

Defining Developmental Dyslexia: An Overview of Etiological Theories and the
Neurobiological Basis of the Disorder

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Abstract

Defining Developmental Dyslexia: An Overview of Etiological Theories and the Neurobiological Basis of the Disorder

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Researchers have long been debating the most accurate definition for developmental dyslexia, likely due to its complicated and unknown etiology, and its variability in expression. The phonological deficit theory, the cerebellar theory, and the magnocellular theory each argue the primary characteristics of dyslexia that inform their varying definitions. In this review, I explore what is known about developmental dyslexia, the research that supports its etiological theories as well as the interventions that address them. Though the evidence is correlational, new research indicates potential answers in the genetic association of dyslexia. Perhaps the causality of dyslexia is not as imperative as the proper intervention of its consequential reading difficulties.

Introduction

Historically, developmental dyslexia was perceived as a hereditary defect, one that affects some aspects of the visual processing of words without influencing intellect, or verbal skills (Stein, 2018). In 1878, renowned German physician Adolf Kussmaul, observing his stroke patients, noted selective deficiencies in reading but otherwise unaffected non-verbal reasoning and oral skills, thus introducing the term “word blindness” (Stein, 2008). Rudolph Berlin later renamed this condition to dyslexia in 1884, though the term only gained popularity almost 50 years later. Physician Pringle Morgan published the first academic paper on dyslexia, noting the earliest case of what he called “congenital word blindness,” a visual processing issue with written words that had no bearing on intelligence (Snowling, 1995). By the mid to late 20th century, dyslexia was established as a hereditary defect that maintained oral and verbal intelligence but affected visual processing of reading. However, this was later rejected, and dyslexia was redefined as a language disorder—an inability to develop the necessary phonological skills involved in reading (Stein, 2008). The general public has maintained this view of dyslexia and most existing research supports this interpretation, though it is certainly not the only viable explanation. Recent developments in technology have revealed the neurological component of the disorder and corresponding theories, like the visual processing deficit, have been reconsidered, piquing the curiosities of many researchers in the field. Experts continue to deliberate as to what characteristics are universal to dyslexics and thus essential to its definition. Despite the significant progress in this endeavor, the understanding of dyslexia remains in constant flux, and causality remains a mystery.

Methods

The following literature review aims to present a summary of what is known and agreed upon by researchers regarding the nature of dyslexia. The goal is to highlight the major extant theories explaining the etiology of dyslexia as revealed by recent neurological research. Several procedures were taken to ensure that a high quality of literature was compiled and reviewed. Databases such as EBSCOHOST, ERIC, Google Scholar, Jstor, ScienceDirect and PubMed were utilized during a widespread search for peer reviewed journals and medical publications. The reference section of each article was thoroughly explored and served as another valuable source of literature. During the collection process, a general search of studies on dyslexia and other literature reviews that comprehensively describe the diagnosis were accumulated for research. After realizing that the controversy was rooted in the definition, search criteria was refined to scholarly educational and medical journals, specifically on the theories that frame dyslexia and the numerous studies that have attempted to prove them. For the sake of focusing recent developments in the field, any source not published in well-known, peer-reviewed journals after 1990 were excluded from the search, though a few exceptions have been made in an effort to holistically approach particular aspects of the theories and definitions. The studies chosen focused predominantly on children with dyslexia, though this scope severely limited the research available, and thus adults with dyslexia were also taken into account. Keywords for the search included dyslexia, reading disabilities, neurological, phonological deficits, the brain, vision pathways, magnocellular, brain abnormalities, genetics and interventions. Of the aggregated search results, I found 70 references to be most relevant.

Overview: What do we know?

Under current definitions, individuals with dyslexia account for 7% of the population, (Peterson and Pennington, 2012), roughly 3% -17.5% of all school-age children (dependent on definition and diagnostic criteria) and 80% of all reading disabilities (Saviour and Ramachandra, 2006). Children with dyslexia are often diagnosed early in their elementary school experience, as difficulties in reading begin to surface and hinder their academic performance. Boys are often affected more drastically than girls, possibly due to fetal testosterone levels in late pregnancies (Saviour and Ramachandra, 2006) or referral bias in the classroom (Shaywitz, 1990). This can also be explained by higher prevalence of comorbid externalizing disorders such as attention deficit hyperactivity disorder (ADHD) in boys (Peterson and Pennington, 2012). Other comorbidities of dyslexia include language impairment (difficulties in syntax and vocabulary development), motor skills, executive functioning and speech sound disorder (difficulties in intelligible and accurate reproduction of sounds in speech) (Gooch, 2014; Peterson and Pennington, 2012). These conditions are typically detected far earlier than dyslexia and could therefore be important indicators for later reading complications (Peterson and Pennington, 2012).

In the past, only a handful of research has focused on cross-cultural manifestations of dyslexia. Cross-cultural research hoped to discover whether dyslexia existed only in languages with complex orthographies like English. Ziegler's 2003 study determined that children from both Australia and Germany exhibited non-word reading deficits, indicating the universality of non-word decoding speed deficiency in dyslexics, regardless of the language's orthography. Studies like that of Lindgren (1985) found the frequency of dyslexia to be higher in the United States than in Italy; while both were similar in their association with verbal processing issues,

English dyslexics were unique in their visual-motor deficits. Overall, the research tends to agree on a common neurocognitive basis for dyslexia, though the severity and variation of reading behaviors seen among different countries is dependent on the complexity of different orthographies (Paulesu et al., 2001).

The Defining Controversy

The definition of dyslexia has proven to be the most controversial aspect of the disorder. For decades, researchers in every related field have attempted to set definitional parameters, yet there is still no universal agreement for its essential characteristics. The DSM V no longer provides a definition for dyslexia, instead grouping it with other disorders placed under the category of Specific Learning Disabilities (American Psychiatric Association, 2013). Some argue that dyslexia is neurodevelopmental in nature, characterized by slow and inaccurate word recognition (Peterson and Pennington, 2012). Others argue that it is “unexpected, specific and persistent” in its inability to facilitate proficient reading skills (Démonet, 2004), independent of factors such as learning opportunity, instruction, and motivation (Critchley, 1970; World Health Organization, 1993). Yet another definition with more historical acceptance emphasizes the impairment in vision processing that produces the visual confusion of written words (Stein, 2000). To many, dyslexia is more complicated than a specific reading disability (Catts, 1989), though the general public tends to use dyslexia and reading disability interchangeably, partly due to the vague and conflicting definitions used to describe both.

The more common classification of dyslexia is that it is a language disorder involving deficits in phonological processing (Castles and Coltheart, 2004). Still, the vision pathway deficit theory has made a recent comeback, attempting to explain the apparent movement of words and letters that some individuals experience. The trouble with having a conglomerate of competing

definitions is that they are often exclusionary, addressing only some of the experienced symptoms. These parameters are therefore discernibly limited, as many people who have the diagnosis report deficiencies that are not always highlighted. Arguably, the biggest downfall of the current diagnosis process is a heavy reliance on the presence of reading difficulties, meaning that affected children must fail before being identified (Catts, 1989). The complicated and varied nature of the disorder and the way it affects each individual, makes it difficult to characterize a universal definition, and research that attempts to do so will prove futile (Stein 2018).

Despite the lack of consensus, there are a few fundamentals of dyslexia that researchers tend to agree on. For example, dyslexia is widely assumed to be genetic in origin, with some elements of neurological abnormalities at play (Smith et al., 1998), though the extent of those abnormalities is debated. A large majority of people with dyslexia demonstrate cognitive issues in phonological processing such as encoding speech sound information into long term memory (Kamhi, 1988). In other words, people with dyslexia need multiple encounters and depictions of words before they can reliably reproduce them (Catts, 1989). Individuals with dyslexia also have difficulty retaining verbal information in short term memory, causing poor performance on activities depending on the short term recall of lists, whether they be letters, words, numbers or phrases (Cohen and Netley, 1981). Similarly, there is a general complication with retrieving any phonological information from long term memory (Wagner and Torgesen, 1987). Lack of sensitivity to the speech sound structure of language also characterizes one of the neurological abnormalities, though this may stem from issues with limited exposure to reading, as reading promotes improved experience with speech sound and phoneme awareness (Catts, 1989).

One of the most widely accepted and relatively inclusive definitions is that of Catts (1989), who outlines dyslexia as a developmental language disorder present at birth and

persisting into adulthood; the specific reading disability is characterized by difficulties in phonological processing such as coding, retrieving and code use of phonological information in memory, extending to lack of awareness of speech sound structures (Catts,1989). However, even this definition leaves many questions unanswered. A major controversy surrounding dyslexia lies in the perceived importance of limited phonological processing, namely that it may not be the only defining characteristic and many experts in the field argue that this should not be a primary component of the definition. In contrast to Catts, Saviour and Ramachandra (2006) base their definition of dyslexia more broadly in the difficulties in processing new words and single word decoding, while struggling with the reversal and rearranging of words and letters. They also note an association between dyslexia and delayed milestones in development and motor skills (Saviour and Ramachandra, 2006). The neurological basis of dyslexia is undeniably present in the diagnosed population and definitions modeled around Saviour and Ramachandra's (2006) framework maintain the reality that cognitive abnormalities should be leading contenders in the primary characterization of dyslexia. This leads us to the heart of the definition controversy: what is universally experienced by individuals with dyslexia? Researchers have generated several theories that seek to explain the indicators of dyslexia by turning to observable abnormalities in the structure and function of brain.

Structural and Functional Brain Abnormalities

Data gathered from MRI scans of individuals with dyslexia reveals the absence of typical asymmetry of the planum temporale, in the center of the Wernicke's area—an important area for language processing, analyzing sounds, naming objects and recalling words (Duara, 1991;

Galaburda, 1979). In the average brain, the larger side of the Wernicke's area, specifically the planum temporale on the left, is possibly related to the language dominance being localized in the left brain hemisphere (Duara, 1991). Due to enlargement of the right

hemisphere, this area appears to be symmetrical in dyslexic brains (Velluntino, 1996), yielding poor performance on non-word reading tasks (Saviour and Ramachandra, 2006). The parietal lobe area in dyslexic brains also shows more symmetry, correlating with poor phonological performance (Saviour and Ramachandra, 2006). Additionally, the posterior part of the corpus callosum, referred to as the splenium, is larger in dyslexic brains, but little is understood about what that may mean for cognitive and functional abilities (Duara, 1991). Also larger in dyslexic brains is the angular gyrus and posterior pole in the posterior region of the parietal lobe, which is involved in a multitude of processes related to language processing (Duara, 1991).

The connectivity between the middle and temporal gyrus, superior temporal sulcus and the supramarginal and angular gyri show reduced activity in dyslexics and is generally correlated with the underdevelopment of reading skills in children (Darki, 2012). The occipitotemporal cortex, which holds the area for visual word form, along with the temporoparietal and inferior frontal cortices, which play a role in phonological and semantic processing of words, and the inferior frontal cortex, involved in the formation of speech sounds, are all areas of the brain associated with reading that are altered in people with dyslexia (Richlan et al, 2011; IDA 2015).

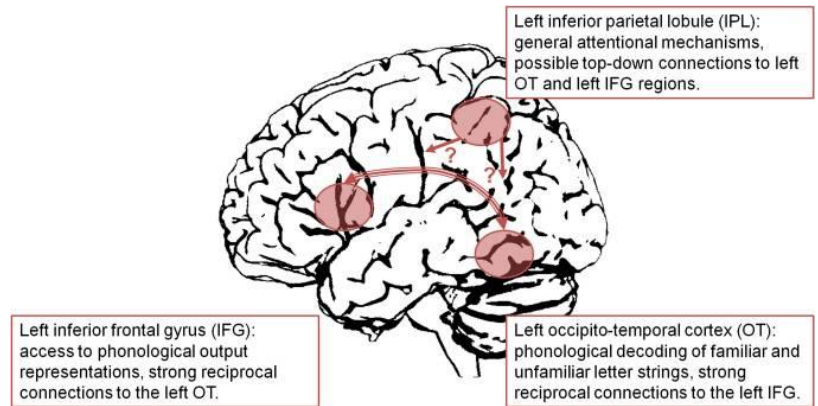


Figure 1. Areas of the left hemisphere involved in reading that are altered in dyslexic brains. Reprinted from "Developmental Dyslexia: dysfunction of a left hemisphere reading network" by F. Richlan, 2012, *Frontiers in human neuroscience*, 6, 120.

Microscopic cortical malformations have also been discovered in language areas of dyslexic brains, such as the interior frontal gyrus and the temporoparietal area (Galaburda, 1985). These malformations appear as ectopias, misplaced collections of neurons caused by disturbances in neural migration that results in changes in both the white and gray matter structure. They can also appear as microgyrias and dysplasia: lost structural organization among cortical neurons (Saviour and Ramachandra, 2006; Darki 2012). This could be an indication of abnormal cortical development in dyslexic brains (Saviour and Ramachandra, 2006), possibly dictated by an undercurrent of genetic risk factors.

Additionally, PET scans show white matter density and connectivity between posterior and frontal regions to be significantly altered in dyslexic brains (Saviour and Ramachandra, 2006). Variability in white matter structure is correlated with variability in reading ability since connectivity between language regions in the brain is critical for literacy (Darki, 2012). In children and adults with dyslexia, white matter structure is altered in the left temporoparietal area and in the left interior frontal gyrus of the Broca's area, which is responsible for language processing and speech production (Peterson and Pennington, 2012; Stein, 2018). According to Hoeft (2006), the fMRI data analysis revealed that children with dyslexia exhibited less activation in their left parietotemporal lobe than age-matched children in the control group with the same reading ability. Other regions that exhibit reduced activation include the right occipitotemporal cortex and the bilateral frontal cortices (Hoeft, 2006). The results of the study indicate that the reduced activation in these brain regions is characteristic of dyslexia, rather than a consequence of low reading proficiency (Hoeft, 2006).

Silani (2005) noted that decrease of gray matter density among dyslexics corresponds with under-activation of the left medial temporal gyrus, affecting the performance in tasks

requiring phonological retrieval such as reading. While reduced grey matter density is observed in the left middle temporal region, a higher density of grey matter surrounds the area down to the inferior temporal cortex; this phenomenon was associated with greater impairment in reading tasks performed by the subjects (Silani, 2005). Significant reduction of white matter density in dyslexic brains was seen within the frontal and parietal section of

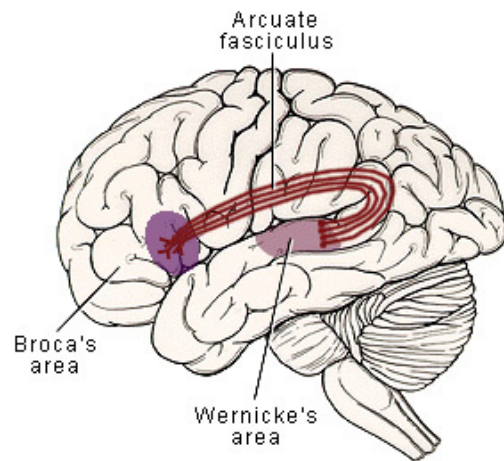


Figure 2. Connectivity between the Broca's Area and Wernicke's Area commonly affected in dyslexic brains.
Reprinted from "Treatment of Learning Disabilities: EEG Neurofeedback for Connectivity Problems" by M. Horst, 2003, *Edmonton Neurotherapy*.
http://www.edmontonneurotherapy.com/treatment_of_learning_disabilities.html.

the arcuate fasciculus, a bundle of axons that connect the Broca's area (speech production) to the Wernicke's area (comprehension of written and spoken language) (Silani 2005). Furthermore, Raschle (2011) reported that the decrease of grey matter in the occipitotemporal, temporoparietal and lingual gyrus is present in dyslexic brains before reading instruction, therefore indicating that the atrophy is not a result of reading failure. Naturally, the extent of these structural and functional abnormalities vary in each individual and thus cannot be universal markers of the diagnosis, though they do provide a deeper understanding of the major etiological theories rooted in it's neurological irregularities.

Etiological Theories

Of the current prevailing theories in the literature, the phonological deficit theory, which re-brands dyslexia as a language based disorder, is the most widely supported and researched. In addition to sub-par reading performance, individuals with dyslexia tend to perform poorly on

tasks that require phonological awareness such as manipulating speech sounds (Ramus, 2003). According to Démonet (2004), people with dyslexia have difficulties in the representation, retrieval and storage of phonological information from long term memory. In other words, there is an inability to separate sounds that match with the letter itself, a skill known as phonemic awareness (Stein, 2018). It exists as part of a larger process of learning known as phonological awareness, which is the ability to identify and manipulate units of sound in language such as words and syllables, vital to the foundation of reading and fluency. Without this skill, children are unable to efficiently read and understand text. The phonological processing deficit interferes with their phonemic awareness and subsequent phoneme-grapheme mapping, the process of matching sounds to the corresponding written letter (Peterson and Pennington, 2012).

Essentially, the lack of phonological awareness experienced by children with dyslexia explains the root of their reading incompetence. Any impairments in processing are associated with abnormalities in the neural connectivity and structure of the left hemisphere language network, including the temporal, occipital and parietal lobe (Stein, 2018).

This theory, though highly probable, remains undeniably incomplete. Critics note that it is impossible to differentiate dyslexia from other factors that yield reading failure, such as lack of resources, low general ability, poor teaching, stress, etc. (Stein, 2018). No singular deficit in phonological processing is enough to entirely explain the causation of dyslexia, and in chicken-or-egg fashion, it remains unclear whether phonological awareness problems are a cause or effect of dyslexia. This is especially muddled because the relationship between phonological skills and reading is bidirectional; over time, poor reading can cause poor phonological awareness (Peterson and Pennington, 2012). There are also studies of individuals who have difficulties in non-word reading yet demonstrate adequate phonological awareness (Castles and Coltheart,

2004). Interventions that aim to remediate the deficits of dyslexia via phonological training often neglect the possible visual impairments experienced by many dyslexics. Though this theory dominates current understanding of the diagnosis, the recent renewal in interest regarding the visual explanation for dyslexia seems to fill in ignored gaps, but produces many more questions of its own.

On the other hand, the magnocellular theory takes into account the notable visual impairments associated with dyslexia. According to this theory, sensory stimuli of both visual and auditory varieties cannot be processed efficiently and are therefore distorted in time due to abnormalities in the neurons of the magnocellular pathway (Petkov, 2005). Anatomically, the magnocellular pathway consists of neurons that connect the lateral geniculate nucleus of the thalamus to the primary visual cortex where information received from the eye is processed (Demb, 1998). These large neurons are sensitive to motion perception and are important for the control of eye movements and rapid transmission of signals throughout the magnocellular layer. Impairments in this pathway can lead to visual confusion and letters may “appear to move around,” a phenomenon experienced by some individuals with dyslexia. As a result, the consequential unsteadiness of the eyes can explain visual reading errors, qualifying it as another marker of the disorder (Saviour and Ramachandra, 2006).

Both the slow recognition of letters and a general inability to sequence the order of those letters and sounds are also outcomes of impairments in temporal processing—the timing of visual information during reading and of sounds in the word while hearing it (Breznitz, 2003). Typically, magnocellular neurons mediate temporal processing when the visual system first senses a letter and directs the eyes’ focus in order to identify it (Stein, 2018). In dyslexic brains, the magnocells in this pathway are abnormal in shape; size is important in the rapid processing

and transmission of signals concerning timing, in which the larger the magnocells, the better. As a result, dyslexics with impaired magnocells cannot maintain the speed of reading, as their eyes simply cannot appropriately fixate on the letters due to slow motion processing.

Due to this impairment, their eyes compensate with saccades—quick and simultaneous involuntary movement of both eyes between two or more points of fixation in the same direction (Adler, 1978; Stein, 2018). Saccades serve as a mechanism for fixation by signaling any slip in focus to bring the eyes back to target (Fischer, 2000). In non-dyslexic brains, this feedback is relayed to the retina, but in dyslexic brains, this process is slowed, and fixation on the letters when reading is unstable. Consequently, people with dyslexia struggle with fixation stability, as they find difficulty keeping their eyes focused on the same points, since they jump around rather than glide through letters and words smoothly. This lack of stability renders inaccurate saccades, causing letters to move or appear distorted, thus leaving no choice but to fixate on words longer in an attempt to make sense of them (Tiadi, 2016). Abnormal magnocellular layers are also a consequence of impaired function of the dorsal stream, which plays a critical role in the control of focus of visual attention and eye movements in reading (Stein, 2018). Individuals with dyslexia traditionally have low levels of Docosahexaenoic acid—an Omega-3 fatty chain imperative to the flexibility of the membrane in magnocellular neurons which accounts for their sensitivity and efficient functionality in processing and sending information— in their blood (Haag, 2003; Muskiet, 2004). Research reveals that supplementing Docosahexaenoic acid in the diet of children with dyslexia can improve reading behavior, reaffirming the significant role of magnocellular functioning in reading (Cyhlarova, 2007; Richardson, 2005).

While the most famously reported side effects of dyslexia are distorted and moving letters, most individuals with dyslexia experience auditory impairments as well. Some have impairments

in auditory temporal processing which manifest as difficulties with auditory sequencing of written words—an important cause of reading failure (Stein, 2018). Accompanied by visual sequencing of written letters, reading also requires auditory sequencing of phonemes in spoken word (Stein, 2018). Differentiation of phonemes relies on the ability to sense the frequency and amplitude changes that characterize them. A specialized set of neurons, often referred to as the auditory magnocellular neurons, detect these subtle changes in frequency and amplitude modulations. Therefore, sensitivity to these modulations is indicative of one's phonological awareness and reading ability (Stein, 1995). Lab studies show that individuals with dyslexia are less sensitive to frequency and amplitude modulations (McAnally, 1996) because the specialized cells found in the medial geniculate nucleus of the thalamus are smaller in size (Galaburda, 1994). To address this, studies like that of Thomson (2012) demonstrate that musical and rhythmic training interventions are strong options to improve phonological skills by increasing a dyslexics person's sensitivity to frequency and amplitude modulations. According to Witton (1998), auditory sensitivity modulations correlate quite strongly with visual magnocellular sensitivity, possibly indicating similar genetic and environmental influences.

Most research in support of this theory argues that the magnocellular pathway manages to account for all known manifestations of dyslexia, whether it be visual, auditory, tactile, motor and phonological (Ramus, 2003). However, this theory is not as recognized as the phonological deficit theory. In actuality, only a small percentage of people with dyslexia experience visual problems in reading tasks, which means it cannot be a universal indicator, nor generalizable extension. Children with impairments in the magnocellular pathway still learn to read despite the neurological abnormality and thus cannot provide a sufficient baseline condition for the understanding of dyslexia (Skoyles, 2004). Additionally, the dorsal stream and visual motor

pathway are not entirely magnocellular as much of its function includes the parvocellular pathway and thus cannot be solely attributed to difficulties in reading (Skottun, 2015; Stein, 2018). Lastly, most studies regarding this theory are correlational in nature, and magnocellular deficits can in fact be a consequence of reading impairment rather than the cause (Gori, 2016). With regards to the auditory deficits in the magnocellular pathway, it is debatable whether they are capable of adequately predicting phonological deficits, and the failure in the replication of study findings on auditory disorders in dyslexia renders the data unreliable (Ramus, 2003).

Another alternative, the cerebellar theory, seeks to explain the motor function deficit associated with dyslexia. Fundamentally, there is an experienced impairment in the cerebellum, affecting the ability to perform skills automatically (Nicholson, 2001). Though it is not widely regarded or studied, there is evidence indicating poor performance in motor tasks, (Fawcett et al., 1996), impaired balance automatization (Nicolson and Fawcett, 1990) and time estimation among people with dyslexia (Nicolson et al., 1995). Of its many functions, the cerebellum is responsible for the automatization of learned tasks such as texting, writing, reading, etc. A weak capacity to automatize in reading would affect the learning of grapheme and phoneme correspondences (Ramus 2003). This theory also provides an explanation for the poor quality of handwriting, as it is essentially a motor skill contingent on timing and coordination of muscle movement, a challenging feat for individuals with dyslexia with cerebellar dysfunction (Nicholson, 2001).

From a structural level, the functional deficit of the right side of the cerebellum is evident in the decreased blood flow (proxy of activation) in response to learned and new motor skills (Nicholson, 1999). The ratio of left grey matter to right grey matter is greater in the cerebella of dyslexic individuals (Saviour and Ramachandra, 2006). Cerebral laterality, or the dominance of

one side of the brain in controlling organs and activities, is ipsilateral to handedness (Saviour and Ramachandra, 2006) in that there is a larger portion of cerebral grey matter in the dominant hemisphere. This balance tends to be irregular in dyslexic brains; the ratio of left grey matter to right grey matter is greater in the dyslexic cerebella (Saviour and Ramachandra, 2006). Higher density of gray matter in regions of the brain are associated with higher ability in the corresponding functions. Other abnormalities include the altered ratios of choline and creatine in the right cerebellum, indicating altered development of this region and subsequent motor skills, including reading (Rae, 1998). Studies have indicated that choline is associated with white matter density and abnormal levels affect connectivity and myelination in dyslexic cerebella (Pugh, 2014). Some children with dyslexia also struggle with postural balance and motor coordination in attention-demanding tasks (Fawcett, 1999). Despite the soundness of these findings, cerebellar abnormalities are not commonly reported in dyslexia diagnoses and are less prevalent than visual and phonological deficit impairments.

Genetics

Developing research in genetics has succeeded in revealing both the genetic influences on dyslexia and the underlying deficits in the language areas associated with the disorder in the brain (Saviour and Ramachandra, 2006). According to Velluntino (2004), the risk of dyslexia is eight times higher in children with parental history of reading difficulties. Similarly, Hallgren's 1950 genetic study on the inheritance of dyslexia concluded that this disorder is familial and autosomal dominant, though data also noted the possibility of sex-related autosomal transmission. Identifying the gene for dyslexia is complicated because it affects multiple and varying aspects of reading, with no sole source. Any changes in the structure or function of

corresponding brain regions typically implies genetic mutations; in the case of dyslexia, these changes tend to affect the development of areas of the brain involved in reading (Saviour and Ramachandra, 2006). Still, the varying manifestations of dyslexia are more likely a combination of many genetic and environmental factors, not just genetic mutations.

Comprehensive genetic research on dyslexia is scarce and not enough studies have been conducted to associate chromosomes to the expression of dyslexia. Still, a locus on chromosome 6 has been discovered to possess a role in phonological awareness and some degree of single-word reading, and a locus on chromosome 15 seems to affect single word reading (Fisher, 1999). A handful of genetic studies have identified corresponding chromosomal loci for different dyslexia related phenotypes, suggesting that many genes contribute to the predisposition for the disorder. Common genes associated with dyslexia are *DYX1C1*, *DCDC2*, and *KIAA0319*. Their involvement in neural migration, the method by which neurons travel from their birthplace to their final position in the brain, affects brain connectivity between the temporal and parietal regions which in turn affect reading ability (Darki, 2012). Additionally, a protein expressed by *DCDC2* affecting cell polarity has been found to have an influence on neuronal migration, though the research remains unclear as to why these genes affect neuronal migration specifically in the temporal and parietal cortex (Massinen, 2011).

According to Darki (2012), the *DYX1C1* gene may influence both early brain development and additional pathways that affect later myelination of neurons, affecting white matter density (Darki, 2012). Darki's (2012) study on gene susceptibility of dyslexia concluded that both the *DCDC2* and *KIAA0319* genes affect early brain development, specifically the neuronal migration altering white matter density in the left temporoparietal region of the brain, which has been shown to impact reading scores and reading accuracy. Another study also found

that single nucleotide polymorphisms affect the expression of the KIAA0319 gene, associated with dyslexia (Pinel, 2012). Specifically, the natural asymmetry in activation of the superior temporal sulcus, an area of the temporal lobe linked to phonological processing, is reduced in the left hemisphere in participants with the KIAA0319 gene variant (Pinel, 2012; Cope, 2012).

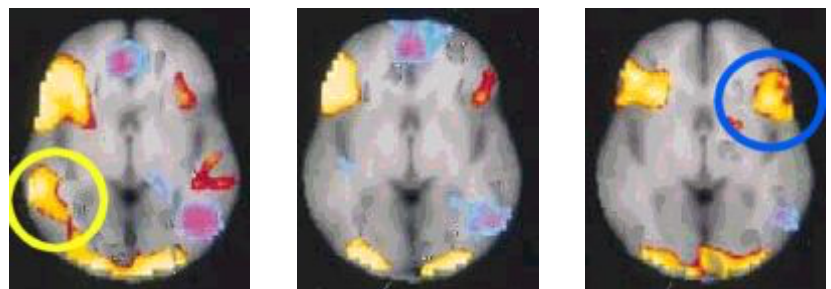
Unfortunately, most of the existing research on genetics has been inadequately replicated, if at all. A genome-wide study of dyslexia has yet to be conducted, and most of the known loci fail to explain the heritability in dyslexia that has been described in twin studies (Peterson and Pennington, 2012). It remains unclear as to what loci are shared or not shared between comorbidities of dyslexia. Whether dyslexia shows any genetic mechanisms that other neurodevelopmental disorders present, like epigenetic effects, is also unknown (Peterson and Pennington, 2012). Lastly, the role of the environment in the development of dyslexia has yet to be established.

The Importance of Informed Interventions

Phonological Interventions:

In the last 50 years, efforts have been focused on interventions that can address the learning deficiencies that are characteristic of the diagnosis. Research from Yale university discovered different

activation patterns in the brains of individuals with dyslexia who learn to read compared to those with poor reading ability (Marshall, 2003). While people with dyslexia typically show less



Non-dyslexic readers use left temporal area for sounding out words.

Dyslexic readers do not use a particular area for sounding out words.

Improved dyslexic readers depend on right hemisphere for sounding out words.

Figure 3. Brain imaging of activation during reading in both dyslexic and non-dyslexics. Adapted from "Brain Scans Show Dyslexics Read Better with Alternative Strategies," by A. Marshall, 2015, Davis Dyslexia Association International.

activation in the left posterior and temporal areas of the brain during reading tasks, those who develop reading skills via phonological trainings and interventions show increased activation in the right temporal areas and left frontal areas, as noted in Figure 3 (Marshall, 2003).

Phonological interventions, like the Phono-Graphix technique, are based on the notion that poor reading skills are due to a child's inability to utilize the phonemic level of language and is most effective for dyslexic children with some word-reading skills. Phono-Graphix aims to teach the alphabetic principle via letters and letter combinations as pictures of sounds and its evaluation study noted meaningful improvements in word recognition and nonsense word decoding tasks (McGuiness, 1996; Simos, 2006). After eighty hours of one-on-one instruction, the Woodcock Johnson's "Word Attack" scores of students with dyslexia increased from the 20th percentile to the average range of their corresponding age group (Papanicolaou, 2003). Figure 4 illustrates the activation changes in the temporoparietal lobe following the Phono-Graphix intervention as

noted in Papanicolaou (2003). For dyslexic children with more severe phonological processing deficits, the Lindamood program is unique in its simpler focus on pictures and modeling of lip and tongue movements. This technique improves sound awareness in children, and helps them move towards more structured understanding of letter sound pairings (Simos, 2006).

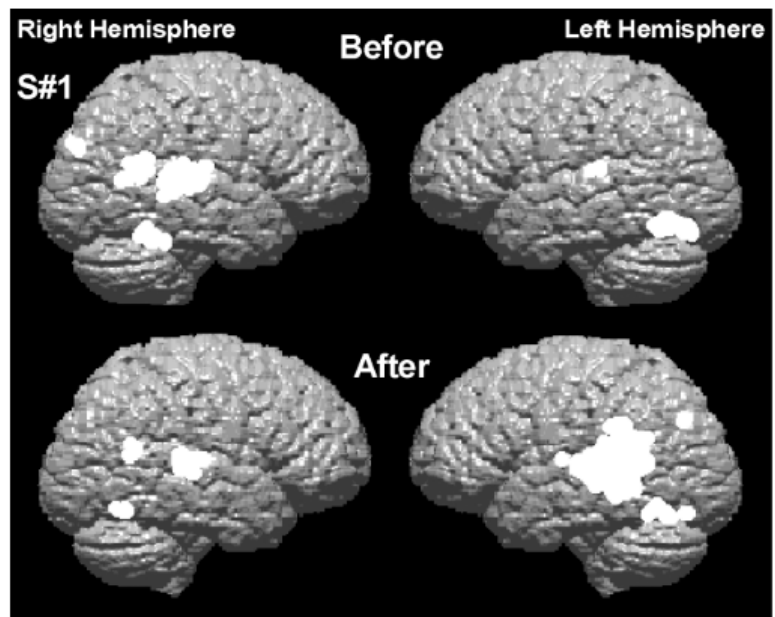


Figure 4. The activation maps for a 9-year-old with dyslexia before and after the Phono-Graphix intervention program. Significant increases in activation of temporoparietal areas after 8 weeks of enrollment in the program. Reprinted from "Brain mechanisms for Reading in children with and without dyslexia: A Review of Studies of Normal Development and Plasticity," by A. Papanicolaou, P. Simos, J. Breier, 2003. *Developmental Neuropsychology*. 24(2-3).

Alternatively, the Auditory Discrimination in Depth program is effective in developing stronger analytic decoding skills and phonological awareness by consciously focusing on the sensory—especially auditory—feedback from the mouth, ears and eyes in distinguishing and labeling vowels, constants and common phoneme pairings (Alexander, 1991). Most, if not all, researched phonological based interventions emphasize training in phonemic awareness, and all are successful in remediating these deficits., However, these methods do not address other reported impairments, such as visual and auditory confusion.

Magnocellular Interventions:

There has been progress in the last 20 years in interventions targeting the magnocellular visual pathway, with the assumption that training the pathway to improve its detection of fast movements can further reinforce its connection to reading ability and dyslexia. Chouake et al. (2012) proved that continuous magnocellular based visual training improved reading ability by strengthening neural connections critical for lexical decision and reading accuracy. This result showed that training the magnocellular pathway to detect progressively faster movements was followed by improved lexical decision and reading accuracy. Unique interventions like that of Gori et al. (2016) demonstrate that video games encourage the continued use of the pathway to process the speed of moving scenes and objects. This improved ability, coupled with simultaneous motor engagement, improves the visual magnocellular function and subsequent reading behavior in children with dyslexia. Eye movement training aimed at increasing the accuracy of saccades has also been shown to drastically improve reading fluency and comprehension (Leong et al., 2014).

Davis Learning Strategies:

Of all the aforementioned interventions, the Davis Learning Strategies, also referred to as the Davis Dyslexia Correction, remains the most popular. Created by Ron Davis, who overcame his own severe dyslexia by this visual, meaning-based method, this intervention does not follow any of the previously outlined theories. In fact, Davis emphasizes that his methodology lies “not [with] phonics or a phonetic theory; it is simply letter and word recognition” (Marshall, 2009).

According to Davis’ hypothesis and personal experience, individuals with dyslexia think in pictures. Therefore, breaking down words into their phoneme-grapheme units is inherently difficult for them. Using a visual and meaning-based approach to reading, he argues, is not only easier to learn but also incredibly effective, as demonstrated by a quick progress in reading development, which is almost always guaranteed. By using clay to model letters and the concepts related to the word’s meaning, students can expect to learn 200 or so common sight words by using clay to model the letters. The Davis Spell Reading and Sweep-Sweep-Spell exercises train the eye and brain to develop instant word recognition, a naturally occurring process among non-dyslexic readers. During training, students read a passage out loud in the presence of a teacher or parent, and if an unfamiliar word is encountered, the student spells out the word letter by letter until they recognize it and move on. If they cannot identify the word by the last letter, the parent or teacher says the word, and the student continues reading after repeating said word (Marshall, 2009). It is important to note that students engaged in this intervention are discouraged from using phonics and decoding sounds because this is what confuses and disorients the reader. These strategies aim to strengthen the neural pathways in the Visual Word Form Area in the occipital lobe which identifies words from memory instantaneously before one is aware of seeing the word, improving reading speed, accuracy and fluency (Marshall, 2009).

Ron Davis himself, with the addition of a few colleagues, conducted a longitudinal study on first graders with dyslexia in two San Francisco Bay area elementary schools. The participants were divided equally: 48 participants in the control group and 48 participants receiving instruction in the Davis Learning Strategies (Pfeiffer et al., 2001). The results noted that those who received the Davis Learning Strategies intervention in class demonstrated significant increases in accuracy on sight word recognition tests and by the third grade, none of these children required special services (Pfeiffer et al., 2001). Most of the students in the intervention group also benefited in their sight-word reading accuracy, though more research is required in evaluating the benefits of the intervention in earlier age groups. The data was inconclusive when the study was conducted on kindergartners in the same schools, requiring a more discriminating assessment tool that highlights early word recognition and reading ability (Pfeiffer et al., 2001).

Implications for Future Study

While leading experts disagree on the principle characteristic of the disorder, these theories may not be as mutually exclusive as they are currently perceived (Démonet, 2004). Figure 5 illustrates the many potential pathways that yield dyslexia and the intertwining nature of its etiological theories. Perhaps it is not as crucial to determine causation as it is to

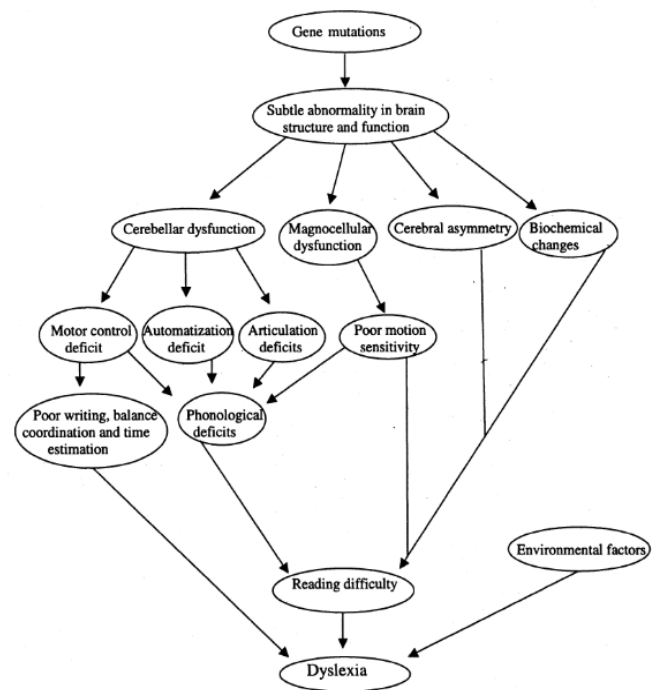


Figure 5. Possible Pathways of causation that lead to dyslexia. Descriptive phrase that serves as title and description. Reprinted from "Biological Basis of Dyslexia: A Maturing Perspective," by P. Saviour and N.B. Ramachandra, 2006, *Current Science*, 90(2), 173. <https://doi.org/10.2307/24088978>

understand its various forms and create informed strategies that address them. Nonetheless, continued study in the field can only widen our scope of knowledge. There is much to be uncovered in the cerebellum's role in dyslexia and to what extent its deficits co-exist with magnocellular deficits (Nicholson, 2001). Additionally, dyslexia has primarily been studied in individuals with affluent backgrounds, and little research has been done on lower socioeconomic groups (Peterson and Pennington, 2012). Such investigation would prove important, as it could possibly reveal the importance of environmental factors on the individual experience of dyslexia. In this capacity, a more thorough investigation on the interplay of these factors could broaden our understanding of genetics and environmental influence. In regards to genetics, future advancement in technology may aid in uncovering specific loci on chromosomes for heightened risk of dyslexia (Démonet, 2004). Replication of genetic studies on particular chromosomes can make that data more reliable. As noted previously, magnocellular irregularities in temporal processing can be diagnosed earlier than dyslexia. This will allow for earlier testing in temporal processing in children, before they experience reading failure (Stein, 2018). As some questions are answered and more surface, it is essential that the focus of research remains on the proper interventions and trainings that can motivate children towards academic success despite the struggle they experience with reading. We may never fully understand dyslexia, or narrow it down to a definite origin. Perhaps it is a necessary reminder of the natural variance in learning behavior and the individuality of the human experience.

References

- Adler-Grinberg, D., & Stark, L. (1978). Eye movements, scanpaths, and dyslexia. *American Journal of Optometry and Physiological Optics*, 55(8), 557-70.
- Alexander, Ann W., & Others. (1991). Phonological Awareness Training and Remediation of Analytic Decoding Deficits in a Group of Severe Dyslexics. *Annals of Dyslexia*, 41, 193-206.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Association, T. I. D. (2014). Dyslexia and the Brain. *Just the Facts*, 5.
- Breznitz, Z.; Meyler, A. (2003). Speed of lower-level auditory and visual processing as a basic factor in dyslexia: Electrophysiological evidence. *Brain Lang*, 85, 166–184.
- Catts, H. W. (1989). Defining dyslexia as a developmental language disorder. *Annals of Dyslexia*, 39(1), 50–64.
- Castles, A., & Coltheart, M. (2004). Is there a causal link from phonological awareness to success in learning to read? *Cognition*, 91(1), 77–111.
- Cohen, R. L., & Netley, C. (1981). Short-Term Memory Deficits in Reading Disabled Children, in the Absence of Opportunity for Rehearsal Strategies. *Intelligence*, 5(1), 69-76.
- Cope, Eicher, Meng, Gibson, Hager, Lacadie, . . . Gruen. (2012). Variants in the DYX2 locus are associated with altered brain activation in reading-related brain regions in subjects with reading disability. *NeuroImage*, 63(1), 148-156.
- Cyhlarova, E.; Bell, J.G.; Dick, J.R.; MacKinlay, E.; Stein, J.F.; Richardson, A.J. (2007). Membrane fatty acids, reading and spelling in dyslexic and non-dyslexic adults. *European Neuropsychopharmacology*, 17(2), 116-121.

- Darki, F., Peyrard-Janvid, M., Matsson, H., Kere, J., & Klingberg, T. (2012). Three dyslexia susceptibility genes, *DYX1C1*, *DCDC2*, and *KIAA0319*, affect temporo-parietal white matter structure. *Biological Psychiatry*, *72*(8), 671–676.
- Demb, J. B., Boynton, G. M., & Heeger, D. J. (1998). Functional magnetic resonance imaging of early visual pathways in dyslexia. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience*, *18*(17), 6939–6951.
- Démonet, J.-F., Taylor, M. J., & Chaix, Y. (2004). Developmental Dyslexia. *The Lancet*, *363*, 1451–1460.
- Duara, R., Kushch, A., Grossglenn, K., Barker, W. W., Jallad, B., Pascal, S., ... Lubs, H. (1991). Neuroanatomical differences between dyslexic and normal readers on magnetic-resonance-imaging scans. *Archives of Neurology*, *48*(4), 410–416.
- Fawcett, A., Nicolson, R., & Dean, P. (1996). Impaired Performance of Children with Dyslexia on a Range of Cerebellar Tasks. *Annals of Dyslexia*, *46*, 259-283.
- Fawcett, A., & Nicolson, R. (1999). Performance of Dyslexic Children on Cerebellar and Cognitive Tests. *Journal of Motor Behavior*, *31*(1), 68-78.
- Fischer, B., & Hartnegg, K. (2000). Stability of gaze control in dyslexia. *Strabismus*, *8*(2), 119-122.
- Fisher, S., Marlow, A., Lamb, J., et al. (1999). A Quantitative-Trait Locus on Chromosome 6p Influences Different Aspects of Developmental Dyslexia. *The American Journal of Human Genetics*, *64*(1), 146-156.
- Galaburda, A., & Kemper, T. (1979). Cytoarchitectonic abnormalities in developmental dyslexia: A case study. *Annals of Neurology*, *6*(2), 94-100.

- Galaburda, A., Sherman, G., Rosen, G., Aboitiz, F., & Geschwind, N. (1985). Developmental dyslexia: Four consecutive patients with cortical anomalies. *Annals of Neurology*, *18*(2), 222-233.
- Galaburda, A., Menard, M., & Rosen, G. (1994). Evidence for aberrant auditory anatomy in developmental dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, *91*(17), 8010-3.
- Gooch, D., Hulme, C., Nash, H. M., & Snowling, M. J. (2014). Comorbidities in preschool children at family risk of dyslexia. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *55*(3), 237–246.
- Gori, S., Seitz, A., Ronconi, L., Franceschini, S., & Facoetti, A. (2016). Multiple Causal Links Between Magnocellular–Dorsal Pathway Deficit and Developmental Dyslexia. *Cerebral Cortex*, *26*(11), 4356-4369.
- Haag, M. (2003). Essential Fatty Acids and the Brain. *The Canadian Journal of Psychiatry*, *48*(3), 195-203.
- Hallgren, B. (1950). Specific dyslexia: a clinical and genetic study. *Acta Psychiatr Neurolog Scand*, *65*.
- Hoefl, F., Hernandez, A., McMillon, G., Taylor-Hill, H., Martindale, J. L., Meyler, A., ... Gabrieli, J. D. E. (2006). Neural Basis of Dyslexia: A Comparison between Dyslexic and Nondyslexic Children Equated for Reading Ability. *Journal of Neuroscience*, *26*(42), 10700–10708.
- Kamhi, A., Catts, H., Mauer, D., Apel, K., & Gentry, B. (1988). Phonological and spatial processing abilities in language- and reading-impaired children. *The Journal of Speech and Hearing Disorders*, *53*(3), 316-27.

- Leonard, C., Lombardino, L., Mercado, L., Browd, S., Breier, J., & Agee, O. (1996). Cerebral Asymmetry and Cognitive Development in Children: A Magnetic Resonance Imaging Study. *Psychological Science*, 7(2), 89-95.
- Lindgren, Scott D., et al. (1985). Cross-National Comparisons of Developmental Dyslexia In Italy and the United States. *Child Development*, 56(6), 1404-17.
- Marshall, A. (2003). Brain Scans Show Dyslexics Read Better with Alternative Strategies. Retrieved June 14, 2012 from Dyslexia, the Gift.
URL: http://www.dyslexia.com/science/different_pathways.htm
- Marshall, A. (2009). Davis Reading Exercises: Spell Reading & Sweep-Sweep-Spell. *Davis Dyslexia Association International*.
- Massinen S, Hokkanen ME, Matsson H, Tammimies K, Tapia-Paéz I, Dahlström-Heuser V, et al. (2011). Increased expression of the dyslexia candidate gene DCDC2 affects length and signaling of primary cilia in neurons. *PLoS One*, 6(6), E20580.
- Mcanally, K., & Stein, J. (1996). Auditory Temporal Coding in Dyslexia. *Proceedings: Biological Sciences*, 263(1373), 961-965.
- McGuinness, C. (2016). Phono-Graphix™ : A New Method for Remediating Reading Difficulties Author (s): Diane McGuinness , Carmen McGuinness , Diane McGuinness and Geoffrey Stable URL : <http://www.jstor.org/stable/23769456> Phono-Graphix™ : A New Method for Remediating Reading , 46(1996), 73–96.
- Muskiet, F., Fokkema, M., Schaafsma, A., & Crawford, M. (2004). Is Docosahexaenoic Acid (DHA) Essential? Lessons from DHA Status Regulation, Our Ancient Diet, Epidemiology and Randomized Controlled Trials. *The Journal of Nutrition*, 134(1), 183-6.

- Nicolson, R.I., Fawcett, A.J. (1990). Automaticity: A new framework for dyslexia research? *Cognition*, 35(2), 159-182.
- Nicolson, R., Fawcett, A., & Dean, P. (1995). Time Estimation Deficits in Developmental Dyslexia: Evidence of Cerebellar Involvement. *Proceedings of the Royal Society B: Biological Sciences*, 259(1354), 43-47.
- Nicolson, R., Fawcett, A., & Berry, E. (1999). Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *The Lancet*, 353(9165), 1662-7.
- Nicholson, R. I., Fawcett, A.J., & Dean, Paul. (2001). Developmental dyslexia: The cerebellar deficit hypothesis. *Trends in Neurosciences*, 24(9), 508-11
- Papanicolaou, A., Simos, P., Breier, J., Fletcher, J., Foorman, B., Francis, D., . . . Davis, R. (2003). Brain Mechanisms for Reading in Children With and Without Dyslexia: A Review of Studies of Normal Development and Plasticity. *Developmental Neuropsychology*, 24(2-3), 593-612.
- Paulesu, E., Demonet, J., Fazio, F., McCrory, E., Chanoine, V., Brunswick, N., et al. (2001). Dyslexia: cultural diversity and biological unity. *Science*, 291, 2165–2167.
- Peterson, R. L., & Pennington, B. F. (2012). Developmental dyslexia. *The Lancet*, 379(9830), 1997–2007.
- Petkov, C., O'Connor, K., Benmoshe, G., Baynes, K., & Sutter, M. (2005). Auditory perceptual grouping and attention in dyslexia. *Brain Research. Cognitive Brain Research*, 24(2), 343-54.
- Pfeiffer, S., Davis, R., Kellogg, E., Hern, C., McLaughlin, T.F., Curry, G. (2001). The Effect of the Davis Learning Strategies on First Grade Word Recognition and Subsequent Special Education Referrals. *Reading Improvement*, 38(2).

Pinel, P., Fauchereau, F., Moreno, A., Barbot, A., Lathrop, M., Zelenika, D., . . . Dehaene, S.

(2012). Genetic variants of FOXP2 and KIAA0319/TTRAP/THEM2 locus are associated with altered brain activation in distinct language-related regions. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 32(3), 817-25.

Pugh, K., Frost, S., Rothman, D., Hoeft, F., Del Tufo, S., Mason, G., . . . Fulbright, R. (2014).

Glutamate and choline levels predict individual differences in reading ability in emergent readers. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 34(11), 4082-9.

Rae, C. et al. (1998). Metabolic abnormalities in developmental dyslexia detected by 1H

magnetic resonance spectroscopy. *The Lancet*, 351(9119), 1849-1852.

Raschle, Chang, & Gaab. (2011). Structural brain alterations associated with dyslexia predate

reading onset. *NeuroImage*, 57(3), 742-749.

Ramus, F., Rosen, S., Dakin, S. C., Day, B. L., Castellote, J. M., White, S., & Frith, U. (2003).

Theories of developmental dyslexia: Insights from a multiple case study of dyslexic adults. *Brain*, 126(4), 841–865.

Richardson, A., & Montgomery, P. (2005). The Oxford-Durham study: A randomized, controlled

trial of dietary supplementation with fatty acids in children with developmental coordination disorder. *Pediatrics*, 115(5), 1360-6.

Richlan, F., Kronbichler, M., & Wimmer, H. (2011). Meta-analyzing brain dysfunctions in

dyslexic children and adults. *Neuroimage*, 56(3), 1735–1742.

Richlan F. (2012). Developmental dyslexia: dysfunction of a left hemisphere reading

network. *Frontiers in human neuroscience*, 6, 120. doi:10.3389/fnhum.2012.00120

- Saviour, P., & Ramachandra, N. B. (2006). Biological basis of dyslexia: A maturing perspective. *Current Science*, *90*(2), 168–175.
- Shaywitz, S.E., Shaywitz, B. A., Fletcher, J. M., & Escobar, M. D. (1990). Prevalence of Reading Disability in Boys and Girls: Results of the Connecticut Longitudinal Study. *JAMA*, *264*(8), 998-1002.
- Silani, G., Frith, U., Demonet, J.F., et al. (2005). Brain abnormalities underlying altered activation in dyslexia: A voxel based morphometry study. *Brain*, *128*(10), 2453-2461.
- Simos, P. G., Fletcher, J. M., Denton, C., Sarkari, S., Billingsley-Marshall, R., & Papanicolaou, A. C. (2006). Magnetic source imaging studies of dyslexia interventions. *Developmental Neuropsychology*, *30*(1), 591–611.
- Skottun, B. (2015). The need to differentiate the magnocellular system from the dorsal stream in connection with dyslexia. *Brain and Cognition*, *95*, 62-66.
- Skoyles, J., & Skottun, B. C. (2004). On the Prevalence of Magnocellular Deficits in the Visual System of Non-Dyslexic Individuals. *Brain and Language*, *88*(1), 79-82.
- Smith, S., Kelley, P., & Brower, A. (1998). Molecular Approaches to the Genetic Analysis of Specific Reading Disability. *Human Biology*, *70*(2), 239-256.
- Snowling, M. (1995). Phonological processing and developmental dyslexia. *Journal of Research in Reading*, *18*(2), 132-138.
- Stein, J., & Mcanally, K. (1995). Auditory Temporal Processing in Developmental Dyslexics. *The Irish Journal of Psychology*, *16*(3), 220-228.
- Stein, J., Talcott, J., & Walsh, V. (2000). Controversy about the visual magnocellular deficit in developmental dyslexics. *Trends in Cognitive Sciences*, *4*(6), 209-211.

- Stein, J. (2018). What is developmental dyslexia? *Brain Sciences*, 8(2).
- Thomson, J.M., Leong, V., Goswami, U. (2013). Auditory Processing Interventions and Developmental Dyslexia: A Comparison of Phonemic and Rhythmic Approaches. *Reading and Writing: An Interdisciplinary Journal*, 26(2), 139-161.
- Tiadi, A., Gérard, C.L., Peyre, H., Bui-Quoc, E., Bucci, M.P. (2016). Immaturity of Visual Fixations in Dyslexic Children. *Front. Hum. Neuroscience*, 10.
- Vellutino, F. R., Fletcher, J. M., Snowling, M. J., & Scanlon, D. M. (2004). Specific reading disability (dyslexia): what have we learned in the past four decades?, 1, 2–40.
- Wagner, R., Torgesen, J., & Masters, John C. (1987). The Nature of Phonological Processing and Its Causal Role in the Acquisition of Reading Skills. *Psychological Bulletin*, 101(2), 192-212.
- Witton, C., Talcott, J.B., Hansen, P.C., Richardson, A.J., Griffiths, T.D., Rees, A., Stein, J.F., Green, G.G.R. (1998). Sensitivity to dynamic auditory and visual stimuli predicts non-word reading ability in both dyslexic and normal readers. *Current Biology*, 8(14), 791-797.
- Ziegler, J. C., Perry, C., Ma-Wyatt, A., Ladner, D., & Schulte-Körne, G. (2003). Developmental dyslexia in different languages: Language-specific or universal? *Journal of Experimental Child Psychology*, 86(3), 169–193.