The evolution of research on dyslexia

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1 History of reading disability

From Cleopatra to Cher, dyslexia has probably been present always during the history of humankind, even before writing systems were developed. We can imagine a caveman unable to understand the paintings in a cave depicting predators, venturing outside to be devoured by a beast. The consequences of dyslexia in our current society may not be as dramatic or life-threatening as I have imagined them in the beginning of humankind, but they are socially important, since they involve learning problems at an early age that can affect the cognitive and emotional development of a child.

The causes of dyslexia are yet unknown, although there exist many theories, some more popular than others among scientists. Perhaps the greatest burden to find the causes of dyslexia has been the lack of a concise definition of this deficit, but recent efforts have tackled this issue. For many years, scientists studying dyslexia have considered a large variety of symptoms as representative of dyslexia, contributing to the lack of convergence on the fundamental causes of this disorder, and to the proliferation of theories describing possible causes to each specific group of dyslexics. In this introductory chapter, I will present a historical perspective of some of these theories.

Dyslexia can not be considered as a qualitative disease, that you may have or not, but as a syndrome, that is, a range of symptoms on which dyslexic subjects experience some relative, quantifiable, difficulty. Obviously, the etiology and diagnosis of dyslexia depends essentially on those symptoms that are considered critical. Nowadays, although there are many plausible theories, many dyslexia researchers agree that dyslexia is a type of learning disability, affecting specifically language and reading skills, more than math

skills, attention deficits, or motor problems. It is important to remember that although dyslexics exhibit quite a variety of different symptoms, they seem to experience deficits in certain skills (i.e., phonological, reading) more often that expected in the population as a whole. Therefore, the current denomination of dyslexia as reading disability.

Given the broad scope of dyslexia research, it is impossible to cover everything in this introduction, and therefore I have decided to discuss some issues but avoid others. For example, although there is evidence of comorbidity between reading deficits, math deficits, attention problems, etc. I have focused entirely on the study of reading and language problems, which are considered the most representative symptom of dyslexia. Indeed, comparative analysis of several definitions of dyslexia revealed that reading deficits was the only common symptom among all of them (Doyle, 1996). Moreover, I have focused on developmental dyslexia, in which reading skills has not matured properly, as opposed to acquired dyslexia, which originates as a brain insult causing disruption of an already established skill, reading in this case.

As an overview of the history of dyslexia, we can propose four stages which, although not clearly differentiated, allow us a tidier review of these historical events. The first stage, the origins of dyslexia, identified the first subjects with reading and language deficits, who were generally acquired aphasic patients, and lasted until the end of the nineteenth century. During the beginning of the study of developmental dyslexia (1895-1950), this condition was discovered and its causes and characteristics started to be analyzed. Next, there was an evolution stage (1950-1970) in which the field of dyslexia opened up to a variety of clinical, research, and educational approaches. Finally, the modern theories (1970-2000) have created the foundations of our current knowledge

about dyslexia.

1.1 Origins

The first writing systems were established perhaps ten thousand years ago and have evolved through the classic cultures (Babylonian, Asian, Greek, Egyptian, Roman) and the Middle Ages. Nonetheless, this means of communication has always been reserved to a very exclusive group of society. Perhaps the invention of the press by Gutenberg in 1493 helped towards the popularity of reading and writing, but still only among an elite group of intellectuals and erudites. The origins of mass reading are much more recent, linked to the birth of an institutionalized public education. Although this idea was already considered during the Illustration, it was never implemented until approximately the end of the nineteenth century. For example, in Great Britain (GB) in 1870, the Forster Education Act guaranteed a basic level of education to all children. This event meant that from then on, educators could observe a large number of children at school, allowing them to identify those with reading problems (i.e., developmental dyslexics). Nevertheless, during the history of humankind there are isolated cases of persons, mainly famous characters, who exhibited certain learning or reading difficulties, although there is no evidence that they could be considered dyslexics, such as: Leonardo da Vinci, Italian inventor (1452-1519); Santa Teresa de Jesús, Spanish nun and saint (1515-1582); Galileo Galilei, Italian scientist (1564-1642); Karl XI, King of Sweden (1655-1697); Hans Christian Andersen, Danish writer (1805-1875); Mr. Krook, character of Charles Dickens' novel "Bleak House" (1852); Napoléon Eugène Louis Jean

Joseph Bonaparte, the Imperial Prince of France, only child of Napoleón III (1856-1879); Auguste Rodin, French sculptor (1840-1917); Thomas Alva Edison, American inventor (1847-1931); Thomas Woodrow Wilson, American politician (1856-1924); Sir Winston Churchill, English politician (1874-1965); Carl Jung, Swiss psychiatrist (1875-1961); Albert Einstein, German scientist (1879-1955); Cher, American actress (1946-) (Adelman & Adelman, 1987; Aaron, Philipps, & Larsen, 1988). Nonetheless, it has not been until the twentieth century when, among a large number of readers, the existence of a small group that exhibits a great difficulty in learning to read has been detected.

The origins of dyslexia in the scientific literature are due to the first findings of language problems, mainly due to acquired aphasia. These aphasic patients sometimes suffered from a loss of reading ability too. Some scientific breakthroughs were necessary before aphasia and dyslexia were related to brain lesions. It was around the sixteenth century when philosophers and physicians decided that the body localization of thought was not the heart but the brain. Of course, we have to credit the work of the Austrian doctor Franz Joseph Gall who, in the beginning of the nineteenth century, suggested that each specific part of the brain had a precise function. Moreover, Pierre Paul Broca (1861, 1865) localized the specific brain areas where language functions might reside.

The first case of loss of reading ability was described in 1676 by the physician John Schmidt. Also interesting was the experience of Professor Lordat, of Montpelier, France, who described how in 1825, he suffered a temporal crisis which make him lose the ability to understand written symbols. To these famous clinical cases followed other cases of aphasics with reading problems: Gendrin (1838), Forbes Winslow (1861), Falret (1864), Peter (1865), Schmidt (1871) and Broadbent (1872). Wilbur (1867) and Berkan (1885)

have been considered sometimes pioneers in the history of developmental dyslexia, but it seems now that their patients were mentally retarded, and their reading problems were simply an aspect of a more general disability (Critchley, 1964).

The first reference of the term dyslexia occurred in 1872 by the physician R. Berlin of Stuttgart, Germany, who used the term to describe the case of an adult with acquired dyslexia, that is, loss of reading ability due to a brain lesion. Short afterwards, Dr. A. Kussmaul (1877) suggested the term "word blindness" to describe an adult aphasic patient who had lost his reading ability. Similarly, Charcot (1887) defined alexia as the total loss of reading ability. Finally, Bateman, in 1890, defined alexia or dyslexia as a form of verbal amnesia in which the patient has lost the memory of the conventional meaning of graphic symbols (Critchley, 1964).

The work of Dejerine was more innovative, and in 1892 he localized the lesion causing these reading problems to the parietal lobe and the middle and inferior segments of the left occipital lobe, including the fibers connecting both occipital lobes. At this time, dyslexia was considered a disability of essentially neurological origin caused by a cerebral trauma, what is nowadays commonly called acquired dyslexia. Nevertheless, there exists another form of dyslexia which is not caused by a sudden brain insult, as the ones described above, but it develops during the growth of the child. In order to discover this developmental dyslexia, it was necessary the existence of physicians or educators who paid attention to the cognitive development of children and adolescents.

1.2 Beginnings

The history of developmental dyslexia began more than one hundred years ago, and it happened in Great Britain. The scientific atmosphere in Britain was very exciting at the end of the nineteenth century, due to a great academic culture, intellectual curiosity, and human and practical resources pushed by a rising economy. The proliferation of academic and professional journals incited scientist to publish articles and create intellectual debates to increase and improve scientific knowledge. Developmental dyslexia saw the light for the first time in one of these journals, and it did it among physicians, specially ophthalmologists. For this reason, during the first stages of the study of developmental dyslexia, it was always described as a disease of the visual system.

On December 21th 1895, James Hinshelwood, an optic surgeon from Glasgow, Scotland, published an article in the journal "The Lancet" on the issue of visual memory and word blindness. This article inspired W. Pringle Morgan, a general doctor of the seashore town of Seaford, to describe the case of an intelligent fourteen years-old boy who could not learn how to read. His article, published in the British Medical Journal on November 7th 1896, is considered as one of the first reports about congenital word blindness, if not the first. In this sense, Morgan is recognized as the father of developmental dyslexia.

Nonetheless, James Kerr, Health Medical Secretary of the city of Bradford, had mentioned a child with word blindness who could spell, in an essay which won the Howard Medal of the Royal Statistical Society in June 1896, a few months prior to Morgan's article. However, Kerr's essay, published in 1897, was on the topic of school

hygiene, and only mentioned word blindness briefly, while Morgan's article was dedicated exclusively to this issue.

Morgan's famous article marked the beginning of a stage of identification of dyslexics, specially by British ophthalmologists, like C. J. Thomas (1905), J. Herbert Fisher (1905), Treacher Collins, Sydney Stephenson (1907), Plate, and Robert Walter Doyne. Reports about patients with reading problems were also published in other countries, like Lechner in the Netherlands (1903), Wernicke in Argentina (1903), Peters (1903), R. Foerster (1904), y Warburg (1911) in Germany, and Schapringer in the USA (1906).

As much as Morgan is considered the father, the ultimate founder and sponsor of the study of dyslexia was Hinshelwood, who between 1896 and 1911 published a series of reports and articles in the medical press describing clinical cases and suggesting its possible congenital nature. Hinshelwood contributed essentially to create a clinical and social awareness necessary to consider dyslexia as a medical issue of greatest importance. In 1917, Hinshelwood published a second treaty on "Congenital Word Blindness", which summarized the current knowledge on the issue. According to him, the defect involved the acquisition and storage in the brain of the visual memories of letters and words. This defect was hereditary, but remediable, and more common in boys. His classification of dyslexics in three groups is also interesting: Alexia, for cases of mental retardation with reading disability; Dyslexia, for common cases of small delays in learning to read; and word blindness, for severe cases of pure reading disability. In his multiple publications, Hinshelwood described the congenital nature of these reading difficulties and tried to find its biological causes.

Hinshelwood's second monograph (1917) started a second phase of these beginnings of

the history of developmental dyslexia, evolving from a mere identification and description of clinical cases, to the analysis and discussion of the syndrome and its components. At about this time, the main core of the study of dyslexia moved from Great Britain to the United States. Nonetheless, the Scandinavian countries deserve a special note, since they soon created a tradition of the study of reading in general, and dyslexia in particular, that has been maintained until now.

The first theories about the causes of dyslexia ranged from brain structural defects, like an inherited aplasia in one or both cerebral angular gyri as suggested by Fisher (1910), to functional deficits, like the idea of Apert (1924) and Pötzl (1924) of a developmental delay in dyslexics. However, during the 1920s and 30s there was a trend against neurological causes of behavior and in favor of environmental explanations.

One of the most important figures in the history of dyslexia was the American neurologist Samuel Torrey Orton, who between 1925 and 1948 modeled the evolution of the study of dyslexia. As Director of Greene County Mental Clinic, in Iowa, he had the opportunity to study the language problems of mentally retarded patients, and afterwards, not only in Iowa, but also at the Neurological Institute of New York and Columbia University, his research focused on language disabilities, studying about three thousand children and adults with this disorder. He first found the correlation between the delay in learning to read and other factors, such as left-handedness, and even left-eyedness. He also found a great number of ambidextrous among language-delayed patients. He observed a large amount of writing and reading errors that were due to inversions of either isolated letters or letters in words (i.e., b for d, was for saw). From these observations, he proposed his "strephosymbolia", or twisted symbols, theory which focused on

reversal errors. He also showed that these deficits ran in families, which was consistent with a genetic nature of the deficit. Orton suggested that dyslexics had a deficient visual perception of letters, possibly due to a brain malfunction, specially in the cerebral hemisphere dominance of one occipital lobe over the other.

Orton and his contemporaries' emphasis on visual problems and reversal errors mystified the popular legend of dyslexia for many years, but nowadays these theories have been contradicted by more recent experimental data. The Orton Society, later renamed Orton Dyslexia Society was founded in the USA short later after Orton's death in 1948, with the goal of promoting the study of the functional and social problems of dyslexics. This society, along with Orton's pupils, has been greatly influential both in the US and in Europe, and has helped improve our knowledge about dyslexia, as well as improving the quality of life of dyslexics, creating special attention and learning centers for dyslexics, and even fighting for their legal rights. This society has grown considerably and has recently become the International Dyslexia Association, it organizes annual conferences, and publishes a journal "The Bulletin of the Orton Society, name recently changed to Annals of Dyslexia.

Meanwhile in Europe the study of dyslexia continued sporadically. Besides a few studies elsewhere, like Ombredanne's who introduced the term dyslexia in the First Congress of Child Psychiatry in Paris in 1937, or MacMeeken's study on Scottish children (1939), the research on dyslexia was almost exclusive to the Scandinavian countries. Edith Norrie, a dyslexic patient herself, founded in 1938 the Word Blind Institute in Copenhagen to diagnose and teach dyslexic subjects. This center was probably the first one of its kind in the world. Hallgren (1950) organized several studies on which he

established the heritability of dyslexia. Knud Hermann, a neurologist at the University Hospital of Copenhagen, analyzed in great detail the difficulties of dyslexic patients during the 1940s, 50s and 60s, and provided a classical definition of dyslexia: "...a deficit in the acquisition of an age-appropriate level of reading and writing ability; this deficit is due to constitutional (hereditary) factors, it is often accompanied by difficulties with other kinds of symbols (numeric, musical, etc.), it exists in the absence of other cognitive or sensory deficits, and in the absence of inhibitory influences, past or present, in the internal or external environment."

1.3 Evolution

We have seen that until the time of Orton, dyslexia was an almost exclusive field for physicians, specially ophthalmologists and neurologists. After Orton, the study of dyslexia was shared also by psychologists, sociologists, and educators. This competition between clinicians and researchers contributed to the proliferation of new theories about the causes and the symptoms of dyslexia. In contrast to the biological, and possibly genetic, nature of dyslexia suggested by the clinicians, sociologists and educators began to discuss about the adverse environmental factors that can affect dyslexic's difficulties, such as the inefficacy of the educative method. Many psychologist began to analyze dyslexic's abilities and disabilities, like Cyril Burt, who was the first education psychologist in Great Britain (1913), and later was knighted, A. J. Gates, F. Schonell, M. Vernon, M. Monroe, Ch. C. Bennet, W. D. Wall, and H. M. Robinson. These studies revised the concept of dyslexia towards a multifactorial phenomenon that can

originate from many diverse causes. Psychological theories described quite a range of possible symptoms, specially reading, writing, and spelling difficulties, which could happen together or in isolation, and with different degrees of intensity. That is, the clinical classification of a disability of an isolated group of patients was evolving to the more realistic concept of a continuity in reading ability, with dyslexics in the low tail of the distribution. This idea was originally supported, at least with respect to the distribution of intelligence, by psychologists like Monroe and Backus (1937), Meyer (1943), Nørgaard and Torpe (1943), Robison (1947), Larsen (1947), Tordrup (1953), and Gates (1955). Nonetheless, others like Sjögren (1932), Hermann (1959), Jaederholm, Pearson, Roberts (1945), Smith, and Strömgren (1938), have argued against this continuity, saying that the existence of a hump in the low tail of the IQ distribution suggests a different pathological nature. In the distribution of reading ability, this hump would reflect the dyslexic subjects (Hallgren, 1950; Critchley, 1964). This debate about the categorical or continuous distribution of reading ability is still open, and has been the subject of recent studies (Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992).

Regardless of a researcher's opinion in this debate or about the causes and symptoms of dyslexia, there was a general consensus that dyslexia could be remediated, if the appropriate method was used. There were of course different methods proposed. Fernald (1943) wrote a book on teaching procedures for dyslexics, including phonics. Anna Gillingham, Orton's research associate and psychologist, and Bessie Stillman, teacher, wrote a book on remedial techniques for dyslexics (1946): Remedial Training for Children with Specific Disability in Reading, Spelling and Penmanship. Their work was followed by her pupil, Sally Childs at the Scottish Rite Hospital in Dallas, and even later by

Aylett Cox.

Perhaps because of the increasing interest on dyslexia by many different scholars, the study of the psychiatric consequences of dyslexia was also undertaken. Phyllis Blanchard classified reading difficulties in two categories: neurotic, in which emotional problems arose before reading difficulties, and non-neurotic. Blanchard, and also Gates (1941), suggested that three out of four dyslexics had symptoms of emotional problems, although these problems were mainly due to the failure of learning to read. During the 1950s and 60s, R. D. Rabinovitch (1968), of the Howthorne Center in Michigan, tried to identify the possible neurotic reactions of dyslexics in particular, and of anybody with reading problems in general.

In 1957, Magdalen Vernon, Psychology Professor at the University of Reading, England, supported the multifactorial origin of dyslexia, recognizing subgroups with either visual, auditory, or abstract reasoning problems. In 1960, Silver and Hagin tried to set some diagnostic criteria and suggested the term specific language disability. In France, Alfred Tomatis proposed his theory of dyslexia as a problem exclusively of the auditory system .

The idea of subgroups of dyslexia gained popularity since the 1960s, specially when categorized by deficits, such as auditory and visual (Myklebust and Johnson, 1962; Ingram, 1970). In the 1970s, Boder (1976) continued this distinction, although he named these groups dysphonetic and diseidetic, and added a mixed group. Mattis, French, and Rapin (1975) described a new subgroup with motor problems. Simultaneously, the neurologist Martha Denckla suggested that the largest subgroup (more than half of dyslexics) had language deficits, in rapid naming, and some particular motor character-

istics.

In Britain, the study of dyslexia was largely ignored until the early 1960s. Maisie Holt, a psychologist at St. Bartholomew's Hospital, started teaching dyslexic children in 1960 at the instigation of Dr. Alfred White Franklin, a pediatrician at the Hospital who afterwards became Chairman of the Invalid Children's Aid Association (ICAA). Holt, advised by Sally Childs of the Scottish Rite Hospital in Dallas, followed the teaching approach of Gillingham and Stillman. Her work was continued and improved later by Beve Hornsby creating the "Alpha to Omega" method (Hornsby & Shear, 1975).

In 1963, the ICAA took the initiative in setting up the Word Blind Centre in London, to assess, study and teach dyslexic children. In Bangor, Wales, Professor T. R. Miles created the Dyslexia Unit during the middle 1960s to evaluate and study dyslexia. The closure of the Word Blind Centre in 1972 was the occasion for the expansion of several other centers around Britain, such as the Helen Arkell Centre (1971), and the Dyslexia Institute (1972), with Kathleen Hickey as head teacher. In addition, Marion Welchman founded the British Dyslexia Association in 1972, while the European Dyslexia Association was founded in 1987.

Macdonald Critchley, neurologist at King's College Hospital in London, was very influential in the field of dyslexia since he taught the 1961 Doyne Memorial Lecture on "Inborn Reading Disorders of Central Origin". He suggested the term specific developmental dyslexia, characterized by phonological deficits. He made an special effort in distinguishing dyslexics, which he considered as a compact group, from the rest of people with reading deficits, which he denominated with the Spanish term "olla podrida", in reference to the dish in which many different ingredients are mixed together. He was

also a convinced supporter of the constitutional origin, possibly genetic, of dyslexia.

The evolution of of the study of dyslexia also had legal consequences in Great Britain. The Chronically Sick and Disabled Persons Act, 1970, section 27, referenced acute dyslexia, probably the first legal appearance of the term dyslexia in Britain. Later, the Department of Education and Science's Tizard Report (1972) on Children with Specific Reading Difficulties described the small group of children with reading and perhaps writing, spelling, and number difficulties. The Bullock Report (1975) also commented on dyslexics problems and had the merit to shift attention to language. The Warnock Report (1978) considered dyslexic children had special education needs, a recommendation implemented in the 1981 Education Act. The Department of Education and Science's Tansley and Panckhurst (1981) Report advocated use of the expression 'specific learning difficulties'.

Another important aspect of the study of dyslexia, neuroanatomy, regained popularity with the neurologist of Harvard University Norman Geschwind, who is considered one of most important sponsors of neuroscience as a scientific discipline. Geschwind and Levitsky (1968) discovered an asymmetry in the area of the temporal plane of the human brain, an area related to language. In sixty-five percent of normal brains this area is larger in the left hemisphere than the right, while in twenty-four percent this area is similar in size in both hemispheres. Later, this finding would lead to the famous theory of the relationship between the asymmetry in the temporal plane and dyslexia. Geschwind also introduced theories relating the immune system, left-handedness and even left-eyedness, as well as pointing out the larger proportion of males versus females affected by dyslexia (Geschwind and Behan, 1982).

1.4 Modern theories

After the 1970s, theories of dyslexia based on newborn disciplines such as cognitive psychology and neuroscience stole the spotlight and provided the most compelling results. Within psychology, a very important character was Isabelle Y. Liberman, Psychology Professor at the University of Connecticut, and Research Associate of the Haskins Laboratories in New Haven. Liberman and her collaborators, like her husband Alvin Liberman, and Donald Shankweiler, were very influential to the scientific study of dyslexia, and demonstrated the importance of language in general, and speech in particular, to the development of reading skills. Today, the Haskins Laboratories continue studying language and reading, even with state-of-the-art methods like neuroimaging (R. et al., 2000). In 1971, Liberman stated that the linguistic determination of children's reading and language errors is very important. Visual or reversal errors, like those pointed out by Orton, only account for a small proportion of reading errors. Moreover, reversal errors do not have to be visual in nature. Liberman and her colleagues also described the relationship between human speech and phoneme awareness, and claimed that poor-readers' difficulties are usually linguistic in origin, specially rooted in the misuse of phonological structure and segmentation.

This line of reasoning was followed by other researchers. For example, Luria (1971, 1974) suggested that the observed difficulties in naming tasks affected speech, and that reading, writing and speech are all aspects of the same activity. Also Mattingly (1972) stated that good phonological awareness, as measured for example in a phonological segmentation task, is essential for learning to read successfully. However, Naidoo (1972)

observed that dyslexics had memory problems, specifically with storage capacity, claiming that dyslexics could experience other problems besides phonological deficits. Two similar studies (Spring and Capps, 1974; Denckla and Rudel, 1976) showed that dyslexics, in general, have a good vocabulary but they are slow in naming objects. Martha Denckla and Rita Rudel designed a serial rapid naming task (colors, numbers, objects and letters), named "Rapid Automatized Naming," which has become the standard task to measure this skill. Subject's scores on this task are related to phoneme awareness and reading skills, but they are still, to some degree, different cognitive abilities. From this moment on, it became apparent that it was necessary to study all these language and cognitive skills that are related to reading ability. The goal was to find out which of these skills are causally important in the development of reading and the etiology of dyslexia.

Marshall and Newcombe (1966), possibly inspired to some extent by the comparative study of the different sensory and cognitive skills related to reading, classified the mistakes of acquired dyslexic patients. They used these typical mistakes to create subgroups, such as deep, visual, and surface dyslexia. Somewhat later, subgroups of attentional dyslexics (Shallice and Warrington, 1977), and phonological dyslexics (Beauvois and Derouesne, 1979) appeared. This taxonomic system is based on the popular "dual route" of reading (Warrington and Shallice, 1980), although several other theories, with different possible routes of reading, exist (Massaro, 1975; Morton, 1979; Marshall, 1987). These subgroups of acquired dyslexia have been analyzed more recently (for example by John Marshall in Oxford, England, and by Max Coltheart and Anne Castles in Australia, during the 1980s and 90s) and their characteristics have been compared to

developmental dyslexia subgroups (Holmes, 1973; Marshall, 1982).

Due to the proliferation of studies and theories about dyslexia, the National Committee on Learning Disabilities was created in the USA in 1975, with representatives of groups such as the Reading International Association and the Orton Society. This committee stated since the beginning the heterogeneity of the concept of learning disabilities, and suggested the creation of subgroups in order to study the etiology, diagnosis and treatment of each subgroup specifically.

Only one year later, the existence of reading disability, that is, a group of children with specific reading deficits, sometimes questioned by some researchers, was proven. Rutter, Tizard, Yule, Graham, and Whitmore (1976) carried out the epidemiological studies of the Isle of Wight, in the United Kingdom, and showed the existence of intelligent children with specific reading deficits (specific reading retardation), as well as children with simultaneous reading and cognitive deficits (backward readers). Also in England, during the end of the 1970s and beginning of the 80s, Oxford University psychologists Peter Bryant and Lynnette Bradley showed that phoneme awareness at 4-5 years of age predicted reading and spelling skills even 3 and 4 years later, in normal readers. These researchers also discovered a phonological deficit in dyslexics, or at least in the previously mentioned backward readers. These results came in support of the phonological theory of dyslexia. Morais et al. (1979) established that the relationship between reading and phoneme awareness is reciprocal. Vellutino (1979) stated that dyslexics have difficulties establishing verbal associations, perhaps due to phonological decoding problems. Moreover, there exists a relationship between phonological deficits and short-term memory deficits in normal readers. According to Vellutino, dyslexia

was not a visual disorder, but a language deficit, specially involving the phonological processing of words.

Since the 1970s, theories of dyslexia switched gradually from visual to linguistic explanations of the disorder. Professor of Psychology of the University College of North Wales, T. R. Miles, created the "Bangor Dyslexia Teaching System" (1978) to remediate dyslexic deficits. This method was focused on several areas: Orientation, naming or repeating long words, arithmetic difficulties, list of items (forward or reverse), letter reversals, etc. This teaching method gained a lot of popularity in Bangor, Wales, and was used across the United Kingdom during the 1980s. Miles and his colleague, Ellis, also suggested that dyslexic problems were not visual but lexical, specially in the naming of objects and concepts.

At the same time, bolder theories of dyslexia were also proposed. For example, Tallal and Piercy (1973), Tallal (1980) proposed her theory of a deficit in the processing speed of general information, since she observed the relationship between slow auditory processing of words and sounds, and language deficits. Results of Pavlidis (1981) with 14 dyslexic children showed that reading deficits characteristics of dyslexia could be the consequence of abnormal eye movements. In addition, neurobiological studies made some important advances. Hier, LeMay, Rosenberg, y Perlo (1978) analyzed brain scans of twenty-four dyslexics, finding a correlation between asymmetry in the parieto-occipital region and verbal intelligence scores. Alberto Galaburda and Kemper (1979) found symmetry in the temporal plane, where there should be asymmetry, and cellular lesions in the brain of a 20 years-old dyslexic who died accidentally. Galaburda (1989) continued this work with more subjects, but there is not sufficient evidence yet that these brain

abnormalities have a direct relationship with dyslexia.

Gradually, the cognitive abilities more strongly related to reading were identified. Maryanne Wolf (1979, 1984) described the tight relationship between reading and rapid naming. Dyslexics have difficulties finding words accurately and quickly. This researcher, in 1986, found that rapid naming is a precursor, not a consequence, of reading deficits . In a slightly different approach, Crowder in 1982, and later Just and Carpenter (1987), analyzed the characteristics of good readers, trying to discover the most important skills which influence the normal development of reading. During the 1980s, two important researchers in the field of reading, Keith Stanovich, of the University of Toronto, Canada and Charles Perfetti, of the University of Pittsburgh, stated that accuracy and speed of single word identification predicts reading, and it is indeed a crucial skill for efficient reading. Stanovich claims that dyslexics have mainly a phonological deficit that consequently hurts their word recognition skills, and this later affects negatively their reading comprehension, vocabulary, and even intelligence. Stanovich has named this phenomenon as the Matthew effect, in which the richer get richer, and the poorer get poorer. In the case of learning to read, good readers become better, while poor readers get worse.

Theories relating dyslexia with memory or visual deficits continued during this decade, although in a lesser amount than in previous decades. Thomson (1984), of the University of Birmingham, England, pointed out that dyslexics have memory deficits, specifically a smaller storage capacity than normal readers. John Stein and Fowler (1982), of Oxford University, England, considered that dyslexics' deficits originate in a faulty eye convergence and an unstable ocular-motor dominance. From London, the study

of dyslexia received another notable influence, from the researcher of German origin, Uta Frith (1986) who analyzed the development of reading in children and characterized three main stages: logographic, alphabetic, and orthographic. Later, Linnea Ehri (1989) added one more stage, phonetic-cue.

Nonetheless, the most influential theories of this time claimed that the essential deficits that dyslexics face are in phonological skills and in isolated word recognition. John Rack showed that dyslexics are slower than normals in rhyming tasks, and they have difficulties matching speech sounds to their respective letters (Rack, Snowling, & Olson, 1992). Richard Olson, psychologist of the University of Colorado, also showed that dyslexics' deficits have a phonological nature, and that phonological deficits, as well as deficits in orthographic coding and word recognition, are hereditary (Olson, Forsberg, & Wise, 1994). Philip Gough claimed that reading comprehension depends on two factors: word decoding and oral comprehension (Hoover & Gough, 1990). Dyslexia, in this sense, occurs as a consequence of the word decoding deficits. Philip Seymour (1986) added that dyslexics exhibit very diverse patterns of impairments, complicating the creation of subgroups. Nonetheless, Seymour suggested the categorization of dyslexics into 3 subgroups: semantic, phonological and visual. In any case, most dyslexics seem to have phonological deficits which make them inaccurate or slow when reading pseudo-words. Richard Wagner and Torgesen (1987) claimed that phoneme awareness is essential to learning to read. Margaret Snowling, English psychologist, described during the 1980s and 90s dyslexics' deficits in phonological tasks and short-term memory. Snowling claimed, in relation to the dual-route theory, that dyslexics must have been using the direct visual route of reading, because the phonological route must have been impaired

(phonological dyslexics). She also described the characteristics of surface dyslexics, who exhibited the opposite deficits, and must have been using the phonological route (Snowling, 1983).

The most recent advances in dyslexia-related fields, such as cognitive psychology, have also influenced the current linguistic theories of reading. Mark Seidenberg and James McClelland (1989), of the University of Southern California, developed a connectionist model of reading using neural networks. This model allowed them to simulate reading in normal readers or even in different types of reading-disabled subjects. During the 1990s, Frank Manis, psychologist of the same university, has advanced our knowledge of several developmental dyslexia subgroups, and has collaborated with his colleagues to reinterpret dyslexia after the connectionist model findings. This work is being carried out by other researchers too, like Plaut, VanOrden and Pennington. In recent years, and due to the new neurobiological findings, the visual theories of dyslexia have been revisited, specially since the discovery of deficits in the magnocellular pathway of the visual system of dyslexics (Lovegrove, Martin, & Slaghuis, 1986) that could consequently cause reading deficits (Livingstone, Rosen, Drislane, & Galaburda, 1991; Galaburda & Livingstone, 1993).

At Yale University, Sally and Bennett Shaywitz and their collaborators have extensively studied the issue of the correct classification of dyslexics (Shaywitz et al., 1999). Bruce Pennington, a clinical psychologist at the University of Denver, has also contributed to improve the nosology of dyslexia. In addition, he has confirmed many theories, some of them already considered classics, like the importance of phonological deficits, which according to him last for a life-time, and the fact that reading is more

strongly related to speech than vision. Finally, Pennington has shown that reading ability depends on single word recognition, as well as on the ability to process words in a text (Pennington, VanOrden, Smith, Green, & Haith, 1990; Pennington, 1999).

Summarizing, dyslexia is currently an interdisciplinary field of study, involving disciplines as varied as education and neurobiology. Researchers hope that the answers to this complex learning disability lie in the intersection of all these disciplines, and with this goal in mind they foster the collaboration of all dyslexia research.

2 A modern definition of dyslexia

There exist many definitions of dyslexia, like those of the World Federation of Neurology, the International Classification of Diseases, 10th Revision (ICD-10), or the Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV). Nonetheless, a definition that expresses the current state of the field is the following, published in Annals of Dyslexia by Dr. Reid Lyon (1995), the Chief of the Child Development and Behavior Branch of the National Institute of Child Health and Human Development at the National Institutes of Health: "It is a specific language-based disorder of constitutional origin characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing. These difficulties in single word decoding are often unexpected in relation to age and other cognitive and academic abilities; they are not the result of generalized developmental disability or sensory impairment. Dyslexia is manifested by variable difficulty with different forms of language, often including, in addition to problems with reading, a conspicuous problem with acquiring proficiency in writing and spelling."

3 Summary of theories about dyslexia

3.1 Neurological/Sensory

3.1.1 Visual deficits

Visual perception deficit The first observations of dyslexic patients (Morgan, Hinshelwood, Orton, etc.) were made by physicians and ophthalmologists, who used the term word blindness to describe the disorder, so it seems reasonable that the first theories of dyslexia had a visual basis. Dyslexia was considered precisely as a visual perception deficit. This theory, that was widely accepted until the 1960s, and even had remediation techniques like that of Marianne Frostig, started losing adepts by the 1970s (Fischer, Liberman, & Shankweiler, 1978; Vellutino, 1972; Vellutino, Steger, Desetto, & Phillips, 1975; Vellutino, 1977; Arter & Jenkins, 1979).

Intersensory deficit Herbert Birch (1963) suggested the hypothesis that dyslexics had difficulties integrating the information of two or more sensory systems. This theory has been criticized for lack of valid evidence, and with experimental data that could not find these differences between normal readers and reading disabled children (Zigmond, 1966; Bryant, 1968; Vellutino, 1973).

Erratic eye movements Some researchers suggested that dyslexics exhibited erratic eye movements while reading, which causes their reading deficits (Hildreth, 1945; Pavlidis, 1981), but these results have not hold in more recent studies (Olson, Rack, Conners, DeFries, & Fulker, 1991).

Eye convergence deficits Another visual theory claims that dyslexics' problems involve eye convergence and binocular control (Stein & Fowler, 1982, 1985), which could be related to a neurological deficit that impairs efficient information processing. These results have been criticized by many scientists (Newman, Wadsworth, Archer, & Hockly, 1985; Wilsher, 1985; Bishop, 1989).

Color lenses Helen Irlen, of the Irlen Institute in the US, introduced color lenses to ease reading in some children, but her experiments and results have been criticized by many researches (Irlen, 1983; Irlen & Lass, 1989; Whiting & Robinson, 1988; Martin, Mackenzie, Lovegrove, & Mcnicol, 1993).

Magnocellular system There is evidence that a slow or irregular functioning of the magnocellular pathway of the visual system could cause reading deficits (Lovegrove et al. 1986; Livingstone et al., 1991; Galaburda y Livingstone, 1993), but these claims have not been confirmed.

3.1.2 Auditory deficits

Auditory transcription deficit Alfred Tomatis proposed his theory of an exclusive auditory deficit at the end of the 1960s in France. He suggested that dyslexics have difficulties transcribing written words into their phonological representations. Although this notion is similar to current phonological theories, his emphasis was in the auditory system, not in language deficits. Moreover, his remediation method, which consisted in teaching about the ear and motivating the patient to communicate, never became popular .

Auditory perception deficit Uncorrected auditory deficits can impair the normal development of language and speech, and consequently, could cause language and reading deficits. However, although this a possible cause of dyslexics' symptoms, this type of sensory deficits are generally excluded in dyslexia definitions, due to the lack of specificity. Theories claiming, not an auditory perception deficit, but a deficit in the phonological representation of language have much more support (Brady, Shankweiler, & Mann, 1983; Mody, StuddaertKennedy, & Brady, 1997).

3.1.3 Neurobiology and brain structures

Cerebral dominance Orton (1937) had originally suggested the existence of instability in the cerebral dominance of linguistic functions, and also in hand or eye preference. The relative dominance of cerebral hemispheres, left being in general more dominant for language, could be different in dyslexics and normal readers. If the brain areas involved in language are balanced between both hemispheres, dyslexics might need more interhemispheric communication, slowing their language processing.

The vestibular system An interesting theory claims that dyslexic's symptoms are due exclusively to a simple deficit in the inner ear (Frank & Levinson, 1976; Levinson, 1994). The cerebellar-vestibular system is responsible for tuning outgoing motor signals and incoming sensory signals. A deficit in this system can impair the tuning of these signals, which could in turn cause the multiple symptoms of dyslexia.

The corpus callosum Some studies have found anomalies in the size of the corpus callosum in dyslexic brains (Njiokiktjien, Desonneville, & Vaal, 1994; Hynd et al., 1995;

Rumsey et al., 1996), which could result in communication deficits between the cerebral hemispheres, but the implications of these anomalies for dyslexics are not clear yet.

The planum temporale and neuroanatomy Many researchers have studied the neurologic substrate of dyslexia, like the neurologists Drake Duane, of Mayo Clinic, Minnesota, or Martha Denckla in Maryland. Harvard Medical School, through scientists like Norman Geschwind and Alberto Galaburda, who introduced the famous theory of the temporal plane, have contributed in great amount to the advance of this field, analyzing post-mortum dyslexic brains, and more recently with the analysis of magnetic resonance images (MRI) of living dyslexic brains. Many researchers (Pauline Filipek, Frank Wood, and many others) are using these brain imaging techniques to identify the neurologic characteristics of dyslexia. The brain areas analyzed in more detail are those thought to be involved in language: Left parietal and temporal lobes, specially around the temporal plane and angular gyrus. Current neurobiological studies are three-fold: molecular (analyzing post-mortum brains and using mouse and rat brains as animal models), structural (using MRI to compare the morphometry of normal and dyslexic brains), and functional (observing the working brain with techniques such as function MRI (f-MRI) and positron emission tomography (PET). (Duane & Gray, 1991; Filipek et al., 1995; Pennington et al., 1999; Filipek, 1999; Rumsey et al., 1999)

3.2 Cognitives

3.2.1 Speed of information processing

Dyslexics make more mistakes than normal readers in auditory perception tasks that require quick stimulus discrimination. This finding suggested to Tallal and her colleagues that dyslexics have difficulties trying to perceive and process rapid information. This deficit might cause the phonological deficits that dyslexics exhibit while reading. The similarities between these deficits and the visual system (magnocellular) suggest that dyslexics' problem might be one of rapid information processing (Tallal, Miller, & Fitch, 1995).

3.2.2 Memory deficits

Dyslexics have a smaller storage capacity (Naidoo, 1972; Thomson, 1984) that could be due to coding deficits (Cohen & Netley, 1981). Vellutino (1979) suggested a phonological coding deficit. Denckla and Rudel (1976) also described coding or naming deficits. Shankweiler and Liberman (1979) suggested that dyslexics' memory deficits only exist for language information, results confirmed by other studies (Mann et al., 1980).

3.2.3 Language and phonological deficits

Theories claiming language deficits, mainly phonological, that impair learning to read (Orton, 1937; Liberman, 1971) and cause dyslexics' reading mistakes and slowness, are mainstream in current cognitive psychology and linguistics agendas. Phonological awareness at age 4-5 predicts reading at age 9-10. This fact, together with other similar

results, suggests that phonological awareness is a very important precursor of reading, and supports the hypothesis that early phonological deficits could cause later reading disability. This theory has a large number of followers and produces a large number of scientific studies each year. However, it is important to note that different languages have different phonologies. In irregular phonology languages, like English, dyslexic children in general make more mistakes and are slower than normal readers. In more regular languages, like German, Spanish, or Norwegian, dyslexics tend to be slower readers, and only make certain mistakes, but to a lesser degree than in irregular languages, since phonological rules are easier to learn.

4 History of the genetic etiology of dyslexia

A genetic nature of dyslexia is not in disagreement with the neurobiological theories previously discussed. On the contrary, they complement each other. Any structural or chemical disturbance in brain development can be caused by a genetic effect such as a mutation. Moreover, the fact that dyslexia is hereditary does not affect the educational and psycholinguistic theories for remediation of dyslexics' symptoms, as it is the case that many genetic diseases can be treated with environmental interventions (myopia can be corrected with lenses; diabetes can be controlled by regulating the insulin levels in the blood; etc.). In addition, the genetic nature of a disease does not necessarily imply complete determinism, since complex disorders like dyslexia are most likely influence by the interaction of several or many genetic and environmental factors. In fact, one of the more important environmental factors influencing reading skills is reading experience,

that is, the amount of time children spend reading, alone or with their parents, or at school.

Nonetheless, the genetic analysis of dyslexia has many advantages. On one hand, it will help identify the non-genetic, that is, environmental, factors (educational, familial, social) that impair the normal development of learning to read, thus contributing to the advance of psychological theories and remediation methods. On the other hand, the identification of genes with a direct or indirect influence in reading, and the localization of the activity of these genes in the brain, will perhaps allow an early diagnosis of dyslexia, as well as help find more direct treatments, and even help researchers understand the brain mechanisms of cognitive skills (Pennington, 1997; Flint, 1999; Plomin, 2000; Skuse, 2000).

The genetic theory of dyslexia began when researchers observed that it run in families. Familiality is a necessary condition for genetic disorders. The familial nature of dyslexia has been observed since the beginning of the twentieth century. Already in 1905, C. J. Thomas described a family with several members affected, and J. Herbert Fisher (1905) described an uncle and a nephew with reading deficits. S. Stephenson (1907) suggested that dyslexia was inherited as a recessive trait, at least in six cases of dyslexia from a three-generation family. Plate (1910) observed 4 relatives with reading deficits in another three-generation family. Warburg of Colone, Germany (1911) suggested that dyslexia was transmitted genetically through the mother, even if she was a good reader.

Hinshelwood, in 1917, showed that word blindness could be hereditary, since he studied a family with eleven children, of which the first seven were normal readers, but the remaining four had reading deficits, as well as a nephew and niece, progeny of an

older sister who did not exhibit reading deficits.

Illing (1929) claimed hereditary factors in seven other cases of dyslexia, and other researchers, like Laubenthal, from Bonn, Germany (1936), H. Rønne (1936), and Ferguson (1939), reached the same conclusion studying mutigenerational families. Orton, during the 1930s and 40s, also found that dyslexia seems to run in families.

Evidence for the familiality of dyslexia was also found in the Scandinavian countries (Norrie, 1939; Kågén, 1943; Ramer, 1947), even in mutigenerational families (Skydsgaard, 1942). A very influential study was carried out by Bertil Hallgren (1950), physician of the Psychiatric Clinic of the Karolinska Institute, in Stockholm, Sweden, who studied 276 cases of dyslexia and suggested, for the first time, that dyslexia was an autosomal dominant disorder.

During the 1970s and 80s more families with a large incidence of reading deficits were studied (Naidoo, 1972; Ingram et al., 1970; Rutter et al., 1976). Decker and DeFries (1981) compared a sample of dyslexic families with a control group, and their findings supported the familial aggregation of reading deficits. Finucci et al. (1976) and Lewitter et al. (1980) suggested that dyslexia was inherited as a heterogeneous or multifactorial disorder. Stewart (1989) noted that autosomal dominant transmission might be correct for dyslexia, but penetrance was smaller in women.

One of the most convincing experimental methods to determine the genetic origin of a disorder is the behavioral genetic study of twins and relatives. Identical and fraternal twins, siblings and other relatives, can be compared in base of their relative genetic similarity in order to establish the possible genetic nature of a disease or trait. This method was already used during the 1930s by researchers like Hallgren, Norrie, and

Brander (1935), Ley and Tordeur (1936), Jenkins, Brown, and Elmendorf (1937), and Schiller (1937).

Hermann (1959) compared dyslexic identical and fraternal twins and found that dyslexia was heritable. This finding was confirmed by Zerbin-Rüdin (1967) and Bakwin (1973), and more recently, by the large-scale twin studies in Colorado, USA, and London, England. John DeFries is the Director of the Colorado Learning Disabilities Research Center (CLDRC), which has sampled, during more than 20 years, thousands of twins, dyslexics and normal. The CLDRC has obtained convincing results estimating the heritability of dyslexia in about 50 percent (DeFries et al., 1997). These findings have been complemented by the analysis of DeFries' colleagues, Bruce Pennington and Richard Olson, who are interested in which specific reading components, and related disorders like ADHD, are heritable. In London, Jim Stevenson has carried out a similar project, sampling hundreds of twins, arriving to very similar conclusions (Stevenson, Graham, Fredman, & McLoughlin, 1987). Currently, Robert Plomin, of the Institute of Psychiatry in London, is creating a twin registry of all of England, which will allow for another replication of the genetic roots of dyslexia.

Results from these behavioral genetic studies show that approximately 50 percent of individual differences in reading ability are due to genetic factors, the remaining 50 percent attributable to environmental factors. Nonetheless, since the 1960s some researchers are skeptical about any substantial role for genetic factors in the etiology of behavioral disorders (Rutter, 2000), specially arguing that there has not been yet a single gene found that directly affects dyslexia. For this reason, genetic linkage analysis, which allow for the observation of the simultaneous genetic transmission of a marker and a disease,

will become very beneficial, helping to identify the putative genes affecting reading and dyslexia. With classic linkage techniques, Smith, Kimberling, Pennington, and Lubs, in 1983, found a region in chromosome 15 that is related to reading disability. Later, this same group of researchers suggested the existence of candidate regions in chromosomes 6 and 15 (Fulker et al., 1991; Smith, Kimberling, & Pennington, 1991), although a Danish study (Bisgaard, Eiberg, Møller, Niebuhr, & Morh, 1987) did not find evidence of linkage to chromosome 15. Other researchers, like Lubs and Rabin, have suggested other candidate regions, such as chromosomes 1 and 2 (Rabin et al., 1993; Grigorenko et al., 1998; Fagerheim et al., 1999). It is important to note that complex syndromes like dyslexia will have a complex genetic nature, in which perhaps multiple genes interacting among themselves create genetic risk factors, which then can interact with environmental factors. Because of this complex system, the identification of dyslexia genes is a very difficult task. Fortunately, the fields of molecular and statistical genetics have experience great advances thanks to new technological and theoretical paradigms. This experimental revolution has given birth to new methods for the identification of genes, taking advantage of more efficient genetic markers, new sampling methods (i.e., extremely discordant sib pairs), and modern statistical analysis techniques.

Using some of these new techniques, several confirmations of the candidate regions in chromosomes 6 and 15 have been published (Fulker et al., 1991; Smith et al., 1991). Specially important was the article by Cardon et al. (1994) which confirmed the relationship between reading disability and the chromosome 6 region in two more new samples. Until then, linkage studies on dyslexia had used global measures of reading ability, that were not taking advantage of the psychological theories that had decomposed reading

into its major components. The first study that analyzed these reading components (Gayán et al., 1995) showed that the 6p region affected several components of reading, such as word recognition, phonological decoding and orthographic coding. Evidence for association between reading disability and a gene in the short arm of chromosome 6 (6p) was shown by Warren et al. (1996). Specifically, it is the C4B gene of the major histocompatibility complex (MHC), involved in the correct functioning of the immune system. Another linkage study, carried out by researchers at Yale University, suggested a possible specificity of genes, so that the 6p putative gene would affect mainly phonological awareness, and the chromosome 15 putative gene would affect word recognition (Grigorenko et al., 1997). Nonetheless, this specificity theory has been rebutted by the latest findings, since two new studies by the University of Colorado and by Oxford University have confirmed that the 6p region affects several reading components, mainly phonological skills and orthographic coding (Gayán et al., 1999; Fisher et al., 1999). Moreover, a German study has confirmed that the chromosome 15 region also affects spelling (Schulte-Körne et al., 1998). Although some researchers have been unable to replicate these linkage results, having found only weak evidence for a locus affecting reading in the 6p region (Schulte-Körne et al., 1998; Field & Kaplan, 1998; Petryshen, Kaplan, Liu, & Field, 2000), the existence of a QTL on 6p influencing a number of dyslexia-related cognitive deficits has been confirmed in an extended study (Grigorenko, Wood, Meyer, & Pauls, 2000). In addition, another group in the UK have found evidence for association between reading disability and genetic markers in both the 6p21 and 15q21 regions, which will hopefully help refine the positions of the putative genes (Morris et al., 1999, 2000).

These findings allow us to dream with the day, perhaps in the next decade, in which we will know the main genes influencing the development of reading and dyslexia, and this knowledge will allow us design more direct therapies to remediate these deficits. In any case, it seems to soon, since so far we have only identified chromosomal regions, but not genes, affecting reading skills.

5 Conclusion

This short history of dyslexia and its genetic etiology had many limitations. Although I have tried to write a complete and detailed history, I have had to limit many contributions to one sentence or so in order to save space. The early stages of dyslexia are documented better for two complementary reasons: Enough time has passed since these events occurred so that they are already consolidated, but at the same time, their relative recentness has provided us with good historical reference of the facts. However, the recent history of dyslexia has experienced a proliferation of theories and studies, with different degrees of validity. My goal has been to mention as many of them as possible, but probably many more have escaped my attention.

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