http://dx.doi.org/10.23925/2176-2724.2018v30i4p791-801

The historical track of studies and research on dyslexia: the search for the understanding of the phenomenon

A trajetória histórica dos estudos e pesquisas sobre a dislexia: a busca pela compreensão do fenômeno

La história de la carrera de estudios y investigaciones sobre la dislexia: la búsqueda de la fenómeno de entendimiento

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Abstract

The aim of this article is showing historical development of the concept of dyslexia through an analysis of constituted researches. As it is possible to verify, studies about learning disabilities of reading and writing have emerged from Neurology Studies, which moved to the other field of medical area without find the etiology of the phenomenon. This fact may have contributed to the proposition of different theories and hypothesis, which does not confront and also does not complement each other. That promotes controversies and tension to the field. At last, this study shows the relevance of studies, which evaluate external facts to dyslexic children, such as their culture context, experiences with writing, teaching methodology, with the purpose of to construct an overview of this phenomenon of learning.

Keywords: Dyslexia; Learning Disorder; Special Education; Education; Learning Disabilities; Educational Evaluation

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Authors' contributions:
PO -This article is a cut from the studies of the thesis "Portraits of Dyslexia in Brazil: bibliographic analysis of the period from 2002 to 2014" developed by this author.
CBFL - Adviser of the research that resulted in the thesis "Portraits of Dyslexia in Brazil: bibliographic analysis of the period from 2002 to 2014".

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Received: 03/04/2017
Accepted: 15/10/2018

Resumo

O objetivo deste texto é apontar o desenvolvimento histórico do conceito de dislexia por meio de uma breve análise da trajetória dos estudos e pesquisas constituídos. Conforme é possível constatar, os estudos sobre as dificuldades de aprendizagem da leitura e da escrita emergiram a partir de estudos da Neurologia, migrando para outros campos dentro da área médica, sem localizar a etiologia do fenômeno. Este fato pode ter contribuído para a proposição de teorias e hipóteses diferentes entre si, que não se confrontam e não se complementam, necessariamente, trazendo controvérsias e tensões ao campo. Finalizamos apontando a necessidade de estudos que avaliem fatores externos às crianças diagnosticadas como disléxicas, como a cultura na qual estão inseridas, suas vivências com a escrita, a metodologia de ensino aplicada, entre outros, a fim de se construir uma visão integral deste fenômeno da aprendizagem.

Palavras-chave: Dislexia; Transtorno da Leitura; Educação Especial; Educação; Dificuldade de Desenvolvimento da Leitura; Avaliação Educacional.

Resumen

El objetivo de este texto es mostrar el desarrollo histórico del concepto de dislexia a través de una análisis corta de la trayectoria de los estudios y pesquisas constituidos. Como es posible verificar, los estudios acerca de las dificultades de aprendizaje de la lectura y de la escrita emergieron a partir de los estudios de la Neurología, cambiando para otros campos en el área medica sin localizar la etiología del fenómeno. Esto puede haber contribuido para la proposición de teorías e hipótesis diferentes entre si, pero que no se confrontan tampoco se complementan, promoviendo controversias y tensiones al campo. Finalizamos mostrando la necesidad de estudios que evalúen factores externos a los niños diagnosticados como disléxicos, la cultura en la cual están insertadas, sus vivencias con la escritura, la metodología de enseñanza aplicada, entre otros, con la finalidad de se construir una visión integral de este fenómeno del aprendizaje.

Palabras claves: Dislexia; Trastorno de la lectura; Educación Especial; Educación; Dificultad en el desarrollo para leer; Evaluación de la Educación.

Introduction

Dyslexia has been defined as a neurobiological and hereditary disorder or difficulty in the acquisition of written language, whose symptoms emerge within a normal intelligence condition, with no shortcomings, enough schooling, and access to social and cultural opportunities[1]. Such condition is provided in the tenth edition of the International Classification of Diseases - ICD-10[2] under the sub-section F81.0 - Specific Reading Disorder and within the subcategory of Specific Developmental Disorders, which includes the F81 category - Behavioral and Developmental Disorders.

According to the ICD-10[2], this disorder is a specific and significant impairment of reading ability, reading comprehension, word recognition, oral reading, and spelling. The disorders and difficulties are noticeable from the earliest stages of development and extend through adolescence and they may be accompanied by emotional and behavioral problems[2].

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders*[3], which brought together Mathematics, Reading and Writing Disorders in the Specific Learning Disorders category, assumed the same conceptions and concepts of dyslexia as those provided in the ICD-10 (2007) and also provided the following guidelines for diagnosis: at least one of the symptoms must be present for a minimum of six months; inaccurate, slow and laborious reading; difficulties with the sounds of words and in understanding reading; difficulties with spelling; difficulties in producing written expressions; difficulties with numbers, numerals, mathematical calculations and concepts; academic skills below expectations; and increasing difficulties as school demands require more and more the skills involved.

The etiology of dyslexia can be regarded as quite complex. Some researchers have sought to

understand it as an Auditory Processing Deficit[4]. Others have understood it as a Visual Processing Deficit[5]. However, the hypothesis of a deficit in speech-language processing has been the most studied and applied approach in a significant number of research[6].

The lack of consensus among researchers on the possible cause of dyslexia has the reflexes of its historical trajectory of studies and research. As it can be noticed, researchers have analyzed the neurological and cognitive processes in order to locate deficits that may be responsible for learning disorders. And although it is recognized by relevant international documents, the researches related to this field are trying to understand the source of the learning difficulties of written language presented through the understanding of the anatomical and physiological functions of the brain.

Therefore, the aim of this article is showing a brief analysis of the historical development of the concept of dyslexia by indicating the way in which different fields of study entered and were articulated in an attempt to locate the source of the disorders and to find a solution to them.

The search to understand the phenomenon of reading and writing difficulty

Firstly, it should be noted that the description of the historical track of the development of the dyslexia concept required a thorough research work. For this purpose, documents of different natures were analyzed, such as books, theses, dissertations, articles, essays, among others. This was done because the researchers of this object of study chose different historical moments of the constitution process of Neurology as a science to establish the starting point of their research. Most of the time, their choices were based on the role played by their field of research in dyslexia and on the contributions provided to the elucidation of the problem, disregarding the scientific findings of other related areas.

For this reason, dyslexia became a research area with many starting points, advancing to many possibilities that do not face each other and do not complete each other, trying only to explain the phenomenon in a parallel and non-articulated way.

Another relevant aspect that must be mentioned is the possibility of conceiving that the definition of the dyslexia concept can be recorded in the Neurology field, since many studies developed on the relations between the anatomy and physiology of the central nervous system conducted in the development of such field were essential to the design of oral and written language studies.

In a way, the Neurology studies field was driven by studies that sought to understand the sequels presented by patients that survived to brain injuries caused by work-related accidents, war-fighting, and strokes. The tests and studies of these injuries led neurologists to investigate and list the characteristics and symptoms of the reported losses to the regions affected by the injuries, assigning the responsibility for the disturbed processes to these areas[7,8]. Among the many sequels resulting from these injuries, those related to speech and language were the most interesting for physicians, leading to neurological studies in order to understand the functioning of brain processing.

García[8] reported that we can credit the origins of Neurology to the studies of the neuroanatomist and physiologist Franz Joseph Gall (1758-1828), who performed observations and postmortem studies by 1800 on adult aphasic individuals. Gall became known as the author of the brain anatomy by describing its morphology and major nerve structures, as well as the difference between gray and white matter. His work provided a significant advance in the discretization of the different regions of the brain and the characterization of its specific functions stating that certain human capacities have particular and well defined locations[7,9,10].

Gall's pioneering studies provided an opportunity to several researches that brought essential knowledge on the anatomy and physiology of the human brain, such as the research of the French anatomist physician Paul Broca (1824-1880) and the German psychiatrist Karl Wernicke (1848-1905).

According to Luria[7], 1861 should be considered as the date of birth of the studies on mental processes disorders, when Broca described the brain of a patient with a marked motor speech disorder (expressive) and showed that such a problem was due to injuries present in the posterior third of the inferior frontal gyrus of the brain[9]. In this way, Broca indicated that "the left posterior inferior frontal gyrus is the 'center for the motor images of the words' and that an injury in this region leads to a characteristic type of expressive speech

loss"[7], which he called first as aphemia and, later, as aphasia

Twelve years later, in 1873, Karl Wernicke (1848-1905), a German psychiatrist, described the effects of injuries on the left posterior superior temporal gyrus, which caused a condition of opposite nature to that described by Broca, leading to a loss of the ability to understand speech hearing with no impact to the expressive speech (motor)[11]. Based on these studies, Wernicke indicated that "the left posterior superior temporal gyrus is the 'center for the sensory images of the words'", that is, it would be the center for speech understanding[7].

The works conducted by Broca and Wernicke were key to understand the speech and language sequels that resulted from injuries in these brain areas, as well as for the description of their symptoms and characteristics, which led to a growing interest in the field of research.

As for the relationship between brain injury and loss of reading ability, according to[12], there is a record in 1676 of the German physician Johann Schmidt (1649-1690) on the loss of the reading ability in a 65-year-old man after a stroke. However, different researchers assign the pioneering spirit to the work of Joseph Jules Dejerine (1849-1917), a French neurologist, who described the difficulties of an adult who lost the reading ability, but preserved the ability to understand and express verbally, after a brain injury also caused by a stroke. The clinical case described by Dejerine occurred in 1887 and he called the phenomenon as pure verbal blindness, thus suggesting the existence of a visual center for letters located in the left occipitotemporal region[13].

Years later, around 1891, the *pure verbal blindness* term was redefined as *pure alexia* or as *alexia with or without agraphia*, in order to describe the reading impairment without the writing impairment, contributing to the behaviors description of individuals who were able to write, but not to read, or who couldn't write and read[11].

Sally Shaywitz, a researcher and professor of Child Neurology at Yale University (USA), stated in her book *Overcoming Dyslexia: A New and Complete Science-Based Program for Reading Problems at Any Level* (2008) that the observation of subjects with good vision and intelligence and who lost their reading skills due to brain injuries dates back to the second half of the 17th century. In this book, Shaywitz[12] described the first impressions of different physicians with respect to the loss of these abilities, which occurred from studies that occurred in parallel with important findings on the brain anatomy and physiology.

According to the researcher, in 1872 - that is, a year before the publication of Wernick's findings - Sir Willian Broadbent (1835-1907), a British neurologist, reported a case of acquired alexia from a patient who had great difficulty in referring to common objects, in addition to his difficulty to read words. Shaymitz[12] reports that although Broadbent's work provided important contributions on reading difficulties, these difficulties were given a more elaborate description and a term that would define them only in 1877, with the German physician Adolf Kussmaul (1822-1907): *wortblindheit*, which in German literally means *verbal blindness*.

Kussmaul suggested the possibility of a total blindness to the written text even with the vision, intellect and speech integrity verified. The German physician still restricted the clinical question of verbal blindness to only "an isolated condition affecting the ability to recognize words and read text, but with both intelligence and expressive language intact"[12]. He went even further in tracing the cases to lesions in the back of the brain, around the left angular gyrus.

Based on these data, it is possible to note that ten years before the French neurologist Dejerine, the German physician Kussmaul already conducted similar studies and coined the term *word-blindness* in order to define the clinical condition of those who lost written language abilities. The studies conducted by Kussmaul were essential to further researches and the subsequent publication of a monograph entitled "*A Particular Kind of Word-Blindness*" by Rudolf Berlin (1833-1897), another German physician, in 1887, that is, at the same time as Dejerine.

Shaywitz[12] reported that Berlin described six cases that he personally observed over a period of twenty years, and that he became the first researcher to use the term *dyslexia* to refer to a finding in adults who lost their abilities to read secondary to a specific brain lesion. Berlin understood that "[...] if the lesion was complete, there would follow an absolute inability to read, acquired alexia. If the disruption was only partial, however, there may be very great difficult in interpreting written or printed symbols (dyslexia)"[12]. According to Berlin, dyslexia could be considered as a minor part of a language

disorders family - aphasia, in which the difficulties are related to the understanding or production of spoken language, or in the understanding and production, in mixed aphasia cases[12].

The inclusion of ophthalmologists in the field of studies related to dyslexia is credited by Shaywitz[12] to the denomination of verbal blindness, which led many cases to be referred to these professionals. James Hinshelwood (1859-1919) was one of the most prominent ophthalmologists to enter this field of study.

According to Shaywitz[12], in December 1895, Hinshelwood published a research report in *The Lancet*, a prestigious medical journal, on a 58-year-old man with high education level who was not able to read printed texts regardless of the size of the print types, thus showing that his difficulty was not related to the absence of visual acuity. Hinshelwood also prepared another study in which he described problems with reading skills in members of the same family and he discussed the possibility of the presence of a genetic factor associated with the disorder, being thus incurable but adaptable according to the use of certain appropriate methods of teaching[11].

According to Shaywitz[12] and Rodrigues and Ciasca[11], the work prepared by Hinshelwood was essential to the research of W. Pringle Morgan[1], an English ophthalmologist who found a similar condition of alexia in a teenager who presented great difficulties in reading and writing.

Based on Hinshelwood's studies, in 1896 Morgan published an article in the British Medical Journal describing the case of a fourteen-year old boy, Percy F., who, although he had no lower characteristics when compared to his colleagues, he presented significant difficulty as he learned to read and write. According to Shaywitz[12], Morgan was the first researcher to relate Hinshelwood's findings to the learning difficulties in healthy children and adolescents, describing *verbal blindness* - which now is referred to as dyslexia - as we know it today: average intelligence level, no sensory impairment, difficulty in acquiring written code and excellent oral performance in school contents. Therefore, the detailed report provided by Morgan changed the definition of the phenomenon to *congenital verbal blindness*.

Although Hinshelwood's early studies involved patients who acquired the difficulty, this researcher focused on studies on *congenital verbal blindness* following the publications from Morgan, and he published articles and monographs reporting several remarkable cases of children and adolescents who presented similarity of characteristic difficulties in relation to reading acquisition throughout their lives. Shaywitz[12], further reports that Hinshelwood's work has provided what has been considered the central concept of dyslexia in his rather elaborate and detailed studies: the unexpected difficulty of reading for the age.

From a practical perspective, this means that the difficulty in reading is somewhat isolated and limited and, according to Hinshelwood, it reflects a "local", rather than widespread, cerebral dysfunction. A child who is slow in all cognitive abilities would not be classified as dyslexic; a dyslexic child must have strengths with regard to cognition, not just problems in reading functions[12].

The researcher also pointed out that some of the contributions provided by Hinshelwood to the studies related to dyslexia remain to this day, such as the indication of high prevalence in school-aged children, the concern with the proper disclosure of information so that physicians can reach a diagnosis with greater ease, the concern with the clinical character of the diagnosis, based on information on the patient and his/her history, and the access to special education for diagnosed individuals.

Shaywitz's[12] study still mentions the work of the ophthalmologist E. Nettleship[2], which was published in 1901, on *congenital verbal blindness* in children from rich and poor families. In his study, Nettleship reported that the detection of the *congenital verbal blindness* in children of schooling parents was easier, since parents in these cases were more attentive to their children's reading ability. However, the diagnosis can be understood as more difficult in children from disadvantaged situations of schooling, since their difficulties were not even noticed.

1. Data on this researcher with respect to his life and death are quite inconsistent; therefore, it is not possible to confirm specific dates.

2. Data on this researcher with respect to his life and death are quite inconsistent; therefore, it is not possible to confirm specific dates.

From the early 20th century, studies on dyslexia - still referred to as *congenital verbal blindness* - were given a new impetus by reports of studies conducted in the Netherlands and Germany in 1903 and in France in 1906. The first studies in the Americas were published in Argentina in 1903 and in the United States in 1905, with the publications of the ophthalmologists W.E. Bruner[3], Edward Jackson[4], and E. Bosworth McCready[5], who published his research in 1909 (ibid., 2008).

As reported by Shaywitz[12], McCready was the first researcher to relate *congenital verbal blindness* to creativity and mental superiority, when he reported cases in which an individual diagnosed by him reached a profession with high reading demand, and another case with a patient who became a judge. Some researchers are currently linking dyslexia to high levels of intelligence; however, the data is poorly consistent.

A significant change (or reorganization) in the field of knowledge on *congenital verbal blindness* started on the publication of the studies developed by the American physician Samuel T. Orton (1879-1948). There is no consensus among the several researchers on their expertise and the dates of their publications, even so they all consider the value of his contributions and his strong influence on studies related to dyslexia.

In his research on learning disorders, Orton analyzed more than a thousand children in order to understand the genesis of their difficulties and he suggested that these learning disorders were more common than previously thought, reaffirming what had already been suggested by Hinshelwood. According to Orton, written language disorders would be associated with a problem to recognize the orientation and sequence of the letters in the words, although visual perception and spatial orientation were shown to be intact. In this way, he indicated that the difficulties resulted from a deficit in the development of cerebral hemispheric dominance[14,15,16].

According to the Cerebral Dominance Theory proposed by Orton,

> Until lateralization is established in the motor plan, (...), we can find inversions (omissions, substitutions, additions, confusions, repetitions, etc.) in reading. Since words are recorded in the non-dominant hemisphere, inversions arise and as a consequence the individual may change "b" by "d", "q" by "p", "u" by "n", "6" by " 9, "or their combinations, such as reading "*dão*" as " *bão*", or "*pai*" as "*qai*", or even reading "69" as "96", etc. (strephosymbolia cases). With no hemispheric dominance, the child may experience great confusion, and therefore may have difficulties in learning to read[14].

In order to replace the term *congenital verbal blindness*, Orton proposed the term *strephosymbolia* - distorted symbolization - aiming to highlight the main characteristic of the phenomenon in his opinion: inversions, exchanges and omissions of letters. In addition, he intended to replace the existing term, since he believed that this was an anomaly in the predominance of the cerebral hemispheres and not from lesions in specific regions of the brain[16]. According to Fonseca[14], Orton proposed educational pedagogical methods based on the role of language for the development while considering the functional asymmetry functions of the human brain.

Orton suggests that only at six years of age the child would be fit to the language symbolic representation systems due to the reach of the anatomical or physiological maturity of the angular gyrus, which is understood as the center of reading or the "privileged center of sensorineural association located in the first temporal sulcus of the dominant hemisphere"[14]. He postulated the following theorems based on postmortem studies: a) considering that there are specific related to language, the location of an injury is more important than the number of affected tissues; b) lesions in the left hemisphere cause speech or reading disorders, while similar lesions in the right hemisphere (non-dominant for speech and reading) do not cause language disorders[14]. Orton also suggested the heredity of the disorder.

Considering his postulates, the method proposed by Orton suggested the use of the relationship between the sound and the symbol (phoneme-grapheme) from an auditory stimulation (sound/phoneme), followed by visual stimulation (grapheme), seeking a verbal repetition of the sound of the letter. He also used the gesture of tracing the letter with his index finger (tactile-kinesthetic approach) at the same time that he pronounced the sound of the letter.

3. Same as above.
4. Same as above.
5. Same as above.

> First, the sounds of the consonants with the various vowels and their proper associations; then entering the exact sequences from left to right, as in the words. Thus, according to Orton, the child would progress in oral reading using "units" and phonetic combinations, syllables with multiple meanings, word families, prefixes and suffixes, simple derivations and grammatical constructs, etc.[14].

Still as reported by Fonseca[14], Orton and his colleagues criticized the use of the global method of literacy for children with learning disabilities in written language and proposed the early identification and preventive intervention, suggesting their inclusion in school since child education.

The recognition of Orton's work resulted in the creation of the Orton Society, in 1949, in the United States - which is now called the International Association of Dyslexia (IDA). This entity has influenced the creation of many associations and institutions for the study and dissemination of dyslexia and its forms of care in different countries. The Brazilian Dyslexia Association (ABD), which was established in 1983, fits this profile.

During the following twenty years after the establishment of the Orton Society, the understanding that dyslexia was a result of visual deficits (a legacy of the early conceptions based on the testimony of the first "patients") or resulted from problems in cerebral hemispheric dominance influenced research and diagnosis.

Santos and Navas[15] report that Doris Johnson and Helmer Rudolph Myklebust (1910-2008), in the 1960s, were responsible for bringing a new perspective on the causes of dyslexia by relating the reading disorders with deficits in the phonological processing of words. The aforementioned investigators understood that children with reading problems had problems in perceiving the manipulation and use of sounds in speech, as well as in retrieving complex phonological information.[15].

Also in the 1960s, dyslexia was recognized as a specific disorder of written language learning by the National Congress of the United States of America[16]. The recognition by the World Federation of Neurology came in 1968, when it recommended that the term should be applied to the diagnosis of children who couldn't learn to read despite having proper intelligence and education and sociocultural opportunities[12].

The concept that dyslexia would be resulted from a deficit in phonological processing began to be adopted in the 1970s by several researchers influenced by the studies of Ignatius G. Mattingly (1927-2004), Isabelle Yoffe Liberman (1918-1990), and Donald P. Shankweiler, who described the difficulty of children with reading disorders in analyzing the sound components of the spoken word and in mastering the alphabetical concept[15].

In the late 1970s, postmortem studies in the brains of adults who were considered dyslexic conducted by the American physician Albert M. Galaburda and his co-workers showed that the asymmetry of the temporal plane in the left hemisphere tended to be absent in dyslexics, in addition to the presence of an abnormal symmetry in the posterior parietal cortex. The investigators also reported the existence of ectopias (excrescences of neurons) around the temporo-parietal junction[17,18].

According to Galaburda and Kemper[17], the abnormalities found were concentrated in the left hemisphere, presenting a polymicrogyria area (small and multiple turns) in the posterior part of the transverse rotation of Heschl and also in the left temporal plane. This polymicrogyria area is located at the left of the auditory region. They also found dysplasias (abnormal cell growth) throughout the left hemisphere, especially in the parietal, occipital, and temporal lobe regions. The researchers also reported that such abnormalities could be not directly related to dyslexia, since these findings have not been found repeatedly in other studies with dyslexic brains. However, they also suggested that the characteristics of these abnormalities showed that the area related to language was affected, what could imply in relevant dysfunctions.

The auditory processing deficit theory gained in strength from the 1980s. Although it has been studied since the 1960s[19], it was probably driven by the studies of Galaburda and his co-workers in the previous decade.

According to Frota and Pereira[19], a central auditory processing deficit could impair the perception of short and speech sounds, thus causing a deficit in phonological awareness. In this scenario, the deficit in phonological awareness is understood as secondary to the auditory processing deficit, being considered as an effect of the latter[20].

Nowadays, some researchers are trying to understand the auditory processing deficit from the presence of a possible deficit in some of its components. Among the components of auditory processing, Frota and Pereira[19] indicated that the

temporal aspects of sound (duration pattern and sound frequency) have been widely studied due to their possible implications for the development of sound segmentation and comprehension skills, thus influencing the development and the acquisition of written language.

Also in the 1980s, the visual deficit theory - which is considered by many researchers as traditional in studies on dyslexia - has resurfaced, presenting new and more elaborate definitions. As suggested by Ramus et al[20], according to the visual deficit theory, the magnocellular pathway[6] is disrupted, which causes deficits in the information path through the posterior parietal cortex and binocular control. In this perspective, the reduction of the magnocellular sensitivity would lead to instabilities in the binocular fixations.

Breznitz[21] and Oliveira[22] understand that the visual deficit is characterized by less control of eye movements in the voluntary convergence and by the instability in the binocular fixation. Therefore, there would be greater difficulty in the perception of stimulus change and in isolating items during reading.

With respect to the possible relation between the phonological processing and the visual deficit, many researchers relativize the influences of one over the other. While some researchers understand that these deficits are independent, others understand that there is a multimodal relationship between auditory and visual deficits in order to influence phonological awareness[22].

In the 1990s, the relationship of the magnocellular visual deficit theory to reading problems was studied more extensively by Lovegrove and his colleagues[18] due to the development of neuroimaging resources and their application in studies of the functioning brain. In the development of new studies based on the theory of magnocellular deficit, Stein[18] reported that the magnocellular cells are also found in the occipital cortex and can be activated by visual stimuli. According to the researcher, the use of motion sensitivity tests may indicate the presence of deficits that, in this particular case, involve not only peripheral magnocellular neurons, but also the processing center.

Thus, according to Ramus et al[20], the magnocellular dysfunction is not restricted only to the vision, as it can be generalized to other modalities, such as auditory, motor, and, consequently, the phonological processing[7]. In this context and due to its scope, the magnocellular theory is understood by many researchers within a hypothesis of a cerebellar deficit as cause/origin of dyslexia, being called by some as Cerebellar Deficit or Automation Deficit.

The hypothesis of a cerebellar (or automation) deficit, although it has been studied by some researchers since the 1980s, was driven quite significantly from the 1990s with the inclusion of neuroimaging in their research. The cerebellum, which is responsible for balance, muscle tone and voluntary movements, also receives information from the magnocellular cells that bring them from the sensory and motor centers[18, 25]. In this way, when the cerebellum receives information from the sensory pathways, it monitors the movements - including eyes movements - and mediates the interaction of the subject with himself and with

6. According to Couto[23] and Souza, Lacerda, Silveira, Araújo and Silveira[24], the trichromat retina of the primates (those with color vision) have four types of photoreceptors: rods, S cones (sensitivity to blue), M (sensitivity to green) and L (sensitivity to red) cones. These photoreceptors are connected to retinal ganglion cells in the neural circuits in a way that each ganglion cell has a receptor field. Each of these receptor fields is involved in a variable number of photoreceptors in two concentric opposed areas, which are composed by "on" or "off" neurons to the center or periphery of the Central Nervous System. These "on" and "off" designations are related to the depolarization currents (increased chemical permeability of the cell membrane) that photoreceptors receive due to the presence or absence of light. Therefore, those who are stimulated by the presence of light are called as "on", and those who are inhibited by the light are called as "off". Also according to the researchers, the retinal ganglion cells connect to the lateral geniculate body (part of the diencephalon that receives visual information from the optic tract) through the three main processing routes: the parvocellular pathway, which uses 80% of these pathways with midget (very small) cells, whose function is to allow high spatial resolution - although it provides low resolution to moving stimuli; the magnocellular pathway, which uses 10% of the pathways with parasol cells (in the form of webs or umbrellas), whose function is to respond to changes in lighting; and the koniocellular, which uses 9% of the pathways with bistratified and extremely small cells, whose function is to respond to contracting.

7. While both visual deficit and magnocellular deficit theories mention the presence of a disorder in a specific type of cells - in this case, the magnocells - they deal with completely different effects. According to the visual deficit theory, the disorder or dysfunction of the magnocellular pathway promotes the disruption in the binocular fixations, making it difficult to get the stimulus. On the other hand, in the magnocellular deficit theory - also called cerebellar deficit - the effects depend on the location of the affected cells in the cortex, which can lead to deficits in visual processing of contractions/luminance, reaching also areas related to hearing and motor skills, among others.

the environment, and it can interfere in the phonological analysis, participating in this processing[25].

The 1990s were also marked by the genetic studies of dyslexia. According to Svidnicki (2011), several studies have been held in order to identify chromosomal regions that could be responsible for this function through binding studies[8] in affected families[25].

Still as reported by Svidnicki[25], nine chromosomal regions have been identified in the human genome as susceptible to dyslexia and have been studied in studies conducted in different countries. They are the following: DYX1 (15q21), DYX2 (6p21), DYX3 (2p16-p15), DYX4 (6p13-q16), DYX5 (3p12-q12), DYX6 (18p11), DYX7 (11p15), DYX8 (1p34-p36) and DYX9 (Xp27). Genetic research on dyslexia has been developed throughout the world, but its results are still quite superficial.

At present, many other hypotheses have emerged in an attempt to identify the origin and possible remediation of dyslexia. Almeida[25] referred to them in his master's research, reporting that these new studies have led to new perspectives on the etiology of the disorder in the XXI century. In this way, it is possible to consider the following hypotheses: the automation deficit (difficulty in fluent reading); processing rate deficit (difficulty in decoding words); double deficit (combination of the phonological processing deficit with the naming speed deficit); reading balance model deficit (a small disorder in the left hemisphere that may impair the development and understanding of the syntactic and linguistic rules governing language); attention deficit disorder (a deficit in the executive functions that may impair one of the components of the working memory that leads to the attention control); delays in maturation (delayed cortical development); distorted perceptions or disorientation (conceptualizations replace normal sensory perceptions due to a disorientation in cognitive ability); among others[25].

Alves, Siqueira, Lodi and Araújo[28] warned about the perspectives and expectations regarding what several researchers have defined as symptoms of the phenomenon, suggesting that it is essential to broaden the knowledge on dyslexia, but that a single theory could not explain all the complexity of its symptomatology. And while the phonological processing deficit theory is currently presented as the most accepted among the various hypotheses proposed, these researchers reported that it is necessary to consider that this may occur with other deficits, causing the heterogeneity of the symptoms.

In this context, new hypotheses for the etiology of dyslexia will probably arise, making the field of study even more complex and confusing. Therefore, the development of studies and research that try to understand the phenomenon of dyslexia from external factors to the individual is urgent, analyzing the way in which the culture in which it is inserted, its living conditions, its schooling process, among others, may be implying on their acquisition of written language.

It does not mean to deny the existence of neurological disorders; it just means to seek new understandings through perspectives from other fields of knowledge, which can elucidate how certain elements and external factors can negatively influence the teaching and learning processes of written language of healthy children. In addition, these new perspectives may facilitate the establishment of a more comprehensive picture of the phenomenon, thus allowing the construction of truly inclusive pedagogical proposals.

Final Considerations

As it can be noticed, all the historical background of the dyslexia concept is based on the perspective of the presence of neurobiological changes in the individual. Even when subjects without neurological lesions are in the research, the studies turn to the search for biological responses to reading and writing difficulties.

In these studies, the teaching context and the learning status usually are not questioned and most of the theories and hypotheses that try to explain dyslexia end up assuming biological conceptions, keeping the development of the issue within a perspective that is not enough to understand the phenomenon nor able to respond to the educational needs of children.

Many studies in this perspective are still being conducted in the pursuit to understand the etiology

8. Cendes[27] suggested that binding studies are an experimental research methodology that is used to map the locus of a gene which may be responsible for a particular disease. This study methodology is based "on the recombination concept and on the principle that when two genes are close in the same chromosomal pair they do not segregate independently." This methodology also uses the percentage of recombinants in order to identify the chromosomal position of a gene and infer the position of the second.

and the location of the defect that leads to dyslexia, as well as to a description that allows drawing a clear understanding of the phenomenon in order to promote health care actions for the diagnosed children. However, over the years, these actions are becoming only an unfulfilled promise, making children responsible for their school problems, since the difficulty is attributed only to them, excluding the role of the educational system and social relations in the process.

It should be noted that, although many theories and hypotheses have been developed, each one of them does not directly contradict the others. Over more than three centuries of Neurology studies, each new theory or hypothesis on the phenomenon sought to understand it in their own way, trying to find its etiology from the physiological and anatomical study of brain development. However, this diversity of possibilities has shown inconclusive results, failing to explain the plurality of cases that are identified and/or diagnosed as dyslexia.

The absence of conclusive studies and studies for the constitution of a consistent theory that may explain the phenomenon allows the maintenance of dubious and misleading interpretations regarding how subjects relate to the written language, favoring the permanence of this multiplicity of views and hypotheses and associations between them.

In addition, the neurological development study has not proven sufficient to explain the development of typically human behaviors, such as voluntary and intentional actions through the most varied situations promoted by social experiences. In other words, such studies allow us to know the anatomy and physiology of the human brain development; however, they do not explain how we may become part of the culture of the society in which we are inserted and how we humanize ourselves from it.

So, through these aspects, the theories that have been developed and undertaken in the search of the etiology of dyslexia are unable to explain the relations that the subjects establish with the written language, not allowing to reach an understanding on the trajectories assumed by children during literacy, and about how the writing acquisition may change individuals.

These facts give rise to questions on the insistence on the perspective of the biological defect despite the writing social function studies and the way in which they promotes deep social and individual transformations. Therefore, it is essential to analyze the phenomenon according to other scientific perspectives, looking for some answers in the cultural relations of the subjects, and not only in their biological functioning.

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