Chapter

Neuropsychological Perspective on Dyslexia

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Abstract

The aim of this chapter is to offer a neuropsychological approach to dyslexia. Firstly, the definition of dyslexia is addressed, as a specific learning disability that is neuropsychological in origin. Secondly, the clinical manifestations of dyslexia are discussed: academic, cognitive-linguistic, and socio-emotional. Thirdly, the main clinical explanations are explored, based on genetic theories (familial and twin heritability) and neurological theories, mainly neuroanatomical (brain asymmetry, corpus callosum morphology, cerebellar morphology, and variations in grey/white matter) and neurophysiological hypotheses (magnocellular system, connectivity between brain areas, and functional activity of brain areas). Finally, the main bases of an adequate neuropsychological intervention are detailed, such as training in visual perception, auditory perception, phonological processing, and orthographic processing.

Keywords: Definition, clinical manifestations, neuropsychological theories, neuropsychology intervention

1. Introduction

Dyslexia has been studied from various fields such as medicine and psychology, and a number of different explanatory and descriptive approaches can be found in this regard. Scientific and clinical research has provided various definitions of dyslexia, describing deficits and their origin, giving rise to an array of differing approaches that have even offered conflicting explanations, blurring definitions, causes, and interventions [1–4].

In recent decades, the study of literacy and its disorders has sparked interest in understanding the underlying cognitive and psychological mechanisms, as well as the biological bases [2, 5]. Neuropsychology has focused on providing a comprehensive explanation for the genetic and neurological foundations of reading and writing mechanisms, and putting forward different theories about brain structures and cortical functioning involved in reading and writing disorders, which characterise dyslexia.

Dyslexia affects between 5 and 17.5% of the population in compulsory education, depending on criteria, definitions, and classifications [2, 4]. Prevalence also depends on the transparency and granularity of spelling systems, more frequent in opaque languages. In transparent languages such as Spanish, it can range from 3 to 7% of the population.

The aim of this chapter is to provide a neuropsychological approach to dyslexia that makes several relevant contributions in relation to this disorder. Firstly, the conceptualisation of dyslexia is addressed, reviewing the different definitions provided by major scientific and professional organisations. Secondly, the clinical manifestations of dyslexia at the academic or school level, in cognitive, linguistic and socio-emotional terms, are discussed, focusing particularly on the description of deficits that can occur according to the different subtypes of dyslexia. Thirdly, the main clinical explanations of dyslexia are examined, according to genetic, neuroanatomical, and neurophysiological hypotheses, with the aim of synthesising and integrating the different neuropsychological theories. Finally, we review early intervention in cases of dyslexia, proposing certain tasks to target the processing deficits that occur in this pathology.

2. Definition

In recent decades, the definition of Dyslexia has gradually changed, specifying certain aspects that have been controversial over the years. During this time, not only inclusion criteria but also exclusion criteria have been considered when defining this pathology. Some relevant definitions of this disorder are provided below, such as those proposed by the World Federation of Neurology (WFN), the International Dyslexia Association (IDA), the International Classification of Diseases (ICD-11) of the World Health Organisation (WHO), and the American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-5).

The **World Federation of Neurology** states that dyslexia is a disorder manifested by difficulty in learning to read and write despite conventional instruction, normal intelligence, and adequate learning opportunities [6]. According to this definition, dyslexia depends on fundamental cognitive disabilities, which are constitutional and structural in origin. Dyslexia, therefore, is a disorder that appears in childhood and is characterised by failing to achieve language skills in reading, writing, and spelling in accordance with the child's intellectual abilities.

For its part, **International Dyslexia Association** defines dyslexia as a specific learning disability that is neurobiological in origin, characterised by difficulties in the accuracy and/or fluidity of word recognition, and problems in spelling and decoding skills [7]. These difficulties typically result from deficiencies in the phonological components of language that are often unexpected in relation to other cognitive skills and reading instruction within the classroom. The consequences or side effects are reflected in problems of understanding and poor experience with printed language that impede vocabulary development.

From a more descriptive and nosological perspective, the Diagnostic and Statistical Manual of Mental Disorders 5 [8] and the International Classification of Disease 11 [9] point to dyslexia as a Specific Learning Disorder, within the Neurological Development Disorder axis with onset in childhood, along with other disabilities such as Intellectual Disability, Autism Spectrum Disorder, or Attention Deficit Hyperactivity Disorder, among others.

ICD-11 considers dyslexia a developmental learning disorder (code 6A03) in the classification of Developmental Disorders in axis 06 (**Table 1**), considering different exclusion criteria in its definition. Dyslexia is defined as a specific developmental disorder of school skills characterised by a specific and significant deterioration in the development of reading skills [9]. This deficit may be accompanied by difficulties in reading comprehension, spelling, and is often associated with emotional and behavioural disturbances during school age. ICD-11 establishes

Developmental learning disorder is characterised by significant and persistent difficulties in learning academic skills, which may include reading, writing, or arithmetic. The individual's performance in the affected academic skill(s) is markedly below what would be expected for chronological age and general level of intellectual functioning, and results in significant impairment in the individual's academic or occupational functioning. Developmental learning disorder first manifests when academic skills are taught during the early school years. Developmental learning disorder is not due to a disorder of intellectual development, sensory impairment (vision or hearing), neurological or motor disorder, lack of availability of education, lack of proficiency in the language of academic instruction, or psychosocial adversity.

Table 1.
Definition of learning development disorder according to ICD-11 [9].

differential diagnoses with intellectual disability, visual acuity problems, and inadequate teaching. The definition states that Dyslexia is produced by some kind of neurobiological dysfunction and is usually preceded by a history of speech or language disorders.

The DSM-5 [8] notes that dyslexia is a type of neurodevelopmental disorder and, in particular, a type of Specific Learning Disorder (code 315.00.F81.0) (**Table 2**).

A. Difficulty learning and using academic skills, evidenced by the presence of at least one of the following symptoms that have persisted for at least 6 months, despite interventions targeting these difficulties: 1. *Difficulty reading (e.g., inaccurate, slow* and only with much effort, in other words, reads individual words aloud incorrectly or hesitantly, often guesses words, difficulty expressing words well). 2. Difficulty *understanding the meaning* of what is read (e.g., can read a text accurately, but does not understand the sentence, relationships, inferences, or deeper meaning of what has been read). 3. *Spelling difficulties* (e.g., might add, omit, or replace vowels or consonants). 4. Difficulties with *written expression* (e.g. makes multiple grammatical or punctuation errors in a sentence; poor paragraph organisation; written expression of ideas is unclear). 5. Difficulties in mastering *numeric sense, numeric data, or calculation* (e.g. misunderstands numbers, their magnitude, and their relationships; uses their fingers to add single-digit numbers instead of remembering mathematical operations as their peers do; becomes lost when performing arithmetic calculation and might swap procedures around). 6. Difficulties with mathematical reasoning (e.g., has great difficulty in applying mathematical concepts, facts or operations to solve quantitative problems).
B. Academic skills are substantially affected, quantifiably below expectations according to the individual's chronological age, and *significantly interfere* with academic or occupational performance, or with activities of daily living, as confirmed by standardised, individually administered measurements (tests) and a comprehensive clinical evaluation. In individuals aged 17 and older, the documented history of learning disabilities can be replaced by standardised assessment.
C. Learning disabilities *begin at school age* but might not fully manifest until the demands of the affected academic skills exceed the individual's limited abilities (e.g., in set examinations, reading or writing complex and long reports for an non-deferrable deadline, very large amounts of school work).
D. Learning disabilities *are not best explained* by intellectual disabilities, uncorrected visual or hearing disorders, other mental or neurological disorders, psychosocial adversity, lack of proficiency in the language of academic instruction, or inadequate educational guidelines.

Table 2.
Nosological description of specific learning disorders, according to DSM-5 [8].

Specific learning disorders cause deficiencies in personal, social, academic, or occupational performance, such as school dropout, mental health problems, and high levels of psychological distress, or high unemployment. Based on exclusion criteria, it is not explained by intellectual disability, global developmental disorder, visual, auditory, or motor disorders, other mental or neurological disorders (stroke, brain trauma), psychosocial adversity (economic difficulties, absenteeism), inappropriate academic instruction, or lack of opportunities to learn. Dyslexia is a type of specific learning disorder characterised by specific deficits in the ability to perceive or process information efficiently and accurately that impede the learning of reading (accuracy and speed) and writing (accuracy), resulting in problems of reading comprehension and written expression, and which persist for at least six months despite intervention. It may be associated with a known medical or genetic condition or environmental factor. The DSM-5 notes that it is not clear whether cognitive processing difficulties are a cause, correlate, or consequence.

The DSM-5 definition of Learning Disorder (**Table 2**) presents new developments with respect to previous editions. One of them is the term "specific" disorder, to emphasise the importance of attributing the diagnosis to a specific area or aptitude, either in the sublexical processes of literacy (accuracy or fluency in reading and/or writing), supralexical processes (reading comprehension and/or writing composition), or mathematics (mathematical calculation and reasoning). Furthermore, for the first time, the concept of dyslexia has been included as a term used to refer to the learning disorders of reading and writing accuracy and fluency, which may present other additional difficulties, such as deficits in reading comprehension or mathematical reasoning.

In short, the definitions indicated above point to dyslexia as a specific learning disorder that presents deficits in learning accuracy, reading fluency and orthographic fluency, mainly in the sub-lexical processes of reading and writing (**Table 3**). There is also consensus regarding its neurobiological origin and ruling out the socio-educational environment or socio-educational deprivation as a cause. They point out that individuals with dyslexia present cognitive problems and agree that this disorder begins in childhood, when children first begin to learn the written code. They also point to other disorders that require a differential diagnosis, such as intellectual disability, general developmental disorders, brain injuries, and others.

	WFN (1968)	**IDA (2002)**	**WHO (2018)**	**APA (2013)**
Reading accuracy and fluency	✓	✓	✓	✓
Spelling	✓	✓		✓
Neurobiological origin	✓	✓	✓	✓
Cognitive components	✓	✓		✓
Other secondary deficits		✓	✓	✓
Onset during childhood	✓		✓	✓
Differential diagnosis (intellectual or sensory disability, general developmental disorders, psychosocial adversity)		✓	✓	

Table 3.
Main coincidences between the different definitions of dyslexia.

3. Major clinical manifestations of dyslexia

Dyslexia presents a pattern of specific characteristics at the academic, cognitive-linguistic, and finally, socio-affective levels [1–4].

3.1 Academic manifestations

The academic manifestations of dyslexia are presented in the accuracy and speed of reading words and/or pseudo-words, as well as spelling and spelling correction [2]. The most common reading and writing errors that may occur depend on the type of dyslexia [2–4].

There are two types: phonological dyslexia (difficulties with the phonological route) and visual or dyseidetic dyslexia (difficulties with the visual route) (**Table 4**). On the one hand**, phonological dyslexia** affect the grapheme-phoneme conversion mechanism, and in particular, it causes errors in the decoding of reading/writing and reading speed. The most frequent academic manifestations are: slow and sounded-out reading; lexicalisations (turning pseudo-words into words); errors in reading long, unfamiliar, or pseudo-words; derivative errors (maintains the root, but modifies the suffix); inappropriate separations and joins in writing; and visual errors, such as rhotacism, where words that are orthographically and visually similar are interchanged. On the other hand, **visual dyslexia** is characterised by subjects' inability to do global reading/writing and making errors primarily in reading and writing accuracy. Its most common academic manifestations are: errors in reading short, frequent and familiar words; errors in understanding homophones; errors in the task of lexical decision-making with pseudo-homophones; phonological errors (errors due to phonetic similarity); and frequent errors in conventional spellings and exception words.

3.2 Cognitive-linguistic manifestations

The main manifestations of dyslexia are found in the cognitive and linguistic areas, in particular in visual processing, auditory processing and speech discrimination, auditory and phonological memory, knowledge of letters, prosody, phonological knowledge, rapid automatic naming, and executive functions [10–12].

One of the most widely debated issues surrounding the study of dyslexia is its relationship to intelligence [1, 2]. Today, the general consensus is that subjects

	Phonological or dysphonetic dyslexia	**Visual or dyseidetic dyslexia**
Errors depending on the type of words	• Long, infrequent, or pseudo-words	• Short, frequent and familiar
Common errors in reading	• Lexicalisations • Slow reading speed • Sounding out	• Understanding homophones • Pseudo-homophones lexical decision-making
Common errors in writing	• Incorrect separations and joins in words	• Conventional spellings and exception words

Table 4.
Main characteristics of phonological and visual dyslexia.

with dyslexia do not present relevant deficits in intelligence, i.e., they display a standardised intellectual capacity; however, dyslexic subjects can have high intelligence quotients, resulting in the phenomenon of double exceptionality [13, 14], or medium-low intelligence quotients. Furthermore, it appears that subjects with dyslexia do not present differences between their total intellectual quotient (IQ) and manipulative IQ (perceptual reasoning), but may have differences or discrepancies between verbal IQ (verbal comprehension) and manipulative IQ (perceptual reasoning), with low working memory indices. Children with dyslexia often have deficits in crystallised intelligence, but not in fluid intelligence, that is, they may manifest problems in practical intelligence [1].

One of the cognitive manifestations that dyslexics may present is a deficit in **visual perceptual processing** (**Table 5**). Problems in visual perception are often manifested in terms of difficulties distinguishing stimuli presented sequentially over short intervals of time, so the subject may have problems in differentiating between the stimuli presented, as the different stimuli interfere with each other and are not processed separately [15, 16]. Visual perceptual deficits may also occur due to difficulties in processing quick time sequences in time order judgement tasks. Therefore, the subject does not properly process the order in which stimuli are presented and is less sensitive to the order in which visual stimuli are presented sequentially [17]. Other deficits that dyslexia subjects typically experience are that they have longer execution times in visual perceptual processing tasks, because they are slower and require more time to acquire information during vision fixation, performing more saccade and shorter movements [18, 19]. Another feature is that they need larger time gaps between stimuli to perceive two different low spatial frequency, low contrast, or low brightness sequential stimuli as separate stimuli [20]. Another characteristic deficit is difficulty in processing position in the left-to-right spacing of words, and in the beginning part of the word and in letters that look similar (for example, m-n; F-E). Ultimately, the deficits presented by subjects with dyslexia in visual processing do not originate in problems of attention in general, but rather in perceptual processes.

Other deficits are manifested in relation to **auditory perceptual processing and speech discrimination** (**Table 5**). Subjects with dyslexia have alterations in auditory temporal processing related to verbal sounds or non-verbal elements, resulting in a lack of integration of auditory sensory information [21]. They also have difficulties in automatically discriminating between phonemes presented sequentially, causing interference between the two. Dyslexics have difficulty discriminating certain frequencies and amplitudes of sounds within a single tone presented sequentially and separated at short intervals of time. For example, they have difficulties with auditory stimuli that are phonetically and acoustically similar (da-ba), but do not present deficits when it comes to very different phonemes (ba-sa). Dyslexic subjects do not perceive in the acoustic signal the basic characteristics of phonemes (sound, place, and point of articulation) that are necessary to discriminate phonetic sounds, which could result in a deficit when it comes to identifying phonemes and syllables [22]. Finally, it should be emphasised that these difficulties would be related to deficits in phonological memory and phonological knowledge.

Subjects with dyslexia also have deficits in **auditory and phonological memory** (**Table 5**). Dyslexics have difficulties storing phonological items and retrieving or repeating them immediately [23–25]. Difficulties in phonological memory can justify difficulties in phonological knowledge for awareness and in the grapheme-phoneme association, which requires retention and retrieval of phonological information. These deficits occur mostly in languages with greater morphological complexity.

Another cognitive variable where dyslexics may present problems is **knowledge of letters** [26, 27] (**Table 5**). Subjects with dyslexia have difficulties learning the

	Manifestations:
Visual perceptual processing	• Difficulties in differentiating stimuli presented over short intervals of time • Difficulties in processing fast time sequences • Slow visual processing • They need longer intervals between stimuli depending on presentation conditions • Left–right processing deficits
Auditory perceptual processing and speech discrimination	• Deficits in sequential perception of phonemes or non-verbal sounds • Low discrimination of frequencies and amplitudes with the same tone in fast time sequences • They do not perceive sound, place, and point of articulation
Auditory and phonological memory	• Deficits in storage and retrieval of verbal information • Related to difficulties in phonological knowledge and grapheme-phoneme conversion
Knowledge of letters	• Problems learning the name and/ or sound of the letter • Deficits in distinguishing capital letters or similar letters • Difficulties in creating representations between phonemes and possible graphemes
Prosody	• Difficulties perceiving tonic syllable or accent • Difficulties in the prosody of pseudo-words • Difficulties repeating sequences of syllables
Phonological knowledge	• Difficulties perceiving and manipulating speech segments • Difficulties in identification, counting, omission, addition and substitution of syllables and phonemes • In early stages and in less consistent languages
Rapid Automatised Naming	• Slow in naming alphanumeric and non-alphanumeric items • Verbal or visual deficits
Executive function	• Deficits in use and control of cognitive skills such as attention and memory • Poor flexibility of thought • Deficits in productivity and verbal fluency

Table 5.
Cognitive and psychological manifestations of dyslexia.

alphabetical code, in particular, learning the names of letters, case sensitivity, or between similar letters (m-n, F-E) or symmetrical letters (p/q). They also exhibit problems in identifying the sound of letters, i.e. creating phoneme representations so that the phoneme can be matched to the grapheme (t––/t/). And finally, in creating consistent representations between phonemes and graphemes when there are several possible associations between them.

Prosody is another psychological variable where dyslexics can manifest serious problems [28–31] (**Table 5**). In particular, they present difficulties in perceiving the tonic syllable, according to the accent or stress of the word (differentiating the intonation MA-ma vs. maMA), difficulties in the prosody of pseudo-words (repetition of POga, RuPA), or in the reading of syllables (repetition of syllable sequences: Mamama/mamama).

Phonological knowledge for awareness is another of the most frequent deficits found in dyslexia [1, 2, 24, 32] (**Table 5**). Dyslexics present difficulties perceiving and/or consciously manipulating linguistic units (syllables and/or phonemes) in different tasks such as identification, counting, omission, addition, and substitution. These deficits are usually present mostly at younger ages and in less consistent languages.

Rapid Automatised Naming (RAN) is another variable where dyslexics often present deficits, as it is related to sublexical reading and writing processes (**Table 5**). These difficulties manifest themselves when the subjects are asked to quickly name a sequence of highly frequent elements, which can be colours or objects (non-alphanumeric) or letters and numbers (alphanumeric). Dyslexic children are often slower to name non-alphanumeric items (colours and drawings) and alphanumeric items (letters and numbers), with greater deficits observed in the latter. Subjects must access a phonological label from a graphic symbol [33]. This ability could therefore refer to both phonological and non-phonological skills [34], as they need access to information of a visual nature (detection and discrimination of visual traits) and also phonological (integration of visual information with stored phonological patterns and retrieval of phonological labels). These deficits may be due to phonological or verbal memory difficulties or may also be related to visual processing deficits [32, 35–37].

Dyslexics also present serious deficits in **executive functions** [38–41] (**Table 5**) that would justify their difficulties in reading and writing. The most relevant deficits are in working memory, planning, organising and switching attention. There are also deficits in the inhibition of distractors and in the sequencing of elements, deficits in the flexibility of thought, as well as difficulties in productivity and verbal fluency.

3.3 Socio-emotional manifestations

Subjects with dyslexia also have other socio-emotional clinical manifestations, such as anxiety and depression problems, maladaptive attributional styles, low self-concept and self-esteem, as well as low motivation (**Table 6**).

Subjects with dyslexia are more likely to present **anxiety**, which is more frequent among boys than in girls, and in situations of greater stress such as tests. They also manifest more internalising emotions associated with **depressive symptoms**, although their relationship with dyslexia is not precise [42–44].

Maladaptive attributional styles are also common in subjects with dyslexia [45, 46]. In particular, dyslexics often present maladaptive patterns, attributing their successes to luck, the quality of help and attention given by their teachers (external locus and uncontrollable), and to a lesser extent to their own effort (unstable internal locus). Their achievements are not attributed to their interest

	Manifestations:
Anxiety and depression	• Greater anxiety found among boys and in test situations • Internalising emotions and depression
Maladaptive attributional styles	• Attribution of success to unstable external causes (luck, teachers) • Attribution of failure to stable internal causes (ability)
Self-concept and self-esteem	• Negative self-esteem patterns • Inconclusive studies
Motivation	• Poor reading motivation • Low persistence in tasks

Table 6.
Socio-emotional manifestations of dyslexia.

in the activities or their capabilities (stable internal locus). Instead, based on the experience of repeated failure, they perform causal attributions of their failures to their own capacity (stable internal locus) compared to normal subjects, who attribute their successes to internal causes and failures to non-stable external or internal causes (such as effort).

Subjects with dyslexia often have problems of **self-concept and self-esteem**. Dyslexics present dependence, insecurity, lack of confidence, and feelings of helplessness more frequently than normal subjects [47, 48]. However, it is unclear whether self-concept is negative in all cases, with very few studies conducted on adults. In addition, the self-perception of subjects with dyslexia seems similar to that of control groups.

Another common problem is the **low motivation** of students with dyslexia. Children with dyslexia are not oriented towards motivation for achievement and performance, and show low perseverance in achieving their goals [47].

4. Clinical explanations of dyslexia

The various definitions have shown that Dyslexia is a developmental disorder that is biological in origin [2.10]. These definitions include interaction between genetic, epigenetic (e.g. embryonic development, proteins or enzymes) and environmental (e.g. premature or low birth weight, prenatal exposure to nicotine) factors that affect the brain's ability to perceive or process verbal or non-verbal information, efficiently and accurately. Neurological and genetic alterations in dyslexic subjects are the basis for cognitive processing problems, although there are no known universal markers for the individual diagnosis of a patient with Dyslexia.

At present, there are different neuropsychological explanatory theories of dyslexia, such as genetic explanations (familial and twin heritability, and genetic iteration) and neurological explanations (neuroanatomical and neurophysiological).

4.1 Genetic explanations

Numerous studies relate genetic predisposition and the development of dyslexia [49, 50]. Etiological research has carried out two types of studies around genetics, namely studies of family heritability and studies with twins; and studies of molecular genetic alterations.

4.1.1 Family and twin heritability

One of the findings indicating that dyslexia may have a genetic explanation is that it is more common in boys than in girls [49, 51]. Similarly, family inheritance studies have found that dyslexic children often have parents who have also had reading disabilities, so there is a greater likelihood of developing reading problems when family members have a history of dyslexia. Some studies indicate heritability levels of between 18 and 65%, with up to eight times the probability of developing dyslexia when one of the parents is dyslexic [49, 52], and even greater if both the father and mother are dyslexic [53]. In contrast, this probability drops to 5% when the parents have no history of dyslexia [54]. This research supports genetic predisposition; however, these data are not sufficient, as in addition to genes, families share a cultural and socio-educational environment as well as parenting patterns that can also influence [2].

Studies conducted with twins also propose a genetic explanation for dyslexia. Brothers who share the same genetic load (monozygotic) have been found to show

greater concordance of reading deficits than twins with different genetic (dizygotic) loads, so the probability that one monozygotic twin presents dyslexia when the other presents it is between 70 and 100%, a figure that it is reduced to 32% when they are dizygotic [55]. Variation in prediction also appears according to the domain subtype we are dealing with, being higher in phonological competency or spelling [55].

4.1.2 Molecular genetic alterations

Genetic alterations in dyslexia have been investigated using molecular genetic techniques, trying to isolate the genes responsible for reading and writing problems [49, 56]. However, studies have not concluded that there is a single chromosome responsible for dyslexia, but instead there are several possible chromosomes that could explain it, depending on the type and characteristics (**Table 7**).

Chromosome 1 is involved in visual processing deficits, and more specifically in processing speed, which would justify literacy problems [49, 57].

Chromosome 21 (short arm) is related to the functioning of cognitive processes that depend on the hippocampus, affecting the way words are processed [52].

Likewise, research has also shown that *chromosomes 13 and 7* (short arm) are involved in the circuits that interconnect the cortex, thalamus, and the striatum [58, 59]. This circuit would be responsible for linguistic processing, and its mutation would give rise to a phonological deficit related to verbal working memory [60].

Chromosome 15 and specifically Gene DYX1C1, located at locus DTYX1 (long arm), was among the first to be isolated [61], identified through high concurrency among members of a Finnish family [62]. This gene would be related to the different tissues, including the brain, involved in the radial migration of neurons and linked to the development of the cortex. It is located in the nucleus of certain neurons and glial cells of the cerebral cortex, so it is postulated that the gene acts indirectly and would maintain cell functionality [63]. Its role seems to be related to reading isolated words and spelling [61].

Chromosome 6, and in particular genes DCDC2 and KIAA0319, located in region DYX2, is related to the development of the temporal cerebral cortex and the cingulate gyrus. Its function is unknown, but it appears to be involved in neuronal migration [64, 65], and may be involved in mediating the interaction between glial cells and neurons. This chromosome would influence phonological and orthographic reading processes, although other studies do not find such an association [66].

Chromosome	Gene	Structure	Deficits
1	—	—	• Visual processing
21	—	Hippocampus	• Cognitive processing
13 and 7	—	Cortex circuit, thalamus and striatum	• Phonological processing (verbal working memory)
15	DYX1C1	Cerebral cortex	• Isolated words and spelling
6	DCDC2 KIAA0319	Temporal cerebral cortex and the cingulate gyrus	• Phonological and visual processes
3	ROBO1	Cerebral cortex and thalamus	• Discrimination of sounds of speech and phonological processing

Table 7.
Genetic alterations of dyslexia.

Chromosome 3, and specifically gene ROBO1, located in region DYX5, appears to be related to the development of the cerebral cortex, the growth of the thalamus, and the origin of dyslexia [67]. It is involved in the growth of axons and neuronal migration, as well as the growth of the corpus callosum. Specifically, the ROBO1 gene is linked to disorders in speech and the phonological processing of sounds [68].

4.2 Neurological explanations

Neurological theories have grown substantially in recent decades in the field of dyslexia study. This boom is motivated by an interest in brain function and, above all, by advances in neuroimaging techniques [69, 70].

Neurology has taken an interest in the description of brain areas and structures, as well as the functioning and organisation of brain activity according to different reading patterns and their deficits. To this end, technological progress has been key, with the emergence of techniques that allow for neurological studies to be conducted while these processes are taking place.

Neuroimaging techniques are the most widely used in neurological research into dyslexia. These include MRI (Magnetic Resonance Imaging), PET (Positron Emission Tomography), and fMRI (Functional Magnetic Resonance Imaging). *MRI* performs high-precision imaging of the brain, providing information about the brain structure, and being able to identify and locate areas with high precision. *PET* improves on the previous technique by imaging the brain through metabolic activity and changes in blood flow, so contrast is required. The image shows the areas that are active during a cognitive process, so it offers a high degree of precision regarding the brain structures involved. However, it does not provide temporal data, that is, regarding the sequence of a process. fMRI outperforms the above, providing spatial and temporal information on brain structures involved in a cognitive process. In addition, it does not require the use of contrasts and is less invasive than the previous technique.

Below we discuss two types of complementary neurological explanations: neuroanatomical and neurophysiological hypotheses.

4.2.1 Neuroanatomical hypotheses

Neuroanatomical theories refer to abnormalities in the different brain structures involved in reading and writing. Some of the most relevant studies from this perspective are listed below.

4.2.1.1 Cerebral asymmetry

Studies on cerebral asymmetry have been very relevant in the explanation of dyslexia [71, 72]. Two types of explanatory hypotheses have been developed about cerebral asymmetry and dyslexia (**Table 8**).

The first hypothesis assumes that dyslexics have a pattern of cerebral symmetry between the two hemispheres. Some studies indicate that this non-asymmetry is shown in dyslexic children in the temporal plane responsible for receptive language, since the upper part of the temporal region of the left hemisphere is not more developed than that of the right hemisphere [73]. This occurs in types of dyslexia associated with deficits in phonological processing and reading comprehension. Other studies find that non-asymmetry is shown in the parietal and frontal areas, and in particular in the left inferior frontal gyrus [74]. Dyslexics with these deficits present difficulties in speech perception processes, auditory and phonological processing implicit in word reading.

Hypothesis	Description of structures	Deficits
Cerebral hemispheric symmetry	Superior temporal region	• Phonological processing and reading comprehension
	Parietal and frontal regions	• Speech perception, phonological and auditory processing
Different cerebral hemispheric asymmetry	Parietal-occipital region	• Verbal difficulties
	Parietal–temporal region	
	Hippocampus, Lenticular Nucleus and Amygdala	• Visual difficulties

Table 8.
Neuroanatomical hypotheses about cerebral asymmetry.

The second hypothesis assumes that dyslexics have an inverted cerebral asymmetry pattern, that is, different from the pattern presented by those with normal reading development [75, 76]. Some studies find that, in dyslexics, asymmetry is higher in *the parietal-occipital region* and lower in the parietal–temporal region [77]. This pattern is related to verbal difficulties and, in particular, to phonological processing displayed by dyslexics. Other studies find a different asymmetry in *the hippocampus, parahippocampal gyrus, lenticular nucleus (putamen and Globus pallidus) and amygdala* [78, 79] with greater asymmetry between the right and left hemisphere.

Jiménez, Hernández and Conforti [80] investigated the relationship between cerebral asymmetry and dyslexia. Three experimental groups participated in the research: the first group consisted of dyslexic subjects; the second group consisted of subjects with a reading performance similar to the previous group, but with a lower chronological age; and the third group consisted of subjects of the same chronological age as the subjects with dyslexia. The results indicate that there are significant differences in the pattern of verbal and spatial cerebral asymmetry between children with dyslexia and the two control groups. More specifically, there are no differences in the lateralisation of linguistic functions, since in all cases it occurs in the left hemisphere, with bilateralisation being more pronounced in the case of children with dyslexia and those who showed equal reading performance at a younger age. However, significant differences are found in the lateralisation of spatial functions, which occurs in the right hemisphere in the case of control groups, but not in subjects with dyslexia, where it occurs in the left hemisphere. Regarding the hemispheric confluence of linguistic and spatial functions, it was found that the group of subjects with LD as well as the matched group in terms of reading performance presented a convergence of both functions in the left hemisphere, showing significant differences with the matched group in terms of chronological age [80]. This convergence of linguistic and spatial functions in the left hemisphere in subjects with LD stands in contrast to the hemispheric specialisation found in subjects without difficulties and makes higher-order psychological processes less effective, since they require a great deal of synchrony and execution of all available resources and in the case of LD they are concentrated exclusively in a single hemisphere. Thus, the authors conclude that dyslexic subjects do not develop this hemispheric specialisation, concentrating both spatial and linguistic functions in the left hemisphere [80]. Dyslexic subjects show a symmetrical pattern due to a convergence of verbal and spatial functions in the left hemisphere, which sets them apart from subjects with normal reading development. A similar pattern is found when investigating lower-age, normalised subjects matched in reading performance to dyslexic subjects, i.e. in the process of learning literacy, as reading functions are not yet fully specialised [80].

Hypothesis	Description of structures	Deficits
Corpus callosum	Larger posterior portion, and similar anterior-middle portion	• Poor lateralisation of functions
	Smaller anterior portion	• More brain tissue in the temporal–parietal region
	Thin, rounded shape	
	No differences	

Table 9.
Neuroanatomical hypotheses on the morphology of the corpus callosum.

4.2.1.2 Morphology of the corpus callosum

Some studies have observed differences in **the corpus callosum** of dyslexics, presenting a larger posterior portion of the corpus callosum [74], while the anterior and middle part is similar to those with normal reading development. These anatomical variations could be associated with a non-lateralisation of functions (**Table 9**).

Other research indicates that dyslexic subjects have *a smaller anterior portion of the corpus callosum* than subjects in the control groups [81].

Other alterations in dyslexic subjects would be *the more rounded and thinner shape* and sometimes a larger middle third of the corpus callosum. This finding is consistent with the fact that most symmetric brains have more brain tissue in the temporal–parietal region connected to the corpus callosum. The difference in the size of the corpus callosum may reflect hormonal influence during the critical development period of inter-hemispheric connections [82].

Finally, other research suggests that *there are no morphological differences* between controls and dyslexic subjects in the corpus callosum [83].

4.2.1.3 Morphology of the cerebellum

One of the structures that present a different morphology in subjects with dyslexia is the cerebellum, involved in psychomotricity, development of motor skills, and their automation (**Table 10**).

Some studies indicate that while subjects with normal reading development have cerebellar asymmetry, with a larger right anterior lobe size, dyslexic subjects have *cerebellar symmetry* [84]. Its role in language processing, speech perception, and reading has also been discovered [85]. Children with cerebellar symmetry make more errors than those with cerebellar asymmetry in writing, associated with motor difficulties, and reading accuracy difficulties, associated with automation and articulation problems that would justify the phonological knowledge and memory problems that dyslexics present.

Other research indicates that there are *slight cerebellar abnormalities*, since the cerebellum is smaller in subjects with dyslexia. This leads to deficits in postural

Hypothesis	Description of structures	Deficits
Cerebellar symmetry	RH = LH	• Motor difficulties, in language skills, in speech perception, in reading and writing
Cerebellar abnormality	Slightly smaller size	• Difficulties in postural stability, tone, articulation problems, and phonological problems

Table 10.
Neuroanatomical hypotheses on the morphology of the cerebellum.

tone and stability, as well as articulation problems. These difficulties would lead to speech difficulties and problems in the auditory and phonemic processing of words [86], and as a consequence difficulties in the awareness of rhyme and the phonemic structure of language and phonological deficit [86].

4.2.1.4 Variations of grey and/or white matter

Another neuroanatomical explanation shown in neurological studies refers to variations in grey and/or white matter in certain brain regions presented by dyslexics [87]. Using the VMB (Voxel-based morphometry) technique developed by Ashburne and Fristonn [88], the density of grey and/or white matter in various regions of the brain and cerebellum (**Table 11**) has been shown to be different in dyslexics and those with normal reading development.

Some research indicates that there is a variation in the volume of grey matter in the brain. On the one hand, studies indicate that there is a lower volume of grey matter in the brain of subjects with dyslexia, compared to subjects with normal reading development, in particular in two regions.

The temporal–parietal region, and in particular, in the superior temporal supramarginal gyrus of both hemispheres, has a lower density of grey matter in dyslexic subjects. This alteration would be related to deficits in speech perception (production and auditory discrimination of phonemes) and phonological processing, as well as deficits in integrating the auditory processing of linguistic stimuli [87].

Lower levels of grey matter have also been found in bilateral occipital-temporal regions, related to visual processing deficits or letterforms in dyslexic children [89, 90].

Other research indicates that dyslexic subjects present a lower level of grey matter in the right cerebellum and right lentiform nucleus [91, 92], which is related to phonological and lexical difficulties. Subjects with lower volumes of grey matter display a poorer performance in pseudo-word reading and phonological tasks (phoneme omission) than those with a higher volume.

In contrast, other studies have shown variation in white matter in the cerebral hemispheres of dyslexics, finding a lower volume of bilateral white matter in frontal lobes [93] and temporal–parietal lobes [92], which is associated with phonological and/or visual processing deficits.

Neurocognitive research has indicated that grey and white matter variations would lead to changes in brain function, as they generate lower left hemisphere activity and compensatory overactivation in the right hemisphere. In addition, subjects with dyslexia have a reduced gyrification index, and a lower volume of grey matter in the left temporal lobe and ectopia, suggesting a gestational defect in origin or abnormal prenatal brain development [94, 95]. Other authors note that variations in grey and white matter are due to the absence of cerebral asymmetry [70].

Hypothesis	Description of structures	Deficits
Grey matter.	Parietal–temporal brain regions	• Auditory and phonological processing
	Occipital-temporal brain regions	• Visual processing and letterforms
	Right cerebellum and right lentiform nucleus	• Pseudo-words and phonological tasks
White matter	Frontal and temporal–parietal regions (bilateral)	• Visual and phonological processing

Table 11.
Neuroanatomical hypotheses about variations in grey/white matter.

Hypothesis	Description of structures	Deficits
Magnocellular system	Alteration of the lateral geniculate nucleus of the thalamus	• Difficulties in processing short stimuli • Difficulty in motion sensitivity • Difficulties in low contrast and low frequency stimulation
Connectivity	Angular gyrus disconnection LH	• Phonological processing.
	No synchronisation between	• Phoneme recognition
	Broca's Area and Wernicke's Area	• Word recognition
	No transcallosal inhibition	• Deficits in processing speed of letters and words
Functional activity	Dorsal route (left parietal–temporal (angular gyrus, supramarginal, and superior temporal gyrus)	• Deficits in phonological processing
	Dorsal route (middle left occipital-temporal (fusiform and lingual gyrus)	• Deficits in visual processing
	Inferior left frontal area	• Compensation of visual deficits

Table 12.
Neurophysiological hypotheses of dyslexia.

4.2.2 Neurophysiological hypotheses of dyslexia

Neurophysiological hypotheses describe the organisation and activity of the areas of the brain as a whole. Neurophysiological explanations of dyslexia are divided into those related to the magnocellular system, to the connectivity of brain areas, and to the functional activity of brain areas (**Table 12**).

4.2.2.1 Magnocellular system

Magnocellular deficit theory [78, 79] postulates that there are physiological and anatomical deficiencies in the magnocellular system of dyslexics, mainly in the size and organisation of the cells of the lateral geniculate nucleus (LGN) of the thalamus. This structure would be responsible for rapid processing of visual information (spatial perception, selection, planning and hand-eye coordination). It plays an important role in orthographic processing and could be the cause of dyseidetic or visual dyslexia [79].

The LGN would be connected to the parietal lobe and would be critical to RAN skills [96]. Some of the manifestations that dyslexics would present as a result of these deficits would be difficulties in processing short stimuli, movement difficulties, difficulties in low-contrast stimulation and low spatial frequency [18, 97]. However, there would be no difficulties when stimuli are presented at low speed or with high contrast. These difficulties would justify errors in the visual coding of letters, programming of saccadic movements during reading, and selective attention during visual search [98].

Stein [79] notes that the LGN would not only be key to visual processes, but would also extend to perceptual deficits – auditory, sensory, tactile, and motor, and therefore also phonological. Cuetos [99] also points out that it would explain auditory perceptual deficits, since both have the same origin, and subjects with dyslexia have problems in the processing of visual or auditory stimuli presented in a fast and changing manner.

4.2.2.2 Connectivity of brain areas

The explanations for a deficit in the connectivity of brain areas are varied. One of these points out that reading problems in dyslexic subjects occur because of a functional disconnection between the angular gyrus of the left hemisphere and the occipital and temporal areas when phonological tasks are performed [82, 100].

Another theory of connectivity points to the lack of synchrony among dyslexics between Broca's Area and Wernicke's Area. Dyslexics and those with normal reading development activate the same areas during reading: on the one hand, Broca's Area, specifically the lower left frontal area (AB 6/44), responsible for articulation and the mental representation of the sound of the word; and, on the other hand, Wernicke's Area, specifically the upper left temporal gyrus (AB 21/22), which is responsible for relating the processing of phonemes and the recognition of words [101]. However, in subjects with dyslexia, these areas are not activated synchronously, so there is a disconnection between these two regions, due to a dysfunction of the insula (responsible for connecting the anterior and posterior regions responsible for language).

Theory regarding deficits in transcallosal inhibition [102] points out that in subjects with dyslexia, the corpus callosum is unable to inhibit the right hemisphere, interfering with the activity of the left hemisphere. These deficiencies in transcallosal inhibition would justify the poor transfer of information between the hemispheres. This would lead to a loss in processing speed with letters and words [103].

4.2.2.3 Functional activity of brain areas

Another line of research has been related to the functional activity of various brain areas involved in reading in people with dyslexia. Deficits in the functional activity of various neurological structures related to reading circuits have been found in those with normal reading development [75, 104]. Thus, in dyslexics, different areas of the brain activate in comparison to normal readers [105]. In general, it has been found that there is less activity in the left hemisphere and more activation in the right hemisphere, which would justify problems of hearing perception found among dyslexic subjects [106].

In particular, subjects with dyslexia have low parietal–temporal activity in the left hemisphere (supramarginal and angular gyrus in the interior part of the parietal lobe and superior temporal gyrus), which would justify phonological deficits (dorsal route) [60].

A second region with low activity is the left occipital-temporal zone (fusiform and lingual gyrus), related to visual deficits, in particular, the rapid and automatic recognition of words (ventral route) [60].

Overactivity has also been found in dyslexic adults in the inferior left frontal area, responsible for articulation and phonological analysis of words. It is suggested that this overactivity is caused by the overuse of the articulation and grapheme-phoneme conversion systems, to compensate for deficits in visual processing [107, 108].

These investigations have been carried out in languages with different spelling consistency, achieving different results depending on the transparency and granularity of languages [107, 109]. In opaque languages there is low activity in the left inferior frontal gyrus, right superior temporal gyrus and left precuneus, and high activation in the left anterior insula. In contrast, the activity pattern in transparent languages shows low activation in the left fusiform gyrus, left temporal–parietal cortex, right frontal operculum, and high activity in the left pre-central gyrus.

5. Neuropsychological intervention in dyslexia

Neuropsychological intervention in dyslexia requires a neuropsychological evaluation of the clinical variables and manifestations described so that early detection of academic, cognitive-linguistic, and socio-emotional problems can be achieved. Following evaluation, early intervention should be initiated, applying scientifically validated and proven neuropsychological techniques and programmes.

It has been shown that when intervention is carried out at an early age, the neuropsychological results are surprising, as they improve psychological, cognitive, and academic skills, and avoid associated problems such as frustration, internalising problems, and reading rejection. Early intervention is based on evidence of brain modification due to cerebral plasticity since, following early quality intervention, subjects with dyslexia show greater brain activity in regions with neurological deficits [75].

Adequate intervention in dyslexic children has also been shown to lead to improvements in literacy processes, which are manifested following the brain changes generated by the intervention [105, 110]. Following neuropsychological intervention, fluency (precision and speed) was improved, and neurophysiological and neuroanatomical changes were observed. In particular, greater activation was observed in several cortical areas, mainly in the occipital-temporal area, along with an increase in the volume of grey matter in several brain areas (hippocampus, left fusiform gyrus, and right cerebellum).

Yet, studies that design and validate treatments for the improvement of dyslexia are scarce. In recent years, however, efforts have been made to test the effectiveness of intervention programmes based on different theoretical models. This allows intervention programmes to be tailored according to whether this pathology is considered the result of a specific cognitive deficit (e.g. phonological knowledge or processing speed) or the result of a primary general deficit that would explain the cognitive deficit (e.g. auditory and/or visual perceptual processing). The most successful intervention programmes are those responsible for improving accuracy (learning the rules of grapheme-phoneme conversion, phonological awareness, naming speed) and speed (automation of grapheme-phoneme conversion rules and the formation of orthographic representations by repeating words and texts).

Finally, in view of the neurological explanations found, tasks are proposed below for the design of effective interventions, to improve the cognitive processes affected in dyslexia. Neuropsychological intervention focuses on performing tasks that would have to be targeted and adjusted to the characteristics of each case, in order to activate or compensate the areas of the brain that present a malfunction or alteration. In addition, it is generally recommended in all cases to practice repeated reading with and/or without a model (teacher, partner, CD, computer) and provide feedback by recording responses.

Below are some activities designed to improve perceptual processing (visual and auditory), phonological processing, and orthographic processing. Visual and auditory perceptual processing tasks are aimed at improving deficits in the detection and discrimination of graphic and verbal signals.

Tasks for improving visual perceptual processing include [1, 111–113]:

- Pair matching series of signs (letter, syllable, word, pseudo-word)
- Finding a sign (letter, syllable, word, pseudo-word) in an array
- Same-Different: deciding whether pairs of signs are the same or different

- Finding the different sign in an array

Tasks for improving auditory perceptual processing include [1, 12, 112, 114]:

- Identifying the tone of a sound from a series of given tones that vary in sound
- Playing a series of tones of emitted sounds
- Identifying emitted phonemes that vary in articulation point or mode
- Reproducing series of phonemes
- Identifying the location of a syllable/phoneme emitted from a sequence of syllables/phonemes
- Identifying a syllable/phoneme between the emission of two that differ in terms of the mode of articulation
- Matching two syllables/phonemes, which differ in articulation mode, with the drawing that contains them
- Deciding whether or not two auditory sequences formed by a syllable/phoneme differ in rhythm
- Identifying the drawing that begins with a certain syllable/phoneme from two spoken syllables/phonemes

Another area of intervention would be phonological processing, aimed at optimising deficits in the elaboration and interpretation of phonological information, such as phonological knowledge and the improvement of phoneme-grapheme correspondence. Activities to improve this area include [1, 12, 111–113]:

- Counting words in a spoken phrase
- Identification of syllables and/or phonemes in spoken words
- Finding rhyming words with a model
- Counting syllables and/or phonemes
- Sorting words by their syllables and/or phonemes, located in different positions
- Omitting syllables and/or phonemes in a spoken word
- Combining sequences of spoken syllables or phonemes to form words
- Adding syllables and/or phonemes to a spoken word
- Replacing a syllable or phoneme and pronounce the resulting word
- Reversing the order of spoken syllables or phonemes

Finally, intervention in orthographic processing deficits would also be necessary, improving visual word identification [1, 12, 111–113, 115]. Activities would include:

- Differentiating homophone words (understanding homophones)
- Choosing a word from a pair of homophone words according to the stated meaning
- Choosing the word written correctly between a word and a pseudo-homophone
- Completing words by adding vowels or consonants
- Matching a word with its drawing, giving a set of words (flash-card)
- Matching word and pseudo-homophone
- Selecting words represented in drawings from an array of words and pseudo-words
- Word search
- Identifying the model word from a sequence of words
- Forming words with given syllables

6. Conclusion

This chapter has addressed dyslexia from a neuropsychological perspective, specifically tackling the definition, main clinical manifestations, genetic, neuroanatomical and neurophysiological explanations, and finally neuropsychological intervention options.

Firstly, the main definitions of dyslexia [7–9] were analysed, highlighting some key considerations. In general, it is defined as a neurodevelopmental disorder that is biological in origin and which begins in childhood, with deficits in accuracy and/or fluidity in word recognition, pseudo-word recognition, and spelling. These difficulties are not consistent with the child's intellectual level or the school instruction received. They may or may not be accompanied by supralexical deficits, such as reading comprehension and written expression, and cognitive-linguistic manifestations and/or socio-emotional problems. The relevance of differential diagnosis with other deficits, such as intellectual disability, ADHD or sensory-motor problems, has also been indicated. Some definitions identify the psychological and cognitive origin of dyslexia, mainly in phonological components [7].

Secondly, the main clinical manifestations in three areas have been described. Academic deficits occur in terms of problems with the accuracy and speed of reading words and/or pseudo-words, and spelling. The problems are different depending on whether the dyslexia is phonological or visual. Phonological dyslexia is characterised by deficits in the reading of long, infrequent words or pseudo-words, with errors in lexicalisation (converting pseudo-words into words), slow reading speed, and sounding out of words. Writing deficits are gaps and improper joins of words. In contrast, visual dyslexia presents deficits in reading short, frequent, and

familiar words, with misunderstandings of homophones and in lexical decision-making with pseudo-homophones. Writing deficits occur in conventional spellings and exception words [2–4, 10–12]. The different academic manifestations mean that adequate neuropsychological assessment is necessary in order to establish appropriate intervention based on the phonological and/or visual deficits presented by the subjects. The manifestations of dyslexia in the linguistic cognitive areas have also been reviewed. It has been pointed out that the role of intelligence in dyslexia is not relevant, as subjects usually present an average IQ, with discrepancies between verbal and manipulative IQ, and low working memory indices. Subjects with dyslexia may also present deficits in visual processing with difficulties in discriminating between stimuli presented sequentially and temporarily, with slow response times and the need to increase intervals in order to perceive two stimuli independently [15–19]. Similarly, subjects with dyslexia may present deficits in auditory perceptual processing and speech perception, problems in the sequential perception of phonemes, low discrimination of frequencies and amplitudes in rapid temporal sequences, and deficits in perceived sound, place and point of articulation in phonemes [21, 22]. Dyslexics may also present deficits in auditory and phonological memory, with problems in the storage and retrieval of verbal information, which sustain deficits in phonological knowledge and problems in grapheme-phoneme conversion [23–25]. Other manifestations of dyslexia occur in knowledge of letters (the name and sound of the letter, and establishing the relationship between grapheme and sound), prosody (deficits in the perception of the tonic syllable of the word, pseudo-words and in sequences of syllables), phonological knowledge (deficits in perceiving and manipulating speech segments), RAN (slow naming of alphanumeric and non-alphanumeric elements) and executive function (use and control of cognitive and metacognitive abilities). Undoubtedly, the diversity of cognitive and linguistic manifestations makes it necessary to establish different typologies and characteristics in each of the areas identified, since they can translate into different approaches of evaluation and intervention. Subjects with dyslexia may also present clinical manifestations of a socio-emotional nature, with symptoms of depression or anxiety [42–44], maladaptive attributional styles [45, 46], problems of self-concept or self-esteem [47, 48], and low motivation [47]. These secondary or concomitant problems of dyslexia cause great psychological distress in the subject and in their immediate family environment, and are often the reason neuropsychological assistance is requested.

Thirdly, the different clinical theories of dyslexia have been reviewed, according to various genetic, neuroanatomical and neurophysiological factors. Genetic explanations have highlighted the relevance of family heredity, as dyslexia is more frequent when parents have also presented delays in their reading development, and given the concordance of reading deficits in monozygotic twins [55]. These findings have encouraged molecular research to focus on discovering potential candidate genes related to dyslexia deficits, with different genes responsible for different cognitive deficits (chromosome 21), phonological processes (chromosomes 3, 7, 13, and 15), or visual processes (chromosome 1). These findings promote research on the biological foundations of dyslexia and genetic programming of tissue development and structures responsible for reading and writing functions. However, the lack of conclusive results means that further research is required to investigate the specific typologies and deficits of dyslexia in order to find concrete genetic bases. We should also highlight the various neurological explanations put forward in recent decades, thanks to the development of neuroimaging techniques and interest in the functioning of the brain. Neuroanatomical hypotheses have developed theories based on deficits in different brain structures among dyslexic

subjects, such as cerebral asymmetry [71–80], corpus callosum [81–83], cerebellum [84–86], and volume of grey and white matter [87–95]. Undoubtedly, these brain structures present a different anatomical pattern to subjects with normal reading development patterns that serve as a basis for explaining the academic deficits presented by different dyslexic subjects. The different neuropsychological investigations must be unified to provide a neuroanatomical explanation of the different subtypes of dyslexia and the neuroanatomical bases that support them. Neurophysiological hypotheses, on the other hand, have sought to explain the functioning and organisation of the different areas of the brain that develop in the reading and writing of subjects with dyslexia, referring to the functioning of the magnocellular system and, in particular, the lateral geniculate nucleus of the thalamus, associated with auditory sensory perceptual processing deficits, motion sensitivity, and difficulties in discriminating low contrast and frequency stimulation [96–99]. Theories have also been presented that point to deficits in connectivity between different areas [100–103], between cerebral hemispheres (phonological deficits), asynchrony between Broca's Area and Wernicke's Area (recognition of phonemes and words), and deficits in transcallosal inhibition (low processing speed of letters and words). Finally, theories on the functional activity of neurological reading structures in dyslexic children in two circuits, dorsal and ventral, have also been discussed [104–109]. In conclusion, neuropsychological theories have identified the functioning of different brain structures during reading among subjects with normal reading development patterns, children who are learning to read, and in subjects with dyslexia. However, the findings of the different investigations need to be integrated in order to establish the role of the different structures of the Central Nervous System involved in subjects with dyslexia at different moments of learning, the subtypes of dyslexia, and in different languages, in order to establish an integrated and universal theory.

Finally, the chapter has addressed the importance of early neuropsychological intervention, as well as the need to establish scientifically tested and validated intervention methodologies. Early intervention in dyslexia has proven to be particularly effective when programmes target deficits in reading and/or writing, as they are more relevant to the specific needs of subjects, rather than focusing on general cognitive deficits, which are more non-specific and difficult to modify, making them therefore less sensitive to neuropsychological intervention. Finally, a number of neuropsychological tasks have been presented, aimed at improving visual and auditory perceptual processing, phonological processing, and orthographic processing [1, 12, 111–115]. Early evidence-based interventions are required so that the benefits of neuropsychological treatments and the validity of programmes can be quantified according to the deficits of each subtype of dyslexia.

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