/10/9. Analysis of the dyslexic readers revealed no left frontal response to the rapid, relative to the slow, stimuli. This brain imaging study shows both a disrupted neural response to rapid auditory stimuli and its location in dyslexic adults [35]. By using fMRI, Brown et al., 2001 have shown that there were hypointense gray matter in most of the left temporalcortex by voxel-based morphometry or anisotropy in white-matter fibers [36]. Helenius, showed that prelexical processing in left inferior occipitotemporal regions was sometimes absent in people with dyslexia [37]. Using fMRI, Shaywitz et al.,2002 found that the brain activation in dyslexic opposite during reading task where frontal part was more active in comparison with back regions [34].

Johanna Pekkola 2006 et al., found that dyslexic readers' use more of motor-articulatory and visual strategies during phonetic processing of audiovisual speech, possibly to compensate for their difficulties in auditory speech perception [38]. Martin Kronbichler et al., 2008 found that there was less gray matter volume for dyslexic readers in the left and right fusiform gyrus, the bilateral anterior cerebellum and in the right supramarginal gyrus. There was decrease volume mass in gray matter in right and left fusiform gyrus which highlight the importance of this brain regions in developmental dyslexia [39]. Quaglino V et al. 2008 during their fMRI study i a phonological deficit in developmental dyslexia [40]. Vera Blau et al. 2009 showed that dyslexic readers has under activation of superior temporal cortex for the integration of letters and speech sounds. They also showed that there was reduction of audio-visual integration the fundamental deficit in auditory processing of speech sounds, which in turn predicts performance on phonological tasks and account for developmental dyslexia, in which phonological processing deficits are linked to reading failure through a deficit in neural integration of letters and speech sounds and IQ [41]. Fabio Richlan et al., 2010 found that there was dysfunction of the region in the developmental cases who failed to exhibit responsiveness of left OT regions to the length of words and pseudo- words [42]. Rimrodt SL et al., 2009 found that the dyslexic group show more activation in the linguistic processing areas such as left middle and superior temporal gyri as well as in the attention and response selection areas such as bilateral insula, right cingulate gyrus, right superior frontal gyrus, and right parietal lobe [43]. Li Liu et al., 2012 showed that the dyslexic has less activation for both tasks in right visual (BA18, 19) and left occipito-temporal cortex (BA 37), suggesting a deficit in visuo-orthographic processing. It also has abnormalities in frontal cortex and in posterior visuo-orthographic regions may reflect a deficit in the connection between brain regions [44]. In the recent neurobiological study of dyslexia from India dyslexic where compared with healthy matched control and BOLD acquisition using fMRI was done with three different paradigms (semantic, picture and auditory), the study show that it was an important contribution in beginning to understand how higher level language processing impacts reading comprehension, especially in disabled readers. Healthy controls show greater activation within left occipito-temporal region (visual word form area). The Dyslexic group demonstrated right hemispheric dominance for language and exhibit increased articulation and planning as compared to control, in performing the semantic tasks. The BOLD cluster activation and signal intensity were greater in dyslexic patients as compared to control [45]. The further description of BOLD activation during the above mention tasks are given in detail in the tabular form and brain imaging using fMRI (**Tables 2–4** and **Figures 1–3**).

No clusters	Z-score	mni coordinate	Talairach coordinates	Hemi-sphere	Area of activation	Brodmann area
Patient vs. control during rhyming task (p<0.001)						
29	3.21	44 -46 -28	40 -42 -24	Right	Cerebellum-Culmen	
39	3.17	24 -2 -36	22 -1 -28	Right	Uncus	BA 36
14	3.08	20 -30 30	17 -32 29	Right	Cingulate Gyrus	
65	3.01	54 -18 34	49 -22 34	Right	Postcentral Gyrus	BA 2
45	3.01	-4 -46 22	-5 -47 20	Left	Posterior Cingulate	BA 30
Controls vs. patients during rhyming task with respect to meaningless stimulus (P<0.001)						
11	3.81	58 -48 20	52 -48 19	Right	Supramarginal gyrus	BA 40

During intergroup comparison of dyslexic group with respect to control for auditory phonological rhyming task BOLD activation was observed in the right post-central gyrus, right cerebellum and uncus and the left posterior cingulate gyrus. Thus the decoding of language occurs in left superior temporal gyrus that was not observed in dyslexic group.
During Intergroup comparison of controls with respect to dyslexic group during auditory phonological task, rhyming with respect to meaningless baseline BOLD activation was observed more in right superior temporal gyrus where the right hemisphere is dominant for visuo-spatial auditory processing. No such activation was observed in dyslexic group with similar condition. This finding concord with the previous study in which the middle and posterior part of superior temporal sulcus was activated by silent speech-reading, and also by audio-visual speech. This region usually constitutes the principal focus of activation in fMRI studies of speech-reading.

Table 2.
BOLD Activation in Intergroup Comparison during Rhyming Task.

No clusters	Z-score	mni coordinates	Talairach coordinates	Hemisphere	Area of activation	Brodmann area
Controls						
10	3.49	32 -52 68	28 -56 61	Right	Superior Parietal Lobule	BA 7
29	3.29	-52 -70 28	-50 -69 22	Left	Middle Temporal Gyrus	BA 39
Patients						
102	3.15	8 -34 50	6 -38 46	Right	Precuneus	BA 7
28	3.88	-32 38 -14	-30 35 -6	Left	Middle Frontal Gyrus	BA 47
51	3.87	58 0 -34	53 1 -25	Right	Middle Temporal Gyrus	BA 21
58	3.81	-64 -40 34	-61 -42 30	Left	Supramarginal Gyrus	BA 40
238	3.74	-18 -36 4	-18 -36 4	Left	Thalamus	
62	3.66	2 -34 72	0 -40 65	Left	Paracentral Lobule	BA 5
48	3.61	6 -44 20	4 -45 18	Right	Posterior Cingulate	BA 30
14	3.42	-20 66 10	-19 59 19	Left	Superior Frontal Gyrus	BA 10
39	3.39	18 -18 62	15 -24 58	Right	Medial Frontal Gyrus	BA 6
11	3.36	34 -26 66	30 -32 61	Right	Postcentral Gyrus	BA 3

No clusters	Z-score	mni coordinates	Talairach coordinates	Hemisphere	Area of activation	Brodmann area
13	3.33	-46 -20 2	-44 -20 3	Left	Insula	BA 13
16	3.29	-50 -74 28	-48 -73 22	Left	Middle Temporal Gyrus	BA 39
10	3.25	54 -32 40	48 -35 38	Right	Inferior Parietal Lobule	BA 40
Patients **vs.** Controls						
30	3.82	-32 34 -12	-30 31 -4	Left	Inferior Frontal Gyrus	BA 47
Controls **vs.** Patients						
No Activation						

During picture task, in the control group BOLD activation was observed more in left middle temporal gyrus which are involved in visual encoding and memory processing as well as in the right superior parietal lobule (visual processing area). However in dyslexic group BOLD activation was observed more in right precuneus, right posterior cingulate gyrus (visuospatial processing), right medial frontal gyrus (involved in planning and co ordination of movement), right paracentral lobule (voluntary motor function and motor planning) and bilateral middle temporal gyri (right hemispheric dominance). BOLD activation was also observed in left supramarginal gyrus (association area) and left thalamus (sensory motor coordination). Thus the dyslexic group used more memory component of brain.

Table 3.
BOLD Activation during Picture Naming Task in Controls, Dyslexic and the Intergroup Comparison (P<0.001, Cluster Threshold=10).

No clusters	Z-score	mni coordinates	Talairach coordinates	Hemisphere	Area of activation	Brodmann area
Controls (p<0.001)						
363	4.18	-16 -90 -14	-16 -84 -17	Left	Cerebellum - Declive	
99	3.62	22 -88 -10	19 -83 -12	Right	Lingual Gyrus	BA 18
86	3.52	12 -98 0	10 -93 -4	Right	Lingual Gyrus	BA 17
90	3.42	-20 -102 -6	-20 -96 -11	Left	Lingual Gyrus	BA 17
Patients (p<0.001)						
357	4.10	-12 -92 -6	-12 -87 -10	Left	Lingual Gyrus	BA 18
1160	4.36	44 -54 24	39 -54 21	Right	Superior Temporal Gyrus	BA 22
94	4.22	-20 -40 26	-20 -41 23	Left	Cingulate Gyrus	BA 31
218	3.61	-14 -56 36	-15 -57 31	Left	Precuneus	BA 31
141	3.83	-44 20 -30	-41 20 -22	Left	Superior Temporal Gyrus	BA 38
137	3.78	20 -86 4	17 -82 0	Right	Lingual Gyrus	BA 17
57	3.68	2 16 -16	1 15 -9	Right	Anterior Cingulate	BA 25
48	3.66	-30 -74 -16	-29 -69 -17	Left	Cerebellum Declive	
38	3.42	34 -66 -20	31 -62 -19	Right	Cerebellum Declive	

No clusters	Z-score	mni coordinates	Talairach coordinates	Hemisphere	Area of activation	Brodmann area
Patients Vs. Controls (p<0.005)						
25	3.61	-20 -40 26	-20 -41 23	Left	Cingulate Gyrus	BA 31
346	3.18	18 -52 28	15 -53 25	Right	Cingulate Gyrus	BA 31
63	3.14	46 -54 26	41 -55 23	Right	Superior Temporal Gyrus	BA 39
16	3.03	32 -46 6	28 -45 6	Right	Temporal Lobe	Hippocampus
Controls Vs. Patients (p<0.005)						
13	2.73	42 -44 -22	38 -41 -19	Right	Fusiform Gyrus	BA 20

It show bilateral lingual gyrus (left side greater than right) activation. Bilateral superior temporal gyrus (right hemispheric dominance) involved in auditory processing and left precuneus (visuo-spatial imagery, episodic memory retrieval and self-processing operations). Bilateral cingulated gyrus was also activated that plays role in visual spatial processing. Bilateral cerebellum was activated for motor speech articulation as the subject had to verbalize the response. However in control group only bilateral lingual gyri were involved. The finding suggested that reading disable group showed significantly more activation than typical reader in areas associated with linguistic processing (left middle/superior temporal gyri), and attention and response selection (bilateral insula, right cingulate gyrus, right superior frontal gyrus, and right parietal lobe). However during intergroup comparison BOLD activation was observed in control group with respect to dyslexic in right fusiform gyrus (visual word form area). In dyslexic group with respect to control, BOLD activation was observed in bilateral cingulate gyrus involved in visuo-spatial processing (right cerebral dominance). Right superior temporal gyrus (word processing) and right hippocampal (memory encoding and retrieval) were activated. Thus the dyslexic group used different pathway and greater areas of activation as compared to that of control group.

Table 4.
BOLD Activation during Complex Sentence Reading Task (Semantic Task 3) in Controls, Dyslexic and the Intergroup Comparison.

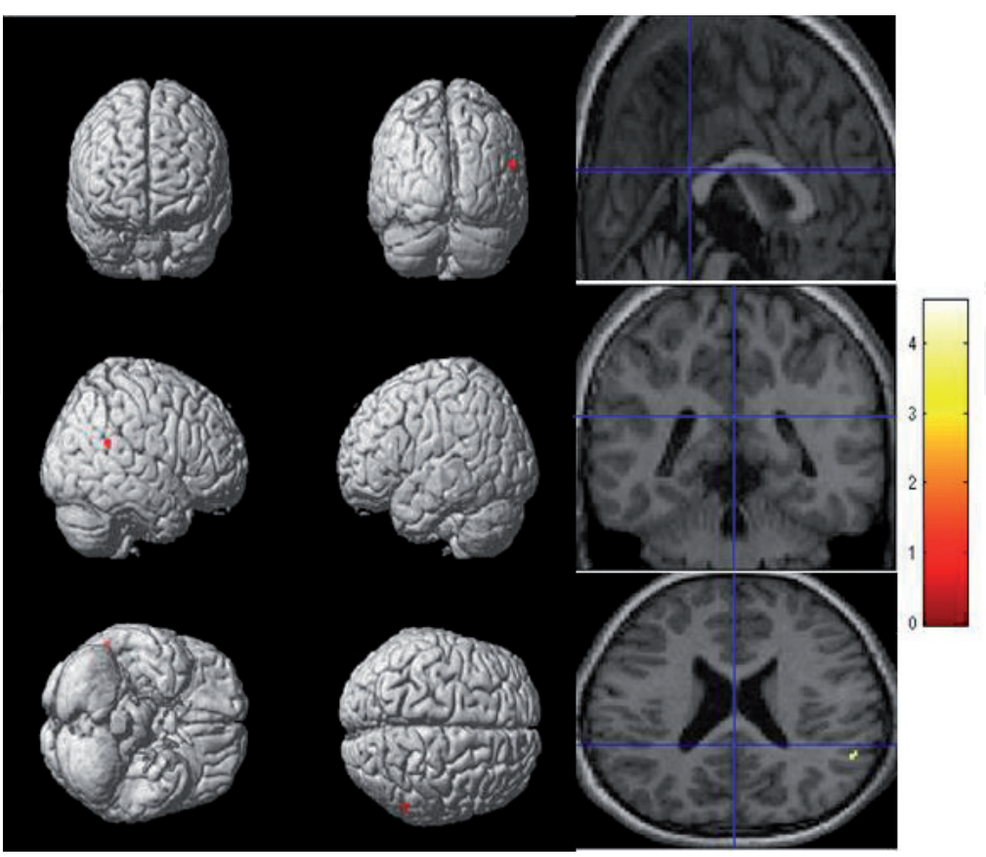


Figure 1.
BOLD activation in intergroup comparison Patient vs. Control during rhyming task (p < 0.001).

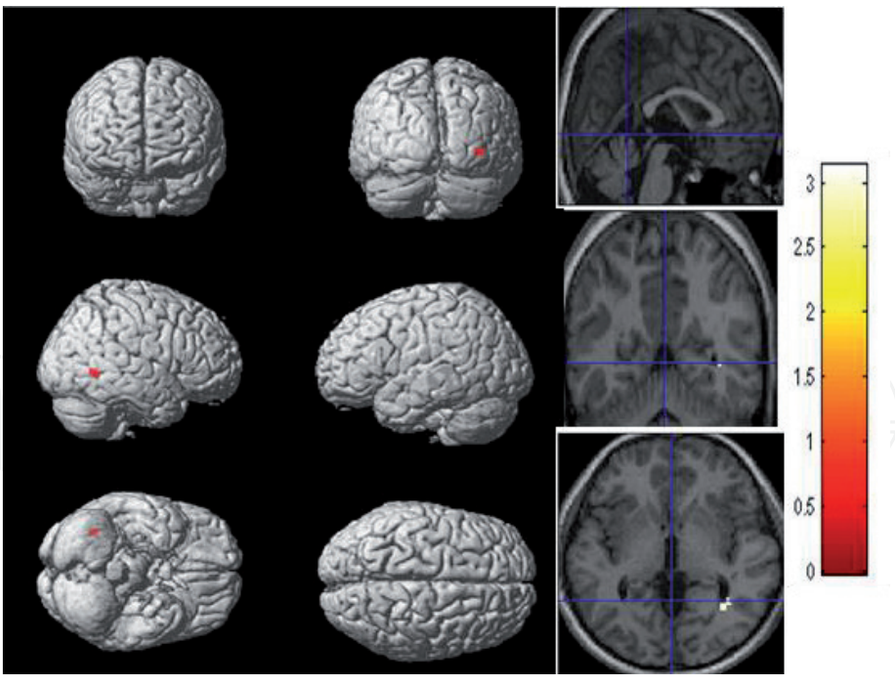


Figure 2.
BOLD activation in intergroup comparison between Patients vs. Controls during picture naming task ($p < 0.001$, cluster threshold = 10).

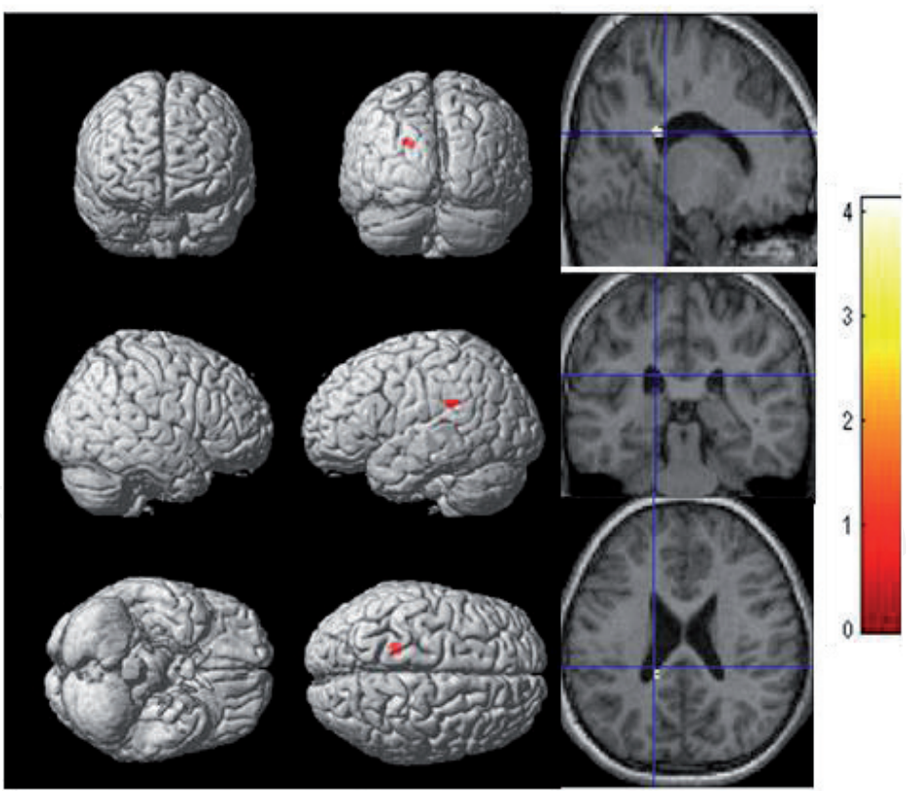


Figure 3.
BOLD activation in intergroup comparison between Patients vs. control during complex sentence reading task ($p < 0.005$, cluster threshold = 10).

10. Management strategies

There are various educational intervention and programs are available to address dyslexia which include regular teaching in small group, a learning support assistant like a specialist teacher, policy interventions etc. The basic strategies of intervention

focus on phonemic skill which include the ability to identify and process word sounds. It include recognize and identify sounds in spoken words such as recognize that even words such as 'RAT' are actually made up of 3 sounds: 'R', 'A', and 'T'. It also include combining letter to create words, and over time, use the words to create more complex sentences, practice reading words accurately to help them read more quickly, monitor their own understanding while they read. The Orton-Gillingham program include special skill which teaches the patient how to match letters with sound and also to recognized letter sound in the words. In the multisensory instruction process the patients were instructed how to use all the senses (touch, sight, hearing, smell, and movement) – to learn new skills. For example, they might run their finger over letters made out of sandpaper to learn how to spell. There are some laws in in school which priorities these children called Individualized Education Plans (IEP). This IEP outlines special services the child needs to make school easier. These might include extra time to finish tests, audio books or text-to-speech—a technology that reads words out loud from a computer or book [46].

11. Besides these there are structural remediation techniques

1. Alphabetic orthography (Henry MK, 1998) [47]

It has multisensory design in which instruction has visual, auditory, and kinesthetic or tactile elements. It is generally believed that such forms of instruction are more effective for such patients. Birsh (2005) [48] and Connor (2007) [49] highlighted the importance of "explicit instruction for remediation as well as the need for intensity that is completely different from regular classroom instruction". The dyslexic needs structured and sequential interactive activities, close monitoring, connecting the known with the new and sufficient time for practice of new skills which would be in use to build automaticity and fluency. They found that the ideal size of the instruction group would be 1:! And 1:3.

2. Academic remediations [50, 51]

It includes appropriate remedial instruction in a structured literacy approach

- Phonology: developing skill in form of rhyming, counting words, clapping syllables in spike words.

- Sound-Symbols Association: In include developing skill to map the phonemes to symbols which could be taught into two direction such as visual to auditory (reading) and auditory to visual (spelling).

- Syllabus Instruction: The concept behind is that a syllabus is a unit or a written language with one vowel sound. So, the instructions must include the 6 basic syllable types in English which include closed, vowel-consonant-e, open, consonant-le, r-controlled, and vowel pair.

- Morphology: In morpheme (the smallest unit of meaning in any language) the base of the words, roots, prefixes and suffixes are considered.

- Syntax: The set of principle which help in sequencing and functioning the words in sentences with concepts of grammar, sentences variation and mechanics of language.

Besides these in include semantics, systematic, cumulative and explicit instruction which in teaching with through interaction with students.

3. **Class room recommendation** (Dyslexia Style guide from the British Dyslexia Association Archived 2013) [52]

The oral testing, untimed tests, audiobooks, eliminate or reduce spelling tests, accept dictated homework. It emphasis on such activities in which students are more active such as sports stories, biographies famous persons, inventors, musicians etc. Recommendation were using appropriate layout, large front size, line space (1.5) and a clear font (sans-serif fonts).

Now there is the concepts that each country has adopted and developed a writing system of choice which are convenient to their people. They have their own statutes relating to the provision of education, and special educational needs. There are various Non-Government Organization (NGOs) and agencies provide many independent and voluntary support in this regards.

12. Conclusion

SLD is a disabling academic problem in children with neurobiological origin. The entity of reading disorder is heterogeneous with respect to its a etiology as understood by the number of biological and neuropsychological theories postulated for the explanation and also with respect to the extent and type and manifestation either because of the different types of disabilities. Many reasons have been cited for its causation starting from genetic defects, perinatal insults, and metabolic abnormality to deficits in the information processing of the central nervous system. According several studies conducted in the past the entity of reading disorder is widely unrecognized and the affected children lacked earlier detection and appropriate intervention.

Author details

Sambhu Prasad[1*] and Rajesh Sagar[2]

1 Department of Psychiatry, All India Institute of Medical Sciences, Patna, India

2 Department of Psychiatry, All India Institute of Medical Sciences, New Delhi, India

*Address all correspondence to: sambhu3011@gmail.com

IntechOpen

© 2021 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. CC BY

References

[1] Shapiro BK, Gallico RP. Learning disabilities. Pediatr. *Clin. North Am.* 1993 Jun;40(3):491-505.

[2] Shaywitz SE. Dyslexia. New England. *Journal of Medicine.* 1998;338(5):307-312.

[3] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders 4th ed Washington, DC (1994).

[4] Morgan WP. A case of congenital word blindness. BMJ. 1896;2:378

[5] Castles A, Coltheart M. Varieties of developmental dyslexia. Cognition. 1997;47:149-180.

[6] Dejerine J. Sur un cas de cécité verbale avec agraphie, suivi d'autopsie. C. R. Société du Biologie. 1891; 43:197-201.

[7] Dejerine J Contribution a l'etude anatomo-pathologique et Clinique des differentes varietes de cecite verbale. Memoires de la Societe de Biologie.1892;4:61-90.

[8] Lewis C, Hitch G J, Walker P. The prevalence of specific arithmetic difficulties and specific reading difficulties in 9 to 10 year old boys and girls. Journal of Child. *Psychology and Psychiatry.* 1994; 35 (2): 283-292.

[9] Roongpraiwan R, Ruangdaraganon N, Visudhiphan P, Santikul K Prevalence and clinical characteristics of dyslexia in primary school students. J Med Assoc Thai. 2002; 85(4):1097-1103.

[10] Nilesh Shah. Attention Deficit Hyperactivity Disorder. Pediatric Clinics of India, July 2005, Vol. 40, No. 3, 205-212.

[11] Sadhu R, Mehta M, Kalra V, Sagar R, Mongia M et al. Neurological Soft Signs in Indian Children with Specific Developmental Disorders of Scholastic Skills. *J. Indian Assoc. Child Adolesc. Ment.* Health 2008; 4(3):62-66.

[12] Agarwal KN, Agarwal DK, Upadhyay SK, Singh M. Learning disability in rural primary school children. Indian J Med Res 1991. 94:89-95.

[13] The ICD-10 classification of mental and behavioural disorders, clinical descriptions and diagnostic guidelines, WHO, Geneva 1993

[14] Diagnostic And Statistical Manual Of Mental Disorders (DSM-5) 5Ed. American Psychiatric Association, 2013

[15] Doehring DG, Hoshko IM, Bryans BN. Statistical classification of children with reading problems. Journal of Clinical Neuropsychology. 1979;1(1):5-16.

[16] Bakker DJ. Neuropsychological Classification and Treatment of Dyslexia. J Learn Disabil. 1992; 25(2):102-109.

[17] Rourke, B P. Socio-emotional disturbance in learning disabled children. *Journal of Consulting and Clinical Psychology*. 1988; 56: 801-810

[18] Rourke B P. Nonverbal learning disabilities, socioemotional disturbance, and suicide: A reply to Fletch, Kowalchuk and King, and Bigler. J Learn Disabil. 1989; 22:186-187.

[19] Rourke B P, Ahmad S A, Collins D W et al. Child clinical/pediatric neuropsychology: Some recent advances. Annu Rev Psyehol. 2002; 53: 309-339.

[20] Fisher S, DeFries JC. Developmental dyslexia: Genetic dissection of a complex cognitive trait. Nat Rev Neurosci. 2002; 3:767-780.

[21] Nicolson RI, Fawcett AJ, Dean P. Developmental dyslexia: the cerebellar deficit hypothesis. *Trends Neurosci*. 2001;Sep 1(24):508 –511.

[22] Stein J. Visual motion sensitivity and reading. Neuropsychologia. 2003;41(13):1785-1793.

[23] Ramus F, Rosen S, Dakin S, Day B, Castellote J, White S, et al. Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults. Brain. 2003;26:841– 865.

[24] Tallal P, Miller S, Fitch R Neurobiological basis of speech: a case for the preeminence of temporal processing. Ann NY Acad Sci. 1993; 682: 1451-1460.

[25] Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci USA*.1991; 88: 7943-47.

[26] Galaburda AM, Menard MT, Rosen GD. Evidence for aberrant auditory anatomy in developmental dyslexia. PNAS. 1994 Aug 16;91(17):8010-8013.

[27] Pugh KR, Mencl WE, Jenner AR, et al. Functional neuroimaging studies of reading and reading disability (developmental dyslexia). Ment Retard Dev Disabil Res Rev. 2006; 6: 207-213.

[28] Cohen L, Lehericy S, Chochon F, et al. Language-specific tuning of visual cortex? Functional properties of the visual word form area. Brain. 2002; 125**:** 1054-1069.

[29] Price CJ. The functional anatomy of word comprehension and production. Trends Cogn Sci. 2001; 2: 281-288.

[30] Paulesu, E., Demonet, J.F., Fazio, F., McCrory, E., Chanoine, V., Brunswick, N., Cappa, S.F., Cossu, G., Habib, M., Frith, C.D., Frith, U.,. Dyslexia: cultural diversity and biological unity. Science. 2001;291: 2165-2167.

[31] Laasonen, M., Tomma-Halme, J., Lahti-Nuuttila, P., Service, E., & Virsu, V. Rate of information segregation in developmentally dyslexic children. Brain & Language. 2000;75(1):66-81.

[32] Vellutino FR, Fletcher JM, Snowling MJ and Scanlon DM. Specific reading disability (dyslexia): what have we learned in the past four decades? J Child Psychology and Psychiatry. 2004; 45(1):2-40.

[33] Erin D. Bigler, Sherstin Mortensen, E. Shannon Neeley, Sally Ozonoff, Lori Krasny, Michael Johnson, Jeffrey Lu, Sherri L. Provencal, William McMahon & Janet E. Lainhart Superior Temporal Gyrus, Language Function, and Autism, *Developmental Neuropsychology.* 2007; 31:2, 217-238.

[34] Shaywitz B A, Fletcher J M, Gonzales, J E J and Espinel A I G. Strategy choice in solving arithmetic word problems: Are there differences between students with learning disabilities, G-V poor performance and typical achievement students?. Learning Disability Quarterly. 2002; 25: 113-122.

[35] Temple, E., Poldrack, R. A., Protopapas, A., Nagarajan, S., Salz, T., Tallal, P., et al. Disruption of the neural response to rapid acoustic stimuli in dyslexia: evidence from functional MRI. *Proc Natl Acad Sci USA*.2000; 97(25): 13907-13912.

[36] Brown WE, Eliez S, Menon V, Rumsey JM, White CD, Reiss AL. Preliminary evidence of widespread morphological variations of the brain in dyslexia. Neurology. 2001 Mar 27;56(6):781-783.

[37] Helenius P, Tarkiainen A, Cornelissen P, Hansen PC, Salmelin R. Dissociation of Normal Feature Analysis

and Deficient Processing of Letter-strings in Dyslexic Adults. Cereb. Cortex. 1999 Jul 1;9(5):476-483.

[38] Johanna Pekkola,,Marja Laasonen, Ville Ojanen, Taina Autti, Iiro P, Teija Kujala, Mikko Sams Neuroimage Perception of matching and conflicting audiovisual speech in dyslexic and fluent readers: An fMRI study at 3 T 2006; 1 February (29)Issue 3:797-807.

[39] Martin Kronbichler, Heinz Wimmer, Wolfgang Staffen, Florian Hutzler, Alois Mair, and Gunther Ladurner. Developmental Dyslexia: Gray Matter Abnormalities in the Occipitotemporal Cortex. Human Brain Mapping. 2008;29:613-625

[40] Quaglinoa V, Bourdina B, Czternastya G, Vrignaudb P, Fallc S, Meyerc M E, Berquind P, Devauchellee B, de Marcoc G, Differences in effective connectivity between dyslexic children and normal readers during a pseudoword reading task: An fMRI study. *Clinical Neurophysiology* 2008; : 73—82.

[41] Vera Blau, Nienke van Atteveldt, Michel Ekkebus, Rainer Goebel, and Leo Blomert Reduced Neural Integration of Letters and Speech Sounds Links Phonological and Reading Deficits in Adult Dyslexia. *Current Biology* 2009; March 24(19): 503-508.

[42] Richlan F, Sturm D, Schurz M, Kronbichler M, Ladurner G, et al. A Common Left Occipito-Temporal Dysfunction in Developmental Dyslexia and Acquired Letter-By-Letter Reading? *PLoS ONE.*2010; 5(8): e12073.

[43] Rimrodt S L, Clements-Stephens A M, Pugh K R, Courtney S M, Gaur P, Pekar J J, and Cutting L E, Functional MRI of Sentence Comprehension in Children with Dyslexia: Beyond Word Recognition. Cereb Cortex. 2009; 19(2): 402-413.

[44] Li Liu , Wenjing Wanga, Wenping You, YiLi , Neha Awati, XuZhao, James R. Booth, Danling Peng Similar alterations in brain function for phonological and semantic processing to visual characters in Chinese dyslexia. Neuropsychologia. 2012; 50: 2224-2232.

[45] Prasad S, Sagar R, Kumaran S, Mehta M: Study of Functional Magnetic Resonance Imaging (fMRI) in Children and Adolescents with Specific Learning Disorder (dyslexia): Asian Journal of Psychiatry 50 (2020) 101945

[46] Ritchey KD, Goeke JL. "Orton-Gillingham and Orton-Gillingham Based Reading Instruction: A Review of the Literature". The Journal of Special Education. 2006; **40** (3): 171-183.

[47] Henry MK. "Structured, sequential, multisensory teaching: the Orton legacy". Annals of Dyslexia. 2007; 48: 3-26.

[48] Birsh JR. Research and reading disability. In Judith R. Birsh (Ed), "Multisensory Teaching of Basic Language Skills" Paul H. Brookes Publishing Co. Baltimore, Maryland. 2007; 16-17.

[49] Connor CMD, Morrison FJ, Fishman BJ, Schatschneider C, Underwood P. "THE EARLY YEARS: Algorithm-Guided Individualized Reading Instruction". Science. 2007; **315** (5811): 464-465

[50] Reid G. Dyslexia 2nd edition. New York: Continuum International. (2007); 41-95

[51] Kuster S M, van Weerdenburg M, Gompel M, Bosman A M T. "Dyslexie font does not benefit reading in children with or without dyslexia". Annals of Dyslexia. 2018; 68 (1): 25-42.

[52] Dyslexia Style guide from the British Dyslexia Association Archived, Wayback Machine 2013.