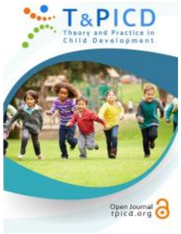

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Dyslexia and the Brain: Understanding the Neuroscience of Dyslexia

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ABSTRACT

Learning can be characterized as altering and strengthening neural connections and networks in the brain. Cognitive neuroscience provides us with a large literature related to the structures and functions of the brain. As our knowledge of neuropsychology and learning increases, so does the opportunity to pattern more successful educational practices. However, taking advantage of these scientific developments requires integrating understanding from many fields, from sociology, psychology, developmental and learning sciences, and linking them to knowledge of emerging successful approaches in education. In addition, the brain's incredible ability to change and adapt (neuroplasticity), that is, the real physiological adaptation ability that occurs in the brain when it interacts with the environment, can have a huge impact on special education, especially in the area of learning difficulties. This review summarizes neuropsychological studies on dyslexia in general.

KEYWORDS

Brain; neuroscience; neuroplasticity; dyslexia.

INTRODUCTION

How complex thoughts and behaviours emerge from the complex neural networks in the brain has been the main subject of brain research. This complex network of relationships between brain-thought-behaviour determines individual characteristics (Fisher & DeFries, 2002). As a result of this complex network of relationships, the cognitive, affective and motor changes experienced by the individual consciously or unconsciously are generally defined as learning. Three fundamental changes occur in the brain to support learning (Tommerdahl, 2010). In chemical change, the processes of transmission, storage and recall of information particles are performed by triggering a series of actions and reactions by secreting chemical substances between brain cells and neurons. This change triggers a series of reactions (Lenroot & Giedd, 2006). The brain supports learning by increasing the concentration of this chemical signal between neurons. As this happens quickly, a series of improvements in short-term memory and motor skill performances are likely to be seen. Another response of the brain to support learning is changes in the brain structure at the micro level (Thomas & Knowland, 2009). This change is supported by making new connections between brain neurons during learning. As the physical structure of the brain changes at the micro level, learning takes place over a long period of time. These changes in the physical structure of the brain at the micro level are usually associated with long-term improvements in long-term memory and motor skills. With the increase in the frequency of use of the area responsible for carrying out some tasks, it becomes easier for the brain to adapt to new learning. An example of this is that an individual who constantly solves math problems gains practice in solving math problems after a while (Tommerdahl, 2010).

Genes that determine individual characteristics form the skeletal structure necessary for the cognitive processes that help the individual to shape his own behaviour by enabling the individual to acquire and store information such as language, memory and learning (Fisher & DeFries, 2002). Studies conducted with identical twins (with the same genetic) structure show that the personality, reading abilities and mathematical abilities of these individuals are more similar than twins with different genetic structures, but genetic predisposition alone cannot shape an individual's learning ability; genetic susceptibility interacts with environmental factors at all levels (Hogarth et al., 2010). Although it is generally agreed that individual differences may have a genetic basis, the effect of genetic traits on brain development and brain function has not yet been fully discovered (Howard-Jones, 2014). For example, although genetic predispositions may partially explain differences in reading ability, there is no single gene that makes an individual a good or poor reader. Instead, there are multiple genes with little specific effects. In addition, environmental factors such as diet, exposure to toxins and social interactions are also cited as situations that can cause differences in reading ability (Thomas & Knowland, 2009). In addition, in the current situation, it seems somewhat impossible to tell whether an individual is a good or a bad reader by measuring activity in brain regions in terms of neurobiological (biology of the brain and central nervous system) (Geake, 2004). While there are genetic conditions that affect an individual's learning skills and cause some extreme abnormalities, most changes in an individual's learning capacity are due to multiple genetic and environmental influences and each of which can have a small impact. In this sense, "neuroscience has the potential to help us understand the genetic predispositions that affect cognitive skills for each individual and how these predispositions can be developed through education and upbringing" (Lenroot & Giedd, 2006).

In the 19th century, advances in imaging technologies such as Magnetic Resonance Imaging (MRI), functional Magnetic Resonance Imaging (fMRI), Electroencephalogram (EEG) led scientists working in the field of neuroscience to interpret the microstructure and function of the brain (Koizumi, 2004). These technological developments have brought new findings on the brain regions responsible for learning, the relationship between neurons and hormones with brain development, the biological responses of the brain to certain physiological stimuli, and the interrelationships of cognitive process skills (Howard-Jones, 2014). These findings contributed to the improvement of all fields, including education, and encouraged the emergence of various teaching methods related to brain-based teaching methods. Brain-based learning (Caine et al., 1999; Tang, 2017), cognitive neuropsychology (Ansari & Coch, 2006; Fischer-Baum & Campana, 2017), neuroscience (Ansari et al., 2011) and educational neuroscience (Immardino-Yang & Damasio, 2007; Zadina, 2015) are among these teaching methods.

Both educators and neuroscientists are constantly interested in how teaching and learning activities can be efficient (Goswami, 2004). While education aims to make teaching accessible to all individuals, neuroscience examines the biological brain and the cognitive processes involved in learning such as attention, memory, information processing, reasoning, and language (Gabrieli, 2016). Especially, educational neuroscience, a newly developing branch of science, combines studies on neuroscience, psychology and education to examine the neurocognitive processes underlying educational practice and theory as a whole. The ultimate goal of the field is to develop teaching methods and curricula on a neurocognitive basis and to develop children's high-level cognitive skills in a democratic classroom environment (Lee & Mudaliar, 2009). In this sense, advances in neuroscience have the potential to have significant effects on the future of education. Studies in neuroscience are expected to contribute to the development of learning-teaching strategies on an individual basis, to ensure emotional and social participation in the individual, to develop cognitive processing skills and learning behaviours (Tommerdahl, 2010). In addition, neuroscience studies can provide a broad perspective to the educational psychology literature on social, emotional and internal factors affecting the research and application environment in education and effective teaching methods in the context of teaching-learning (Clement & Lovat, 2012; Prickaerts et al., 2004).

Findings from neuroscience studies can help identify the specific educational needs of individuals related to difficulties that affect learning, such as specific language and speech disorders, dyscalculia, and dyslexia (Zamarian et al., 2009). It is hoped that studies conducted in the field of neuroscience will develop effective screening methods and inclusive educational supports for individuals with learning disability, and a wider literature on brain development of individuals with autism and pervasive developmental disorders (Stoodley, 2016). For example, scientific studies about the cognitive processes of an individual with dyslexia will provide us with unique opportunities to make educational adaptations and develop appropriate teaching methods and strategies (Van der Lely & Marshall, 2010). In this study, after evaluating the developmental processes of the brain in general, the functional properties of the brain lobes were explained and the plasticity feature of the brain was examined and the neurocognitive characteristics of the students with dyslexia were analysed in all these contexts.

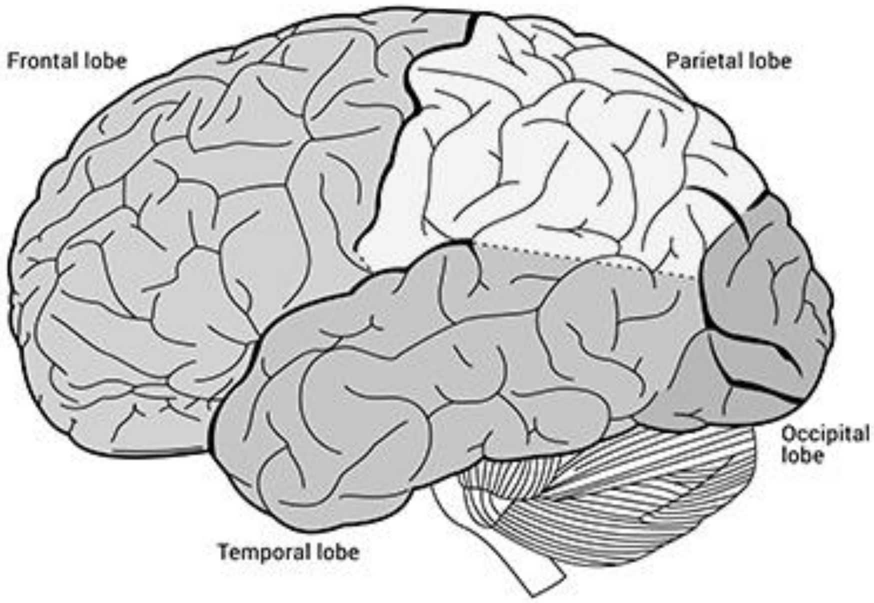
The Brain and Its Development

As seen in Figure 1, the cerebral cortex (Temporal lobe, Frontal lobe, Occipital lobe, Parietal lobe) consists of many lobes and each of them play more active roles in carrying out specific tasks (Meschyan & Hernandez, 2006). The frontal lobe determines communication skills, as well as executing planning and reasoning functions, and controls the type and degree of emotional response to events or situations. The temporal lobe basically executes functions with memory, hearing, language and object selection. The parietal lobe controls our sense of touch and carries out spatial processing and perception functions. Occipital lobe controls vision. Although these basic structures in the cerebral cortex are similar in all adults, they may differ greatly from individual to individual with the differentiation of a number of possible developmental steps in the early stages of age (Cantlon et al., 2011). Even in genetically identical twins, there may be striking differences in the number of neurons responsible for carrying out the same functions. Culture and gene pools and possibly the impact of the environment on current development can be cited as the main causes of individual differences (Raschle et al., 2011). Cognitive differences can sometimes have remarkable beneficial effects on basic functions. For example, neurocognitive imaging studies show that adults with visual impairment are more practical in processing auditory information than adults with visual ability, and adults with congenital hearing impairments are faster than normal adults in processing visual information in the peripheral field (Meschyan & Hernandez, 2006).

Many critical stages of brain development are actually completed before birth. While neural development shows similarity between genders, the rate of brain maturation usually varies (McDonald al., 2008). Brain development is completed a little later in boys than girls on average. Brain development begins with the formation of the cells that make up the brain in the first weeks of pregnancy (Hoffman & Mcnaughton, 2002). Before birth, these cells move from different regions of the fetal brain to regions where they will develop in the mature brain. At seven months of pregnancy, almost all of the neurons that will form the mature brain are formed (Stromberg, 2013).

Figure 1

Brain Lobes



Alcohol and drug addiction have very specific effects on brain development. For example, the parietal lobe is less developed in babies with fatal alcohol syndrome. Since the parietal lobe is critical for arithmetic operations, individuals with fatal alcohol syndrome experience certain problems in number processing and mathematical operations (McDonald et al., 2008). Since most aspects of brain development are completed in the uterus, postnatal brain plasticity development (adaptation of the brain to the situation) is little affected by environmental factors (Kolb et al., 2003). After birth, brain development consists almost entirely of connections between neurons and synapse growth through processes called 'synaptogenesis'. After birth, the intensity of vision and hearing (visual and auditory cortex) synaptogenesis begins early (Stoodley, 2016). In other areas, such as the prefrontal cortex (brain region involved in planning and reasoning), developmental density is slower and peaks after the first year after birth, and it can take at least 10-20 years for the intensity level to decrease. Therefore, significant brain development may occur in the frontal regions even during adolescence (McDonald et al., 2008).

The left parietal-temporal (where the frontal lobe connect with the temporal lobe) area handles word analysis processes. This area matches written letters and words with sounds (letter sounds and spoken words). This area also handles the tasks of understanding written and spoken language. The second area important for reading skill is the left occipito-temporal area (where the occiptal lobe connect with the temporal lobe). The area in question is a critical area for fluent reading skills as it manages automatic and fast access to words (Hudson et al., 2007).

Brain metabolism is above adult levels in the first years after birth. Glucose intake is approximately 150% of adult levels in the fourth and fifth years (Palmer et al.,2004). By the age of about ten years, the brain metabolism in the cortical region drops to adult levels. Brain development subsists of bursts of synaptogenesis, condensation, and subsequent rearrangement and stabilization of synapses in the next process (Liu & Cull-Candy, 2000). This developmental cycle is seen at different times and at different rates for different brain lobes. This means that there are critical periods for the development of contrasting types of knowledge, considering Bloom's knowledge dimensions (factual knowledge, conceptual knowledge, operational knowledge and metacognitive knowledge). During this time, the brain volume nearly quadruples between postnatal and adulthood (Gabrieli, 2016).

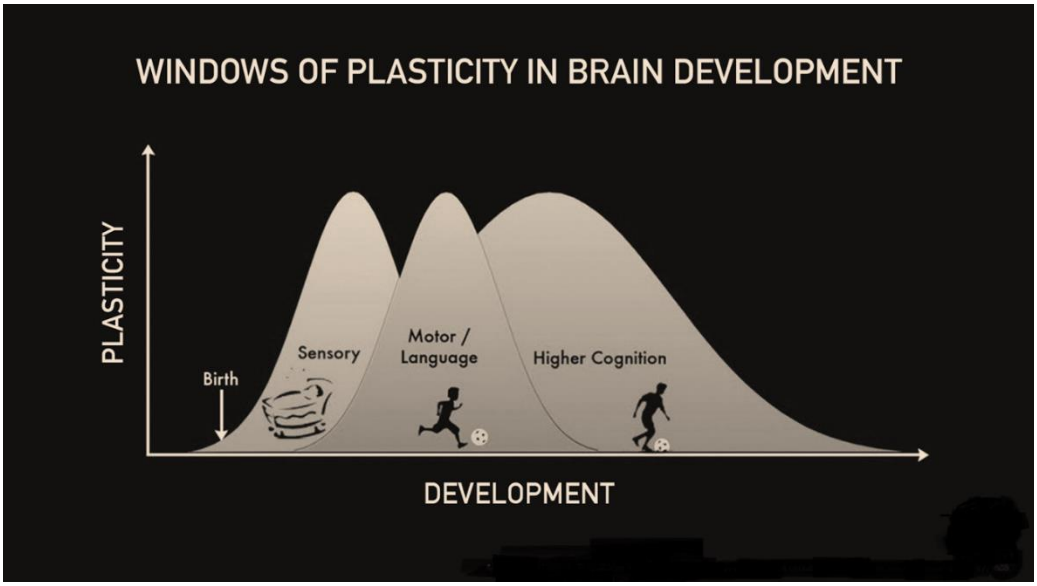
As the brain is highly flexible both developmentally and functionally, it constantly creates new connections to respond to new learning or environmental events (fall or motorcycle accident) even in adulthood (Malinow & Malenka, 2002). Likewise, critical periods are not subject to all-or-none law (Bredt & Nicoll, 2003). In this sense, developed sensitivities do not "turn off" and the brain's inability to be exposed to critical stimuli in critical periods does not completely adversely affect the brain's enhanced sensitivity. For example, the lack of visual input in the early period does not mean that the visual system cannot be fully developed (Gabrieli, 2016). The effects of critical stimulus deficiency in the early period vary depending on visual function (Palmer et al., 2004). Delayed functions (for example, depth perception) are more affected by relatively normally maturing functions due to the lack of critical stimuli, and because the sensitive period is missed, these abilities are less likely to reach their full potential (Malinow & Malenka, 2002).

The extraordinary adaptability of the brain, called 'neuroplasticity', is due to the simultaneous activation of the connections between neurons. Neuroplasticity is the ability of the brain to respond to internal or external stimuli by restructuring its function and connections (Hotting & Roder, 2013). Micro-level plasticity enables the brain to learn new behaviours and skills (Austin et al., 2014). Likewise, plasticity itself can alter the structural functions of the brain and strengthen the brain's response to stimuli (Krafnick et al., 2011). Plasticity based on experience continues for life. Changes in the structure and connections of the brain, depending on the situation, can be seen more frequently in sensitive periods from childhood to adolescence (Bartha & Benke, 2003). Naturally, plasticity contributes to decrease with age. This is particularly evident in a second language learning process (Hotting & Roder, 2013). The mastery of speech sounds and grammar structure can generally be better in individuals who try to learn a second language before adolescence compared to individuals in the post-adolescent period (Meschyan & Hernandez, 2006).

During adolescence, specific parts of the brain undergo further transformation than other parts (Krafnick et al., 2011). As can be seen in Figure 2, areas that undergo priority change with development are listed as neural development, motor and language development and finally metacognitive skills. The skill areas of the brain that can change the most before adolescence can be listed as self-awareness, internal control, perspective taking, control skills such as responses to feelings such as guilt and shame (Austin et al., 2014). Neural connections related to these changes in the brain can change gradually throughout development. Neural connections in impulse control and other 'executive' functions in the frontal lobe in the anterior part of the brain may change depending on environmental factors starting from pre-adolescence and post-adolescence period (Hotting & Roder, 2013). Even after these developmental periods, activity-dependent plasticity can persist throughout life. For example, it has been found that taxi drivers who have tried to adapt to the complex city layout of London for years have a large volume of gray matter in the brain due to memory and navigation activities. It has been determined that there may be a reversal in the brain plasticity of these drivers after retirement periods when they do not use their spatial memory and navigation skills (Krafnick et al., 2011). In another example, individuals with visual impairment can be shown to distinguish sounds and smells better. Therefore, changes due to neuroplasticity that occur in the brain after the acquisition of certain skills also depend on environmental factors that determine our experience (Prickaerts et al., 2004).

Figure 2

Age-related growth and plasticity (Hensch, 2005)



In terms of neurocognitive learning, on the other hand, learning is explained by the physical and chemical change in our brain cells through cell communication and synaptic mechanisms (connecting one brain cell to another through a synapse) (Clement & Lovat, 2012). The transmission of available information occurs through electrical signals that pass through the brain cells (or neurons), synapses, and trigger the release of neurotransmitters (chemical messengers). There are approximately 90 billion neurons in the left part of the brain, 200 million more than the right side, and each neuron has a connection with another neuron around it (Austin et al., 2014).

Neural activity patterns are thought to correspond to certain mental representations. Therefore, learning mainly corresponds to changes in connectivity in synapses (Paulesu et al., 2014). In this sense, a successful learning program is directly related to the context provided to the student by the teacher, the classroom and the family, as well as to the changes in brain function by changing the existing connections at the synapses (Paulesu et al., 2014). In addition to all these factors, environmental factors also cause changes in the neural level in the brain and affect the individual's learning. For example, it has been found that children who have been maltreated and grow up in unfavourable family environments but carry high levels of the MAOA gene (monoamineoxide A) do not have anti-social behaviours (Caspi et al., 2002). Protection from these antisocial behaviours can occur by alleviating neural responses to stress. In addition, it has been observed that various drugs used in children with some disabilities have positive effects on cognitive functions. For example, the drug methylphenidate (Ritalin), which is frequently used in children with ADHD (Attention Deficit Hyperactivity Disorder), contributes positively to their learning by increasing attention skills in children against auditory and visual stimuli (Kotaleski & Blackwell, 2010). Neurocognitive imaging techniques provide us with a large literature on the effects of different drugs, food additives and potential toxins on children's educational performance.

Dyslexia

Learning disabilities are usually grouped according to academic skills. The types of learning disabilities can be listed as reading (dyslexia), writing (dysgraphia) and mathematics (dyscalculia). Children with learning disabilities may experience problems in memory, motor processing, attention, perception, planning, information processing speed, and problem-solving skills (Judge & Watson, 2011). These disabilities arise from developmental brain injuries. Therefore, remedial education (attention, memory, working memory, processing speed) plays an important role in the intervention of individuals with learning disabilities (Judge & Watson, 2011).

Recent studies show that dyslexic individuals have less plasticity in their brain activity than 'normal' individuals. When brain scans of individuals with and without learning disabilities were compared, it was found that rehearsal teaching in individuals with learning disabilities caused a decrease in neural adaptation (Cooper & Mackey, 2016). That is, when the previously shown information was given again, the individuals examined and processed the information as if it was completely new. Accordingly, the sign of reduced plasticity can mean a decrease in repetition ability both socially and academically (such as word repetition). Reading involves the plasticity elements that need to be transformed into sounds and then blended with words (Knowland & Thomas, 2014). This may help explain why static learning strategies such as memorization and rote learning are not so effective for children with learning disabilities.

Dyslexia and Neurocognitive

Studies of children and adults with a history of dyslexia suggest that neural differences may be a cause or effect of dyslexia (Cantlon et al., 2011; Gabrieli, 2016). Neuroimaging data for infants and young children at risk of dyslexia show that there are some differences in the brain even before they start learning to read. The approaches adopted by dyslexia studies focus on studies of neural structure and function in children and infants with a family history of dyslexia and therefore at high risk of dyslexia (Raschle et al., 2011). Dyslexia is inherited, and the clues that family history of dyslexia and parents' phonological awareness give us about future reading competence in children increase the accuracy of the model established for the relationship between dyslexia and the analysis of early neural structure (Gabrieli, 2016). Other studies examine the neural relationships of behaviours (such as poor phonological awareness) that are known to be highly associated with dyslexia diagnoses (Bowers, 2016). Determining the neural relationships of these risk factors is particularly important in identifying the high risk for dyslexia early, because in the current situation, the presence of dyslexia in the child is diagnosed only after the initiation of learning to read at school (Raschle et al., 2011).

In addition to neurocognitive studies, behavioural studies can provide predictions about whether the phonological awareness, receptive-expressive vocabulary and rapid naming deficits observed in the individual before starting reading instruction in children with and without a family history of dyslexia may be a sign of dyslexia in the future (Cantlon et al., 2011). The performance of the individual in reading skill is generally evaluated with rapid automatic naming skills. It has been stated that 60-75% of dyslexic individuals may have limitations in their rapid automatic naming skills (Meyer & Felton, 1999). Both phonological awareness and fluency are important predictors of an individual's future reading skills and contribute to reading success at different levels (Meyer & Felton, 1999). Therefore, both phonology and fluent reading skills should be considered in the diagnosis of dyslexia. Also, although historically dyslexia have been defined as a discrepancy between IQ level and a low-level reading skill, there is much evidence in the literature that dyslexia is not related to IQ (Aparicio et al., 2007). Generally speaking, dyslexic individuals have the same brain development as typically developing individuals. This shows us that reading skill is a phenomenon independent of intelligence measured using IQ tests (Powell, Stainthorp et al., 2007).

According to neurocognitive studies, neural function and structure differences in the brain stand out as the cause of dyslexia in children (Olulade et al., 2013). In pre-reading age children at risk, the reading network in the left hemisphere shows consistent structural and functional differences compared to “normal” individuals (Raschle et al., 2011). These differences are more pronounced in infancy. For example, differences were observed in the left hemisphere neural responses to speech sounds of 6-month-old babies who are inherently at risk of dyslexia due to their family (Stoodley, 2016). In addition, differences were observed in the white matter structure associated with dyslexia in babies, children and adults at risk of dyslexia (Olulade et al., 2013). Many individuals with dyslexia have less white matter in the left parietal-temporal area than average readers. This data is important in terms of showing that there is a correlation between white matter content and reading ability. In addition, the presence of below-normal white matter may reduce the ability or efficiency of different regions in the brain to communicate with each other (Aparicio et al., 2007). Booth and Burman (2001) also state that dyslexic individuals have less gray matter in the left parietal-temporal region than individuals without dyslexia. Low gray matter in this part of the brain may cause problems in processing the phonological structure of language (phonological awareness) (Aparicio et al., 2007). These structural differences in babies at risk of dyslexia may be the root cause of educational problems related to both existing and future neurobiological and behavioural differences (Goswami, 2004). With the tests performed on neural responses to speech and non-speech sounds in babies 36 hours after birth, the diagnosis of dyslexia in these individuals in the following years and the evaluations of white matter density in the curved fasciculus in babies between 5 and 18 months, and predictions of these children's expressive language skills related to reading skills in the following years, neuroscience is possible with the developments in the field (Bowers, 2016).

Most children initially acquire receptive and expressive language skills through hearing and then learn to associate spoken or heard words with meaning (Olulade et al., 2013). Reading is a cognitive process that involves decoding the symbols in order to grasp and understand the text being read, that is, associating the written text with verbal words and therefore with meaning (Booth & Burman, 2001). The situation in question depends on phonological awareness or the ability to recognize and use word-forming sounds. These phonemes are processed into written words (spelling) during reading (Richlan et al., 2009). In this context, reading is a complex of actions that occur with the successful development of cognitive functions related to language, vision, attention, and thinking as well as multiple brain structures (Zamarian et al., 2009). By theorizing the anatomical structure of the neural networks in the brain, scientists have been able to establish a basis for how children recognize phonemes and words (Bowers, 2016). In other words, with the analysis of brain architecture, evaluations and interventions on brain areas that may cause problems in the development of reading skills in individuals, including dyslexic children, can be made with the help of neuroscientific studies (Cantlon et al., 2011).

The neural network in the left hemisphere is thought to support reading with certain neural activation patterns (Richlan, 2012). With the active participation of the left temporo-parietal region, it contributes to the acquisition of reading action by contributing to the development of phonological skills (Heim et al., 2003). Accordingly, activation increases in the left temporo-parietal cortex were observed in children with dyslexia due to improvements in advanced language and reading skills (Kirby et al., 2008). Good reading skill occurs with the cooperation of the left hemisphere language regions of the brain, including the frontal, temporo-parietal, and occipito-temporal regions (Cantlon et al., 2011). While all regions must work together as a network to support reading, each region is individually responsible for functions required for the reading process, such as phonological processing, visual word recognition, or semantic determination. Localized damage in any of these regions has been associated with acquired dyslexia (Gabrieli, 2016).

Neuroscientific studies on dyslexic individuals generally measure the individual's cognitive responses to a single word read using fMRI or ERP. Occipital-temporal areas often become more active in visual stimuli, that is, when perceiving letter shapes or during the writing process. The lower occipital-temporal area makes electrophysiological distinctions between meaningful (such as "**b**lock") and meaningless (such as "**d**lock, **q**lock or **p**lock") words in about 180 ms that not only helps to perceive the word seen or read visually but also decides that it is semantically incorrect (Noble & McCandliss, 2005). While brain activation in the temporo-occipital region increases during reading in individuals with “normal” reading skills, this activation generally does not increase in dyslexic individuals. In children with hyperlexia (the combination of advanced reading skills and poor understanding), this region may be activated at an advanced level during reading (Goswami, 2004). In addition to the insufficient activation recorded in the left occipito-temporal and temporo-parietal regions during reading in dyslexic individuals, studies show that it may be more closely related to dyslexia in the left lower frontal region (Stoodley, 2016). Increased activation in this area is through occult or subvocal reading (subvocal reading or silent speech is typically internal speech during reading and helps to perceive the sound of the word as it is being read. This is a natural process that should happen in the act of reading and potentially cognitive as it helps the mind access meanings. While typical readers have age-related decreases in activation in this region, readers with dyslexia may show hyperactivation for many years (Olulade et al., 2015). However, activation in this region may be related to the ability to read rather than dyslexia. Studies show that a large part of the left lower frontal cortex is more actively involved in phonological processing in individuals with phonological difficulties than those without (Downie et al., 2005).

Reading acquisition studies often emphasize the importance of phonological awareness (the ability to recognize and use the sounds that make up words). Brain imaging studies show that the phonological process focuses on the temporo-parietal junction (Olulade et al., 2013). This area appears to be the main center supporting the letter-sound recording process and plays a role in spelling disorders (Sireteanu et al., 2005). Typically, in dyslexic children with phonological processing difficulties, insufficient activation at the temporo-parietal junction was found in the task of deciding whether or not words rhyme. In targeted and purposeful readings, it was observed that activation in this area increased (Heim et al., 2003). In addition, inadequate activation in the left occipito-temporal cortex in response to words or word-like materials was observed in dyslexic individuals (Nicolson et al., 2001). Decreased activation in this area differs in children and adults with dyslexia. This difference may be related to early failure in the developmental process in children. In other words, activation differences in the region may be related to rapid naming and word definition deficits in children with dyslexia. This shows us that special education programs should be brain-based (Richlan et al., 2011).

Consequently, in behavioural terms, dyslexia are more often characterized by incomplete phonological awareness, inadequate identification and ability to manipulate spoken language units (Kızılaslan & Tunagür, 2021). Students can develop reading skills in dyslexic individuals with phonologically based education. Because this education improves children's ability to play with words by directly manipulating (changing) sounds (Torgesen, 2000). Beyond phonological shortcomings, dyslexic individuals often have difficulties in reading fluently. While phonological awareness is developed through explicit instruction, reading fluency difficulties can sometimes be permanent (Hughes et al., 2017). Explicit instruction is systematic, direct, engaging, and success-emphasizing, and it is stated that it increases success in most students (Archer & Hughes, 2011). Reading acquisition may become more difficult for children with dyslexia as the reading skill becomes more complex as the grade level increases (Gabrieli, 2016). In this case, explicit instruction can be a useful method (Moats & Dakin, 2008). Because many practical and accessible resources developed for direct teaching allow teachers to apply explicit teaching strategies for any classroom level or content area (Archer & Hughes, 2011). In explicit instruction, clear guidelines are prepared to define the basic concepts, strategies, skills and routines to be taught, effective teaching environment is designed and students are offered opportunities to apply new material. In addition, sample lesson plans and repeatable checklists and teacher worksheets increase the efficiency of teaching (Hughes et al., 2017).

RESULTS

Dyslexia is defined as the disability faced by individuals in learning to read and achieving normal reading proficiency, despite teaching at a sufficient level to read. Neuroscience studies show that dyslexia are associated with structural differences seen in the areas responsible for normal reading development in the brain. Nervous symptoms that can be defined as symptoms of dyslexia can now be detected in infancy. This may make it possible to identify reading problems before a child begins teaching reading and leaves behind his peers. In addition, neuroscientific studies investigating the effects of different teaching programs on brain functions in literacy education can offer educational methods for the special educational needs of dyslexic students.

However, neuroscientific studies on the learning processes of dyslexic students generally focus on 'neuromites' (common misconceptions about the brain and learning) in these individuals. From the MRI measurements of the neural connections of the brain or "white matter" (the part consisting of large bundles or parts of myelinated axons connecting regions of the brain), the researchers found that the neural cycle of dyslexic individuals strengthened after an individualized curriculum for eight weeks and the individual's reading performance improved. The study in question is the first to correlate children's learning with the flexibility of the brain by measuring the amount of white matter during an intensive educational intervention (Huber et al., 2018).

Although neuroimaging studies show that dyslexia are due to functional and structural differences in the brain that begins in infancy and typical brain development, currently developed reading teaching methods do not fully target specific nervous systems (Richlan, Kronbichler & Wimmer, 2009). On the contrary, teaching methods for teaching reading target behavioural skills such as phonological awareness as a component of one-word decoding. The one-word decoding referred to here is the ability to correctly pronounce letter-sound relationships over words, including knowledge of letter patterns. For example, when a child reads the word 'Daisy', he or she must be able to understand what the letters are, the sound that makes up each letter, and how these sounds come together to form words. In this context, grammar should be carried out with an approach that teaches students the principles of letter-sound relationships and how to vocalize words (Bowers, 2016).

Another contribution that neuroimaging studies can provide to the field of special education is to predict the individual's response to individualized education. In a study of children aged 10-14 years with dyslexia, none of the 17 traditional reading and reading-related skills tests could provide useful predictions of which individuals would or would not respond to educational interventions over a 30-month period (Gabrieli et al., 2015). In addition, neuroimaging methods have been able to predict whether each child will acquire reading at the same time. Similarly, a large parallelism was found between the brain measurements of the kindergarten children and the reading levels and the brain imaging results when compared to the behavioural measurements of the same children in the fifth grade (Gabrieli, 2016). These types of neuroimaging findings may contribute to pre-planning to make the neurocognitive differences between children benefit more or less from certain types of education (Jordan et al., 2014). In addition, these findings can provide us with useful ideas about determining the appropriate education for the needs of dyslexic children and making instructional changes when necessary. Otherwise, it can only be determined if the teaching given to dyslexic children is effective or not after long-term failure of the children (Torgesen, 2000).

Examining the underlying processes and causes of dyslexia, it would be helpful for teachers to consider the suggestions below (Mortimore & Crozier, 2006);

- Dyslexia is a difficulty arising from the language processing area in the brain. Therefore, it is important to know what weaknesses each student has in determining the appropriate teaching to meet their educational needs.
- In order to measure children's perception of speech sounds and their level of letter sounds in words and their fluent word recognition, screening, progress and monitoring procedures should be applied in the early period. Performing these procedures regularly throughout a child's school career can help us understand what skills will be taught to the child and whether relevant skills are developing in the child.
- Imaging studies found marked differences in the brain activation of dyslexic students. Therefore, interventions should often focus on open, intense, long-term, and particularly on phonological processing, phonics, and fluency.
- Students are generally prone to giving up early in the struggle. Dyslexic individuals require more intensive instruction than their peers due to the difference in their brain structure. Students may show low motivation when trying to avoid a difficult and painful process. At this point, the child should be motivated by showing a high level of awareness.

Consequently, neuroplasticity can be associated with dyslexia in two ways. First, neuroplasticity explains how past experiences can affect learning disabilities. Research shows that learning difficulties have a genetic component, but can be intensified by false stimuli. For example, punishing the child for not meeting the standards or because of a history of failure may worsen the inadequacy and increase the fear stimulus in the child. However, educational environments rich with positive stimuli can help children function better. Second, neuroplasticity can be an effective response to learning difficulties. Children and adults with these difficulties lack academic success and a sense of doing the best. These individuals only have cognitive structures that have gone through different developmental stages. The effects of dyslexia can be reduced by a combination of neuroplasticity-based counselling services and cognitive exercises. Dyslexic individuals can cognitively construct a new path that leads them to the desired goal by actively using and developing areas of the brain associated with reading (Kizilaslan & Avşar Tuncay, 2022). Neuroplasticity provides us with a promising literature to meet the educational needs of individuals with brain damage or learning difficulties by defining the brain as dynamic, not static (Stoodley, 2016).

Neuroscience studies are also decisive in understanding the neural mechanisms of dyslexia, early diagnosis and development of targeted therapies. In addition, these studies can show whether the education in question can be successful at the beginning of appropriate education for children with dyslexia. In this case, children may be directed to alternative forms of healing that are more likely to be beneficial early in the process. Although current traditional educational interventions are not effective in determining variation related to teaching, it is possible to define the individual and determine the appropriate educational intervention with the help of neuroimaging studies.

REFERENCES

Ansari, D., & Coch, D. (2006). Bridges over troubled waters: Education and cognitive neuroscience. *Trends in Cognitive Sciences, 10,* 146-151. https://doi.org/10.1016/j.tics.2006.02.007

Ansari, D., De Smedt, B., & Grabner, R. (2011). Neuro education: A critical overview of an emerging field. *Neuroethics, 5,* 1-13. https://doi.org/10.1007/s12152-011-9119-3

Aparicio, M., Gounot, D., Demont, E., & Metz-Lutz, M.-N. (2007). Phonological processing in relation to reading: An fMRI study in deaf readers. *NeuroImage, 35*(3), 1303–1316. https://doi.org.10.1016/j.neuroimage.2006.12.046

Archer, A.L., & Hughes, C.A. (2011). *Explicit instruction: Effective and efficient teaching*. New York, NY: Guilford Press.

Austin, M.W., Ploughmana, M., Glynnb, L., & Corbett, D. (2014). Aerobic exercise effects on neuroprotection and brain repair following stroke: A systematic review and perspective. *Neuroscience Research, 87*, 8-15. https://doi.org.10.1016/j.neures.2014.06.007

Bartha, L., & Benke, T. (2003). Acute conduction aphasia: An analysis of 20 cases. *Brain and Language, 85*, 93-108. https://doi.org.10.1016/s0093-934x(02)00502-3

Booth, J. R., & Burman, D. D. (2001). Development and Disorders of Neurocognitive Systems for Oral Language and Reading. *Learning Disability Quarterly, 24*(3), 205-215. https://doi.org/10.2307/1511244

Bowers, J. S. (2016). The practical and principled problems with educational neuroscience. *Psychological Review, 123*(5), 600–612. https://doi.org/10.1037/rev0000025

Bredt, D. S. & Nicoll, R. A. (2003). AMPA receptor trafficking at excitatory synapses. *Neuron 40*, 361–379. https://doi.org.10.1016/s0896-6273(03)00640-8

Caine, G., Nummela-Caine, R., & Crowell, S. (1999). *Mindshifts: A Brain-Based Process for Restructuring Schools and Renewing Education* (2. Baskı). Tucson, AZ: Zephyr Press.

Cantlon, J. F., Pinel, P., Dehaene, S., & Pelphrey, K. A. (2011). Cortical representations of symbols, objects, and faces are pruned back during early childhood. *Cerebral Cortex, 21*(1), 191–199. https://doi.org/10.1093/cercor/bhq078

Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*(5582), 851–854. https://doi.org/10.1126/science.1072290

Clement, N. D., & Lovat, T. (2012). Neuroscience and education: issues and challenges for curriculum. *Curriculum Inquiry, 42*(4), 534–557. https://doi.org.10.2307/23324016

Cooper, E. A., & Mackey, A. P. (2016). Sensory and cognitive plasticity: implications for academic interventions. *Current Opinion in Behavioural Science, 10*, 21-27. https://doi.org/10.1016/j.cobeha.2016.04.008

Downie, A. L., Frisk, V., & Jakobson, L. S. (2005). The impact of periventricular brain injury on reading and spelling abilities in the late elementary and adolescent years. *Child Neuropsychol, 11*(6), 479-495. https://doi.org.10.1080/09297040591001085

Fischer-Baum, S., & Campana, G. (2017). Neuroplasticity and the logic of cognitive neuropsychology. Cognitive *Neuropsychology, 34*(7-8), 1-9. https://doi.org.10.1080/02643294.2017.1389707

Fisher, S. E., & DeFries, J. C. (2002). Developmental dyslexia: genetic dissection of a complex cognitive trait. *Nat Rev Neurosci, 3*(10), 767-780. https://doi.org.10.1038/nrn936

Gabrieli, J. D. E. (2016). The promise of educational neuroscience: Comment on Bowers (2016). *Psychological Review, 123*(5), 613–619. https://doi.org/10.1037/rev0000034

Gabrieli, J. D. E., Ghosh, S. S., & Whitfield-Gabrieli, S. (2015). Prediction as a humanitarian and pragmatic contribution from human cognitive neuroscience. *Neuron, 85*(1), 11–26. https://doi.org.10.1016/j.neuron.2014.10.047

Geake, J. G. (2004). How children's brains think: not left or right but both together. *Education, 32*, 65–72. https://doi.org.10.1080/03004270485200351

Goswami, U. (2004). Neuroscience, education and special education. *British Journal of Special Education, 31*, 175–183. https://doi.org/10.1111/j.0952-3383.2004.00352.x

Heim, S., Eulitz, C. & Elbert, T. (2003). Altered hemispheric asymmetry of auditory P100m in dyslexia, *European Journal of Neuroscience, 17*, 1715–1722. https://doi.org.10.1046/j.1460-9568.2003.02596.x

Hensch, T. K. (2005). Critical period plasticity in local cortical circuits. *Nature Reviews Neuroscience, 6*(11), 877–888. https://doi.org/10.1038/nrn1787

Hoffman, K. L., & Mcnaughton, B. L. (2002). Coordinated reactivation of distributed memory traces in primate neocortex. *Science, 297,* 2070–2073. https://doi.org.10.1126/science.1073538

Hogarth, L., Chase, H. W, & Baess, K. (2010). Impaired goal-directed behavioural control in human impulsivity. *Q J Exp Psychol 10*,1–12. https://doi.org.10.1080/17470218.2010.518242

Hotting, K., & Roder, B. (2013). Beneficial effects of physical exercise on neuroplasticity and cognition. *Neuroscience and Biobehavioral Reviews, 37*(9), 2243-2257. https://doi.org.10.1016/j.neubiorev.2013.04.005

Howard-Jones, P. (2014). *Neuroscience and education: A review of educational interventions an approaches informed by neuroscience*. Bristol, UK: Education Endowment Foundation.

Huber, E., Donnelly, P. M., Rokem, A., & Yeatman, J. D. (2018). Rapid and widespread white matter plasticity during an intensive reading intervention. *Nature Communications, 9*(1) https://doi.org.10.1038/s41467-018-04627-5

Hudson, R. F., High, L. Al-Otaiba, S. (2007). Dyslexia and the brain: What does current research tell us? *The Reading Teacher, 60*(6), 506-515. https://doi.org.10.1598/RT.60.6.1

Hughes, C. A., Morris, J. R., Therrien, W. J., & Benson, S. K. (2017). Explicit instruction: Historical and contemporary contexts. *Learning Disabilities Research and Practice, 32*(3), 143. https://doi.org.10.1111/ldrp.12142

Immardino-Yang, M. H., & Damasio, A. (2007). We feel, therefore we learn: The relevance of affective and social neuroscience to education. *Mind Brain and Education, 1*, 3-10. https://doi.org.10.1111/j.1751-228X.2007.00004.x

Jordan, J. A., McGladdery, G., & Dyer, K. (2014). Dyslexia in higher education: Implications for Maths anxiety, statistics anxiety and psychological well-being. *Dyslexia, 20*(3), 225–240. https://doi.org.10.1002/dys.1478

Judge, S., & Watson, S. M. R. (2011). Longitudinal outcomes for mathematics achievement for students with learning disabilities. *The Journal of Educational Research, 104*(3), 147–157. https://doi.org.10.1080/00220671003636729

Kirby, J. R., Roth, L., Desrochers, A., & Lai, S. S. V. (2008). Longitudinal predictors of word reading development. *Canadian Psychology, 49*(2), 103–110. https://doi.org/10.1037/0708-5591. 49.2.103

Kızılaslan, A., & Tunagür, T. (2021). Dyslexia and working memory: understanding reading comprehension and high level language skills in students with dyslexia, *Kastamonu Education Journal, 29*(5), 941-952. https://doi.org.10.24106/kefdergi.741028

Kizilaslan, A., & Avşar Tuncay, A. (2022). A study of perceptions as expressed through metaphors what do preschool teachers think about their inclusive students. *European Journal of Special Needs Education*, *38*(1), 32-47. https://doi.org/10.1080/08856257.2022.2037824

Knowland, V. C. P., & Thomas, M. S. C. (2014). Educating the adult brain: How the neuroscience of learning can inform educational policy. *International Review of Education, 60*, 99-122. https://doi.org.10.1007/sl 1159-014-9412-6

Koizumi, H. (2004). The concept of 'developing the brain': A new natural science for learning and education. *Brain and Development 26*, 434–441. https://doi.org.10.1016/j.braindev.2003.09.011.

Kolb, B., Gibb, R., & Gorny, G. (2003). Experience-dependent changes in dendritic arbor and spine density in neocortex vary with age and sex. *Neurobiology of Learning and Memory, 79*, 1–10.

Kotaleski, J. H., & Blackwell, K. T. (2010). Modelling the molecular mechanisms of synaptic plasticity using systems biology approaches. *Nature Reviews Neuroscience 11*(4), 239-251. https://doi.org/10.1038/nrn2807

Krafnick, A. J., Flowers, D. L., Napoliello, E. M., & Eden, G. F. (2011). Gray matter volume changes following reading intervention in dyslexic children. *NeuroImage, 57*(3), 733–741. https://doi.org/10.1016/j.neuroimage.2010.10.062

Lee, S. S., & Mudaliar, A. (2009). Medicine. Racing forward: The Genomics and Personalized Medicine Act. *Science, 323*(5912), 342. https://doi.org.10.1126/science.1165768

Lenroot, R. K., & Giedd, J. N. (2006). Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioral Reviews 30*(6), 718–729. https://doi.org.10.1016/j.neubiorev.2006.06.001

Liu, S. Q., & Cull-Candy, S. G. (2000). Synaptic activity at calcium-permeable AMPA receptors induces a switch in receptor subtype. *Nature 405*, 454–458. https://doi.org.10.1038/35013064

Malinow, R. & Malenka, R. C. (2002). AMPA receptor trafficking and synaptic plasticity. *Annu. Rev. Neurosci. 25*, 103–126. https://doi.org.10.1146/annurev.neuro.25.112701.142758

McDonald, R. J., Craig, L. A., & Hong, N. S. (2008). Enhanced cell death in hippocampus and emergence of cognitive impairments following a localized mini-stroke in hippocampus if preceded by a previous episode of acute stress. *Eur J Neurosci, 27*, 2197-2209. https://doi.org.10.1111/j.1460-9568.2008.06151.x

Meschyan, G., & Hernandez, A. E. (2006). Impact of language proficiency and orthographic transparency on bilingual word reading: An fMRI investigation. *NeuroImage, 29*(4), 1135–1140. https://doi.org/10.1016/j.neuroimage.2005.08.055

Meyer, M. S., & Felton, R. H. (1999). Repeated reading to enhance fluency: Old approaches and new directions. *Annals of Dyslexia, 1*, 283–306. https://doi.org/10.1007/s11881-999-0027-8

Moats, L., & Dakin, K. (2008). *Basic facts about dyslexia and other reading problems*. Baltimore, MD: International Dyslexia Association.

Mortimore, T., & Crozier, R. (2006). Dyslexia and difficulties with study skills in higher education. Studies in Higher Education, 31(2), 235–251. https://doi.org/10.1080/03075070600572173

Nicolson, R. I., Fawcett, A. J., & Dean, P. (2001). Developmental dyslexia: The cerebellar deficit hypothesis. *Trends in Neurosciences, 24*(9), 508–511. https://doi.org/10.1016/S0166-2236(00)01896-8

Noble, K. G., & McCandliss, B. D. (2005). Reading development and impairment: Behavioral, social, and neurobiological factors. *Journal of Developmental and Behavioral Pediatrics, 26*(5), 370–378. https://doi.org.10.1097/00004703-200510000-00006

Olulade, O. A., Flowers, D. L., Napoliello, E. M., & Eden, G. F. (2015). Dyslexic children lack word selectivity gradients in occipito-temporal and inferior frontal cortex. *NeuroImage: Clinical, 7,* 742-754. https://doi.org.10.1016/j.nicl.2015.02.013

Olulade, O. A., Napoliello, E. M., & Eden, G. F. (2013). Abnormal visual motion processing is not a cause of dyslexia. *Neuron, 79*(1), 180–190. https://doi.org/10.1016/j.neuron.2013.05.002

Palmer, M. J., Isaac, J. T. & Collingridge, G. L. (2004). Multiple, developmentally regulated expression mechanisms of long-term potentiation at CA1 synapses. *J. Neurosci. 24*, 4903–4911. https://doi.org.10.1523/JNEUROSCI.0170-04.2004

Paulesu, E., Danelli, L., & Berlingeri, M. (2014). Reading the dyslexic brain: Multiple dysfunctional routes revealed by a new meta-analysis of PET and fMRI activation studies. *Frontiers in Human Neuroscience, 8*, 830. https://doi.org/10.3389/fnhum.2014.00830

Powell, D., Stainthorp, R., Stuart, M., Garwood, H., & Quinlan, P. (2007). An experimental comparison between rivaltheories of rapid automatized naming performance and its relationship to reading. *Journal of Experimental Child Psychology, 98*(1), 46–68. https://doi.org/10.1016/j.jecp.2007.04.003

Prickaerts, J., Koopmans, G., Blokland, A., & Scheepens, A. (2004). Learning and adult neurogenesis: Survival with or without proliferation? *Neurobiology of Learning and Memory, 81*(1), 1-11. https://doi.org/10.1016/j.nlm.2003.09.001

Raschle, N. M., Chang, M., & Gaab, N. (2011). Structural brain alterations associated with dyslexia predate reading onset. *NeuroImage, 57*(3), 742–749. https://doi.org/10.1016/j.neuroimage. 2010.09.055

Richlan, F. (2012). Developmental dyslexia: dysfunction of a left hemisphere reading network. *Front Hum Neurosci, 6*. https://doi.org.10.3389/fnhum.2012.00120

Richlan, F., Kronbichler, M., & Wimmer, H. (2009). Functional abnormalities in the dyslexic brain: A quantitative meta-analysis of neuroimaging studies. *Human Brain Mapping, 30*(10), 3299–3308. https://doi.org/10.1002/hbm.20752

Sireteanu, R., Goertz, R., Bachert, I., & Wandert, T. (2005). Children with developmental dyslexia show a left visual "minineglect". *Vision Research, 45*, 3075-3082. https://doi.org.10.1016/j.visres.2005.07.030

Stoodley, C. J. (2016). The cerebellum and neurodevelopmental disorders. *Cerebellum, 15*(1), 34–37. https://doi.org.10.1007/s12311-015-0715-3

Stromberg J. (2013). *How Growing up in Poverty May Affect a Child's Developing Brain*. Washington, DC: Smithsonian.

Tang, Y. Y. (2017). *Brain-based learning and education: Principles and practice*. London: Elsevier Academic Press.

Thomas, M., & Knowland, V. (2009). Sensitive Periods in Brain Development – Implications for Education Policy. *European Psychiatric Review, 2*(1), 17–20.

Tommerdahl, J. (2010). A model for bridging the gap between neuroscience and education, *Oxford Review of Education, 36*(1), 97-109, https://doi.org.10.1080/03054980903518936

Torgesen, J. K. (2000). Individual differences in response to early interventions in reading: The lingering problem of treatment resisters. *Learning Disabilities Research and Practice, 15*(1), 55-64. https://doi.org/10.1207/SLDRP1501_6

Van der Lely, H. K. J., & Marshall, C. R. (2010). Assessing component language deficits in the early detection of reading difficulty risk. *Journal of Learning Disabilities, 43*(4), 357–368. https://doi.org.10.1177/0022219410369078

Zadina, J. N. (2015). The emerging role of educational neuroscience in education reform. *Psicologia Educativa, 21*, 71-77. https://doi.org/10.1016/j.pse.2015.08.005

Zamarian, L., Ischebeck, A., & Delazer, M. (2009). Neuroscience of learning arithmetic evidence from brain imaging studies. *Neuroscience and Biobehavioral Reviews, 33*(6), 909-925. https://doi.org.10.1016/j.neubiorev.2009.03.005