Developmental dyslexia: Neurocognitive theories and challenges for educators

Reading is a recent, culturally mediated, cognitive acquisition. This complex cognitive process depends upon the concerted contribution of several cognitive processes of varying complexity levels. Neural circuits originally specialized in the processing of auditory, visual, sensory, and motor functions, together with complex brain networks involved in language and executive functions, are engaged in reading.

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Executive summary

- Developmental dyslexia (DDx) is the most common of the developmental learning disorders and frequently co-occurs with other neurodevelopmental disorders. Its prevalence ranges from 5–17%, depending on language and exact definition.
- DDx children exhibit significant and persistent difficulties in learning academic skills related to reading, including word reading accuracy, reading fluency, and reading comprehension, that cannot be better explained by intellectual, neurological or motor disorders, sensory impairment or poor instruction.
- Typically, a left-lateralized complex brain network is repurposed for reading acquisition. Children with DDx exhibit significant differences in the anatomical and functional properties of this network.
- The phonological deficit hypothesis presents DDx as an impairment in phonological awareness: the ability to perceive and manipulate the sounds of spoken words. This is the most researched neurocognitive hypothesis of the disorder. Other influential hypotheses address visual and/or auditory dysfunctions influencing reading acquisition.
- Typically, DDx is diagnosed around second grade or later. As a result, children with DDx lag significantly behind typically developing peers in reading ability and experience. Reading deficits are persistent and continue into adulthood, despite accumulating experience.
- DDx has a negative impact in individual access to higher education across cultures and leads to a severe cost for societies. The early detection of children at risk of DDx is essential, in order to allow the design of individualized pedagogical and neurocognitive stimulation, remediation, and intervention strategies.

Introduction

Reading is a recent, culturally mediated, cognitive acquisition. This complex cognitive process depends upon the concerted contribution of several cognitive processes of varying complexity levels. Neural circuits originally specialized in the processing of auditory, visual, sensory, and motor functions, together with complex brain networks involved in language and executive functions, are engaged in reading[1].

Reading development has been directly associated with phonological awareness: the ability to perceive and manipulate the sounds of one's language[1]. To learn to read in any alphabetic language, it is necessary to connect individual letters to the corresponding sounds in order to produce and access whole-word phonological representations of familiar words. Initially, serial identification of the letters forming the words is carried out, as the early reader learns the grapheme-to-phoneme mappings, where graphemes are graphic symbols representing sounds and phonemes are the smallest units of sound that change the meaning of a word[2]. This process of "decoding" is the foundation of word identification. This improving ability of "sounding out" words allows the reader to establish high-quality lexical representations and to connect the phonological, orthographic, morphological, and semantic representations of words "stored" in memory. Finally, once lexical representations are available, the reader no longer needs to rely on phonics but can automatically identify familiar words[1]. In this type of decoding, words can be treated as special kinds of objects, and the corresponding "visual word forms" activate the corresponding phonological and semantic representations.

Oral reading fluency, the ability to translate written text to spoken words with speed and accuracy, is considered an overall indicator of reading development and competence[3]. It is influenced by domain general cognitive mechanisms, such as attention and temporal synchronization[1]. Oral reading fluency requires lexical and general knowledge, proper identification and use of linguistic cues, and reasoning skills. Additionally, the language itself and the degree of "orthographic transparency" of the language, in terms of the grapheme-to-phoneme mappings, also influences reading performance[4]. For instance, in alphabetic languages, like Spanish, phonological awareness is essential; however, in logosyllabic languages, like Chinese, visual memory and visual processing are more relevant[1].

The "reading brain"

Using neuroimaging techniques, a core "reading network" has been revealed that is located primarily in the left hemisphere of the brain. This network includes three main specialized regions[5]:

- A circuit around "Wernicke's area," in the left temporo-parietal cortex, including the posterior superior temporal gyrus and supramarginal and angular gyri of the inferior parietal lobule. This system is thought to be involved in phonology-based reading processes (i.e., grapheme–phoneme conversion and phonological assembly).
- A left inferior frontal circuit, around "Broca's area," including inferior frontal and precentral gyri. This system is thought to be involved in processing speech sounds.
- A left ventral occipito-temporal circuit including lateral extrastriate, fusiform, and inferior temporal regions and the so-called "visual word form area" (VWFA). This system is specialized in recognizing print as a special kind of visual percept.

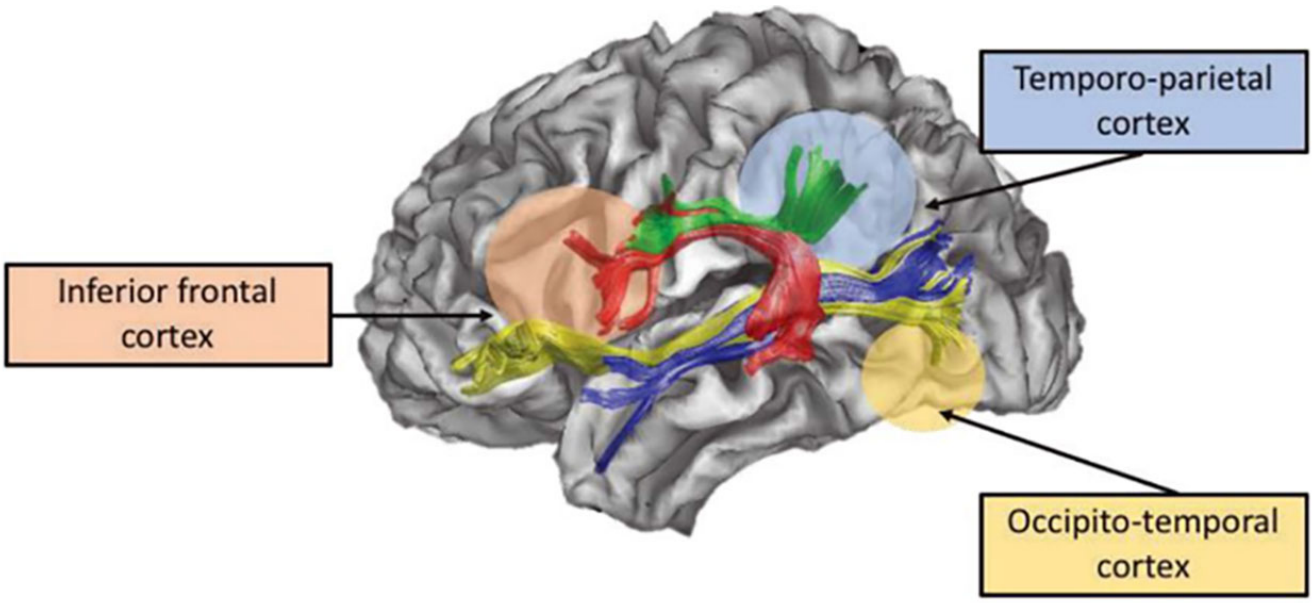

Figure 1. Brain regions and white matter tracts (arcuate fasciculus-*red,* inferior frontal occipital fasciculus-*yellow,* inferior longitudinal fasciculus-*blue,* and superior longitudinal fasciculus-*green)* important for reading that are commonly found to be associated with atypical function or structure in developmental dyslexia.

Figure 1. The reading network[6].

Converging evidence across functional magnetic resonance imaging (fMRI) studies (using functional images of the variations of blood flow to the active areas) shows that, in the early stages of reading development, children exhibit bilateral activations (in both brain hemispheres), in temporo-parietal, temporo-occipital, and inferior frontal regions. However, during early elementary school years, typically developing children shift from bilateral to left lateralized activation of these areas. This pattern remains relatively stable into adulthood[7].

These core regions, and other areas that interact to support reading, communicate through three left-hemisphere pathways: (1) the left arcuate fasciculus, connecting temporo-parietal cortex with frontal regions, supporting phonological processing; (2) the inferior longitudinal fasciculus, connecting the temporal and occipital lobes, including the visual word form area, and implicated in connecting print to meaning; and (3) the superior longitudinal fasciculus, connecting parietal and frontal lobes and important for mapping phonemic representations to motor representations. These tracts' properties are associated with phonological awareness and reading performance in typically developing children[6] (see Figure 1).

Defining developmental dyslexia

According to ICD-11, the developmental learning disorder with impairment in reading (or DDx) is characterized by significant and persistent difficulties in learning academic skills related to reading, including word reading accuracy, reading fluency, and reading comprehension, not better explained by intellectual, neurological or motor disorders, sensory impairment, or poor instruction[8].

DDx is the most common neurodevelopmental disorder, found in about 80% of children with learning disorders[2]. Its prevalence ranges from 5–17%. Familial studies suggest DDx is heritable. It has been reported in up to 68% of identical twins

and up to 40–60% of individuals with a first-degree relative with dyslexia[9-11]. Several candidate genes for dyslexia susceptibility have been suggested (e.g., ROBO1, DCDC2, DYX1C1, KIAA0319), which all seem to be significantly involved in brain development[1].

DDx seems to be associated with morphological and functional atypicalities within the complex network of brain regions underlying reading. Scientists have developed a hypothesis attempting to reconcile genetic effects, developmental brain changes, and perceptual/cognitive deficits in dyslexia. This suggests that changes in the functioning of any number of genes involved in cortical development implicated in dyslexia may lead to subtle cortical malformations involving neural migration (the process by which neurons reach their final destination in specific brain regions) and axonal growth (growth of the structures in the neurons that connect the body of the cell with the next neuron to which it communicates in the circuit). Hence, these genetic changes result in atypical circuits among brain cortical regions (cortico-cortical) and in atypical circuits connecting areas in the cortex to deep (subcortical) brain areas, including the thalamus (a key relay station for incoming sensory information). The alterations in these circuits may explain the range of sensorimotor, perceptual, and cognitive deficits reported in dyslexia[1]. It remains debatable, however, which brain characteristics of DDx are the result of reduced reading practice and which precede the onset of reading instruction[12].

DDx has been reported in all languages studied so far, although its manifestation differs with the orthography of the language[4]. It has been reported that, across cultures, DDx children are disadvantaged, regarding both school learning and later employment. According to an analysis conducted in the United Kingdom, having dyslexia incurs costs of over £80,000 on lifetime earnings[12]. A recent metanalysis conducted in the US included a sample size of 552,729 participants and showed males are 1.83 times more likely than females to present with reading difficulties. The gender ratio is greater when the reading difficulties are more severe[13]. Additionally, other cognitive deficits are also present in DDx, as revealed by a recent metanalysis which has reported executive function deficits in children with dyslexia[14].

Several preliteracy predictors of dyslexia have been consistently reported, including rapid automatized naming (RAN), phonological awareness (PA), letter knowledge (LK), and verbal short-term memory (STM). RAN is the ability to rapidly retrieve the names of familiar items, presented visually, in a serial array (e.g., objects, colors, numbers, letters, or a combination of them). PA refers to the understanding of the sound units of oral language and is measured by the ability to manipulate linguistic sounds independent of meaning. LK includes both, letter name and sound knowledge. STM refers to the capacity to maintain and process information (e.g., digits, pseudowords) for a short period of time[15]. Prereading profiles of these abilities—PA risk, RAN risk, and double deficit (both PA and RAN risk)—have been associated with particular profiles of dyslexia. They are stable across developmental time according to a longitudinal study following 1,215 kindergarten/prekindergarten children until the end of the first grade. Profile membership at the beginning of this study was significantly predictive of later reading performance. Note researchers found a higher frequency of PA and RAN deficits in children from lower socioeconomic status backgrounds[15].

A teacher may suspect children are at risk of reading difficulties, or even DDx, when they exhibit consistently some or all of the following behaviours:

- Read very slowly and make frequent mistakes.
- Confuse similar letters (e.g., p/b) when reading or writing.
- Read infrequent or novel words incorrectly.
- Use fingers to follow the text.
- Read very fast, guessing the words.
- Show difficulties in understanding age-appropriate texts.
- Make many spelling errors while writing.
- Have difficulties in detecting the motion direction of sets of objects.
- Have difficulty in identifying rhymes.
- Are very slow when nominating colors, objects, numbers, or letters.
- Show very poor vocabulary.
- Have difficulty retaining information and processing it for short periods of time.

The "dyslexic brain"

DDx children and adults show both functional and structural brain differences in the reading network compared to typically developing readers. Some fMRI studies have reported DDx children show underactivation compared to typical readers in temporoparietal and occipitotemporal (VWFA) regions while reading and performing phonological tasks, across different cultures and stages of development[16-17]. Others have reported increased activation in the inferior frontal gyrus, in dyslexic

compared to typically developing readers. This has been interpreted as a compensatory mechanism, probably reflecting reliance on memorization of the structure of words rather than phonological processing[18]. Also, in general, struggling readers show a distinct brain activation pattern that relies on right hemisphere homologous regions in the posterior temporo-parietal and temporo-occipital regions[7].

Additionally, some studies focusing on brain structure have reported reduced gray-matter volume or thickness in the same areas showing altered brain activity. Impairments in the white matter cortical connections between these regions and reduced structural connectivity in the three white-matter tracts that integrate the components of the reading network have been reported among dyslexic readers[19-20]. However, a recent metanalysis showed no reliable differences between dyslexics and typical readers, nor association between white matter tracts integrity and reading ability. This challenges the idea that there are clear structural differences underlying reading ability in DDx and suggests its neural correlates might be more subtle than anticipated[18].

Brain differences have been reported in children at risk of reading disorders, before reading instruction even begins. Studies in prereading children (ages 5 to 6) at familial risk for dyslexia have shown reduced gray-matter volume in occipito-temporal and temporo-parietal regions and lower levels of the fMRI signal corresponding to these regions, when presented with auditory tasks requiring phonological awareness or rapid auditory processing of information[21], or just in response to common words in the VWFA, when compared with an age-/gender-matched control group [22]. Studies conducted in newborns have shown that babies from families with a history of dyslexia display altered brain response to language during the first days of life, although only around half of all these children will experience reading disorders in the future[23]. Other studies show that these brain activity differences in early infancy are related to language and reading difficulties in the following years[24]. These findings support the early identification of children at risk of DDx.

Neurocognitive theories: The phonological deficit hypothesis

The dominant view is that DDx is an impairment in phonological awareness[12]. Deficits in phonological awareness are thought to hinder the ability to map speech sounds onto their corresponding visual symbols (letters), preventing fluent reading.

The sounds represented by the alphabet are an abstraction from rather variable acoustic signals. For instance, "p" and "b" sounds are very similar and can be used interchangeably (misrepresented) by young children when starting to recode sound into graphemes. The ability of infants to make categorical phonemic distinctions, putatively by grouping acoustically distinct sounds together and treating them as the same phoneme, is significantly influenced by alphabetic learning. In fact, it has been reported that phonological awareness development follows a hierarchical sequence (in all languages studied so far) and shows a causal relationship with literacy. Illiterate adults and Chinese adults who have learned to read by matching characters to meaning show poor phoneme awareness[12]. In this view, phoneme awareness emerges as a consequence of learning to read an alphabetic orthography, such that learning to read "re-maps" phonology in the brain[25]. An interesting metaphor suggests that acquiring the alphabetic code is like catching a virus which infects all speech processing and, from that moment on, whole word sounds are automatically broken up into sound constituents and language is never the same again[26].

Individual differences in phonological awareness have been reported to predict how rapidly and well phonological recoding skills are learned. Also, difficulties in the phonological recoding of visual letters to the corresponding sounds are considered a core deficit in dyslexia, in all languages studied[12]. These deficits are thought to originate from atypical or impaired representations of spoken word forms in the mental lexicon: the brain's mental dictionary, containing information about the meaning, pronunciation, and grammatical status of words in the spoken language[12].

However, phonological representations are essentially dependent on multimodal input, being supported from early infancy by auditory input, by visual information from the face and lips of the speaker, and by motor information deriving from the process of learning to talk. Hence, visual, auditory, or both sensory dysfunctions could lie at the heart of the phonological deficit; although, so far, the studies' results are mixed. On the other hand, struggling readers will avoid reading and so accumulate less experience and train the corresponding key reading skills less, such as visual scanning, oculomotor control required for exploration of the text, and visuospatial attention skills. In contrast, children who read more tend to automatize most of these processes. Also, they develop better vocabulary, which enhances their phonological recoding skills[12]. Hence, reading experience is another factor to consider in the study of reading deficits.

Sensory theories of DDx:

Several theories suggesting that DDx originates from a sensory dysfunction have been proposed[12]. Auditory deficits have been offered as a cause of the phonological deficits present in DDx. The first auditory theory proposed developmental difficulties in processing auditory information that arrives rapidly and sequentially[27] (as in the frequency variations that occur as a speaker moves from a consonant to a vowel, for example). This was called the "*Rapid Auditory Processing (RAP)*" theory. Children with specific language impairments show lower RAP compared to controls, however the findings are inconsistent in the case of dyslexia. Additionally, phonological performance of DDx children does not improve when the temporal information in speech is slowed down[12]. A more promising theory, coined the "*Rise Time*" theory, proposes that DDx children have deficits in the detection of speech rhythm and prosody that explain the phonological deficits. This, in turn, is determined by a difficulty in natural speech encoding, or in discriminating the rise times of the "amplitude envelope of speech": the patterns of amplitude (intensity) modulation at different temporal rates of utterances that allow the listener to parse auditory input in meaningful units for further analysis[28-29]. This theory has received support from studies measuring rise-time sensitivity in DDx children across languages that have reported impaired discrimination of rise time compared with matched controls[12].

Also, visual deficits have been hypothesised as causes of DDx. One of these theories suggests that impairments in visual attention (the amount of visual individual items that can be maintained across brief disruptions to sensory input, as in blinking) might underlie dyslexia, and that this impairment in the processing of multiple-item arrays limits reading development[30]. However, several studies supporting this theory have been based on tasks using letters, or required oral reporting, which obscures interpretation regarding causality[12]. Another theory suggests an impaired ability to orient spatial attention to relevant external cues causes dyslexia[31]. This theory suggests dyslexic brains cannot move smoothly from letter to letter, successfully suppressing flanking letters when recoding letters to sounds. This hypothesis has been challenged by arguing that reduced reading experience may explain the attentional deficit[12].

Finally, the "*Magnocellular Deficit*" theory suggests that mild dysfunctions in visual motion processing are related to DDx. The magnocellular dorsal and parvocellular ventral pathways (MD and PV pathways) are the major pathways of the visual system, and account for most of the axons leaving the retina. The destruction of these pathways causes loss of vision[32]. Both pathways originate in the ganglion cells of the retina, then the MD projects through the layers 1-2 of the lateral geniculate nucleus (LGN) of the thalamus, and finally reaches the occipital and parietal cortices, while the PV pathway projects to layers 3 through 6 in the LGN and runs ventrally to the occipital cortex. Motion perception is one of the functions of the MD pathway but is the most accepted proxy of MD functioning. The theory stems from the observation that up to 75% of dyslexic individuals show visual MD deficits[2]. A postmortem study showed that magnocellular neurons of the LGN were significantly smaller in individuals with DDx compared to normal readers, whereas the parvocellular neurons did not differ between groups[2]. At the same time, a study has reported a smaller lateral geniculate nucleus volume in individuals with DD compared to typically developing controls[33]. Recently, a study took a comprehensive approach in addressing the relation between the MD deficit and DDX, including comparison of DDx children with reading level-paired controls, prospective longitudinal analysis, and remediation studies targeting MD processes and then measurement of the corresponding effects in reading[2]. This study reported a motion perception deficit in DDx children compared to age-matched and to reading level-matched children, ruling out that the MD deficit is caused by poor reading skills/experience. Additionally, the researchers showed prereading visual motion perception, independent from auditory–phonological skill, predicted future reading development. Finally, they proved that targeted MD training using active videogames and coherent motion detection tasks adapted for stimulation, not involving any auditory–phonological stimulation, led to improved reading skill in children and adults with DDx.

Implications for teachers and policy makers

DDx is considered a complex neurodevelopmental disorder, characterized by multiple interacting deficits that lead to reading impairments. Hence, teachers and policy makers should bear in mind that supporting DDx children will require the implementation of specialized and individualized teaching and neurocognitive interventions, together with the provision of increasing time of reading experience in educational settings and at home. Introducing the neurocognitive foundations of reading in teacher training and professional development programs may support local and global decision-making processes.

DDx has a negative impact in individual access to higher education across cultures and leads to severe cost to societies. The early detection of children at risk of DDx is essential and feasible. In contrast, DDx is usually diagnosed around second grade or later, when children with DDx already lag significantly behind typically developing peers in reading ability and experience. The implementation of sensory deficit assessments in prereaders and infants may contribute to the design of more effective DD remediation and prevention programs. Although reading deficits persist into adulthood, both children and adults may

benefit from the stimulation of the sensory systems underlying phonology, and from the direct stimulation of phonological awareness. However, the promising results presented in the scientific literature still require sufficient replication and appropriate translation into appropriate intervention tools.

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